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THÈSE DE DOCTORAT
DE L'UNIVERSITÉ PSL

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Improving Child Health in Sub-Saharan Africa

THREE ESSAYS IN MICROECONOMETRICS

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Soutenue par

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Improving Child Health in Sub-Saharan Africa

THREE ESSAYS IN MICROECONOMETRICS

Yohan Renard

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“ *Heureux les pays du Nord auxquels les saisons composent, l’été, une légende de neige, l’hiver, une légende de soleil, tristes tropiques où dans l’étuve rien ne change beaucoup, mais heureux aussi ce Sahara où le jour et la nuit balancent si simplement les hommes d’une espérance à l’autre.* ”

— Antoine de Saint-Exupéry,
Terre des hommes, 1939

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*
* *

“ *Je ne suis pour toi qu'un renard semblable à cent mille renards. Mais, si tu m'apprivoises, nous aurons besoin l'un de l'autre. Tu seras pour moi unique au monde. Je serai pour toi unique au monde.* ”

— Antoine de Saint-Exupéry,
Le Petit Prince, 1943

Résumé en français

La santé fait référence à un vaste ensemble de conditions et peut être définie comme *un état de complet bien-être physique, mental et social et ne consiste pas seulement en une absence de maladie ou d'infirmité*. Suivant cette définition, le fait de pouvoir jouir du meilleur état de santé possible a été reconnu comme l'un des droits fondamentaux de tout être humain (OMS, 1946 et article 25 de la Déclaration Universelle des Droits de l'Homme). L'amélioration de l'état de santé dans le monde est considérée, avec l'éducation, comme l'un des piliers de la prospérité partagée. Il est désormais largement admis que la pauvreté est à la fois une cause et une conséquence d'un mauvais état de santé. La pauvreté augmente le risque de mauvaise santé, tandis que la mauvaise santé piège les individus dans la pauvreté. La lutte contre la mauvaise santé est ainsi intimement liée à la lutte contre la pauvreté.

Est-ce uniquement une question de ressources financières ? La relation entre mauvaise santé et pauvreté est en partie due aux contraintes financières qui pèsent sur les individus. Les pauvres n'ont généralement pas les moyens d'investir dans leur santé, tant à titre préventif que curatif, notamment en consommant des quantités suffisantes de nourriture pour satisfaire leurs besoins nutritionnels quotidiens de base. Toutefois, au-delà des contraintes financières, de nombreux facteurs rendent cette relation particulièrement saillante, notamment les faibles niveaux d'éducation, le manque d'information sur les maladies et les moyens de les prévenir, l'information incomplète sur les avantages et coûts des investissements dans la santé, des facteurs environnementaux défavorables entraînant une plus grande exposition aux maladies infectieuses ainsi qu'une plus faible capacité des pauvres à faire valoir leur droit à la santé.

En parallèle, les effets négatifs, y compris économiques, d'un mauvais état de santé sont nombreux et sont notamment observés sur le marché du travail (e.g. Currie et Madrian, 1999; Thirumurthy et al., 2008; García-Gómez et al., 2013; Fletcher, 2014; Lenhart, 2019; Seuring et al., 2019; Jones et al., 2020; Stephens et Toohey, 2022), la productivité agricole (e.g. Dillon et al., 2014; Fink et Masiye, 2015), le niveau d'éducation atteint (e.g. Currie et Stabile, 2009; Field et al., 2009; Simo Fotso et al., 2018) et plus généralement sur le statut socio-économique (e.g. Meyer et Mok, 2019), faisant de l'état de santé un déterminant essentiel de la pauvreté. Les ménages peuvent également basculer dans la pauvreté du fait de dépenses de santé catastrophiques les amenant à épuiser leurs économies et à vendre leurs actifs.

Cette trappe à pauvreté associée à la mauvaise santé appelle une intervention des pouvoirs publics, d'autant plus justifiée que le secteur de la santé souffre d'importantes défaillances de marché [Ghosh, 2008; Dupas, 2014]. Cela renvoie notamment à la présence

d'externalités, à l'existence d'une information imparfaite et au manque d'infrastructures sanitaires. De telles défaillances conduisent à des niveaux d'investissement dans la santé inférieurs à ce qui serait socialement optimal : les individus n'investissent pas suffisamment dans leur santé, soit parce qu'ils n'ont pas la capacité de le faire, soit parce qu'ils n'ont pas conscience de l'entièreté des bénéfices qui en découleraient. Si les pouvoirs publics peuvent intervenir pour pallier ces défaillances de marché, leur participation à la poursuite de l'optimum social s'avère très inégale d'un pays à l'autre.

Malgré des efforts continus, les niveaux de santé restent relativement bas dans plusieurs régions du monde, en particulier en Afrique Subsaharienne. Si de nombreuses causes de maladie et de mortalité, telles que les cancers, sont communes à tous les pays du monde, les pays en développement sont également confrontés à ensemble de pathologies liées à leur environnement ainsi qu'à leur niveau de pauvreté, notamment les maladies tropicales et hydriques. Comme le souligne Dupas [2011], ce fardeau affecte des individus plus jeunes que celui des pathologies dominantes dans les pays développés, et consiste essentiellement en des maladies infectieuses et parasitaires caractérisées par d'importantes externalités en matière de santé publique. L'Afrique Subsaharienne est particulièrement accablée par ce fardeau. En 2019, les maladies infectieuses et parasitaires étaient responsables de 46,5% des décès sur le sous-continent, contre 9,3% dans le reste du monde et 19,7% en Asie du Sud.¹

Si les efforts consentis jusqu'à présent, bien qu'insuffisants, ont permis d'améliorer la situation sanitaire dans le monde, de tels progrès s'avèreront de plus en plus difficiles à réaliser. Ceci renvoie essentiellement à trois défis pour les années à venir. Le premier consiste à atteindre les populations marginalisées qui n'ont généralement pas (ou peu) bénéficié des progrès accomplis jusqu'ici. Le deuxième défi est celui des investissements massifs nécessaires pour, d'une part, garantir des conditions de vie plus saines aux individus grâce à des infrastructures d'eau et d'assainissement adéquates, et, d'autre part, améliorer la qualité des soins. Le troisième défi est lié à l'évolution de la demande en faveur de services de santé plus avancés, tels que la cardiologie et l'oncologie. L'Afrique Subsaharienne fait notamment face à une augmentation de la part des décès attribuables à des pathologies telles que les cancers, les accidents vasculaires cérébraux ou les maladies coronariennes, rendues plus saillantes par le recul des maladies infectieuses et parasitaires observé ces dernières années. Cette évolution nécessitera des investissements considérables en infrastructures, équipements et ressources humaines pour prévenir, diagnostiquer et traiter de

¹ Calculs de l'auteur à partir des données du [Global Burden of Disease \[2019\]](#).

telles pathologies.

Bien que des avancées majeures aient incontestablement été enregistrées au cours des quarante dernières années, la plupart des pays d'Afrique Subsaharienne sont encore loin d'atteindre l'objectif de *santé pour tous* initié en 1978 lors de la Déclaration d'Alma-Ata. Les taux de mortalité infantile y restent dramatiquement élevés, les pathologies dites évitables et soignables y demeurent la première cause de décès, et de nouveaux défis remettent en question la capacité de leurs systèmes de santé à répondre aux besoins de la population.

Les chapitres de cette thèse, résumés ci-dessous, s'intéressent à plusieurs questions liées à l'amélioration de la santé des enfants. Au-delà d'être un objectif de justice sociale en soi, garantir un niveau de santé suffisamment élevé à chaque enfant apparaît comme essentiel au regard des nombreuses conséquences tout au long de la vie d'un mauvais état de santé durant la petite enfance. L'identification des facteurs qui entravent l'amélioration de la santé des enfants dans les pays en développement, notamment en Afrique Subsaharienne, est ainsi primordial.

Chaque chapitre explore indépendamment l'un des multiples aspects liés à la santé des enfants en Afrique Subsaharienne. Bien que les questions de recherche diffèrent d'un chapitre à l'autre, l'objectif général de cette thèse est d'identifier et de mieux comprendre certains facteurs qui favorisent ou ralentissent l'amélioration de la santé des enfants en Afrique Subsaharienne.

Chapitre 1 – Gratuité des soins, recours aux services de santé maternelle et santé des enfants en Zambie

Le premier chapitre porte sur l'effet de la mise en place de la gratuité des soins sur le recours aux services de santé maternelle ainsi que sur la santé des jeunes enfants en Zambie.

Contexte

Bien que la situation se soit améliorée au cours des dernières décennies, un mauvais état de santé général et un faible recours aux soins persistent dans la plupart des pays en développement. Ainsi, en 2017, chaque jour dans le monde, 11 000 enfants décédaient avant d'atteindre leur premier anniversaire et 800 femmes mouraient des suites de leur grossesse. Dans 99% des cas, ces décès ont eu lieu dans un pays en développement. L'accès aux soins et les bénéfices qu'en tirent les individus sont des éléments cruciaux du débat sur

l'efficacité relative des interventions côté offre et côté demande de soins dans l'amélioration de la santé des populations. Malgré l'incidence toujours élevée de décès prématurés dits évitables, relativement peu de travaux empiriques ont exploré le rôle que peut jouer la mise en place de la gratuité des soins pour accroître l'utilisation des services de santé, et *in fine* améliorer la santé des individus.

Une vaste littérature s'est intéressée à l'effet du prix sur la propension des individus à investir dans leur santé. Cette littérature a mis en exergue une très forte élasticité prix, suggérant que même des prix très faibles peuvent décourager les individus à investir dans leur santé. Cependant, cette littérature s'est essentiellement focalisée sur des produits de santé pouvant être utilisés directement à domicile (par exemple [Ashraf et al., 2010](#) pour la chlorine utilisée pour désinfecter l'eau, [Cohen et Dupas, 2010](#) pour les moustiquaires, [Cohen et al., 2015](#) pour des antipaludiques ou encore [Spears, 2014](#) pour du savon destiné au lavage des mains). En revanche, nous disposons de beaucoup moins de preuves s'agissant de l'effet d'une réduction du prix des services de santé dans les infrastructures sanitaires publiques [[Kremer et Glennerster, 2011](#); [Dupas, 2014](#)], et ce malgré le fait que les dépenses de santé curative peuvent représenter une part conséquente du budget des ménages, allant parfois jusqu'à 10% [[Dupas, 2011](#)].

Pourtant, beaucoup de raisons tendent à suggérer que l'effet du prix pourrait ne pas être le même sur la demande de produits et de services de santé, en particulier dans les pays à bas revenu. Réduire le prix des services de santé pourrait encourager l'utilisation des services de santé, et *in fine* contribuer à l'amélioration de l'état de santé de la population si les individus n'avaient pas accès à des soins de qualité pour des raisons financières. Cependant, au-delà du prix des services de santé, de nombreux facteurs tels que la mauvaise qualité des soins, l'éloignement des infrastructures sanitaires [[Thornton, 2008](#)], l'absentéisme du personnel médical [[Banerjee et al., 2008](#); [Chaudhury et Hammer, 2004](#)], ou encore l'information imparfaite sur les bénéfices et coûts associés aux investissements dans la santé [[Banerjee et al., 2015](#); [Jalan et Somanathan, 2008](#); [Rhee et al., 2005](#)] peuvent conduire les individus à ne pas consulter. La réduction du prix des services de santé pourrait même exacerber certains de ces facteurs, tels que la charge de travail du personnel médical, les paiements informels ou encore les ruptures de stock de matériel médical et paramédical [[Hatt et al., 2013](#); [Meessen et al., 2011](#); [Nabyonga-Orem et al., 2011](#)]. Ainsi, la réduction du prix des services de santé pourrait ne pas être suffisante pour réduire le coût marginal d'une consultation en dessous du bénéfice marginal perçu qui y est associé. Par ailleurs, l'effet final sur la santé des individus ne dépend pas uniquement de l'élasticité prix de la demande de soins,

mais aussi des bénéficiaires que tirent les individus d'une visite auprès d'une infrastructure sanitaire.

Ce chapitre apporte un éclairage nouveau sur la manière dont la gratuité des soins joue sur l'utilisation des services de santé maternelle et la santé des enfants dans un contexte où les ressources, tant humaines que financières, demeurent limitées. L'objectif est également d'évaluer l'efficacité d'une telle politique selon d'autres facteurs liés à l'offre de soins, tels que la proximité géographique des infrastructures sanitaires et l'offre de soins qui y est proposée. La Zambie constitue un cadre idéal pour explorer ces questions de recherche. À partir d'avril 2006, le gouvernement décide de rendre les soins primaires gratuits dans les centres de santé publics et confessionnels de 54 districts considérés comme ruraux parmi les 72 que compte le pays. Un an plus tard, en juillet 2007, cette politique est étendue aux zones rurales des districts urbains, jusqu'ici non concernés par cette politique. Les frais d'utilisation des services de santé primaire sont finalement abolis dans l'ensemble du pays à partir de 2012. Les accouchements font partie des services couverts par cette gratuité. À titre d'exemple, avant la mise en place de cette politique, le coût d'un accouchement en milieu médical public dans un district rural de la province du Nord-Ouest coûtait entre 10 000 et 20 000 Kwachas zambiens, c'est-à-dire entre 2,84 et 5,68 dollars américains, soit 15,5% à 31% du revenu mensuel moyen par tête dans cette province en 2006.

Données et stratégie empirique

Ce chapitre mobilise quatre vagues des Enquêtes Démographiques et de Santé menées en Zambie en 1996, 2001, 2007 et 2013, représentatives à l'échelle nationale. Les femmes de 15 à 49 ans interrogées dans le cadre de ces enquêtes fournissent des informations très détaillées sur les naissances et décès de leurs enfants intervenus au cours des cinq années précédant la date d'enquête, y compris sur les conditions d'accouchement et les soins post-partum. Des mesures anthropométriques telles que la taille et le poids sont également recueillies, ainsi que des informations sur les comportements de santé préventifs comme la vaccination des enfants.

Les conditions de naissance renvoient ici au lieu d'accouchement et à la présence de personnel médical pendant la naissance. Toutes choses égales par ailleurs, la mise en place de la gratuité des soins devrait stimuler le recours aux accouchements médicalisés via une réduction du coût marginal qui y est associé. Si tel est le cas, alors les accouchements en milieu médicalisé public devraient augmenter. Si l'absentéisme du personnel médical ne s'aggrave pas du fait de la gratuité, la probabilité d'accoucher avec l'aide d'un personnel médical

devrait également s'accroître. J'explore par ailleurs les effets sur les soins post-partum, qui peuvent refléter la qualité des soins reçus par les mères.

Le risque de mortalité des jeunes enfants est fortement concentré au cours des premiers jours de vie, lorsque les chances de survie du nouveau-né sont particulièrement liées aux conditions de naissance. Dans l'échantillon mobilisé pour ce chapitre, un tiers des décès au cours des 28 premiers jours de vie interviennent en réalité dès le jour de la naissance. Une part importante de ces décès est liée à des complications lors du travail et de la naissance, telles que l'asphyxie du nouveau-né. La présence de personnel médical lors de l'accouchement pourrait permettre de mieux gérer ces complications et de sauver des vies. La santé des enfants est ainsi mesurée par leur risque de mortalité, qui fait ici référence à la mortalité à la naissance, au cours des 28 premiers jours (mortalité néonatale) ou de la première année de vie (mortalité infantile). Le statut nutritionnel est également utilisé comme mesure de santé au moment de l'enquête, et est construit à partir des mesures anthropométriques.

Enfin, une exposition plus régulière au personnel médical du fait de la gratuité des soins peut jouer sur les décisions des ménages en matière d'investissements, notamment préventifs, dans la santé de leurs membres. Ainsi, la vaccination contre la polio, la rougeole, la diphtérie, la coqueluche, le tétanos et la tuberculose est utilisée pour mesurer l'effet sur les investissements dans la santé des enfants.

Le déploiement progressif de la gratuité des soins dans les différentes zones du pays est exploité à travers une approche par double différence. De manière succincte, cette méthode consiste à utiliser l'évolution de la situation dans les zones non concernées par la gratuité (groupe contrôle) pour estimer ce qu'il serait advenu dans les zones concernées par la gratuité des soins (groupe traité) si celle-ci n'avait finalement pas été mise en place. L'effet de la politique est alors obtenu en comparant l'évolution dans ces deux groupes, sous réserve que l'évolution dans le groupe contrôle apparaisse comme une estimation crédible de l'évolution de la situation à laquelle on aurait pu s'attendre dans le groupe traité. Cette hypothèse, dite des tendances parallèles, est longuement discutée dans le cadre de ce chapitre, et apparaît comme tout à fait probable. Ce chapitre utilise également une approche de type *event-study* qui permet d'explorer la temporalité des effets.

L'analyse de l'hétérogénéité des effets selon l'éloignement des infrastructures sanitaires et l'offre de soins au niveau local repose sur des données administratives particulièrement riches issues du recensement national des infrastructures sanitaires mené en 2005. Ce re-

censement contient notamment les coordonnées géographiques de toutes les infrastructures sanitaires publiques et confessionnelles du pays, ainsi que des informations très détaillées sur les infrastructures physiques, les équipements disponibles, les soins proposés ou encore le personnel médical et paramédical présent.

Principaux résultats

Les résultats montrent que la probabilité d'accoucher en milieu médicalisé s'est fortement accrue suite au passage à la gratuité, enregistrant une hausse de 43% par rapport à la situation qui prévalait avant sa mise en place (+13,9 points de pourcentage). Comme escompté, cet effet est exclusivement tiré par les accouchements dans les infrastructures publiques et confessionnelles, le secteur privé n'étant pas concerné par la politique. Ce résultat est rassurant dans la mesure où il indique que cette politique ne s'est pas traduite par un effet de substitution du secteur privé vers le secteur public, mais a bien touché des femmes qui accouchaient en dehors du système de santé. Il apparaît également qu'une part plus importante des accouchements a désormais lieu en présence d'un personnel médical, avec une hausse de 36% (+11,4 points de pourcentage), suggérant ainsi que la gratuité n'a pas conduit à une augmentation massive de l'absentéisme du personnel médical. Cependant, il est à noter que seule la probabilité d'accoucher avec l'aide d'un infirmier ou d'une sage-femme a augmenté, tandis que la probabilité d'accoucher avec l'aide d'un médecin est restée proche de zéro. Ce résultat est cohérent avec la très forte concentration des médecins dans les villes et zones urbaines, qui n'étaient initialement pas concernées par la gratuité.

En revanche, la probabilité pour les mères d'avoir bénéficié d'une consultation post-partum au cours des 24 heures suivant l'accouchement n'a augmenté que de 6,1 points de pourcentage. Si ce résultat est en soi positif, indiquant que davantage de femmes ont pu bénéficier d'une consultation post-partum, il doit également s'interpréter à la lumière de l'effet obtenu sur les accouchements médicalisés mentionné précédemment (+13,9 points de pourcentage). L'écart important entre ces deux effets suggère une mauvaise qualité des soins puisque de nombreuses femmes ayant accouché dans des établissements de santé n'ont pas bénéficié d'un examen médical qui aurait pu prévenir d'éventuelles complications post-partum.

La malnutrition chronique chez les moins de cinq ans, reflétée par une faible taille de l'enfant pour son âge, a quant à elle reculé avec le passage à la gratuité des soins primaires, un effet qui ne se manifeste cependant qu'à partir d'une exposition d'au moins 12 mois à la politique. L'analyse révèle par ailleurs qu'en moyenne la mise en place de la gratuité n'a

pas eu d'effet perceptible sur le risque de mortalité chez les jeunes enfants, quelle que soit la période considérée.

Le chapitre examine ensuite différents phénomènes qui pourraient masquer l'existence d'un effet sur la mortalité, et ainsi expliquer pourquoi aucun effet n'est observé. *(i)* Le type de mère donnant naissance peut avoir été modifié dans les zones traitées par rapport aux zones contrôles : certaines mères possédant des caractéristiques spécifiques (notamment en termes de pauvreté ou de niveau d'éducation) peuvent réagir à la politique en ayant davantage d'enfants, ce qui conduirait à un effet de composition. De manière similaire, le passage à la gratuité a pu aider certaines femmes fortement exposées à des complications pendant leur grossesse et leur accouchement à survivre, en réduisant leur risque de décès. Si tel est le cas, étant donné que les naissances sont reportées uniquement par les mères ayant survécu jusqu'à la date de l'enquête, la composition de l'échantillon peut avoir évolué dans les zones traitées du fait de la politique. *(ii)* La politique peut avoir essentiellement atteint des ménages qui, *ex ante*, font face à un faible risque de mortalité des jeunes enfants, et pour qui les bénéfices en termes de réduction du risque de mortalité sont faibles. Ainsi, les accouchements en milieu médicalisé pourraient s'accroître sans effet perceptible sur la mortalité des jeunes enfants. *(iii)* La gratuité des soins peut avoir aidé certains nouveau-nés particulièrement fragiles à survivre à leur naissance. Cela accroît la probabilité d'observer de tels enfants dans l'échantillon au sein des zones traitées par rapport aux zones contrôles. *(iv)* Enfin, en réduisant le coût marginal à court-terme d'une naissance supplémentaire, la gratuité des soins peut avoir conduit certains ménages à avoir davantage d'enfants avec des intervalles de naissance plus courts, ce qui peut constituer un facteur de risque pour la santé des mères et des enfants. Les analyses révèlent qu'aucun de ces phénomènes ne peut expliquer l'absence d'effet sur la mortalité des jeunes enfants. Plusieurs éléments suggèrent en revanche que la faible qualité des soins pourrait en être à l'origine.

Enfin, les analyses révèlent que l'accès physique aux infrastructures sanitaires et les services médicaux disponibles localement jouent un rôle essentiel dans l'efficacité du passage à la gratuité des soins. L'effet sur les accouchements médicalisés décroît ainsi rapidement avec l'éloignement des infrastructures sanitaires : rendre les soins gratuits n'a plus aucun effet au-delà de 20 kilomètres du centre de santé le plus proche. Si le passage à la gratuité n'a eu en moyenne aucun effet sur les risques de mortalité à la naissance et néonatale, l'analyse révèle que ces risques se sont réduits au sein des ménages vivant à proximité immédiate d'un centre de santé assurant des soins obstétricaux d'urgences et des services de santé orientés vers les jeunes enfants, tels que la réanimation des nouveau-nés.

Implications de politique publique

Ce chapitre suggère que la gratuité des soins permet de stimuler la demande des individus pour des services de santé curatifs, mais qu'elle n'est pas suffisante en tant que telle pour générer les gains escomptés en matière de santé des individus. La qualité des soins apparaît comme une pièce cruciale du puzzle puisque le risque de mortalité chez les nouveau-nés ne diminue qu'à proximité des centres de santé offrant des services obstétricaux d'urgence et de santé infantile.

Ces résultats ont des implications importantes pour les décideurs politiques. Ils illustrent un double défi : rendre les services de santé à la fois financièrement accessibles et de meilleure qualité pour tous. En particulier, l'efficacité d'une politique de gratuité, au demeurant très coûteuse pour les finances publiques, peut s'avérer limitée sans une qualité des soins adéquate. Ces conclusions appellent à des efforts massifs pour améliorer la capacité de ces systèmes de santé à fournir à tous des services de santé financièrement accessibles et de qualité.

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Chapitre 2 – Mères et pères : Éducation, co-résidence et santé des enfants

Le deuxième chapitre, co-écrit avec Élodie Djemaï et Anne-Laure Samson, vise à identifier le rôle respectif joué par l'éducation de la mère et du père sur les investissements effectués pour la santé de leurs enfants ainsi que l'état de santé de ces derniers au Zimbabwe.

Contexte

Depuis les premières formalisations de la *demande de santé* proposées par Grossman [1972], la relation entre éducation et santé des individus constitue un important champ de la littérature économique, aussi bien théorique qu'empirique. Cette relation est complexe : l'éducation d'un individu peut affecter son état de santé, de la même manière que son état de santé durant sa jeunesse peut affecter son niveau d'éducation.

D'importants efforts ont été consentis depuis plusieurs décennies en faveur de l'éducation dans les pays en développement. Les progrès considérables enregistrés ces dernières années sont régulièrement évoqués pour présager une amélioration de l'état de santé de la

population dans les années à venir.

Il existe plusieurs canaux par lesquels l'éducation des individus peut affecter leur santé. *(i)* Le premier est la richesse. Les individus instruits ont tendance à faire face à de meilleures opportunités d'emploi et des salaires plus élevés. Ils peuvent ainsi plus facilement assumer le coût de la prévention, d'une couverture maladie privée et des soins curatifs, ont un meilleur accès aux services de santé, et ont en général des emplois moins pénibles. *(ii)* Les individus éduqués sont aussi plus susceptibles de comprendre les messages de préventions que leurs homologues moins instruits. *(iii)* Les individus éduqués ont également une plus grande incitation à investir dans les comportements de santé préventifs : compte tenu de la différence de salaires, la perte de revenus futurs qu'engendrerait la maladie s'avérerait plus élevée pour les personnes instruites que pour les autres. *(iv)* Enfin, l'éducation peut transmettre des valeurs telles que la discipline, le respect des règles, la nécessité de faire des efforts et l'acceptation des contraintes comme mentionné par [Basu \[2002\]](#). Ainsi, l'éducation en tant que telle peut aider les individus à adopter des comportements préventifs coûteux. La littérature conclut généralement qu'en moyenne les individus les plus instruits sont en meilleure santé et vivent plus longtemps que les autres.

Au-delà de la persistance intergénérationnelle de l'état de santé [[Bhalotra et Rawlings, 2011](#)], ces différents canaux peuvent également expliquer par eux-mêmes pourquoi l'éducation des parents est susceptible d'affecter directement la santé de leurs enfants. Ce chapitre s'intéresse à ces effets intergénérationnels de l'éducation. La littérature existante suggère que l'état de santé d'un enfant est effectivement lié au niveau d'éducation de ses parents. Cependant, les travaux actuels se penchent presque exclusivement sur le rôle joué par les mères, et négligent totalement l'éducation des pères comme facteur d'amélioration de la santé des enfants. Deux raisons pourraient expliquer ce constat. D'abord, ces analyses peuvent refléter l'idée commune selon laquelle les mères comptent davantage que les pères quand il s'agit d'élever les enfants. La seconde raison est purement empirique : les mères étant plus susceptibles de vivre avec leurs enfants que les pères dans la plupart des pays, il est souvent difficile de relier l'état de santé des jeunes enfants au niveau d'éducation de leur père sur la base de données d'enquête. Ce chapitre propose de distinguer le rôle respectif joué par l'éducation de la mère et du père sur les investissements effectués pour la santé de leurs enfants ainsi que l'état de santé de ces derniers dans le cadre d'un pays en développement.

Données et stratégie empirique

Ce chapitre mobilise quatre vagues des Enquêtes Démographiques et de Santé menées au Zimbabwe en 1994, 1999, 2005 et 2010. L'objectif est de comparer investissements et état de santé des enfants de moins de cinq ans selon que leurs parents sont éduqués ou non. L'analyse explore différentes dimensions, à savoir les conditions entourant la grossesse et l'accouchement (visites prénatales, conditions d'accouchement), l'état nutritionnel des enfants (mesures anthropométriques) et les comportements préventifs (vaccination et utilisation d'une moustiquaire).

Estimer simultanément l'effet causal respectif de l'éducation de la mère et du père implique plusieurs défis empiriques.

1. Le premier défi concerne l'endogénéité des variables d'éducation des parents, liée à la corrélation de ces dernières avec des facteurs inobservables susceptibles d'expliquer directement l'état de santé des enfants et les comportements de santé au sein du ménage. Cela renvoie par exemple aux aptitudes et aux préférences temporelles des individus : investir dans l'éducation et la santé implique des investissements coûteux aujourd'hui pour des bénéfices incertains demain. De tels facteurs pourraient biaiser l'estimation de l'effet intergénérationnel de l'éducation sur la santé, et ainsi conduire à des conclusions erronées. Afin de remédier à ce problème, l'accroissement des niveaux d'éducation observé au Zimbabwe suite à une réforme profonde de l'éducation menée en 1980 est utilisé comme source exogène de variation. D'envergure nationale, cette réforme a rendu l'école primaire gratuite et obligatoire, ainsi que l'admission au secondaire automatique. La nature de cette réforme, qui concerne essentiellement les individus nés après 1965, est exploitée dans le cadre d'une approche par variables instrumentales.
2. Le deuxième défi renvoie au fait que tous les enfants ne vivent pas systématiquement avec leurs deux parents. Cela pourrait conduire à un biais dans l'estimation si la probabilité pour un enfant de co-résider avec ses deux parents n'est pas distribuée de manière aléatoire au sein de la population. Dans notre échantillon, seuls 52.7% des enfants de moins de cinq ans vivent avec leurs deux parents, ce qui est susceptible d'influencer leur acquisition de capital humain (Adda et al., 2011; Fitzsimons et Mesnard, 2014). Nous traitons ce problème comme un biais de sélection en suivant la procédure proposée par Heckman [1976], dans la mesure où l'éducation du parent n'est observée que s'il vit dans le même ménage que son enfant. Pour ce faire,

nous exploitons les pratiques au sein de la communauté en matière de divorces, de naissances hors mariage et de polygamie comme sources exogènes de variation pouvant expliquer la probabilité pour un enfant de vivre avec sa mère d'une part, et avec son père d'autre part. Notre analyse de la sélection dans la co-résidence apporte un nouvel éclairage à la littérature actuelle sur la relation éducation-santé qui a jusqu'à présent négligé cette dimension. [Emran et al. \[2018\]](#) documentent cette source de biais, la qualifiant de biais de censure lié à la co-résidence dans les estimations de la mobilité intergénérationnelle.

3. Enfin, nous considérons le phénomène d'homogamie d'éducation, c'est-à-dire la tendance qu'ont les individus ayant des niveaux d'éducation similaires à se marier entre eux, comme source additionnelle de biais dans l'estimation. Ce biais pourrait s'avérer particulièrement important lorsque l'effet de l'éducation de chaque parent est estimé de manière isolée. Si la corrélation entre les niveaux d'éducation des parents est élevée, l'estimation de l'effet du niveau d'éducation de la mère sur les investissements en santé et l'état de santé de l'enfant sans contrôler pour le niveau du père peut, en réalité, capter l'effet de l'éducation de ce dernier. Cette source de biais est mentionnée, bien que non résolue, dans [Carneiro et al. \[2013\]](#). Ce biais peut également être tiré par des facteurs inobservables qui conduisent les individus ayant des niveaux d'éducation similaires à se marier entre eux. Nous tenons compte de ce phénomène en autorisant la corrélation des niveaux d'éducation des parents dans l'estimation de notre modèle.

Ces différentes approches empiriques sont conciliées dans un modèle à équations simultanées.

Principaux résultats

Nous observons à la fois une corrélation très forte de l'éducation des parents avec les comportements de santé au sein du ménage et l'état de santé de leurs enfants, ainsi qu'un phénomène important d'homogamie d'éducation. Lorsque nous considérons de manière isolée le rôle de l'éducation de la mère et que l'endogénéité est prise en compte, nos résultats sont conformes à la littérature existante, à savoir un effet positif significatif de l'éducation sur les investissements en santé et les comportements de prévention.

Toutefois, lorsque l'effet causal du niveau d'éducation des deux parents est estimé simultanément, le rôle de l'éducation de la mère disparaît complètement tandis que le niveau

d'éducation du père continue d'influencer positivement les investissements en santé dans le ménage. Les résultats suggèrent, en effet, que l'éducation du père améliore significativement le recours aux soins prénataux, accroît la probabilité d'accoucher en milieu médicalisé et augmente la vaccination des jeunes enfants. Cette conclusion est robuste à la prise en compte de la sélection dans la co-résidence et à toute une série de tests, portant notamment sur l'échantillon utilisé pour estimer ces effets.

Nos résultats montrent par ailleurs que la sélection dans la co-résidence joue un rôle important dans la détermination de l'état de santé des enfants. En revanche, celle-ci n'interfère pas sur l'estimation de l'effet intergénérationnel de l'éducation des parents sur les investissements en santé dans le ménage et l'état de santé des enfants.

Nous explorons ensuite plusieurs mécanismes par lesquels l'éducation des parents peut influencer la santé de leurs enfants. Le niveau d'éducation du père joue significativement sur les préférences en matière de fertilité au sein du couple, tandis que l'âge de la mère à la première naissance et son utilisation de moyens contraceptifs s'accroissent avec le niveau d'éducation de cette dernière. Enfin, le fait que le père ait atteint le niveau secondaire influence positivement la probabilité de vivre en zone urbaine et le niveau de richesse matérielle du ménage. Cela suggère qu'une partie de l'effet de l'éducation du père sur les soins prénataux et les conditions d'accouchement transite par une plus grande capacité financière des ménages à obtenir des soins et un meilleur accès physique aux infrastructures, en vivant davantage en zone urbaine que rurale. Le niveau d'éducation du père réduit ainsi à la fois la barrière géographique et la barrière monétaire aux soins. En revanche, nous montrons qu'un effet direct de l'éducation du père sur la santé des enfants persiste lorsque le niveau de richesse du ménage et le caractère urbain de sa localité sont pris en compte dans l'estimation.

Implications de politique publique

Nos résultats montrent que ne pas tenir compte simultanément de l'éducation des deux parents dans une seule équation peut conduire à des conclusions erronées : l'effet de l'éducation du père est confondu avec l'effet de l'éducation de la mère en raison de l'homogamie d'éducation.

Les implications de politique publique sont importantes. Nos conclusions suggèrent que des politiques qui cibleraient et impliqueraient davantage les pères pourraient avoir des effets importants et ainsi contribuer significativement à l'amélioration des investissements

en santé dans le ménage, en complément des politiques existantes, très majoritairement axées sur les mères et les futures mères. En particulier, une partie des bénéfices escomptés de l'amélioration des niveaux d'éducation sur la santé transitera probablement par les pères.

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Chapitre 3 – Retenues d'eau et santé en Afrique Subsaharienne

Le troisième chapitre s'intéresse à l'effet causal de la proximité à une retenue d'eau sur la santé des jeunes enfants dans 34 pays d'Afrique Subsaharienne sur la période 1984-2020.

Contexte

Les infrastructures sont régulièrement perçues comme un élément essentiel pour stimuler le développement économique. Les retenues d'eau font partie de ces dernières, en particulier en Afrique Subsaharienne où l'accès à l'eau reste un défi. Des investissements majeurs ont été consentis dans ce domaine depuis une cinquantaine d'années, avec l'appui d'organisations internationales telles que la Banque Mondiale. Que ce soit pour l'irrigation, le bétail, l'utilisation domestique, la prévention des inondations, le secteur minier ou la production hydroélectrique, les retenues d'eau se sont fortement développées sur cette période.

Une part de plus en plus importante de la population devrait dépendre des retenues d'eau dans un futur proche, avec un rôle croissant des solutions de stockage de l'eau face au changement climatique. Leur développement au cours des années à venir est ainsi un objectif affiché de l'Union Africaine. Si leur contribution à la production hydroélectrique – 20.5% de la production électrique totale en Afrique Subsaharienne en 2015 [[Banque Mondiale, 2022](#)] – et à l'irrigation est indéniable, la presse et plusieurs organisations non-gouvernementales se font régulièrement l'écho des effets délétères de la construction de retenues d'eau pour la population locale et l'environnement. Un accroissement du risque infectieux à proximité des barrages a ainsi été reporté, et ce dès le début du XX^{ème} siècle. Toutefois, de tels effets n'ont jamais été explorés de manière causale sur l'ensemble du sous-continent.

L'effet des retenues d'eau sur la santé de la population locale est ambigu.

1. D'une part, les retenues d'eau peuvent avoir des retombées positives, y compris en

matière de santé. La présence d'une retenue d'eau au niveau local offre un accès à une source d'eau supplémentaire, proche des villages, avec un effet potentiellement bénéfique pour la production agricole locale qui pourrait conduire à un effet richesse positif. Cela peut également réduire le coût d'opportunité associé au fait d'aller chercher de l'eau, souvent supporté par les femmes, qui pourraient alors consacrer davantage de temps à des activités génératrices de revenus. Si de tels effets richesse se manifestent, le statut nutritionnel et plus largement l'état de santé moyen pourraient s'améliorer à proximité des retenues d'eau.

2. D'autre part, les retenues d'eau peuvent générer des effets néfastes pour la santé des individus vivant à proximité. Les retenues d'eau conduisent notamment à une augmentation de la salinité des sols, qui, en altérant la productivité agricole, peut conduire à un effet richesse négatif. Elles constituent également des sources d'eau stagnante potentiellement contaminée par des parasites ou des produits phytosanitaires, qui peuvent conduire à des maladies hydriques telles que le choléra ou des diarrhées. Les retenues d'eau créent également des surfaces favorables au développement des moustiques, responsables de la transmission de maladies endémiques comme le paludisme ou la dengue. Enfin, la construction de grandes retenues d'eau peut engendrer le déplacement forcé de la population locale, pouvant conduire à un appauvrissement des ménages en raison de la perte de leurs terres et de leur cercle social, ainsi qu'à des effets néfastes importants en matière de santé mentale. Tous ces effets peuvent sérieusement menacer l'état de la santé de la population vivant aux alentours des retenues d'eau.

Plusieurs scénarios sont alors possibles : ces retenues d'eau pourraient ne générer que des effets positifs, que des effets négatifs, ou bien avoir des effets plus contrastés selon la mesure de santé considérée.

L'objectif de ce chapitre est de mesurer l'effet causal des retenues d'eau sur la santé des enfants de moins de cinq ans vivant aux alentours. L'Afrique Subsaharienne est un terrain particulièrement approprié pour étudier cette question : l'accès à l'eau y demeure problématique, la pauvreté y reste galopante (d'après les données de la [Banque Mondiale \[2022\]](#), 38.3% de la population y vivait avec moins de 1.90\$ par jour en 2019), le statut nutritionnel de la population reste parfois très précaire, et les enjeux liés au paludisme y sont forts. En 2020, le sous-continent enregistrait ainsi 96.3% des cas de paludisme et 97.6% des décès liés au paludisme recensés dans le monde.² Les enfants de moins de cinq

² Calculs de l'auteur à partir des données de l'Organisation Mondiale de la Santé présentées dans le *World*

ans en sont les premières victimes, représentant quatre décès sur cinq liés au paludisme sur le sous-continent cette même année.

Ce chapitre investigate non seulement l'effet agrégé sur le risque de mortalité infantile, mais tire également profit de données très riches permettant de quantifier les effets des retenues d'eau sur le statut nutritionnel des enfants ainsi que leur exposition au paludisme. Plusieurs mécanismes permettant d'expliquer les effets obtenus sur l'état de santé des jeunes enfants sont ensuite explorés et apportent un éclairage nouveau quant aux effets socioéconomiques des retenues d'eau.

Données et stratégie empirique

Ce chapitre combine 108 vagues géolocalisées des Enquêtes Démographiques et de Santé menées dans 34 pays d'Afrique Subsaharienne depuis 1986 avec une base de données unique géoréférençant et datant la construction de plus de 11 700 retenues d'eau sur l'ensemble du sous-continent.

Les données individuelles issues des Enquêtes Démographiques et de Santé couvrent plus de 920 000 enfants de moins de cinq ans et plus de 1.6 million d'adultes âgés de 15 à 54 ans, habitant dans près de 45 500 quartiers et villages en Afrique Subsaharienne. L'état de santé des enfants est mesuré par plusieurs types d'indicateurs : les risques de mortalité à différents âges, des mesures anthropométriques prises au moment de l'enquête (taille et poids) pour capter les effets sur le statut nutritionnel, ainsi que des biomarqueurs relatifs au niveau d'hémoglobine et à la présence de parasites liés au paludisme dans le sang. Des variables déclaratives telles que les cas reportés de fièvre ou de diarrhée au cours des deux semaines qui précèdent l'enquête sont également mobilisées. L'effet sur l'état de santé des adultes, mesuré par leur statut nutritionnel et leur niveau d'hémoglobine, est également exploré.

L'analyse des canaux de transmission se penche ensuite sur les investissements en santé réalisés, les comportements adoptés en matière de fertilité, l'accès aux soins ainsi que sur d'éventuels effets sur le marché du travail et le niveau de richesse matérielle des ménages.

Ce chapitre repose en grande partie sur l'utilisation d'outils issus des systèmes d'information géographique. L'exposition à une retenue d'eau est définie en fonction de sa proximité géographique, de sa date de construction et de la date à laquelle l'état de santé de l'individu est mesuré (information collectée au moment de l'enquête ou de manière rétrospective).

Malaria Report 2021 [OMS, 2021].

Au total, 9.7% des localités présentes dans les Enquêtes Démographiques et de Santé mobilisées dans ce chapitre ont une retenue d'eau à moins de 10 kilomètres au moment de l'enquête.

Le défi méthodologique de ce chapitre réside dans la répartition non-aléatoire des retenues d'eau sur le continent africain. De nombreux facteurs inobservables déterminant la localisation des retenues d'eau peuvent également avoir un effet direct sur la santé des jeunes enfants. Certaines localités peuvent ainsi voir construire des retenues pour tout un ensemble de raisons, notamment politiques et stratégiques, dont il est difficile de tenir compte dans l'estimation. Par exemple, les localités ayant un potentiel de production agricole très élevé ou un secteur industriel en expansion peuvent attirer davantage de retenues d'eau pour l'irrigation et la production d'électricité. À l'inverse, les gouvernements pourraient favoriser les localités les plus pauvres, qui connaissent une faible productivité agricole, pour stimuler le tissu économique local.

Pour faire face à cette localisation non-aléatoire des retenues d'eau et mesurer des effets causaux, l'analyse s'inscrit dans le cadre d'une approche par variables instrumentales. La méthodologie employée repose sur le travail précurseur de [Duflo et Pande \[2007\]](#) basé sur l'ingénierie des retenues d'eau. L'idée est d'utiliser la pente des cours d'eau comme source exogène de variation quant à la capacité de l'environnement local à accueillir une telle infrastructure. En outre, cette approche permet de quantifier les effets des retenues d'eau construites en raison de leur faisabilité technique. À partir de données satellites fournies par la NASA, ce chapitre reconstitue l'ensemble du réseau hydrologique d'Afrique Subsaharienne, et calcule la pente des cours d'eau présents sur le sous-continent.

Cette approche repose sur deux hypothèses : *(i)* les localités ayant des pentes de cours d'eau favorables doivent être plus susceptibles que les autres d'avoir une retenue d'eau ; et *(ii)* la pente des cours d'eau ne doit pas avoir d'effet direct sur la santé des enfants autre qu'au travers de la présence d'une retenue d'eau, conditionnellement aux facteurs pris en compte dans l'estimation. Ces deux hypothèses sont explorées et les analyses menées soutiennent l'interprétation causale des résultats.

Deux extensions à l'analyse principale sont finalement proposées. Celles-ci visent à explorer l'hétérogénéité des effets selon la taille des retenues d'eau, puis les conséquences pour les localités situées en aval.

Principaux résultats

Les résultats obtenus suggèrent un effet contrasté des retenues d'eau : si la malnutrition chronique chez les jeunes enfants recule aux alentours des retenues d'eau, le risque de transmission du paludisme s'y accroît. Les adultes ne sont pas épargnés : leur risque d'être sévèrement anémié, l'un des symptômes du paludisme, augmente également avec la proximité d'une retenue d'eau. Ces conclusions sont en accord avec le risque accru de mortalité post-néonatale (du 28^{ème} jour de vie jusqu'à la veille du premier anniversaire) autour des retenues d'eau, période au cours de laquelle l'immunité des jeunes enfants contre le paludisme acquise de leur mère pendant la grossesse s'étirole.

Cet accroissement du risque d'infection par les parasites responsables du paludisme n'est pas lié à une moindre utilisation des moustiquaires autour des retenues d'eau. Possession et utilisation de moustiquaires apparaissent toutes deux plus élevées aux alentours des retenues d'eau, suggérant d'ailleurs que les individus vivant à proximité sont conscients de leur plus grande exposition aux piqûres de moustique. L'analyse révèle ensuite l'existence d'un important effet richesse des retenues d'eau pour les ménages. Cela reflète notamment les meilleures opportunités observées sur le marché du travail aux alentours des retenues d'eau, les individus ayant une probabilité plus forte d'y occuper un emploi permanent rémunéré. Les résultats suggèrent également une amélioration de l'accès aux soins mais aucun effet perceptible sur les comportements de fertilité, et ce malgré l'effet richesse positif.

Ces effets ne diffèrent pas selon le sexe de l'enfant ou son rang de naissance, suggérant que les retenues d'eau n'affectent pas systématiquement de manière différenciée les enfants d'une même fratrie, que ce soit directement ou indirectement au travers des décisions prises par les ménages en réponse à la proximité d'une retenue d'eau. Les enfants issus de ménages agricoles sont en revanche particulièrement sujets au risque accru de paludisme associé aux retenues d'eau.

Les analyses menées en extension indiquent que la taille de la retenue d'eau est un facteur important, le recul de la malnutrition chronique n'étant observé qu'autour des petites retenues. Enfin, les retenues d'eau situées en amont n'ont globalement pas d'effet sur la santé des enfants.

Implications de politique publique

Ces résultats ont des implications importantes en termes de politique publique. Bien que les retenues d'eau accroissent l'exposition au paludisme, leur construction ne doit pas pour autant être bannie de l'agenda des gouvernements et organisations internationales. Les résultats suggèrent en effet que ces infrastructures génèrent des effets économiques substantiels ainsi qu'une réduction de la malnutrition chronique chez les enfants vivant aux alentours.

En revanche, les localités dotées de ces retenues doivent être la cible de politiques complémentaires visant à atténuer leurs effets néfastes en matière d'exposition au risque de paludisme pour la population locale. Cela peut notamment passer par une intensification des campagnes de prévention dans ces localités, et par le développement de nouveaux outils de prévention en complément des moustiquaires afin d'assurer une protection plus efficace contre les piqûres de moustique.

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General Introduction

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HEALTH refers to a broad set of conditions and can be defined as *a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity*. Following this definition, the enjoyment of the highest attainable standard of health has been recognized as one of the fundamental rights of every human being (WHO, 1946 and Article 25 of the Universal Declaration of Human Rights). Improving health status worldwide is, along with education, considered one of the pillars of shared prosperity. It is now widely acknowledged that poverty is both a cause and a consequence of poor health. Poverty increases the risk of poor health, while poor health traps people in poverty. Tackling poor health is thus intimately linked to the fight against poverty.

Is it all about financial resources? Part of the poor health-poverty relationship is due to financial stress, as the poor usually cannot afford to invest in their health, both preventively and curatively, including through sufficient quantities of food to meet their basic daily nutritional needs. But many factors beyond financial stress make this relationship particularly salient, including low education levels, lack of information on diseases and how to prevent them, incomplete information on the benefits and costs of health investments, adverse environmental factors leading to higher exposure to infectious diseases and weaker capacities of the poor to claim their right to health.

At the same time, adverse effects, including economic ones, of poor health status are widespread and can be observed on the labor market (e.g. Currie and Madrian, 1999; Thirumurthy et al., 2008; García-Gómez et al., 2013; Fletcher, 2014; Lenhart, 2019; Seuring et al., 2019; Jones et al., 2020; Stephens and Toohey, 2022), agricultural productivity (e.g. Dillon et al., 2014; Fink and Masiye, 2015), educational attainment (e.g. Currie and Stabile, 2009; Field et al., 2009; Simo Fotso et al., 2018) and more generally on socioeconomic status (e.g. Meyer and Mok, 2019), making health status a strong determinant of poverty. Households can also be pushed into poverty by catastrophic out-of-pocket health expenditures that lead them to deplete their savings and sell their assets.

This health-poverty trap calls for government intervention, all the more justified as the health sector suffers from important market failures [Ghosh, 2008; Dupas, 2014]. Such market failures include externalities, imperfect information, and under-provision of health infrastructures, making health investments socially suboptimal. Health investments that lead to positive externalities include vaccination, use of insecticide-treated nets to fight mosquito-borne diseases or improved sanitation to limit waterborne diseases. Part of the benefits of such investments extends far beyond the individual who makes them. As the social returns are not accounted for in the private decision to invest or not, the market

equilibrium leads to under-investment compared with what would have been socially optimal. Health behaviors might also generate negative externalities, triggered for example by misuse of health services or over-consumption of drugs that can lead to the development of drug-resistant pathogens. The health sector is also weighed down by imperfect information. Individuals may not have enough information on the relative costs and benefits of health investments to adopt the "right" health behavior, and may suffer from asymmetric information with sellers of health products such as drugs. Individuals' preferences might also be time-inconsistent if they value the expected future benefit of health investment less than its present cost, causing them to under-invest in prevention. Finally, individuals' health behaviors might be shaped by imperfect information on other markets, such as financial ones, leading to credit constraints that could prevent them from investing in their health. Governments might intervene to overcome these market failures, but as discussed further below, they unevenly participate to the achievement of the social optimum.

Despite continuous efforts, health levels remain relatively low in many parts of the World, with a dramatically high concentration of poor health outcomes in Sub-Saharan Africa. While many disorders such as cancers are common to all countries around the World, developing countries face an additional burden of disease resulting from their environment and level of poverty, including tropical and waterborne diseases. As highlighted by Dupas [2011], this additional burden affects people at much younger ages than the one in developed countries and essentially consists in infectious and parasitic diseases with large public health externalities. Sub-Saharan Africa is deeply affected by this burden. In 2019, infectious and parasitic diseases were responsible of 46.5% of all deaths in the subcontinent compared with 9.3% in the rest of the World and 19.7% in South Asia.³

Organized around eight thematic maps, the following pages aim to provide the reader with an overview of the health situation in Sub-Saharan African countries. Section 1 questions the attainment of the *health for all* objective initiated by the Alma-Ata Declaration in 1978, and presents the evolution of the situation since the 1980s. It then depicts where we stand today, while Section 2 focuses on health care resources. This thesis aims to uncover and better understand some factors that either bolster or hinder child health improvement in Sub-Saharan Africa through three independent chapters outlined in Section 3 below.

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³ Author's calculation based on [Global Burden of Disease \[2019\]](#).

1. Health for All?

Thirty years after the creation of the World Health Organization, the Alma-Ata Declaration of 1978 expressed the need for *urgent action by all governments, all health and development workers, and the world community to protect and promote the health of all the people of the world* and declared that *an acceptable level of health for all the people of the world by the year 2000 can be attained through a fuller and better use of the world's resources*. This declaration is historic: for the first time, 134 countries accompanied by many international organizations and non-governmental organizations decided to sit around the table and recognized the urgent need for action to improve global health levels, with an emphasis on primary health care.

1.1. Historical perspectives

1.1.1. *Achieving health for all by the year 2000: from utopian hopes to reality (1978-2000)*

The objective of *health for all by the year 2000* is intrinsically ambitious as it requires governments to ensure a sufficiently high level of health for all people without exception. As of 2000, all countries were expected to develop the appropriate tools and to take the necessary actions to reach this goal. This objective was also particularly ambitious as most of the population in the World was facing very poor health conditions. This is particularly striking when looking at Figure 1. The under-five mortality rate, that is the probability for a child to die before reaching the age of five, was extremely high all over the World, except in North America, Western Europe, some countries in Oceania, and Japan. Achieving health for all thus required considerable effort.

Insufficient financial resources, limited access to technology, and important shortage of qualified medical personnel have rapidly queried the capacity of most countries to achieve this goal. Some progress has been made by the 1990s, but high levels of under-five mortality remained widespread (see Figure 2). More than one child out of four was still dying before reaching the age of five in countries such as Sierra Leone, Liberia, and Niger, the last two of which even experienced an increase in child mortality over the period. Cross-country inequalities were already extremely high, the child mortality rate being up to 50 times higher in low-income countries compared with high-income ones. Two-thirds of countries still had under-five mortality rates above 25 per 1,000 live births. For most countries, the objective was already unachievable, and the spread of HIV/AIDS has put additional

pressure on their health systems.

Twenty-two years after the Alma-Ata declaration, 2000 marks a year of disillusionment. Considerable progress has been made, but it is unevenly distributed across the globe. The *health for all* goal is clearly not reached, and many challenges persist. Sub-Saharan Africa and part of South Asia remain the two regions with the highest under-five mortality rates (Figure 3). Among the 43 countries with an under-five mortality rate above 10%, 37 are in Sub-Saharan Africa, 27 of which had the highest under-five mortality rates in the World that year. Niger appears at the bottom of the ranking, with a child mortality rate of 224.9 per 1,000 live births: a Nigerien child below the age of five died every four minutes in 2000.

1.1.2. A first integrated approach to global health: fifteen years of unprecedented progress (2000-2015)

At the turn of the 21st century, the United Nations Millennium Declaration, signed by 189 countries, renews the hope of ensuring better health for all by 2015 and, more broadly, of eradicating poverty in the World. The resulting Millennium Development Goals (MDGs) have galvanized unprecedented international efforts to achieve a better life for all. Out of eight MDGs, four are explicitly focused on health improvement, and two additional goals make reference to it (see Box 1). The objectives are again ambitious: eradicate hunger (part of MDG 1), reduce child mortality (MDG 4), improve maternal health (MDG 5), and combat HIV/AIDS, malaria, and other diseases (MDG 6) by 2015.

Box 1. Health-related Millennium Development Goals

Out of eight Millennium Development Goals (MDGs), four were explicitly targeted toward the improvement of health status in the World:

MDG 1 - Eradicate extreme poverty and hunger

- *Target 1.C.* Halve, between 1990 and 2015, the proportion of people who suffer from hunger

MDG 4 - Reduce child mortality

- *Target 4.A.* Reduce by two-thirds, between 1990 and 2015, the under-five mortality rate

MDG 5 - Improve maternal health

- *Target 5.A.* Reduce by three quarters, between 1990 and 2015, the maternal mortality ratio
- *Target 5.B.* Achieve, by 2015, universal access to reproductive health

MDG 6 - Combat HIV/AIDS, malaria and other diseases

- *Target 6.A.* Have halted by 2015 and begun to reverse the spread of HIV/AIDS
- *Target 6.B.* Achieve, by 2010, universal access to treatment for HIV/AIDS for

all those who need it

- *Target 6.C.* Have halted by 2015 and begun to reverse the incidence of malaria and other major diseases

Two additional Millennium Development Goals are related to the improvement of health status worldwide:

MDG 7 - Ensure environmental sustainability

- *Target 7.C.* By 2015, halve the proportion of people without sustainable access to safe drinking water and basic sanitation

MDG 8 - Develop a global partnership for development

- *Target 8.E.* In cooperation with pharmaceutical companies, provide access to affordable essential medicines in developing countries



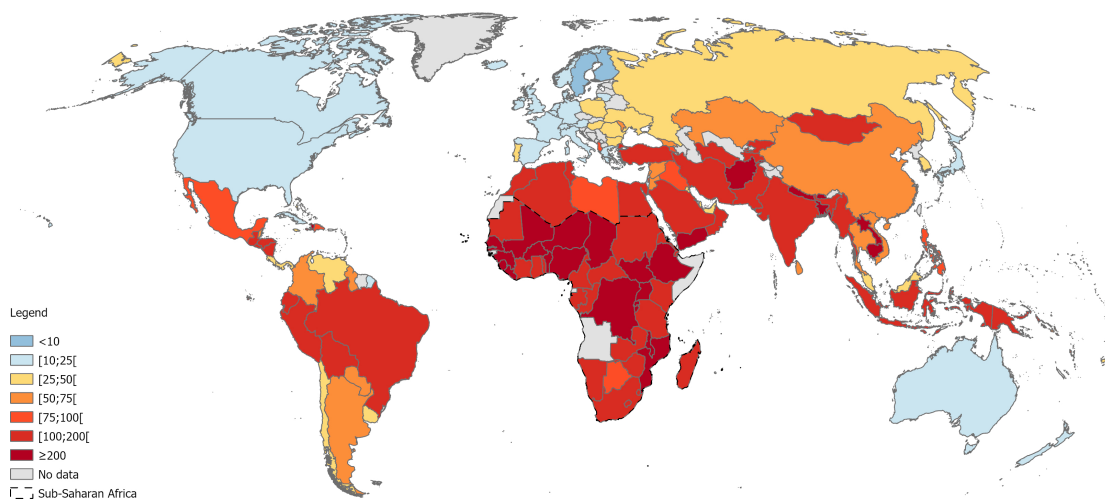
MDG 4 is dedicated to the reduction of child mortality, with the aim of reducing by two-thirds under-five mortality between 1990 and 2015 (Target 4.A). This is particularly challenging for Sub-Saharan African countries, still struck by very high under-five mortality rates and where little progress has been made between 1990 and 2000. Almost everything remains to be done, and considerable efforts are needed to reach this target.

In 2015, fifteen years after the Millennium Declaration, the time has come to take stock. Again, progress towards reaching the MDGs is unequal across countries. The situation has dramatically improved, including in Sub-Saharan Africa. The progress made by some countries is striking. Under-five mortality rate was divided by 3.5 between 1990 and 2015 in Niger,⁴ from 329.6 to 93.8 per 1,000 live births. Eleven Sub-Saharan African countries have recorded under-five mortality rates below 50 per 1,000 live births, including Botswana, Kenya, Rwanda, Senegal, and South Africa.

Nonetheless, the scope for improvement remains high and the situation particularly worrying. Eight Sub-Saharan African countries still have under-five mortality rate above 10% (Burkina Faso, Central African Republic, Chad, Guinea, Mali, Nigeria, Sierra Leone, Somalia) and Sub-Saharan Africa as a whole did not realize any of the health-related MDGs. The proportion of undernourished people was divided by 1.4 instead of 2 (MDG Target 1.C.), the under-five mortality rate was reduced by 52% instead of two-thirds (MDG Target 4.A.), maternal mortality ratio decreased by 49% instead of three quarters (MDG Target 5.A.), the unmet need for family planning remains high, and only half of pregnant women attended at least four antenatal visits as recommended by the World Health Organization (MDG Target 5.B.) based on the MDGs achievement report [[United Nations, 2015](#)].

⁴ As a matter of comparison, over the 1978-2000 period, this rate was divided by 1.5, from 328.6 to 224.9 deaths per 1,000 live births.

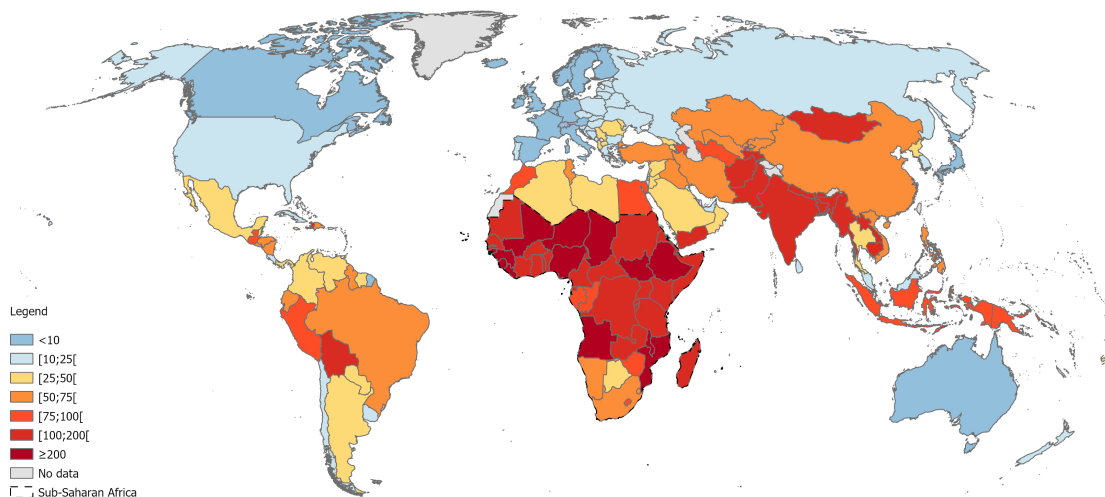
Figure 1. Under-five mortality rate in 1978 per 1,000 live births



Source: Author's computation based on [World Bank \[2022\]](#).

Notes: The map shows the country-specific under-five mortality rate per 1,000 live births in 1978.

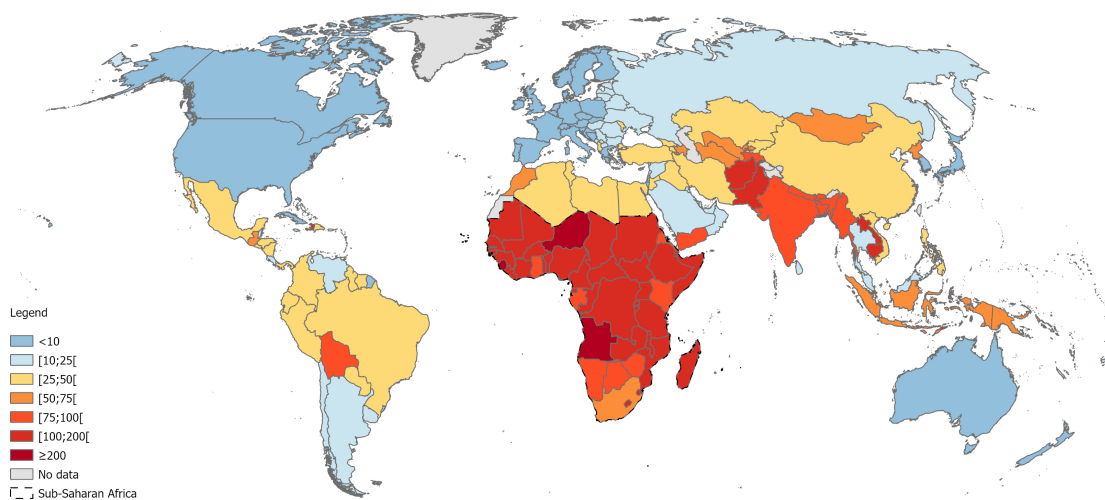
Figure 2. Under-five mortality rate in 1990 per 1,000 live births



Source: Author's computation based on [World Bank \[2022\]](#).

Notes: The map shows the country-specific under-five mortality rate per 1,000 live births in 1990.

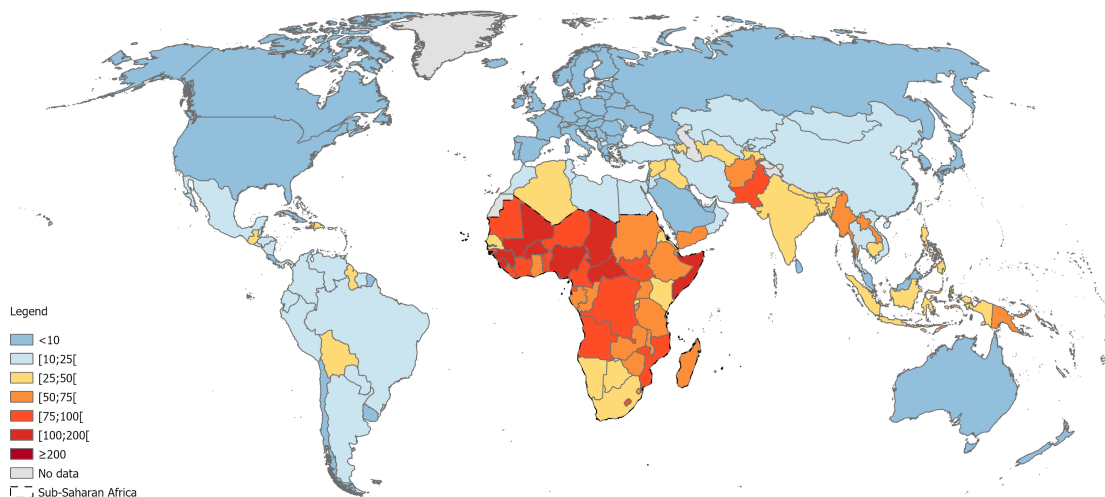
Figure 3. Under-five mortality rate in 2000 per 1,000 live births



Source: Author's computation based on [World Bank \[2022\]](#).

Notes: The map shows the country-specific under-five mortality rate per 1,000 live births in 2000.

Figure 4. Under-five mortality rate in 2015 per 1,000 live births



Source: Author's computation based on [World Bank \[2022\]](#).

Notes: The map shows the country-specific under-five mortality rate per 1,000 live births in 2015.

Sub-Saharan Africa was on its way to achieving MDG Target 6.A., with a 46% reduction in the number of new HIV infections between 2000 and 2015 (instead of 50%). Still, access to treatment for HIV/AIDS was far from universal⁵ (MDG Target 6.B.). Malaria and tuberculosis incidence dropped by 35% and 19% respectively,⁶ well below the expected 50% reduction (MDG Target 6.C.).

1.1.3. A new agenda for increasingly ambitious targets (2015-2030)

As a result, the international community renews its commitment to ensuring health for all, now by 2030 through the Sustainable Development Goals 2 ("zero hunger") and 3 ("good health and well-being") with more comprehensive and increasingly ambitious targets (see Box 2). In particular, Target 3.2 sets the objective of reducing under-five mortality to at least as low as 25 per 1,000 live births by 2030. In 2015, except in four small countries of the subcontinent (Cabo Verde, Mauritius, São Tomé, and Príncipe and Seychelles), no Sub-Saharan African country met this criterion, and some of them have under-five mortality rates four (Burkina Faso, Central African Republic, Guinea, Mali) to five times higher (Chad, Nigeria, Somalia, Sierra Leone), highlighting the significant progress needed to achieve this goal.

Box 2. Health-related Sustainable Development Goals

Out of 17 Sustainable Development Goals (SDGs), two are focused on achieving better health for all:

SDG 2 - End hunger, achieve food security and improved nutrition and promote sustainable agriculture

- *Target 2.1.* By 2030, end hunger and ensure access by all people, in particular the poor and people in vulnerable situations, including infants, to safe, nutritious and sufficient food all year round
- *Target 2.2.* By 2030, end all forms of malnutrition, including achieving, by 2025, the internationally agreed targets on stunting and wasting in children under five years of age, and address the nutritional needs of adolescent girls, pregnant and lactating women and older persons

SDG 3 - Ensure healthy lives and promote well-being for all at all ages

- *Target 3.1.* By 2030, reduce the global maternal mortality ratio to less than 70 per 100,000 live births
- *Target 3.2.* By 2030, end preventable deaths of newborns and children under 5 years of age, with all countries aiming to reduce neonatal mortality to at least as low as 12 per 1,000 live births and under-five mortality to at least as low as 25 per 1,000 live births

⁵ According to estimations from the World Health Organization, antiretroviral therapy coverage only reached 24% of people living with HIV in 2010, 51% in 2015 and 75% in 2020 [WHO, 2022a].

⁶ Author's calculation based on WHO [2021d] and WHO [2021c] databases.

- *Target 3.3.* By 2030, end the epidemics of AIDS, tuberculosis, malaria and neglected tropical diseases and combat hepatitis, water-borne diseases and other communicable diseases
- *Target 3.4.* By 2030, reduce by one third premature mortality from non-communicable diseases through prevention and treatment and promote mental health and well-being
- *Target 3.5.* Strengthen the prevention and treatment of substance abuse, including narcotic drug abuse and harmful use of alcohol
- *Target 3.6.* By 2020, halve the number of global deaths and injuries from road traffic accidents
- *Target 3.7.* By 2030, ensure universal access to sexual and reproductive health-care services, including for family planning, information and education, and the integration of reproductive health into national strategies and programs
- *Target 3.8.* Achieve universal health coverage, including financial risk protection, access to quality essential health-care services and access to safe, effective, quality and affordable essential medicines and vaccines for all
- *Target 3.9.* By 2030, substantially reduce the number of deaths and illnesses from hazardous chemicals and air, water and soil pollution and contamination

One additional goal focuses on ensuring availability of water and sanitation for all:

SDG 6 - Ensure availability and sustainable management of water and sanitation for all

- *Target 6.1.* By 2030, achieve universal and equitable access to safe and affordable drinking water for all
- *Target 6.2.* By 2030, achieve access to adequate and equitable sanitation and hygiene for all and end open defecation, paying special attention to the needs of women and girls and those in vulnerable situations
- *Target 6.3.* By 2030, improve water quality by reducing pollution, eliminating dumping and minimizing release of hazardous chemicals and materials, halving the proportion of untreated wastewater and substantially increasing recycling and safe reuse globally



1.2. Where do we stand?

The important progress made over the last forty years is undeniable but is unevenly distributed across and within countries. Most Sub-Saharan African countries are still lagging behind on the road to *health for all*. I here depict the current situation faced by the subcontinent.

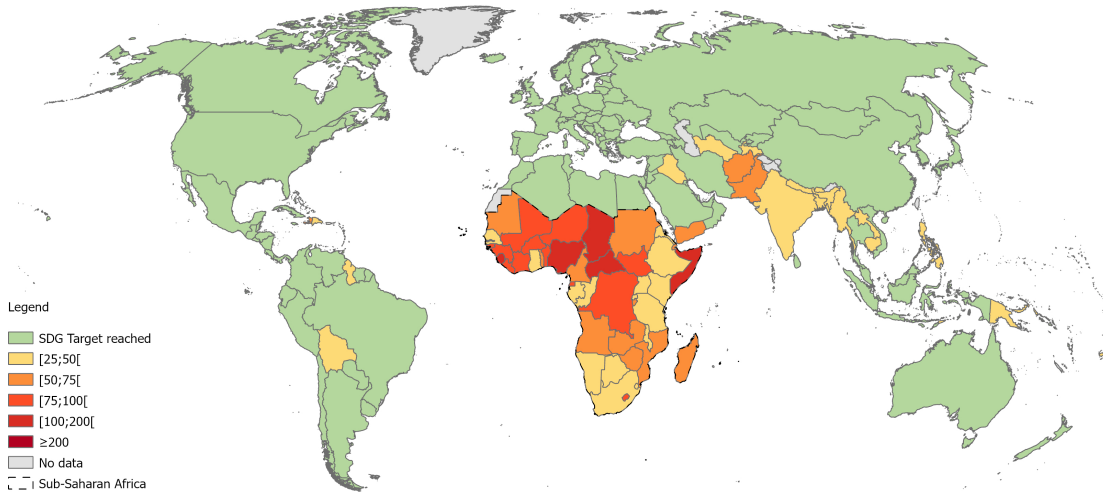
1.2.1. A still tragically high incidence of child mortality

Figure 5 maps the under-five mortality rate in 2020 and symbolizes in green countries with less than 25 under-five deaths per 1,000 live births and thus which have already reached SDG Target 3.2. Of the 70 countries which are still on the way to achieving it, 45 are in Sub-Saharan Africa.⁷ The situation is particularly worrying in Chad, the Central African

⁷ Only Cabo Verde, Seychelles, Mauritius, and São Tomé and Príncipe have already achieved this target.

Republic, Nigeria, Sierra Leone, and Somalia, where the under-five mortality rate is still more than four times higher than this threshold.

Figure 5. Under-five mortality rate in 2020 per 1,000 live births



Source: Author's computation based on [World Bank \[2022\]](#).

Notes: The map shows the country-specific under-five mortality rate per 1,000 live births in 2020. The Sustainable Development Goals (SDG) Target 3.2 aims to reduce under-five mortality to at least as low as 25 deaths per 1,000 live births by 2030.

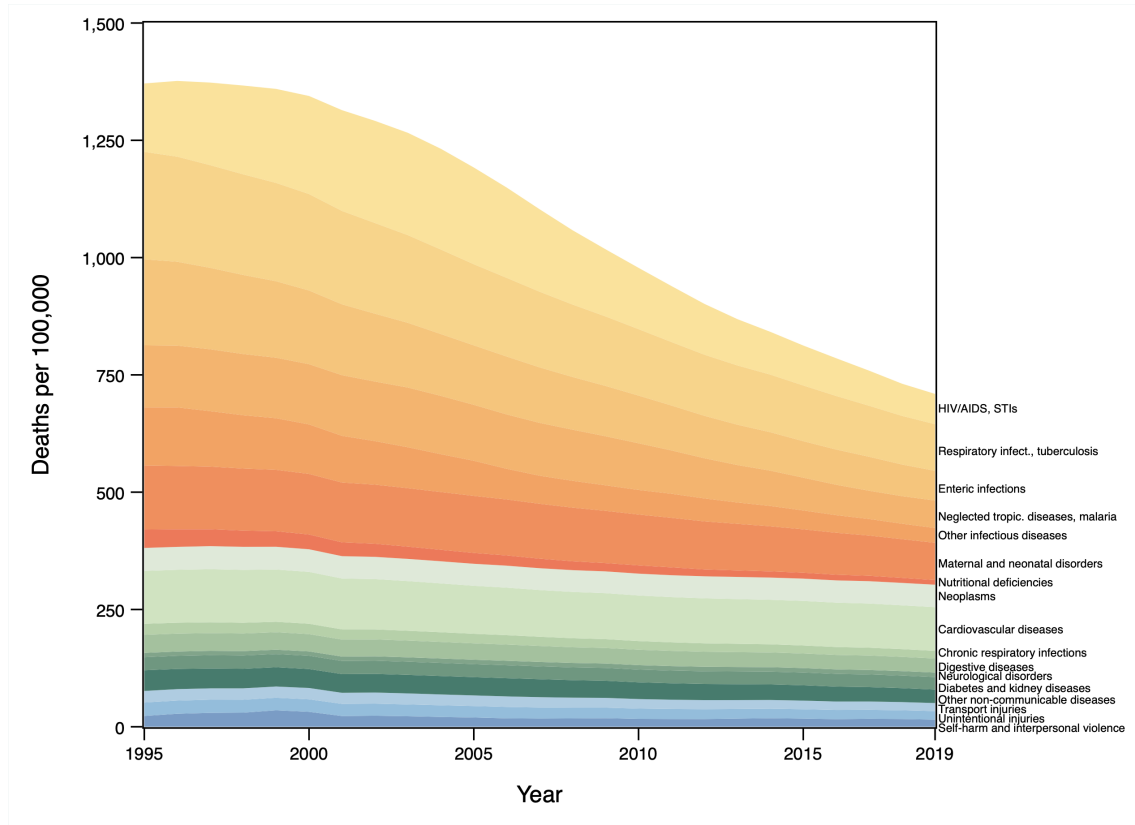
1.2.2. Preventable and treatable conditions remain the first cause of death

Figure 6 shows the evolution of causes of death in Sub-Saharan Africa over the 1995-2019 period, grouped into three mutually-exclusive categories following the International Classification of Disease: Communicable, maternal, neonatal, and nutritional disorders (Group I), non-communicable diseases (Group II) and injuries (Group III). Appendix Figure A.1 replicates this figure for the rest of the World.

Three main patterns emerge from Figure 6. First, the subcontinent has made remarkable progress: the death rate in Sub-Saharan Africa now aligns with the one of the rest of the World. Second, the considerable efforts carried out to fight infectious and waterborne diseases have led to a huge decline in the death rate attributable to Group I disorders, from 990 deaths per 100,000 inhabitants in 1995 to 407 deaths per 100,000 inhabitants in 2019. All components of Group I have seen their death rate more than halved over this period, except maternal and neonatal disorders (-42%). Nevertheless, communicable, maternal, neonatal, and nutritional disorders, many of which are considered easily preventable and treatable causes of death, still account for 57% of all deaths in Sub-Saharan Africa in 2019. This is almost five times higher than in the rest of the World. Finally, non-communicable

diseases (Group II) only account for 36% of all deaths in Sub-Saharan Africa the same year, compared with 80% in the rest of the World.

Figure 6. Evolution of causes of death in Sub-Saharan Africa



Source: Author's computation based on [Global Burden of Disease \[2019\]](#).

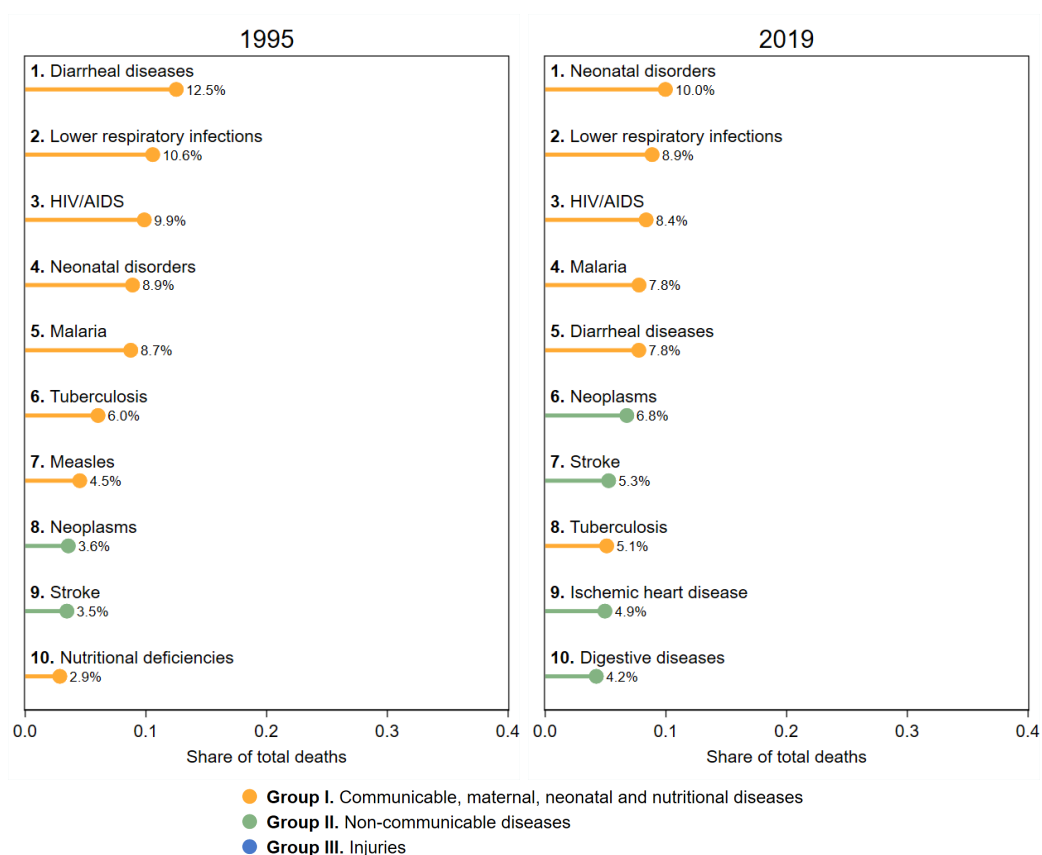
Notes: The figure plots the evolution of the cause-specific number of deaths per 100,000 inhabitants in Sub-Saharan Africa over the 1995-2019 period. Shades of orange stand for deaths caused by communicable, maternal, neonatal and nutritional disorders (Group I). Shades of green stand for deaths attributable to non-communicable diseases (Group II). Shades of blue stand for deaths imputable to injuries (Group III).

1.2.3. The progress made reveals new challenges for still fragile health care systems

If the efforts made so far, although insufficient, have undoubtedly improved the health situation worldwide, such progress will become increasingly hard to achieve. This essentially raises three challenges for the coming years. The first one consists in reaching marginalized populations who have generally not (or not fully) benefited from the improvements mentioned before. Such populations might be difficult to reach because of their remoteness and for security reasons. The second challenge refers to the massive investments needed to create healthier environments through adequate water and sanitation infrastructures and to improve the quality of care. The third challenge is related to a shift in demand for more advanced health services such as cardiology and ophthalmology, for which most

Sub-Saharan African health systems are not prepared. Figure 7 presents the top-10 causes of death in Sub-Saharan Africa in 1995 and 2019.⁸ The number of Group II diseases in this ranking has doubled over the period, with an increase in the share of deaths attributable to neoplasms, stroke, ischemic heart disease, and digestive diseases. Such health issues have always existed but are now more salient as the share of Group I disorders has dropped. This shift will necessitate considerable investments in infrastructures, equipment, and human resources to prevent, diagnose and treat such diseases in order to follow the changing population health needs.

Figure 7. Top 10 causes of death in Sub-Saharan Africa in 1995 and 2019



Source: Author's computation based on [Global Burden of Disease \[2019\]](#).

Notes: The figure plots the top-10 causes of deaths in Sub-Saharan Africa (all ages) in 1995 (left-hand side) and 2019 (right-hand side) expressed as a share of total deaths. Orange dots stand for deaths caused by communicable, maternal, neonatal and nutritional disorders (Group I). Green dots stand for deaths attributable to non-communicable diseases (Group II).

*
* *

⁸ Appendix Figures B.1 and B.2 show the top-10 causes of death in the World excluding Sub-Saharan Africa and in OECD countries, respectively.

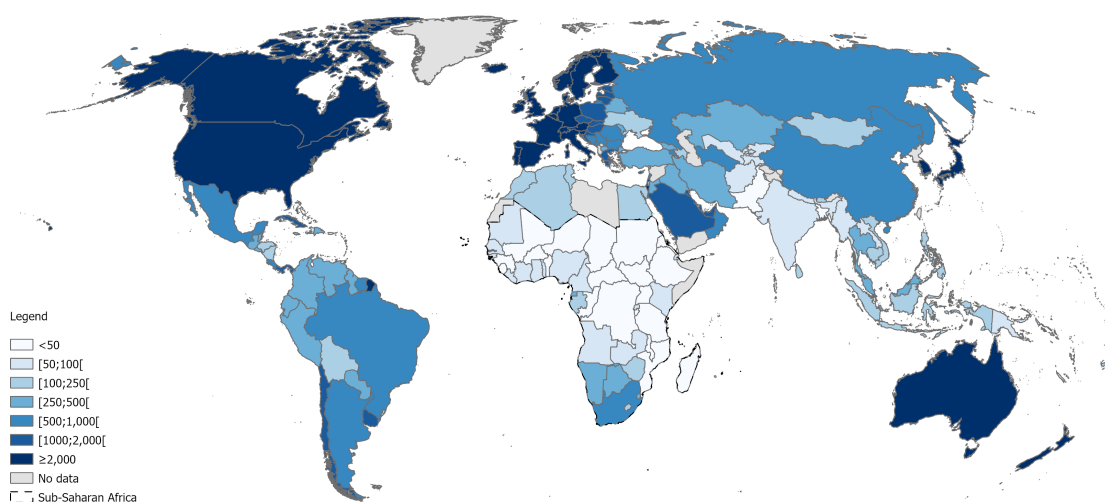
2. Health Care Resources

Most of the progress made over the past forty years has been driven by medical innovations made in high-income countries,⁹ while investments in public health and sanitation remain extremely scarce in most lower-income countries. Health systems in most developing countries, particularly in Sub-Saharan Africa, suffer from a lack of both financial and human resources to meet the actual and future needs of the population.

2.1. Health spending per capita remains low in many Sub-Saharan African countries

Sub-Saharan Africa is home to most of the countries with very low levels of health spending per capita (Figure 8). In countries such as Burundi, the Democratic Republic of the Congo, Madagascar, and South Sudan, the annual health spending is below US\$ 25 per capita in 2019, which is 34 and 180 less than in Brazil and France, respectively. Cross-country disparity is important as health spending exceeds US\$ 400 per capita in countries like Botswana, Namibia, and South Africa.

Figure 8. Health spending per capita in 2019 (US\$)



Source: Author's computation based on the Global Health Expenditure Database [WHO, 2021b].

Notes: The map shows the country-specific health spending per capita in 2019.

Substantial investments are needed to meet the ambitious agenda of the Sustainable Development Goals. Stenberg et al. [2017] estimate that, on average, an additional US\$ 76

⁹ Such innovations cover products like antibiotics, antimalarial drugs, insecticides, oral rehydration salts, treatment for tuberculosis and HIV/AIDS, vaccines or water purification tablets.

health spending per capita per year is needed by 2030 in low-income countries¹⁰ to reach the different targets from Sustainable Development Goal 3 - *Ensure healthy lives and promote well-being for all at all ages*, with substantial investments needed in infrastructures and health workforce. Such a scenario would require a doubling of health spending in many Sub-Saharan African countries, which many of them are currently unable to do, suggesting great difficulties in their road to SDG 3.

2.2. Out-of-pocket spending still accounts for a large part of health spending, and the share of external aid is on the rise

As shown in Figure 9, health spending in Sub-Saharan Africa heavily relies on out-of-pocket spending. In 2019, out-of-pocket spending represents on average 37% of total health spending, down ten percentage points from 2000, a reduction partly driven by a return to free health care policies (see Box 3 for a brief history of user fees policy in Sub-Saharan Africa). However, this is still 16 percentage points more than in high-income countries [WHO, 2021a]. This situation is particularly alarming given the important levels of deprivation observed in Sub-Saharan Africa. Wagstaff et al. [2018] estimate that 11.4% of people in Africa live in households whose out-of-pocket health expenditures represent at least 10% of their total consumption in 2010, a 2.7 percentage points increase compared with 2000.

Box 3. A brief history of user fees policy in Sub-Saharan Africa

Access to basic public services is considered an essential tool to fight poverty and inequalities. Financial stress is a major barrier that usually prevents households from making optimal investments in human capital. Governments are torn between two options: to make access to such services free, at the risk of not being able to ensure their financial sustainability and good quality, or to charge users to drain additional financial resources. As a result, governments are groping around on this issue. Historically, the way African governments and the international community have considered the best pricing policy to be implemented is characterized by three main phases.

Following their independence (1950s-1960s) numerous African countries chose to provide free healthcare for all, considering it as a necessity to improve population health. Their objective was more generally to ensure fair access to human capital for all. Nevertheless, this policy failed to improve population health, mainly because of limited human and financial resources, resulting in poor quality of health services provided. In particular, the economic slowdown of the 1970s and 1980s reduced States' capacity

¹⁰ US\$ 58 in lower middle-income countries and US\$ 51 in upper middle-income countries. Under a more resource-constrained scenario, the amount needed to make progress towards SDG 3 remains high, at US\$ 66 on average in low-income countries [Stenberg et al., 2017].

to invest in their social sector [Akin et al., 1987].

While many developing countries, struck by severe economic difficulties, had to cut back their public health budgets during this period of structural adjustments, international organizations such as the United Nations Children’s Fund (UNICEF), the World Bank, and the World Health Organization rapidly promoted a health care financing scheme based on cost sharing [Akin et al., 1987; Yates, 2009]. The argument to support such a financing scheme, which emerged following the ambitious objectives set by the Alma-Ata Declaration as an attempt to strengthen primary health care, was threefold: (i) to ensure the financial sustainability of national health systems in developing countries with the underlying objective of improving the quality of care; (ii) to reduce the population’s misuse of health services that may contribute to their poor efficiency; and (iii) to improve access to primary health care for the poorest [Akin et al., 1987]. This last argument is based on the fact that when user fees were removed, it benefited almost exclusively the richest, often urban and located close to health facilities. Conversely, the poorest, essentially located in rural areas, did not benefit from free health care. Hence, such a policy seemed to be fair, but in reality, it increased inequities in access to health care between the rich and the poor. By generating additional revenues for the health sector, user fees were expected to finance huge investments in health infrastructures for the most deprived areas. The objective was to reduce spatial inequalities in terms of access to health infrastructures with a cross-subsidize mechanism between urban (rich) and rural (poor) areas. Therefore, during the 1980s and 1990s, many Sub-Saharan countries opted for cost-sharing in the public health sector. In particular, the Bamako Initiative, adopted by African Ministers of health in 1987, and supported by UNICEF and the World Health Organization, called for the design and implementation of local self-financing mechanisms, including cost-sharing policies.

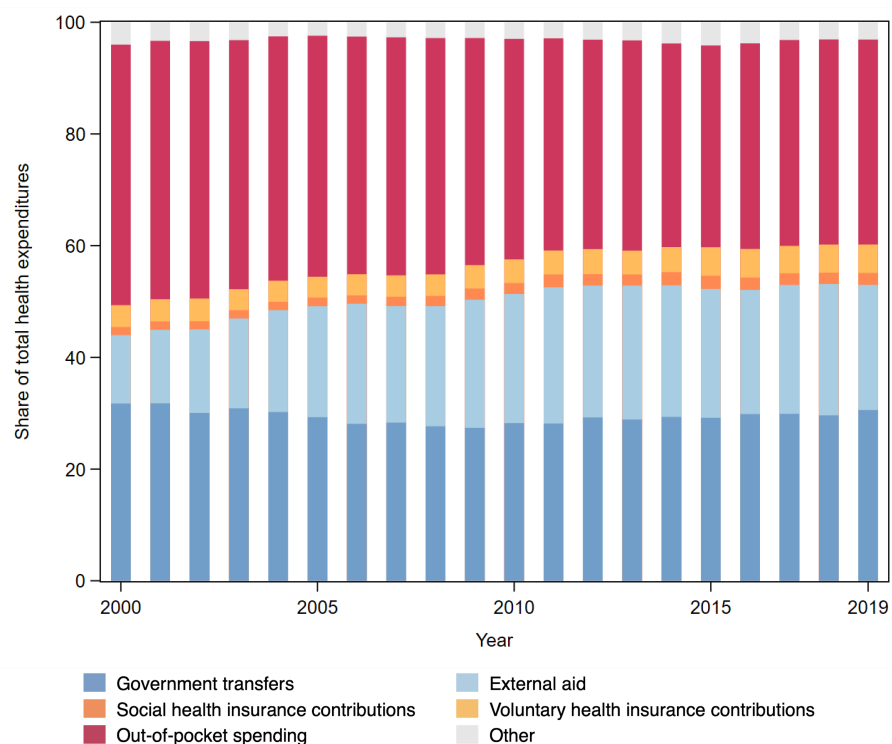
However, a growing literature has emphasized the counterproductive effect of such a policy in terms of public health [Gertler et al., 1987]. It has also been argued that user fees did not raise substantial funds since they only represent between 5% and 9% of public health budgets [Gilson, 1997; Pearson, 2004]. This policy had a negative impact on equity as well as on the health of the poorest since it represents an additional barrier to seeking health care [Yates, 2009; Witter, 2005]. In response to growing inequalities, the World Bank and the World Health Organization decided to update their guidelines in favor of free health care, at least for the most vulnerable populations such as pregnant women and young children [WHO, 2010]. This new strategy is part of an overall effort of the international community to fight against poverty and inequalities, first with the Millennium Development Goals (MDG) and then with the Sustainable Development Goals (SDG).

□

Health insurance schemes remain scarce: in 2019, social health insurance contributions and voluntary health insurance contributions only cover 2% and 5% of total health spending, respectively. As health insurance contributions, the share of government transfers remained very stable over the 2000-2019 period, accounting for 27% to 32% of total health spending depending on the year. Figure 9 reveals a striking pattern: the decrease in the share of out-

of-pocket spending over the period was essentially compensated by an increase in external aid allocated to health, with a 69% increase over the period (covering 85% of the decrease in the share of out-of-pocket spending). If such an increase mostly reflects the international commitments to achieve the *health for all* objective, the situation is particularly critical since more than one-fifth of health financing in Sub-Saharan Africa now relies on external aid, which can be very volatile depending on the international environment.

Figure 9. Evolution of health spending components in Sub-Saharan Africa



Source: Author's computation based on the Global Health Expenditure Database [WHO, 2021b].

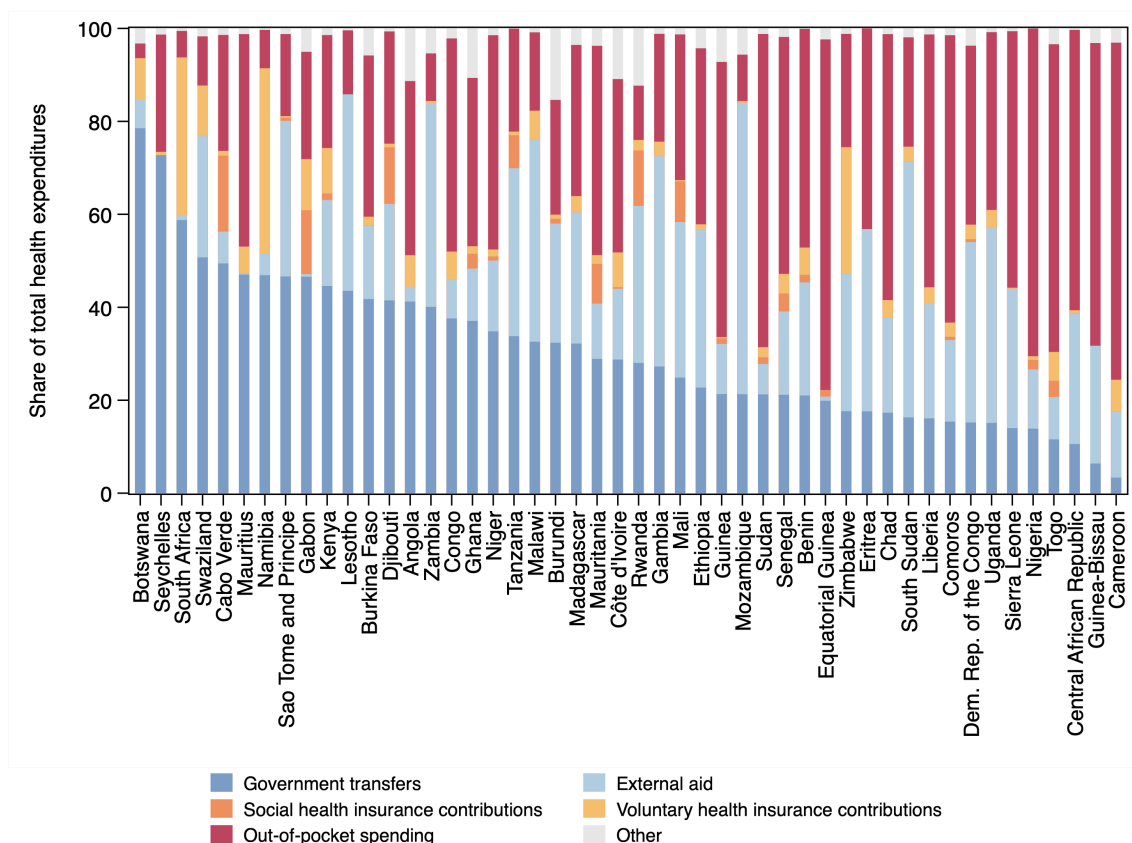
Notes: The figure shows the evolution of health spending components as a share of total health spending in Sub-Saharan Africa over the 2000-2019 period. Other sources include contributions from domestic non-governmental organizations, compulsory private insurance schemes and enterprise schemes including health services provided by enterprises for their employees.

Health financing sources vary greatly from one country to another. Figure 10 shows the structure of funding source of health spending in all Sub-Saharan African countries (except Somalia, for which information is not available) in 2019, while Appendix C reports the country-specific evolution over the 2000-2019 period. In Cameroon and Equatorial Guinea, out-of-pocket spending represents 73% and 75% of total health spending in 2019 while in Botswana and South Africa, their share is below 6%.

Many Sub-Saharan countries heavily rely on foreign aid to fund their health spending.

In 21 countries out of 48, external aid accounts for at least one-quarter of total health spending. The most extreme situation is found in Mozambique and South Sudan, where external aid covers more than half of total health spending.

Figure 10. Health spending components by countries in 2019

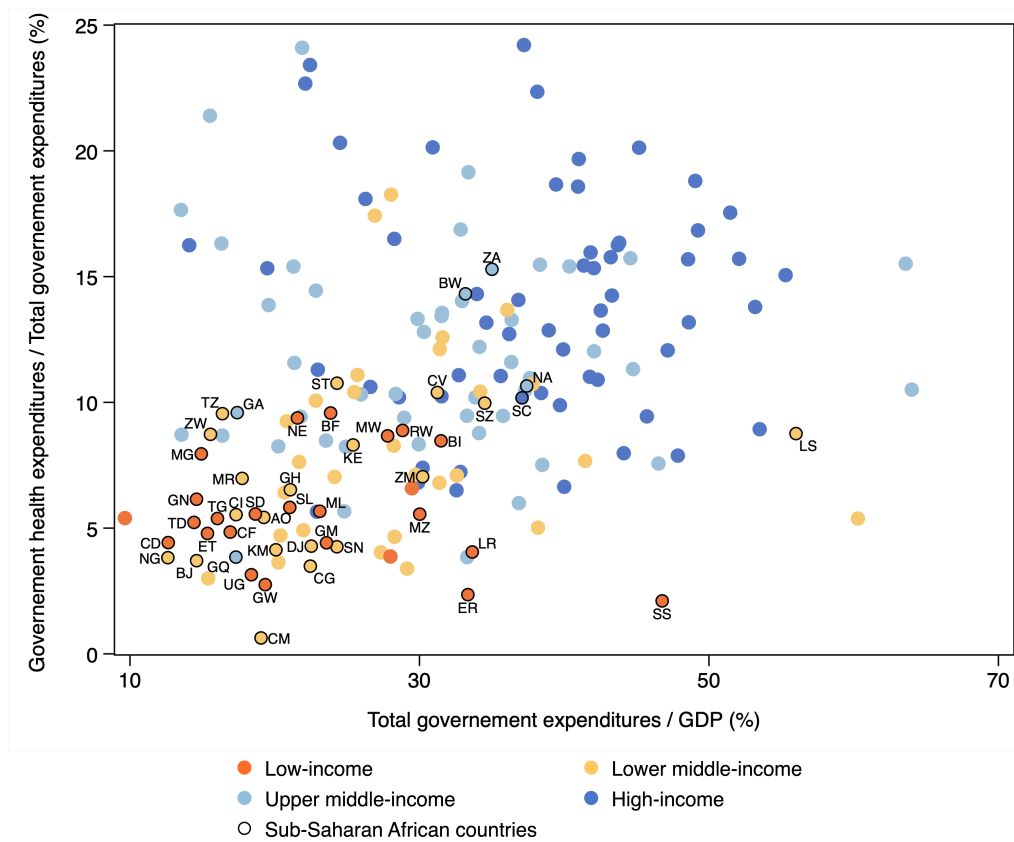


Source: Author's computation based on the Global Health Expenditure Database [WHO, 2021b].
Notes: The figure shows the country-specific health spending components as a share of total health spending in 2019. No information available for Somalia. Other sources include contributions from domestic non-governmental organizations, compulsory private insurance schemes and enterprise schemes including health services provided by enterprises for their employees.

Health insurance schemes are now well established in some countries. Voluntary health insurance schemes account for a significant part of health spending in Namibia (40%), South Africa (34%), and Zimbabwe (27%). Social health insurance schemes are less developed but contribute to a non-negligible part of health spending in countries like Cabo Verde (16%), Djibouti (12%), Gabon (14%), and Rwanda (12%). Nevertheless, such insurance schemes are still nascent, if not non-existent, in many countries. The design of health insurance schemes adapted to the needs and financial capacity of households is a crucial issue for the subcontinent in the years to come.

Figure 10 also illustrates an important heterogeneity in terms of government participation in health spending, which, in 2019, varies from 3% in Cameroon to 79% in Botswana. Such differences might either reflect different fiscal capacities of the governments, a different allocation of resources, or a combination of the two. Figure 11 investigates this point by plotting the share of total government spending devoted to health in 2019 as a function of fiscal capacity the same year proxied by the total government spending-to-GDP (Gross Domestic Product) ratio, following WHO [2021a].

Figure 11. Health priority in government spending



Source: Author's computation based on the Global Health Expenditure Database [WHO, 2021b].
Notes: The figure plots the share of total government spending devoted to health as a function of the total government spending-to-GDP ratio in 2019. Sub-Saharan African countries are highlighted by black circles.

Three main patterns emerge from this figure. First, high-income countries tend to have a higher fiscal capacity and devote a higher share of their budget to health than the other ones, as shown by the concentration of dark blue dots in the upper-right corner of the figure. Second, the scattered distribution of upper-middle income countries, and to a lower extent of lower-middle ones, over the entire quadrant suggests that the government prioritization of health spending along the development process is not straightforward. Third, there are

large variations in health priority within each income group and more generally for a given level of fiscal capacity, suggesting that prioritization of health spending is more of a policy choice than a result of fiscal capacity [WHO, 2021a]. For example, Cameroon has a total government spending-to-GDP ratio similar to the one of Thailand but devotes more than 20 times less of its budget to health (0.6% vs. 13.9%). The average health priority in government spending has slightly decreased in Sub-Saharan Africa since 2000, from 7.5% to 6.8% of total spending in 2019, similar to the share of military expenditures (6.5%).

2.3. Health workers shortage is a pressing issue for Sub-Saharan Africa

Along investments in health infrastructures, an increase in staff expenditures was identified as a key element to meet Sustainable Development Goal 3. Based on data from 67 low- and middle-income countries, Stenberg et al. [2017] estimate that an additional 23.6 million health workers will be needed by 2030 to achieve SDG 3 targets, accounting for 41% of the total projected additional health spending required. WHO [2016b] forecasts that 1.1 million physicians, 2.8 million nurses and midwives, and 2.2 million health workers from other cadres¹¹ will be missing in the WHO African Region in 2030 to meet population health needs.

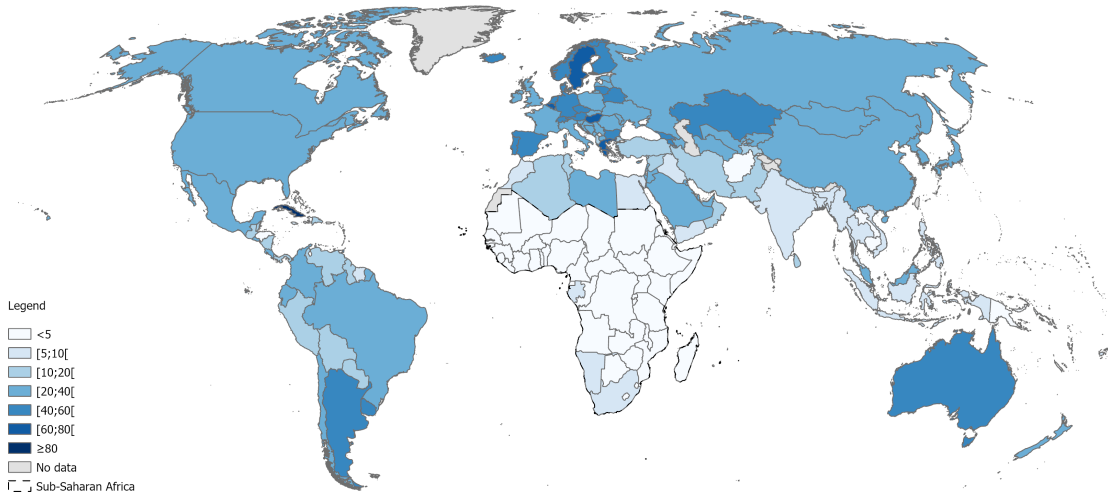
Figures 12 and 13 respectively map the most recent data on the density of physicians and of nursing and midwifery personnel per 10,000 inhabitants. Health workers shortage appears as a pressing issue for Sub-Saharan Africa. In 2006, the minimum recommended medical density, including physicians as well as nursing and midwifery personnel, was established at 22.8 per 10,000 inhabitants by the World Health Organization [WHO, 2006].¹² Many countries have still not reached this threshold, most of which are located in Sub-Saharan Africa. For example, in 2018, the medical density per 10,000 inhabitants stood at 3.28 in the Central African Republic, 4.97 in Madagascar, 5.7 in Mali, and 6.18 in Tanzania.¹³ These figures mask a very inequitable distribution of health professionals between urban and rural areas, the latter being largely underserved. This shortage of trained health professionals is often compensated by informal health providers with little, if any, medical training.

¹¹ Including dentists, pharmacists, environment and public health personnel, laboratory health workers, community and traditional health workers, as well as health management and support staff.

¹² This threshold was then revised to 44.5 per 10,000 inhabitants in 2016 based on 12 health indicators linked to the achievement of Sustainable Development Goal 3 [WHO, 2016b].

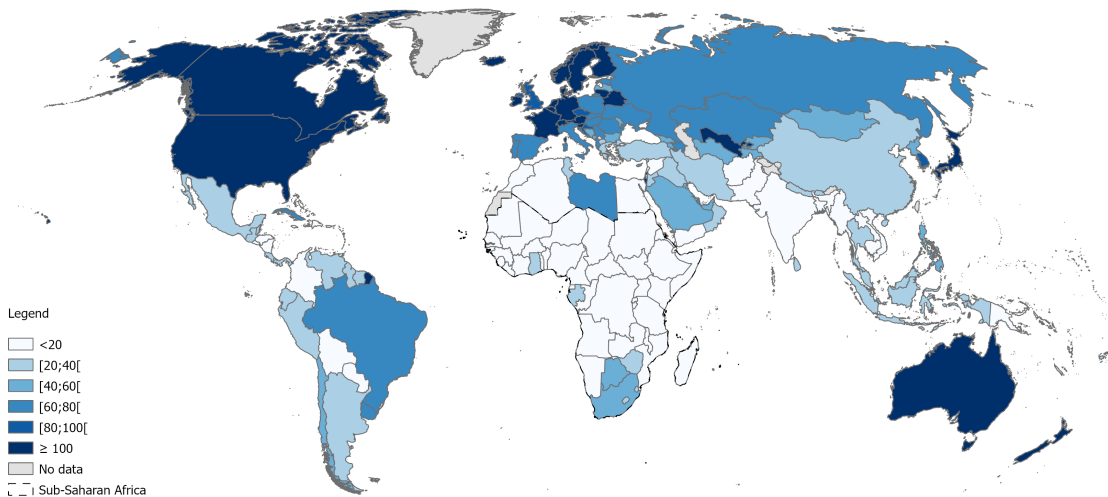
¹³ Author's calculation based on WHO [2022b].

Figure 12. Physicians per 10,000 inhabitants (various years, 2008-2020)



Source: Author's computation based on Global Health Workforce statistics database [WHO, 2022b].
Notes: The map shows the country-specific density of physicians per 10,000 inhabitants. Year of data availability varies slightly from one country to another but most of information dates back to 2017 or later. See Appendix Table D.1 for further information.

Figure 13. Nursing and midwifery personnel per 10,000 inhabitants (various years, 2014-2020)



Source: Author's computation based on Global Health Workforce statistics database [WHO, 2022b].
Notes: The map shows the country-specific density of nursing and midwifery personnel per 10,000 inhabitants. Year of data availability varies slightly from one country to another but most of information dates back to 2017 or later. See Appendix Table D.1 for further information.

All medical professions are understaffed: the subcontinent concentrates most of the countries with less than 5 physicians and 20 nursing and midwifery personnel per 10,000 inhabitants. While some countries like Ghana, Swaziland, and Zimbabwe have met the WHO [2006]’s recommended minimum threshold, much of their health workforce is composed of nurses and midwives but considerably lacks physicians (less than 2 per 10,000 inhabitants in 2020). The situation therefore remains critical. In particular, Okeke [2021] shows in the Nigerian context that mid-level health care providers¹⁴ are lower-quality substitutes for physicians. Most Sub-Saharan African health workers are employed in the public sector (80.5% in 2018), followed by the private for non-profit (16.7%) and the private for-profit (2.8%) ones.¹⁵

Insufficient human resources seriously jeopardize the capacity of such countries to provide basic health care for all. This acute shortage results from many interrelated factors. First, an insufficient number of health professionals is trained every year, leading to an insufficient health worker inflow to meet population health needs [Kinfu et al., 2009]. Intrinsically linked to this point, most Sub-Saharan African countries suffer from inadequate health-care education capacity and pre-service training [WHO, 2016a]. Second, lack of financial resources prevents some countries from absorbing all trained health workers, leading to the coexistence of health workers unemployment and health workers shortage in the health system [WHO, 2016a]. Third, these countries usually have a poor capacity to retain health workers due to unattractive remuneration, poor working conditions, inadequate protection, and little incentives [WHO, 2016a].

As a result, the sub-continent is exposed to an outflow of health professionals. For example, according to OECD [2022] Health Workforce Migration data, 2,406 physicians trained in Sudan emigrated to the United Kingdom between 2010 and 2020. For the sake of comparison, there were 10,683 physicians in Sudan in 2017.¹⁶ Among health workers trained in Nigeria, 5,047 physicians and 1,474 nurses emigrated to the United Kingdom, and 1,205 physicians emigrated to the United States of America over the same period. France, for its part, welcomed 223 physicians trained in Madagascar between 2011 and 2020.

Several pieces of evidence from Sub-Saharan Africa suggest that the medical brain drain is linked to low wages [Antwi and Phillips, 2013; Okeke, 2013b], poor economic conditions [Okeke, 2013a; Lanati and Thiele, 2021], insufficient resources for health [Moullan, 2013;

¹⁴ Including nurses, midwives, and community health workers.

¹⁵ Author’s calculation based on data from Ahmat et al. [2022] for physicians, nurses, and midwives.

¹⁶ Last figure available [WHO, 2022b].

Lanati and Thiele, 2021] and corruption [Okey, 2016] in the home country. Importantly, in addition to its financial cost [Mills et al., 2011], medical brain drain has been shown to be associated with a deterioration of health outcomes, both at the child [Chauvet et al., 2013] and adult level [Bhargava and Docquier, 2008].

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3. Objectives and Outline of the Thesis

Despite important progress since the Alma-Ata Declaration, it turns out that most Sub-Saharan African countries are still lagging behind on the road to *health for all*. Child mortality remains tragically high, preventable and treatable conditions remain the first cause of death, and new challenges question the capacity of their health systems to meet the population health needs.

The chapters of this thesis investigate several issues related to child health. Ensuring a sufficiently high level of health for children is undoubtedly crucial for its own sake,¹⁷ but also given the extensive empirical evidence on the long-run consequences of early life health on later life outcomes, including education (e.g. Currie and Hyson, 1999; Behrman and Rosenzweig, 2004; Case et al., 2005; Almond, 2006; Black et al., 2007; Oreopoulos et al., 2008; Nelson, 2010; Bhalotra and Venkataramani, 2011; Bharadwaj et al., 2013; Almond et al., 2015; Baird et al., 2016; Shih and Lin, 2018; Lo Bue, 2019; Miller and L, 2019; Araújo et al., 2021; Lundborg et al., 2021), health (e.g. Currie and Hyson, 1999; Behrman and Rosenzweig, 2004; Case et al., 2005; Almond, 2006; Black et al., 2007; Oreopoulos et al., 2008; Lindeboom et al., 2010; Bhalotra and Venkataramani, 2011; Venkataramani, 2012; Bharadwaj et al., 2013; Miller and L, 2019; Lundborg et al., 2021), labor market (e.g. Currie and Hyson, 1999; Alderman and Behrman, 2006; Nelson, 2010; Bhalotra and Venkataramani, 2011; Baird et al., 2016; Lazuka, 2020; Araújo et al., 2021) and socio-economic status (e.g. Case et al., 2005; Almond, 2006; Black et al., 2007; Currie and Moretti, 2007; Oreopoulos et al., 2008; Bleakley, 2010; Bhalotra and Venkataramani, 2011; Shih and Lin, 2018; Lundborg et al., 2021; Deng and Lindeboom, 2022),¹⁸ accompanied by significant intergenerational effects (e.g. Currie and Moretti, 2007; Cook et al., 2019). This

¹⁷ In particular, Article 25 of the Universal Declaration of Human Rights states that "*Motherhood and childhood are entitled to special care and assistance. All children, whether born in or out of wedlock, shall enjoy the same social protection.*"

¹⁸ The reader is referred to Currie [2009], Almond and Currie [2011] and Almond et al. [2018] for literature reviews on the long-run consequences of early life health, and specifically to Currie and Vogl [2013] in the context of developing countries.

makes child health a critical form of human capital. Identifying the factors that hinder child health improvement in developing countries, especially in Sub-Saharan Africa, is thus of primary interest. It particularly emphasizes the need for continued efforts to provide evidence and inform public policies.

Each chapter in the following pages independently explores one of the multiple aspects related to child health in Sub-Saharan Africa. Although the research questions differ among the chapters outlined below, the overall objective of this thesis is to uncover and better understand some factors that either bolster or slow down child health improvement in Sub-Saharan Africa. The underlying objective is to provide data-driven evidence of high policy relevance with potentially large real-life implications.

Chapter 1

Access and returns to formal health services are critical elements in the ongoing debate on the relative effectiveness of demand- and supply-side interventions in improving population health in low-income countries. [Chapter 1](#) investigates the effect of removing user fees for primary health care on maternal health care utilization and child health. I focus on the user fee removal policy implemented from 2006 in Zambia. User fees were removed in public and mission health facilities first in 54 districts considered as rural out of 72, and then in rural parts of previously unaffected districts one year later, in 2007. Using birth history from four waves of nationally representative Demographic and Health Surveys reported by more than 18,900 mothers, I exploit this staggered adoption in a difference-in-differences framework to identify causal effects. I find a 43% increase in the probability to give birth in a medical facility following the removal and a 36% increase in the probability of being assisted by a skilled birth attendant during childbirth. The probability for mothers to receive a postnatal check-up in the first 24 hours after giving birth increased by 23%. This result suggests that more women benefited from postnatal check-ups, but it should also be interpreted in light of the large increase in institutional deliveries I found. The large discrepancy in the two effects suggests poor quality of care since many women who delivered in health facilities did not benefit from a medical check-up that could have prevented postpartum complications. Chronic malnutrition decreased by 8 percent after the policy change, but this positive effect only appears for at least 12 months of exposure to free health care. There is however no evidence that user fee removal led to a change in average child mortality risk, a result that is not driven by fertility nor by selection effects potentially set off by the policy. I then use unique administrative data from the 2005 national census of health facilities to further investigate how such policy's effects vary with physical access to health amenities

and the quality of health services available locally. I uncover important heterogeneity. As expected, the positive effect on delivery conditions fades with distance from the nearest health facility, highlighting the importance of considering physical access when estimating the returns to such a policy. While there is no discernible effect on average child mortality, newborn mortality risk did decrease in the direct vicinity of qualified health centers. These findings have important implications for policy makers. They illustrate a twin challenge: making health services both financially accessible and of better quality for all. In particular, returns to formal health services appear to be limited without sufficient quality of care.

Chapter 2

Significant efforts have been made over the last decades to improve education levels in developing countries. The considerable progress made in recent years is expected to translate into a comparable improvement of population health in the years to come, as education has been shown to be a key determinant of health status. Education can affect health through many channels, including wealth, a better understanding of prevention messages, greater incentives to invest in healthy behaviors, and more generally transmits values that can help people to adopt costly preventive behaviors. Such health benefits of education might extend to subsequent generations, either directly through these channels or indirectly through intergenerational transmission of health. [Chapter 2](#) of this thesis, co-written with Élodie Djemaï and Anne-Laure Samson, focuses on the intergenerational effects of parental education on child health. Existing studies suggest that a child's health status is related to the level of education of her parents. However, current evidence focuses almost exclusively on maternal education and has completely overlooked the role played by paternal education as a determinant of child health. There are two main reasons for this. First, these analyses may reflect the common wisdom that mothers matter more than fathers in raising children. The second reason is purely empirical: because mothers are more likely than fathers to live with their children in most countries, it is often difficult to relate the health status of young children to their father's level of education on the basis of survey data. This chapter proposes to estimate the respective causal effect of mothers' and fathers' education on their child's health in a developing country setting. To do so, we use four waves of nationally representative Demographic and Health Surveys collected in Zimbabwe from 1994 to 2010. Disentangling the respective causal effect of both parents' education levels on health investments and child health outcomes is empirically challenging for three main reasons. First, parents' education levels are likely endogenous in the child health equation. We tackle this issue in an instrumental variables framework by using exogenous

variation arising from the age-specific nature of the 1980 Education Reform that has led to an important rise in educational attainment in the country. Second, coresidence between parents and children may bias the estimates as the probability for a child to live with both parents is not randomly distributed in the population, and coresidence might affect child health. We deal with such selection effect by using community-level variation in practices that could affect a child's probability to live with her mother and her father following the Heckman two-step procedure. To date, this dimension has been neglected by the education-health literature. Third, we consider marital education sorting of parents as an additional source of bias. If the correlation between education levels is high, the estimated effect of mother's education on child health may pick up the effect of father's education if it is not controlled for. Unobservable characteristics that drive educated people to match together might also affect child's health. Our results confirm the existence of a high correlation of health investments and child health outcomes with parents' education, and that parental education sorting is high. When we take into account the endogeneity of education, we no longer observe an effect of mother's education, while the significant and positive effect of father's education on prenatal care, birth conditions, and vaccination still holds. We find that coresidence between parents and children affects child health but does not alter the estimated causal effect of education on child health. Overall, our findings suggest that not considering both parents' education simultaneously may produce misleading conclusions. These findings have important public policy implications as they suggest that child health policies targeting and involving fathers could have sizeable effects.

Chapter 3

Dams are among the infrastructures considered essential to bolster economic development, especially in Sub-Saharan Africa, where safe access to water remains an issue. Either for irrigation, livestock, water access, protection from extreme precipitation events, mining waste containment, or hydropower generation, dams have flourished over the continent for over fifty years. A larger share of the population is expected to depend on dams in the near future, with a growing role of water storage solutions in the face of climate change. Nonetheless, the net effect of such infrastructures for the health of the local population is ambiguous. Potential positive effects include access to a more sustainable source of water leading to an increase in agricultural production and a reduction in the opportunity cost of fetching water. Such positive income effects may then translate into higher nutritional intake and an improvement in child nutritional status. Potential negative effects include a rise in soil salinity with a negative effect on agricultural productivity, an

increase in the risk of waterborne disease if water from the dam is used for domestic purposes, a greater exposure to mosquito-borne diseases as stagnant water from the dam constitutes an ideal breeding ground for mosquitoes, and forced displacements of the local population with potentially large detrimental effects on mental health. Such negative effects may seriously threaten the health of the local population. [Chapter 3](#) uses rich microdata from the Demographic and Health Surveys collected in 34 countries and satellite imagery to investigate the causal effect of dams on population health in Sub-Saharan Africa, with a focus on under-five children. Health measures of over 1.7 million individuals are matched to a unique record of dam construction over more than thirty years built for the purpose of this chapter. I use river gradient computed from satellite imagery as an exogenous source of variation in an instrumental variables framework to cope with the non-random placement of dams which may bias the estimates. Results point to a significant reduction of child chronic malnutrition around dams accompanied by a higher risk of malaria transmission. I find a 2.1 percentage point increase in post-neonatal mortality in the vicinity of dams. Importantly, the timing of this increase overlaps with a period in which babies' natural immunity against malaria acquired during childbearing fades away. Adults living near a dam also exhibit a higher prevalence of anemia, one of the main symptoms of malaria infection. Heterogeneity analysis reveals that such effects are equally shared between siblings but that children from agricultural households tend to be more exposed to the increase in malaria risk. Several channels through which dams might impact child health are then explored. Results suggest that households living near a dam tend to be better off, and individuals benefit from important labor-market effects of dams. The results also point to an improvement in access to health care, but I find no discernible effect on fertility behaviors. Health investments are not affected, except those related to malaria prevention as both ownership and utilization of mosquito bed nets are higher near dams. Finally, the chapter proposes two extensions to the main analysis. It first investigates the role played by the size of the dam in shaping these effects and then estimates the impact of dams located upstream from the locality. These findings have important policy implications. They should not be interpreted as evidence against dam construction, as the results point to a positive income effect and a large improvement in child nutritional status. However, they call for complementary policies to mitigate adverse effects set off by the proliferation of mosquitoes.

Although this thesis focuses on Sub-Saharan African countries, conclusions drawn from this work probably extend, at least to some extent, well beyond the frontiers of the subcontinent. In particular, the resulting public policy implications will certainly find a resonance in

most of the World's low-income countries, the majority of which face similar challenges in improving population health.

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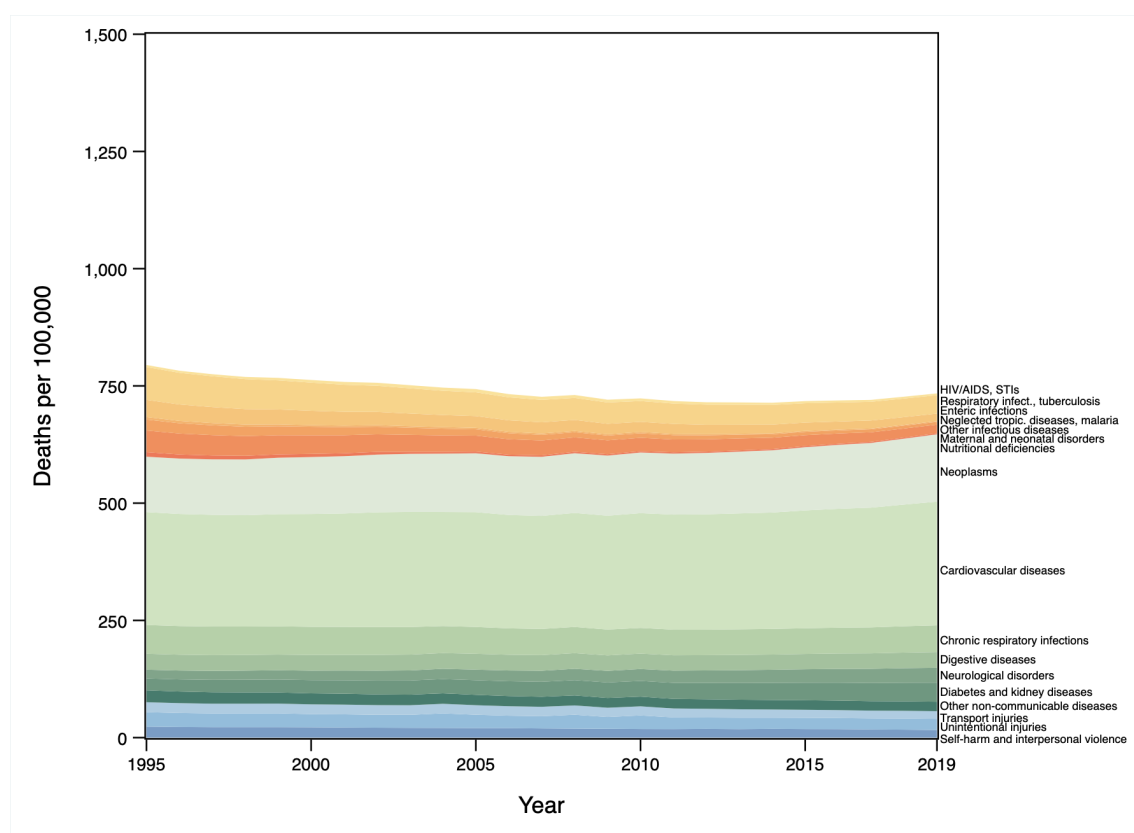
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APPENDICES TO GENERAL INTRODUCTION

Appendix A. Evolution of causes of deaths in the World excluding Sub-Saharan Africa

Figure A.1. Evolution of causes of death in the World excluding Sub-Saharan Africa

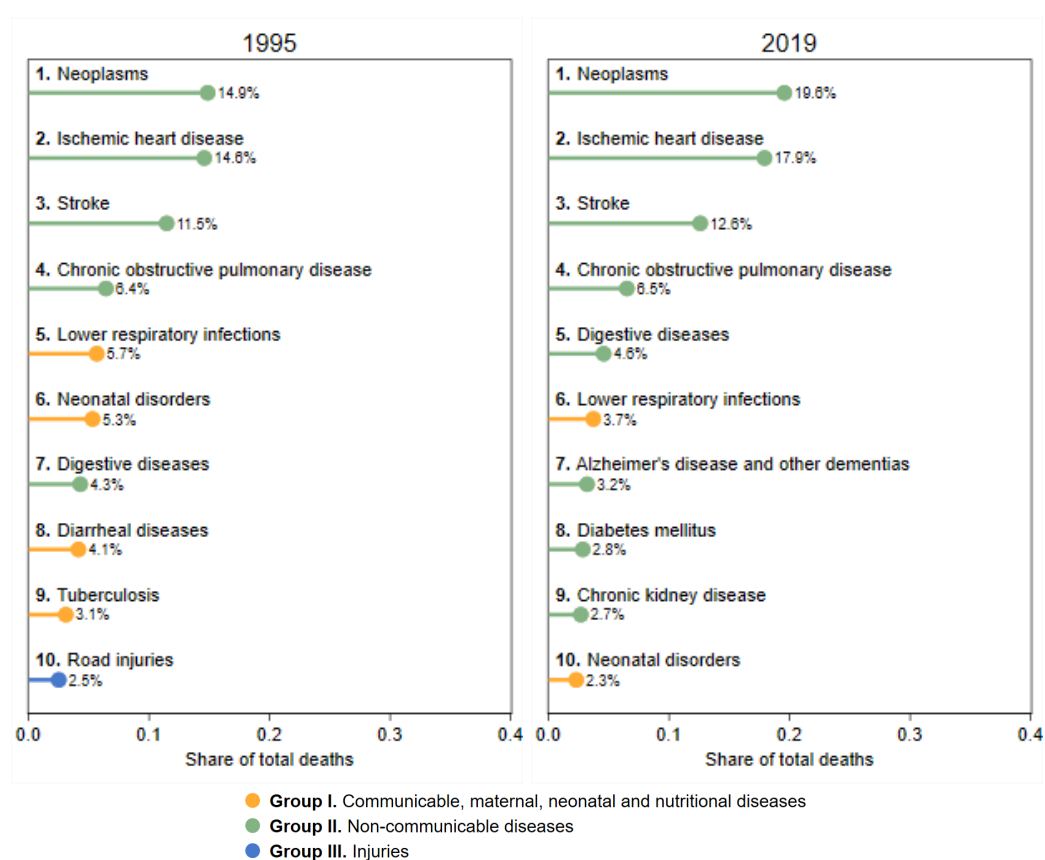


Source: Author's computation based on [Global Burden of Disease \[2019\]](#).

Notes: The figure plots the evolution of the cause-specific number of deaths per 100,000 inhabitants in the World excluding Sub-Saharan Africa@ over the 1995-2019 period. Shades of orange stand for deaths caused by communicable, maternal, neonatal and nutritional disorders (Group I). Shades of green stand for deaths attributable to non-communicable diseases (Group II). Shades of blue stand for deaths imputable to injuries (Group III).

Appendix B. Top 10 causes of death in 1995 and 2019 in the World excluding Sub-Saharan Africa and in OECD countries

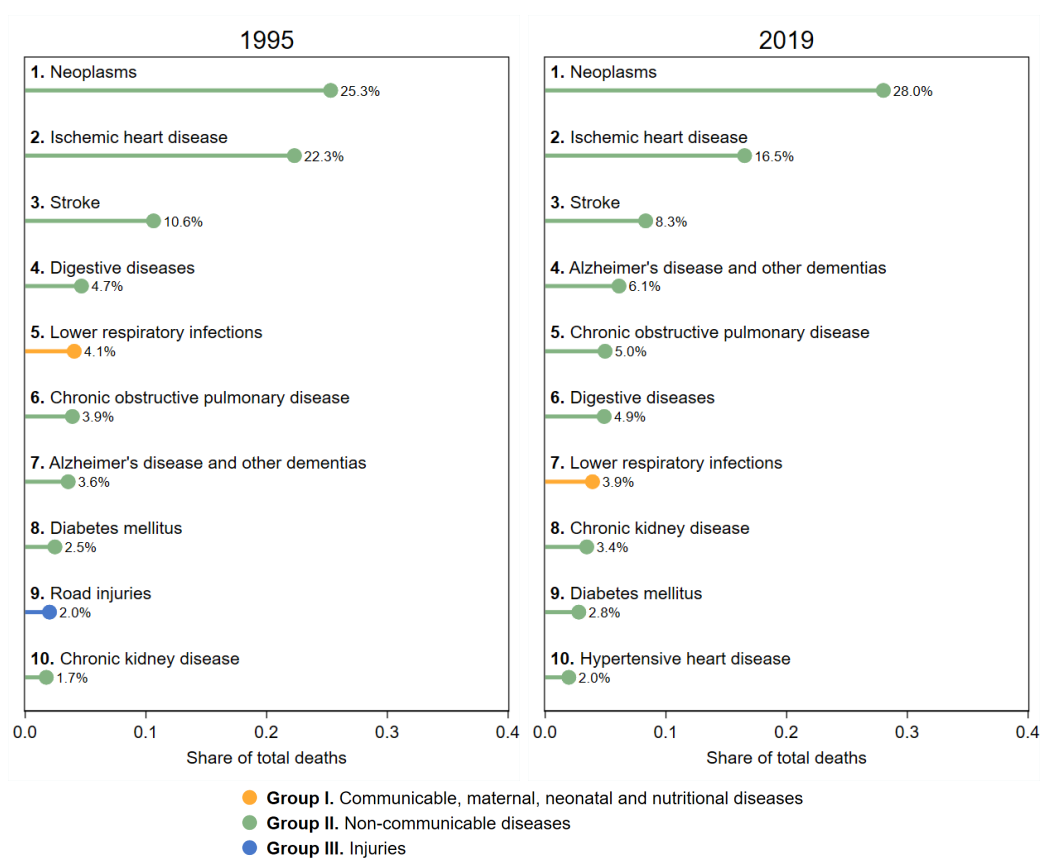
Figure B.1. Top 10 causes of death in the World excluding Sub-Saharan Africa in 1995 and 2019



Source: Author's computation based on [Global Burden of Disease \[2019\]](#).

Notes: The figure plots the top-10 causes of deaths in the World excluding Sub-Saharan Africa (all ages) in 1995 (left-hand side) and 2019 (right-hand side) expressed as a share of total deaths. Orange dots stand for deaths caused by communicable, maternal, neonatal and nutritional disorders (Group I). Green dots stand for deaths attributable to non-communicable diseases (Group II). Blue dots stand for deaths imputable to injuries (Group III).

Figure B.2. Top 10 causes of death in OECD countries in 1995 and 2019

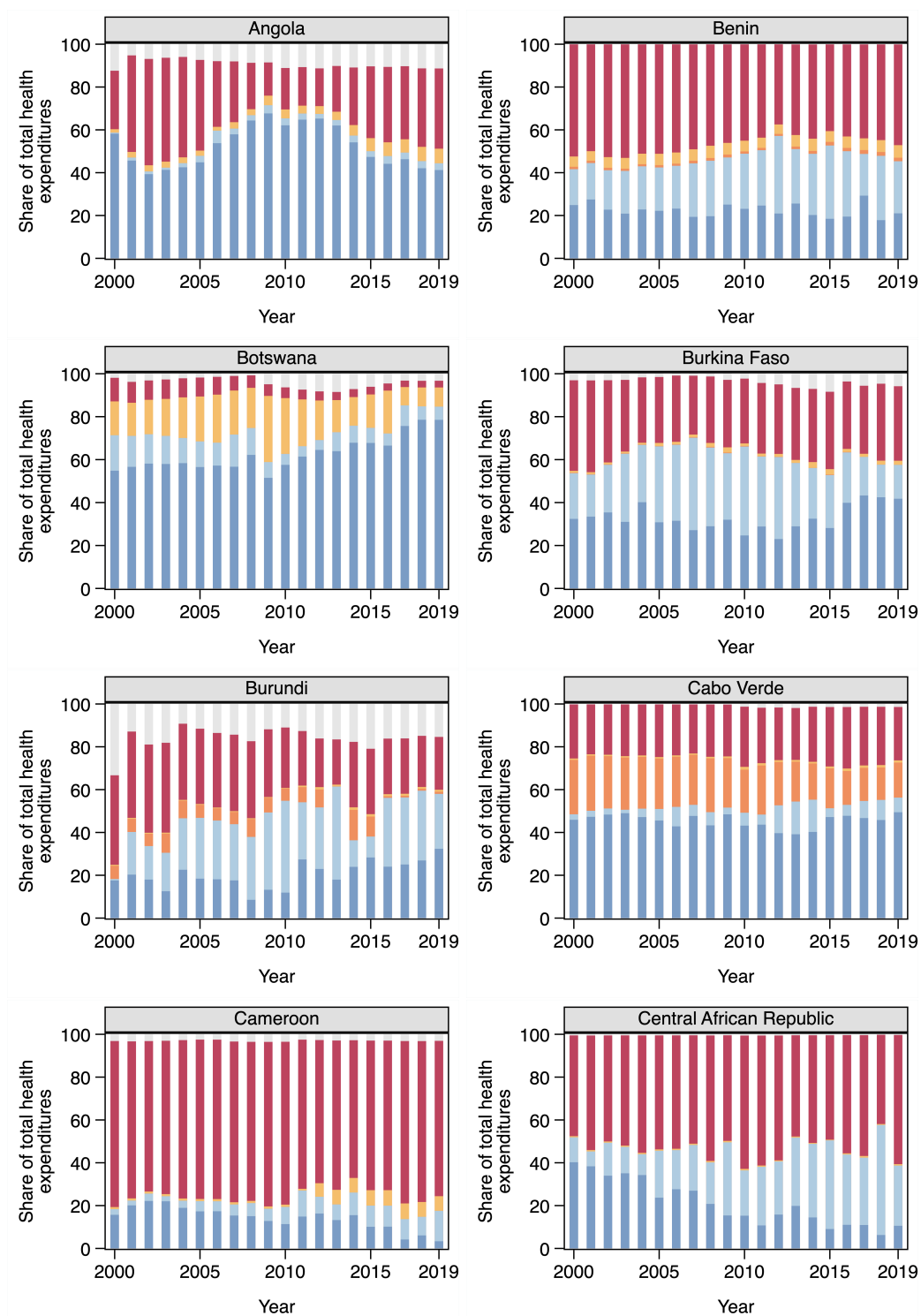


Source: Author's computation based on [Global Burden of Disease \[2019\]](#).

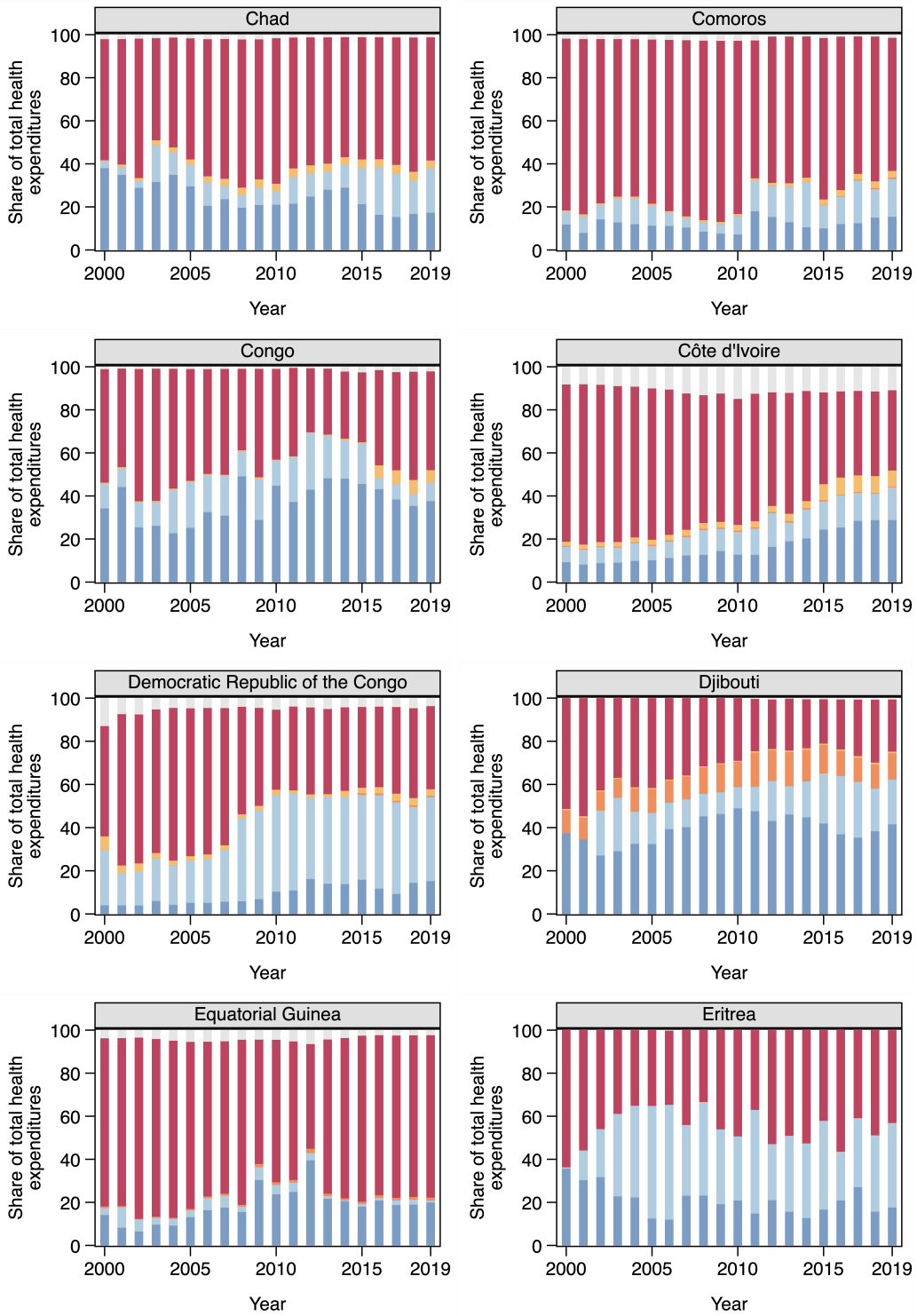
Notes: The figure plots the top-10 causes of deaths in OECD (Organisation for Economic Co-operation and Development) countries (all ages) in 1995 (left-hand side) and 2019 (right-hand side) expressed as a share of total deaths. Orange dots stand for deaths caused by communicable, maternal, neonatal and nutritional disorders (Group I). Green dots stand for deaths attributable to non-communicable diseases (Group II). Blue dots stand for deaths imputable to injuries (Group III).

Appendix C. Evolution of health spending components by country

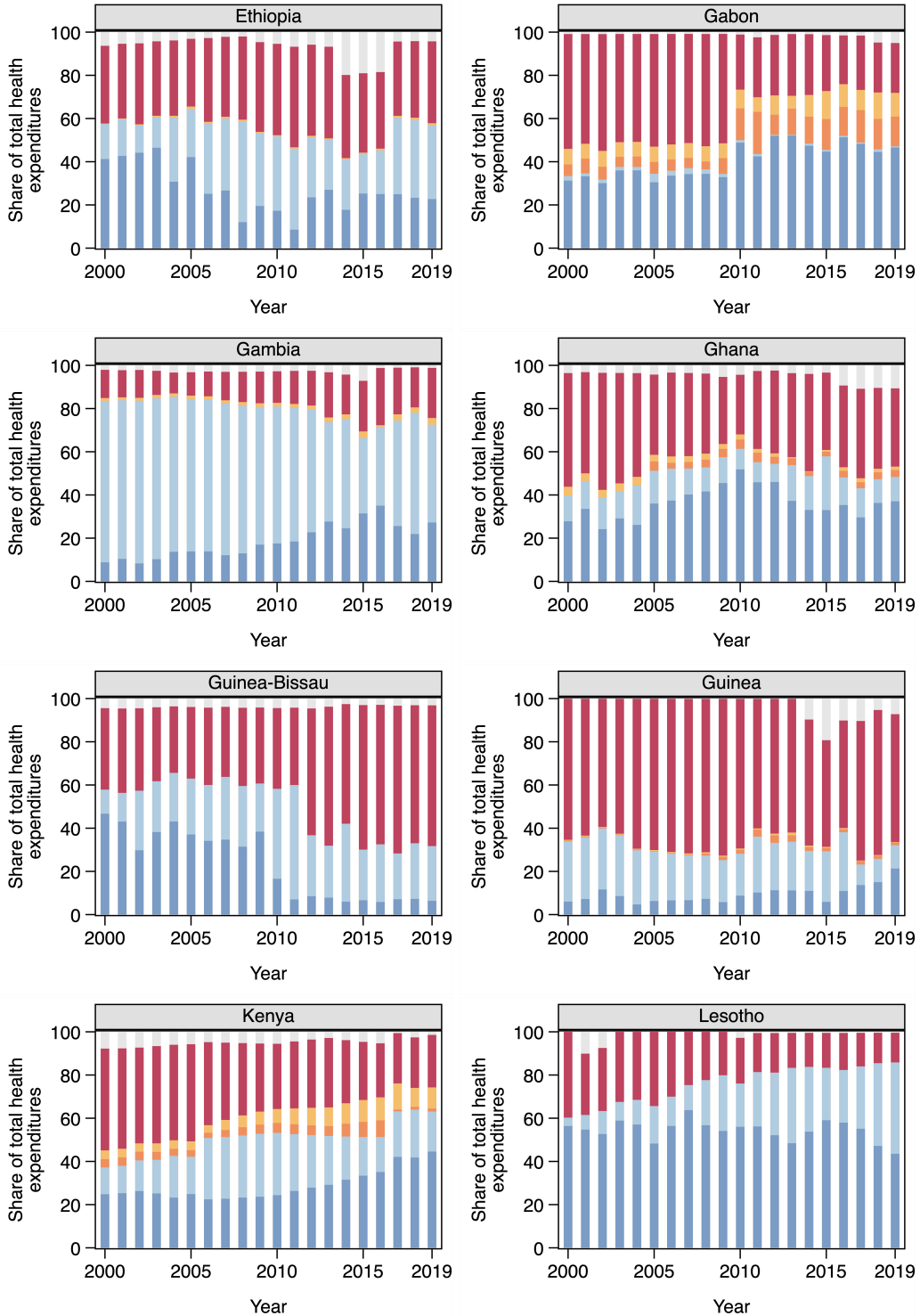
Figure C.1. Evolution of health expenditures components by country



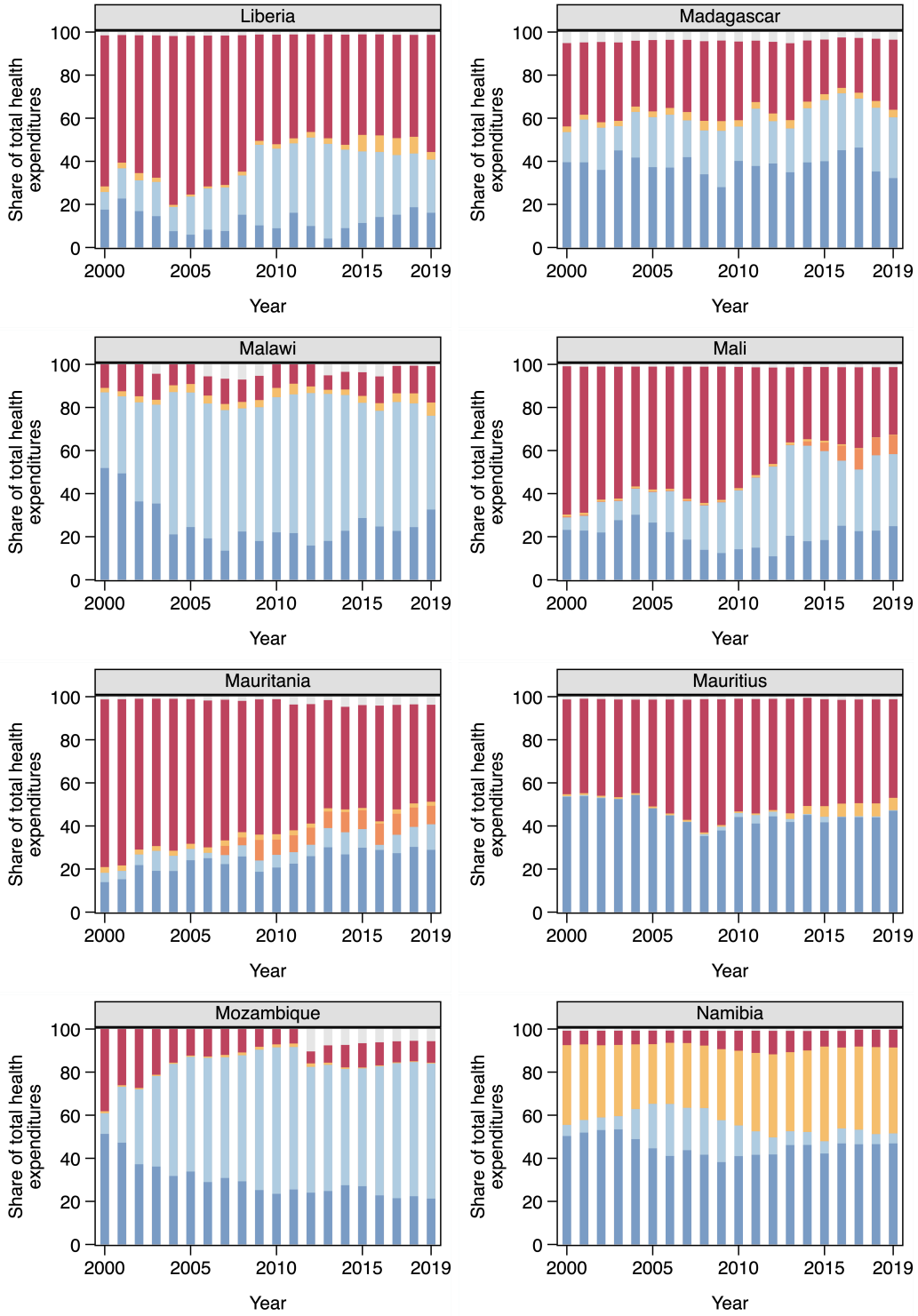
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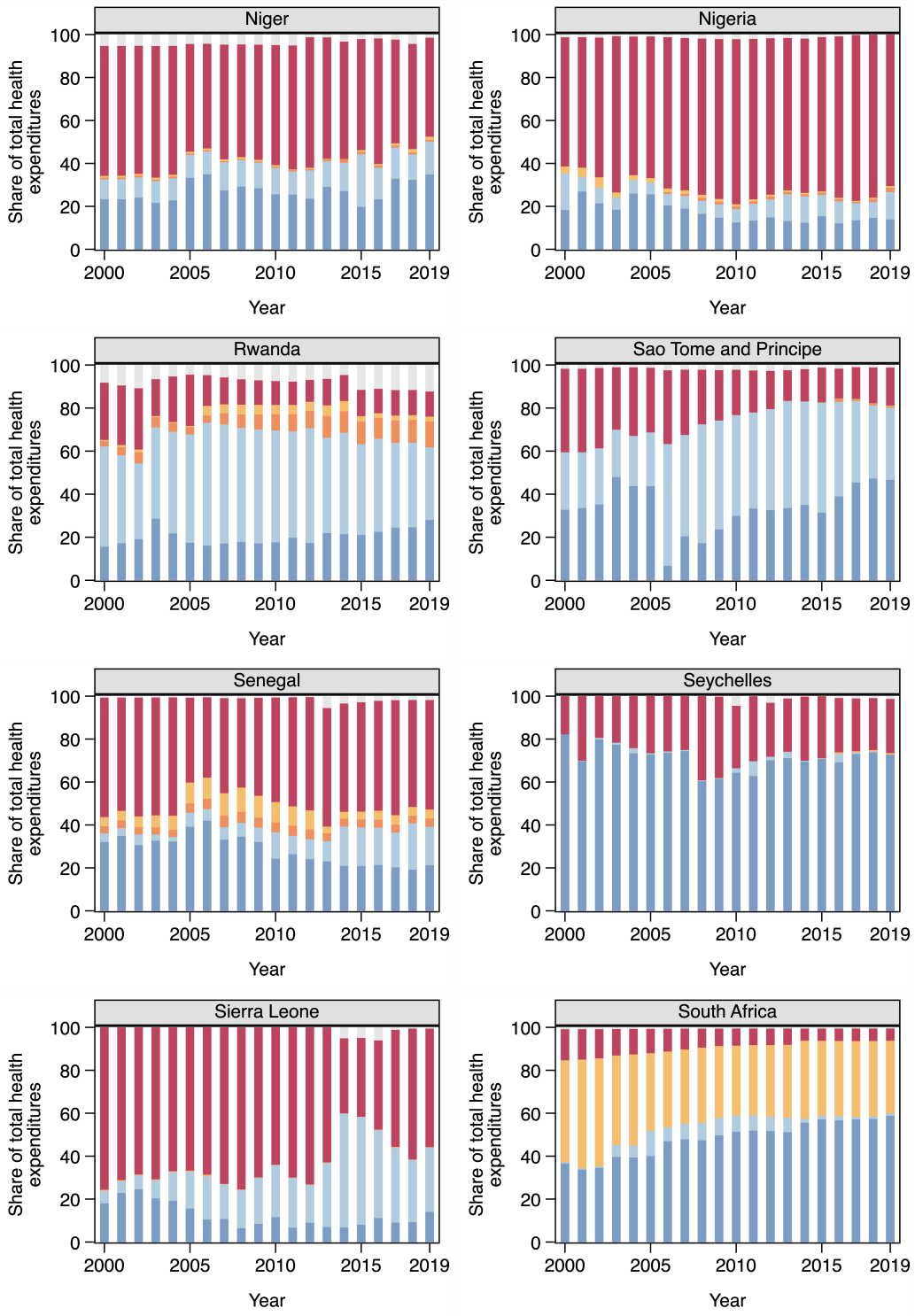
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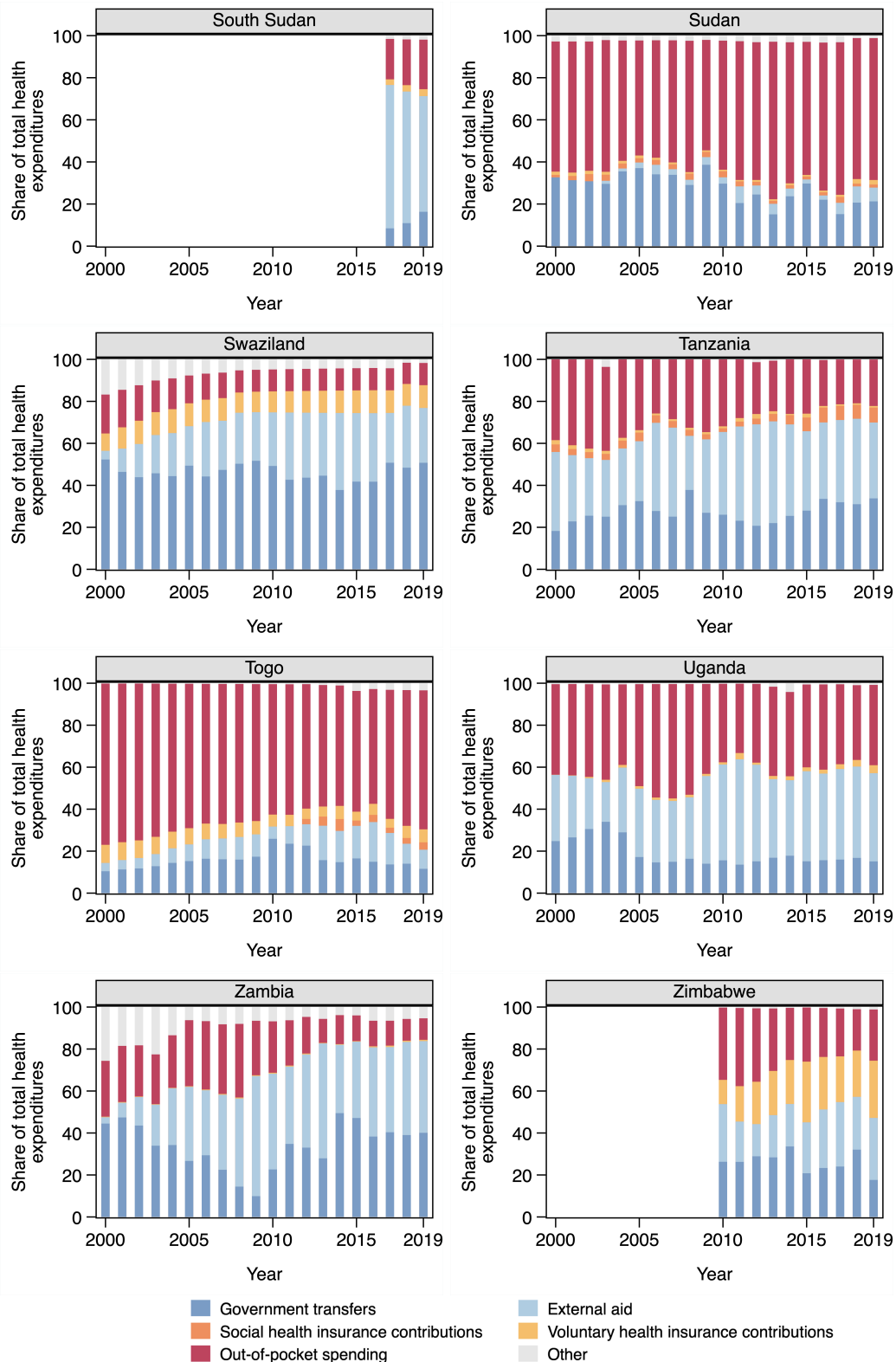
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continued →



Source: Author's computation based on the Global Health Expenditure Database [WHO, 2021].

Notes: The figure shows the evolution of country-specific health spending components as a share of total health spending over the 2000-2019 period. No information available for Somalia. Other sources include contributions from domestic non-governmental organizations, compulsory private insurance schemes and enterprise schemes including health services provided by enterprises for their employees.

Appendix D. Information availability for medical density

Table D.1. Last year for which information on medical density is available

Country	Last year for medical density		Country	Last year for medical density	
	Physicians	Nurses and Midwives		Physicians	Nurses and Midwives
Afghanistan	2020	2018	Dominica	2018	2017
Albania	2020	2020	Dominican Republic	2019	2019
Algeria	2018	2018	Ecuador	2017	2018
Andorra	2015	2015	Egypt	2019	2018
Angola	2018	2018	El Salvador	2018	2018
Antigua and Barbuda	2017	2019	Equatorial Guinea	2017	2018
Argentina	2020	2017	Eritrea	2020	2018
Armenia	2017	2015	Estonia	2019	2019
Australia	2020	2019	Eswatini	2020	2020
Austria	2020	2019	Ethiopia	2020	2020
Azerbaijan	2019	2014	Fiji	2015	2019
Bahamas	2017	2018	Finland	2018	2018
Bahrain	2015	2015	France	2019	2019
Bangladesh	2020	2020	Gabon	2018	2020
Barbados	2017	2018	Gambia	2020	2020
Belarus	2019	2015	Georgia	2020	2020
Belgium	2020	2020	Germany	2020	2019
Belize	2018	2018	Ghana	2020	2020
Benin	2019	2019	Greece	2019	2019
Bhutan	2020	2020	Grenada	2018	2018
Bolivia	2017	2017	Guatemala	2020	2020
Bosnia and Herzegovina	2015	2018	Guinea	2018	2018
Botswana	2018	2018	Guinea-Bissau	2020	2020
Brazil	2019	2019	Guyana	2020	2020
Brunei	2017	2018	Haiti	2018	2018
Bulgaria	2018	2018	Honduras	2020	2018
Burkina Faso	2019	2019	Hungary	2020	2019
Burundi	2020	2020	Iceland	2019	2020
Cabo Verde	2018	2018	India	2020	2020
Cambodia	2014	2019	Indonesia	2020	2020
Cameroon	2018	2018	Iran	2018	2018
Canada	2019	2020	Iraq	2020	2020
Central African Republic	2018	2018	Ireland	2020	2019
Chad	2020	2020	Israel	2020	2020
Chile	2020	2020	Italy	2020	2020
China	2019	2019	Jamaica	2018	2018
Colombia	2020	2020	Japan	2018	2018
Comoros	2018	2018	Jordan	2019	2019
Congo	2018	2018	Kazakhstan	2020	2015
Cook Islands	2014	2019	Kenya	2018	2018
Costa Rica	2020	2020	Kiribati	2013	2018
Croatia	2019	2016	Kuwait	2020	2020
Cuba	2018	2018	Kyrgyzstan	2014	2019
Cyprus	2019	2016	Laos	2020	2020
Czechia	2020	2019	Latvia	2020	2020
Côte d'Ivoire	2019	2019	Lebanon	2019	2018
Democratic Republic of the Congo	2018	2018	Lesotho	2018	2018
Denmark	2018	2018	Liberia	2018	2018
Djibouti	2014	2014	Libya	2017	2017

continued →

Table D.1 (*continued*). Last year for which information on medical density is available

Country	Last year for medical density		Country	Last year for medical density	
	Physicians	Nurses and Midwives		Physicians	Nurses and Midwives
Lithuania	2020	2020	Saint Lucia	2017	2017
Luxembourg	2017	2017	Saint Vincent and the Grenadines	2012	2018
Madagascar	2018	2018	Samoa	2020	2020
Malawi	2020	2020	San Marino	2014	2014
Malaysia	2020	2019	Sao Tome and Principe	2019	2019
Maldives	2019	2020	Saudi Arabia	2020	2019
Mali	2018	2018	Senegal	2019	2019
Malta	2015	2018	Serbia	2016	2016
Marshall Islands	2012	2018	Seychelles	2019	2018
Mauritania	2018	2018	Sierra Leone	2018	2018
Mauritius	2020	2018	Singapore	2019	2017
Mexico	2019	2019	Slovakia	2019	2019
Micronesia	2020	2019	Slovenia	2019	2019
Monaco	2014	2014	Solomon Islands	2016	2018
Mongolia	2018	2018	Somalia	2014	2014
Montenegro	2020	2020	South Africa	2019	2018
Morocco	2017	2017	South Sudan	2018	2018
Mozambique	2020	2020	Spain	2019	2019
Myanmar	2019	2019	Sri Lanka	2020	2020
Namibia	2018	2018	Sudan	2017	2018
Nauru	2015	2018	Suriname	2018	2019
Nepal	2020	2020	Sweden	2019	2018
Netherlands	2020	2020	Switzerland	2020	2019
New Zealand	2020	2020	Syria	2016	2016
Nicaragua	2018	2017	Tajikistan	2014	2014
Niger	2020	2018	Tanzania	2018	2018
Nigeria	2018	2019	Thailand	2020	2019
Niue	2008	2018	Timor-Leste	2020	2020
North Korea	2017	2017	Togo	2020	2020
North Macedonia	2015	2015	Tonga	2020	2020
Norway	2020	2020	Trinidad and Tobago	2019	2019
Oman	2020	2020	Tunisia	2017	2017
Pakistan	2019	2019	Turkmenistan	2014	2014
Palau	2020	2020	Tuvalu	2020	2020
Panama	2019	2019	Türkiye	2019	2019
Papua New Guinea	2019	2019	Uganda	2020	2020
Paraguay	2020	2018	Ukraine	2014	2014
Peru	2018	2018	United Arab Emirates	2019	2019
Philippines	2020	2019	United Kingdom	2020	2020
Poland	2020	2020	United States of America	2018	2018
Portugal	2019	2019	Uruguay	2017	2019
Qatar	2018	2018	Uzbekistan	2014	2014
Republic of Korea	2019	2019	Vanuatu	2016	2019
Republic of Moldova	2020	2020	Venezuela	2017	2018
Romania	2017	2017	Viet Nam	2016	2016
Russian Federation	2020	2020	Yemen	2014	2018
Rwanda	2019	2019	Zambia	2018	2018
Saint Kitts and Nevis	2018	2015	Zimbabwe	2020	2020

Source: Author's computation based on Global Health Workforce statistics database [WHO, 2022].

Notes: The table reports the last year for which information used in Figures 12 (physicians) and 13 (nursing and midwifery personnel) on medical densities is available.

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Chapter 1

From Fees to Free: User Fee Removal, Maternal Health Care Utilization and Child Health in Zambia

A more concise version of this Chapter has been published in World Development [Renard, 2022].

A B S T R A C T

Despite recent progress, about 295,000 women in the World still die each year from pregnancy-related causes, and about 4.1 million children die before reaching the age of one. 99% of these deaths occur in developing countries. In 2006 the Zambian government removed user fees in public and mission health facilities in 54 out of 72 districts and then extended this policy to rural parts of unaffected districts in 2007. I exploit the staggered implementation of the policy to assess its impact on maternal health care utilization and child health outcomes. Using a difference-in-differences estimation strategy, I find a 43% increase in the probability of giving birth in a medical facility following the removal and a 36% increase in the probability of being assisted by a skilled birth attendant during child-birth. These positive effects decrease with household's distance from the nearest health facility. In terms of child health, chronic malnutrition decreased by 8%, and the abolition of user fees reduced newborn mortality risk only for those living close to a health facility providing essential emergency obstetric care and child health services. Access improved, but returns to formal health services remained rather limited, highlighting the importance of addressing supply-side constraints to generate substantial gains in population health.



Location of Zambia

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1. Introduction

Access and returns to formal health services are critical elements in the ongoing debate on the relative effectiveness of demand- and supply-side interventions in improving population health in low-income countries. Despite a still tragically high incidence of preventable, premature deaths, there is little rigorous empirical evidence on whether removing user fees effectively helps increase health care utilization and ultimately improve population health [Dzakpasu, 2013; Lagarde, 2011; Hatt et al., 2013].

An extensive empirical literature has established that even small prices may drastically deter individuals' willingness to invest in their health. However, it focuses almost exclusively on health products that can be directly used at home (e.g. Ashraf et al., 2010; Cohen and Dupas, 2010; Cohen et al., 2015; Spears, 2014). Evidence concerning the impact of reducing fees for health services in public amenities is more scarce [Kremer and Glennerster, 2011; Dupas, 2014], despite the fact that curative out-of-pocket health expenditures may represent 10% of total household's budget [Dupas, 2011].

Theoretically, the effects of user fee removal are unclear, especially in low-income countries. On the one hand, removing user fees may encourage health care utilization and improve population health if individuals were kept out of good-quality health services for financial reasons. On the other hand, many factors beyond user fees may discourage individuals from seeking care. The removal of user fees may have exacerbated some of them, such as health staff workload, informal fees and medical supplies shortages. The final impact on health will depend not only on the price sensitivity of health care use but also on the impact of health facility visits on health. If removing user fees only leads to a drop in households out-of-pocket health expenditures without any effect on individuals' health, it should cast some doubts about the appropriateness of such an expensive policy.¹

Existing studies generally point to an increase in health care utilization [Bagnoli, 2019; Fitzpatrick and Thornton, 2018; Friedman and Keats, 2019b; Leone et al., 2016; Masiye et al., 2010; McKinnon et al., 2015a,b; Powell-Jackson et al., 2014; Ridde et al., 2013] and to a decline in household out-of-pocket health expenditures [Powell-Jackson et al., 2014; Ridde et al., 2015] after a reduction of user fees. The impact on health outcomes has received much less attention and evidence is much more mixed. Exceptions include Tanaka [2014] who finds a significant improvement of child's nutritional status after the removal

¹ The reader is referred to Dupas [2014] for a comprehensive discussion of the issue of user fees for public health services.

of user fees in South Africa and [McKinnon et al. \[2015b\]](#), who find no change in neonatal mortality risk following user fee removal for facility-based deliveries in Kenya, Ghana and Senegal. [Fitzpatrick \[2018\]](#) finds that free caesarean sections and deliveries have resulted in a decrease in maternal mortality and a stagnant or increased neonatal mortality risk in Sub-Saharan Africa depending on the specification used. Finally, [Friedman and Keats \[2019b\]](#) show that making facility births free in Ghana has had no effect on newborn mortality, but has led to substantial reduction in infant mortality risk and improvement of child nutritional status later in life.

This chapter sheds new light on the extent to which abolition of user fees affects maternal health care utilization and child health in a resource-limited setting. It also investigates how physical access to health amenities as well as quality of care shape the effectiveness of such a policy. Zambia constitutes an interesting framework to study these questions. User fees were removed in government-run and mission primary health facilities from April 2006 in 54 districts out of 72, and then in rural areas of previously unaffected districts one year later, in July 2007. Using birth history from four waves of nationally representative Demographic and Health Surveys reported by more than 18,900 mothers, I exploit this staggered adoption in a difference-in-differences framework.

The impact of this policy change has been explored in three recent papers. [Chama-Chiliba and Koch \[2016\]](#) find no effect of the April 2006 removal on deliveries in public facilities, but part of their control group was already exposed to free primary health care at survey time through the second wave of removal. [Lépine et al. \[2018\]](#) find no impact of the April 2006 removal on health care utilization but a strong short-term reduction in out-of-pocket health expenditures. Finally, [Hangoma et al. \[2018\]](#) assess the long-term effects of the policy and find a significant increase in health care utilization but no impact on average out-of-pocket health expenditures. None of these papers investigate how these effects depend on other supply-side factors, nor the resulting impact on health outcomes.

Looking at the effect on child health is important for several reasons. First, under-five children were in theory already covered by targeted fee exemptions since 1995. However, targeted exemptions were poorly implemented in practice, so that one can reasonably expect that under-five children have directly benefited from the 2006 policy change in terms of access to health services, and potentially, health status. Second, even if under-five children were perfectly covered by fee exemptions, they may have been adversely affected by the extension of free health care to the rest of the population. For instance, the increase in health care utilization may trigger supply-side constraints that may result

in a deterioration of health services quality. Third, delivery conditions have been shown to be a strong predictor of newborns' survival chances both in developed [Daysal et al., 2015; Lazuka, 2018] and developing countries [Okeke and Chari, 2018] with important long-term effects on individuals' health [Ahsan et al., 2020; Friedman and Keats, 2019a,b; Lazuka, 2018], including in terms of child nutritional status. For instance, institutional deliveries may result in more interactions with postnatal health services providers and higher child health investments early in life. Fourth, if parents visit health facilities more frequently as a result of the policy, they will be more regularly exposed to health workers, and potentially to prevention messages. Finally, households may benefit from additional resources as they no longer have to pay for primary health services after the removal. This might indirectly affect child health through an income effect. For instance, these resources might be reallocated to invest in preventive health products and to increase food consumption. Overall, it appears that from a theoretical point of view the effect of removing user fees on child health is of interest but is far from clear-cut and must be empirically assessed.

This chapter makes several contributions to the literature. First, I find a large and sustained change in maternal health care utilization, with a 43 percent increase in the probability to give birth in a medical facility after the removal, a result confirmed by the concurrent work of Lagarde et al. [2021]. Second, I assess the final impact of this reform on child health outcomes. Chronic malnutrition decreased by 8 percent after the policy change, but this positive effect is only showing up for at least 12 months of exposure to free health care. There is however no evidence that user fee removal led to a change in average infant mortality risk, a result which is not driven by potential fertility or selection effects set off by the policy. Finally, I use unique administrative data from the national census of health facilities to further investigate how such policy's effects vary with physical access to health amenities and the quality of health services available locally. I uncover important heterogeneity. As expected, the positive effect on delivery conditions fade with distance from the nearest health facility, highlighting the importance of considering physical access when estimating the returns to such a policy. While there is no discernible effect on child mortality on average, newborn mortality risk did decrease in the direct vicinity of qualified health centers. These findings have important implications for policy makers. They illustrate a twin challenge: making health services both financially accessible and of better quality for all. In particular, returns to formal health services appear to be limited without a sufficient quality of care.

The remainder of this chapter is organized as follows. Section 2 depicts the conceptual

framework. Section 3 provides some background on the Zambian health system and the policy of user fee removal. Section 4 presents the data as well as the empirical strategy. Results are presented in Section 5. Section 6 discusses the results and concludes.

*
* *

2. Conceptual Framework

Consider individuals that maximize their utility function over their health stock, determined by their current investment in health as well as their stock of health during the previous period of time. These individuals are subjected to a budget constraint that depends on their resources and a vector of prices, including the prices of preventive and curative health investments. We assume that individuals value health in itself since, other things being equal, they prefer to be healthy than sick and thus invest in their health if they have the opportunity to do it. For example, individuals may decide whether to invest in and use a mosquito net to protect against malaria, whether to be vaccinated, whether to seek care and, if so, when and from which health provider [Dupas, 2011]. We also assume that individuals are not covered by a health insurance scheme since only 4% of Zambians had a health insurance during the period studied in this chapter.

To decide whether to invest in their health, individuals compare the marginal benefit with the marginal cost of making such an investment, given their actual health stock.² It follows a demand for health products and services, which depends negatively on the price, as is usual. For instance, numerous empirical studies have found a high price elasticity of demand for health products (e.g. Ashraf et al., 2010; Cohen and Dupas, 2010), suggesting that household's health-related decisions may be very sensitive to price. It could partly explain the large differences in maternal health care utilization and nutritional status by wealth level observed in Zambia before the removal of user fees,³ both nationally and within rural and urban areas (Appendix Table B.4). Other things being equal, including the perceived quality of care, if households were kept out of health services for financial reasons, a fall in the price of health services should lead to a higher demand for health care.

² In particular, if their health stock reaches its maximum level, then the marginal benefit will be zero for curative care but positive for preventive care. Indeed, the objective of preventive investments is to reduce the likelihood of adverse health shocks in the future, with benefits that extend far beyond the current period of time.

³ For instance, 63% of childbirths were assisted by a health professional among the richest 50%, compared with 24% only among the poorest 50% (42% vs. 23% in rural districts, 36% vs. 22% in rural areas of urban districts, and 91% vs. 69% in urban districts).

By lowering the marginal cost of health investments, user fee removal will *ceteris paribus* increase individuals' demand for health care and result in better health. Moreover, resources released by fee exemption may be reallocated within households towards other virtuous practices such as higher nutritional intakes or investment in preventive health products such as mosquito nets and vaccination. If this is the case, then we should observe an increase in individuals' health following the removal of user fees in health facilities.

However, we might expect to find no evidence of such an effect for different reasons. First, if the quality of care was initially too low, encouraging health facility visits at a reduced cost may not translate into health gains for users. I investigate whether this is the case by looking at the heterogeneous effect of removing user fees depending on the quality of health services available locally.

Second, the overall increase in health services utilization after user fee removal may have led to a deterioration of health care quality because of insufficient funding and human resources to compensate for the increase in utilization [Meessen et al., 2011]. This drop in quality may affect one's health stock in the short term when perceived quality has not changed but effective quality of services offered already did.⁴ In the medium- to long-term, individuals may react and reduce their demand for health care in the public sector since the drop in quality potentially lowers the marginal benefit of investing in their health and increases the marginal cost of doing so. If the quality of delivery care becomes equivalent at home and in a health facility, then we would only observe a price effect on demand for health care without any impact on health outcomes.

Third, the loss of user fee revenue in health facilities and the increased workload may have encouraged health workers not to spread information about the removal and to charge informal fees on users [Hatt et al., 2013; Nabyonga-Orem et al., 2011]. In that case, user fee removal will not (or not fully) translate into lower out-of-pocket health expenditures for households, hence reducing the expected higher health services utilization and potential health gains. Hangoma et al. [2018] find a reduction in the probability of incurring any spending after the removal in Zambia.

Fourth, other barriers may discourage individuals from seeking care, like health staff absenteeism [Banerjee et al., 2008; Chaudhury and Hammer, 2004], distance from health facilities [Thornton, 2008] or imperfect information on the benefits and costs of health

⁴ For example, demand for health care cannot be fulfilled, longer waiting times induce higher risk of birth asphyxia, or going to a health facility may raise one's risk of contracting a disease if sanitary conditions have worsened.

investments [Rhee et al., 2005; Jalan and Somanathan, 2008; Banerjee et al., 2015]. The removal of user fees at the point of services might thus not be sufficient to reduce the marginal cost of health investment below the perceived marginal benefit associated with, leaving individuals' demand for health services unchanged. In particular, I explore how physical access to health amenities shapes the effect of removing user fees.

*
* *

3. Policy Background

Despite having one of the continent's fastest-growing economies between 2000 and 2010, Zambia is also one of the poorest and more unequal countries in Sub-Saharan Africa. According to the World Development Indicators [World Bank, 2022] in 2006, the year of policy adoption, more than 60% of the population lived with less than 1.90 dollars per day. About two-thirds of the poor were located in rural areas of the country, a situation that has barely changed since then. Life expectancy at birth stood at 50 years, and the average fertility rate was 5.7 births per woman. The same year, 75% of all deaths were due to communicable diseases or maternal, perinatal, and nutritional conditions, which are mostly preventable causes of death. In particular, maternal and neonatal disorders represented 7.2% of all deaths occurring in the country in 2006, a share that increased to 8.8% in 2017 [Global Burden of Disease, 2019].

3.1. Zambian health system

Health care provision in Zambia is organized through a three-tier referral system. The first level provides primary health care services and includes health posts, health centers as well as district hospitals. The second level of care corresponds to provincial and general hospitals, while the third one comprises central hospitals and the National University Teaching Hospital. In 2006, 85% of the 1,327 health facilities in the country were government-run, 9% were private facilities, and the remaining 6% were mission facilities that are publicly-supported [Chankova and Sulzback, 2006]. Like many countries in Sub-Saharan Africa, Zambia faces an important health worker shortage. In 2006, there were only 649 doctors, 6,096 nurses, and 2,273 midwives in the country [WHO, 2018]. For a corresponding population of 12.4 million, it gives a density of 7.3 health care professionals per 10,000 inhabitants, far below the World Health Organization's recommendation of 22.8 per 10,000 [WHO, 2006].

3.2. The user fee removal policy

After independence, one of the top priorities of the newly elected government was to improve health equity throughout the country between racial groups. From 1964, health care was provided free of charge at public health facilities. In 1993, during a period of structural adjustments, the government of Zambia decided to introduce user fees at all levels of care to raise additional resources for the health sector, struck by severe economic difficulties. A flat user fee was set by each health facility with the local community and District Health Office, depending on the ability to pay of the population living in its catchment area [Carasso et al., 2010]. Targeted fee exemptions were then introduced in 1995 for children below the age of five and the elderly (65 years old and above), antenatal care as well as chronic diseases, but were poorly implemented in practice [Masiye et al., 2010]. However, delivery services were not exempted from payment [Chama-Chiliba and Koch, 2016]. In a study by Cheelo et al. [2010], the average user fee charged for deliveries in a rural district of the North-Western province prior to user fee removal lied between 10,000 and 20,000 Zambian Kwachas (US\$ 2.84 and US\$ 5.68 in 2006), that is 15.5% to 31% of the average monthly per capita income in this province in 2006.⁵

In January 2006, the Zambian President announced that user fees were to be abolished for registration, consultation, outpatient and inpatient care, treatment, as well as diagnostic services in all publicly-supported primary health facilities of rural areas as a first step towards universal access to health services [Ministry of Health, 2007]. Facilities had to provide free health services to all individuals living in their catchment area, except foreigners. Patients referred to higher levels of care continued to be exempt from paying user fees. From April 1st, 2006, user fees were removed in government-run and mission facilities in 54 districts classified as rural but not in the 18 districts designated as urban.

One year after, in 2007, the government redefined eligibility criteria to extend the policy to rural areas of previously unaffected districts. From July, 1st, publicly-supported facilities located more than 15 kilometers away from the administrative center of urban districts and more than 20 kilometers away in urban districts located along the line of rail (the major Zambian railway) started to provide free primary health care. Such areas were previously excluded from the policy, despite levels of deprivation and poverty equivalent to rural districts.

User fees were finally removed in urban areas of urban districts from 2012, making primary

⁵ Ngulube and Carasso [2010] note that traditional healers are not necessarily cheaper than formal care but are generally more flexible on payment.

health care free in publicly-supported health facilities throughout the country from this date (see Appendix Figure A.1 for a timeline of the policy implementation).

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* *

4. Data and Estimation

4.1. Data

4.1.1. Individual-level data and outcome variables

I use four waves of microdata from the nationally representative Zambia Demographic and Health Surveys (DHS) conducted in 1996, 2001, 2007, and 2013. Appendix B provides a description of the sampling frame. Within sampled households, all women aged 15-49 who were either permanent residents of the household or visitors present on the night before the survey were eligible for survey interview. The Demographic and Health Surveys collect data on birth history,⁶ with detailed information on delivery conditions for births that occurred during the last five years preceding the survey, as well as maternal and under-five health, including anthropometric measurements and child death history.

Place of birth and the presence of a skilled birth attendant during childbirth constitute our main indicators of delivery conditions. Other things being equal, the removal of user fees may stimulate the demand for health services, including delivery services, through a reduction of the marginal cost of doing so. In that case, one should observe an increase in the probability of delivering in a publicly-supported facility. If health worker absenteeism did not increase dramatically as a result of the policy, a higher share of births should in turn be assisted by a skilled birth attendant.⁷ I also explore the effect on postnatal check-ups, which gives an insight into the quality of care received by women, but this information is only available in the last three survey waves.

Child health is proxied by anthropometric indicators and child mortality. Anthropometric indicators refer to being stunted (height-for-age z -score < -2), severely stunted (height-for-age z -score < -3) or wasted (weight-for-height z -score < -2). Stunting and wasting are often referred as indicators of chronic and acute malnutrition, respectively, and are strong predictors of overall health and mortality among under-five children. It is estimated that in

⁶ Interviewers ask women to report only live births. Very limited information on miscarriages, abortions and stillbirths is available and was not collected in 1996 and 2001.

⁷ Note that women may have difficulty in accurately reporting whether the attendant was qualified and in identifying distinct cadres of skilled birth attendants [Radovich et al., 2019].

2006, malnutrition was the main cause of 2.6% of infant deaths in Zambia [Global Burden of Disease, 2019] in addition to being a serious compounding factor in other causes of child mortality. In all waves, anthropometric measures were taken for survivors: of the 23,128 under-five children alive at survey time (90.1% of the original sample), 92.9% were measured (Appendix Table B.2).⁸ I show further below that my results on nutritional status are not driven by selective mortality using both inverse probability weighting and a semi-parametric approach based on survival probabilities. Premature deaths are measured by deaths at birth as well as neonatal and infant mortality risks, which correspond to the probability for a child to die before reaching the age of 28 days and one year respectively.⁹ Infant mortality risk is highly concentrated within the first days of life, when newborn survival is strongly related to delivery conditions. In my sample, about a third of neonatal deaths occurred on the day of birth and more than three quarters within the first week of life. A large part of these deaths is due to labour and delivery complications, such as birth asphyxia which accounts for a quarter of neonatal deaths and one-third of deaths in the first week of life in the country in 2006.¹⁰ The presence of a skilled birth attendant may help manage such complications in a life-saving way. Hence, by improving mothers' access to skilled birth attendants, the removal of user fees may have resulted in lower mortality risks and better health among newborns.

Finally, I explore the effect on health investment in children, proxied by whether child's vaccinations against polio, measles, diphtheria, pertussis, tetanus, and tuberculosis were up-to-date at survey time. One can reasonably expect that a more regular exposure to health workers following the policy can affect household's health-related decisions, including preventive investments.

4.1.2. Assignment to treatment

To identify individuals' district of residence, I obtained from the DHS the name of the district for each household surveyed in the first two waves and made use of the geographic

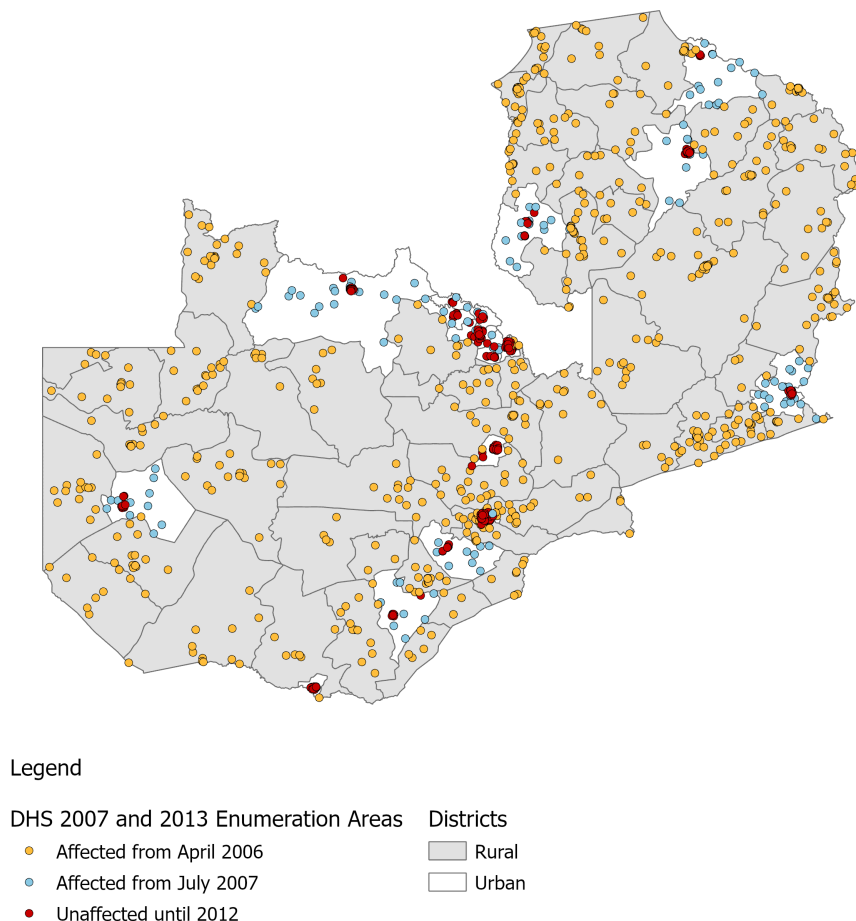
⁸ 2.3% were not measured because they were not present during interview, 0.7% refused to be measured and 4.1% missed anthropometric measurements because they were sick or for an unknown reason. Appendix Table B.2 decomposes these figures by survey wave. Among children measured, some have anthropometric indicators considered biologically implausible by the World Health Organization: height-for-age z -score below -6 or above 6 for stunting, and weight-for-height z -score below -5 or above 5 for wasting [WHO, 2019]. The corresponding 1.8% and 1.9% of measured children falling outside these intervals, respectively, are dropped from the analysis. Results do not change if these children are kept in the sample.

⁹ One concern that arises when using retrospective data is measurement error due to recall bias. I argue that recall bias can be considered low in this setting since the birth and death of a child are milestones in a woman's life, and the recall period of five years is relatively short. However, mothers may have rounded up child's age at death, leading to mismeasurement in child mortality. I show as a robustness check that my results are not sensitive to age-heaping.

¹⁰ Author's calculation from the Global Burden of Disease [2019] data (accessible from <http://ghdx.healthdata.org/gbd-results-tool>).

coordinates of each cluster for the last two (Figure 1). Since administrative boundaries changed after 1996, with some old districts splitting into several new ones, I use a consistent definition of district boundaries over time which respects the staggered implementation of the removal.¹¹

Figure 1. Map of districts and DHS clusters from 2007 and 2013



Source: Author based on DHS 2007 and 2013.

Notes: The map shows the 72 districts of Zambia according to the 2006 classification of the Government. Gray areas are rural districts and white areas represent urban districts. Clusters surveyed in the Demographic and Health Surveys were not georeferenced in 1996 and 2001. Hence, the map only reports DHS 2007 and 2013 clusters. Yellow dots correspond to clusters located in districts where user fees were removed from April 2006. Blue dots denote clusters located in rural areas of urban districts where user fees were removed from July 2007. Red dots represent clusters located in urban areas of urban districts, where user fees were maintained until 2012.

¹¹ 43 districts did not change over time, 10 districts split into 21 new ones with exactly the same exposure to the policy (i.e. for example, a given old district split into two districts that were equally affected by the policy in 2006) and four districts split into eight districts with different treatment status, which hinders accurate assignment of the corresponding DHS 1996 households to treated and non-treated areas. Thus, 911 births reported in 1996 are excluded from the analysis. It gives a total of $43+10+8=61$ harmonized districts, of which 43 are rural and 18 are urban.

Based on the progressive roll-out of the policy, I define three groups, two being affected from different dates and one being the control group. The first treatment group (T1) consists of individuals living in rural districts where user fees were removed as of April 2006. The second one (T2) corresponds to individuals living in rural areas of urban districts, affected from July 2007. Finally, the control group (C) refers to individuals living in urban areas of urban districts which were not affected by the policy until 2012. To very precisely determine the treatment status of an individual living in a rural area of an urban district, one would need to know both to which health facility's catchment area she belongs and the geographical coordinates of the corresponding health facility to compute the distance from the district administrative center. Such information is unfortunately not available. Thus, I consider exposed to the second wave of user fee removal individuals from urban districts who reside in an area classified as rural by the DHS. I show as a robustness check that the results do not change when using a finer assignment to treatment based on the eligibility criteria defined above for households sampled in 2007 and 2013.

I restrict my sample in three ways. First, I exclude children born before 1993 since they were already exposed to a policy of free health care. Second, I drop children born in 2012 or later since there is no more control group as the policy was extended throughout the country from this date. Finally, I exclude visitors since we do not observe their district of residence.¹² The analytical sample consists of 25,678 live births reported by 18,903 mothers, with reliable anthropometric information for 91.3% of children alive at survey time.

4.1.3. Health Facility Census

I complement the DHS with facility-level data obtained from the national Zambian Health Facility Census conducted in 2005. Precise information on the geographic coordinates, physical infrastructures, equipment, services offered, and headcount of health workers were collected from all public and mission health facilities. Figure 2 maps the corresponding location of public health facilities.

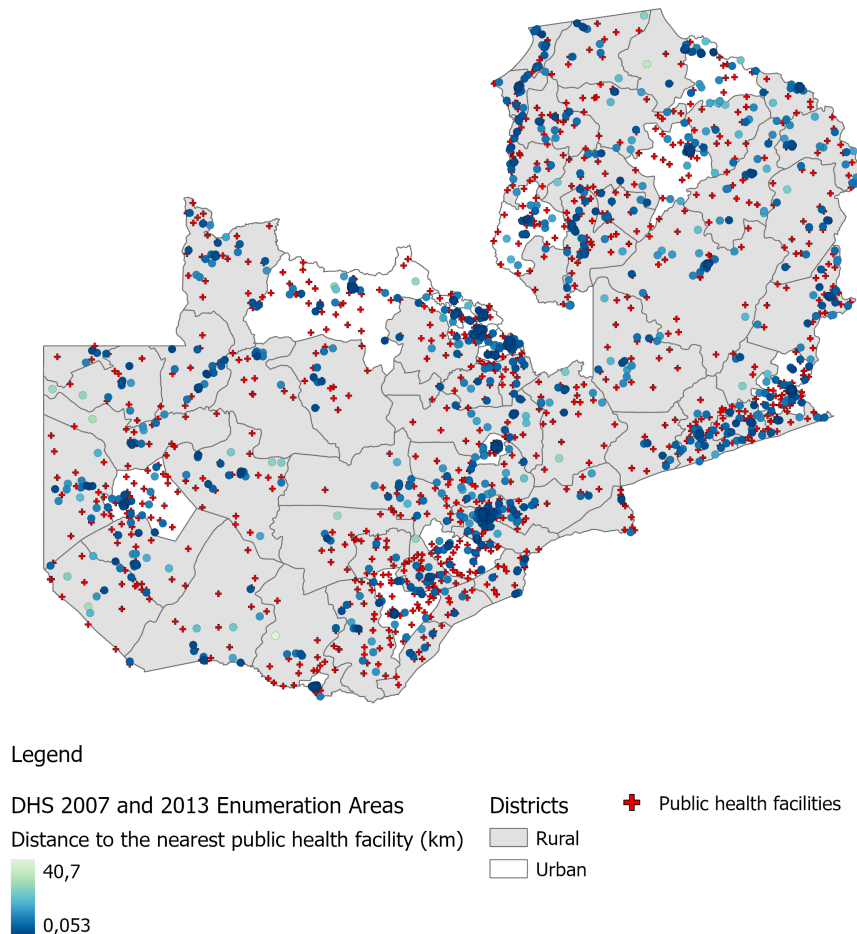
I use the straight-line distance from each DHS cluster surveyed in 2007 or 2013 to the nearest health facility as a proxy for travel time.¹³ This distance varies from 53 meters to

¹² The DHS define visitors as individuals who are not usual residents of the household, that is who usually do not live and eat with the household's members, but who stayed in the household the night before the interview [ICF, 2012]. Following this definition, 2.7% of all eligible adults interviewed in the four DHS survey waves I use are considered as visitors, similar to what is observed in the national census data from 1990, 2000, and 2010 (2.5%).

¹³ Results from Masiye et al. [2010] suggest that 92% of Zambians seek care at the nearest health facility to their home.

40.7 kilometers (see Figure 2). On average, households are located 6 kilometers away from their closest health facility.

Figure 2. Health facilities and DHS clusters from 2007 and 2013



Source: Author based on DHS 2007 and 2013, and 2005 National Health Facility Census.

Notes: The map shows the DHS clusters from 2007 and 2013 along with public health facilities from the 2005 National Health Facility Census. Clusters surveyed in the Demographic and Health Surveys were not georeferenced in 1996 and 2001. Darker color indicates higher proximity to a public health facility present in the 2005 census.

To ensure respondents' confidentiality, the DHS randomly displace cluster location¹⁴ (see Appendix B.4 for more details), creating a measurement error in the distance to the nearest facility which generates an attenuation bias [Arbia et al., 2015]. Corresponding point estimates thus represent lower bounds of the true effects of distance to the nearest health facility on delivery and health outcomes.

Beyond monetary cost and distance, the quality of health services available locally may play

¹⁴ Urban clusters are randomly displaced within a radius of 2 kilometers around their true location. Rural clusters are randomly displaced within a radius of 5 kilometers around their true location and up to 10 kilometers for a further 1% of them.

a crucial role for parents in non-emergency situations when deciding where to give birth or whether to seek care for their child and, if so, where. In particular, perceived quality might be a key determinant of such decisions, and quality itself may improve newborns' survival chances. I construct an indicator for the local availability of essential care based on [Gabrysch et al. \[2011\]](#). It measures the provision of emergency obstetric care and child health services by a publicly-supported health facility within a radius of five kilometers around each enumeration area.¹⁵ One concern is that such data is only available for the year 2005: new facilities may have opened while others may have closed. To limit this problem, I alternatively restrict my sample to births occurring three and four years around the census date as a robustness check. Conclusions presented below remain unchanged.

4.2. Empirical approach

I here consider a simple set-up with two time periods, one before ($t = 0$) and one after ($t = 1$) the abolition of user fees in public health facilities, which corresponds to the treatment. As in the causal model developed by [Rubin \[1974\]](#), let $Y_{i1}(1)$ be the potential outcome of individual i that would be realized at time $t = 1$ if exposed to the policy ($T_i = 1$). Conversely, $Y_{i1}(0)$ denotes the potential outcome for the same period and individual if not exposed to the policy ($T_i = 0$). In this case, $Y_{i1}(0)$ is the counterfactual of $Y_{i1}(1)$ in absence of the policy. Thus, the causal effect of the policy for individual i is given by:

$$\Delta_{i1} = Y_{i1}(1) - Y_{i1}(0)$$

and the average treatment effect on the treated is:

$$\mathbb{E}[Y_{i1}(1) - Y_{i1}(0)|T_i = 1] = \mathbb{E}[Y_{i1}(1)|T_i = 1] - \mathbb{E}[Y_{i1}(0)|T_i = 1]$$

where $\mathbb{E}[Y_{i1}(1)|T_i = 1]$ is the average post-treatment potential outcome of individuals exposed to the policy and $\mathbb{E}[Y_{i1}(0)|T_i = 1]$ corresponds to what would have been observed for treated individuals at time $t = 1$ in absence of the policy.

¹⁵ This proxy is an indicator variable that takes the value of one if there is at least one publicly-supported health facility within five kilometers (1) providing at least 4 out of 6 basic emergency obstetric care signal functions (injectable antibiotics, injectable oxytocics, injectable anticonvulsants, manual removal of placenta, manual removal of retained products, assisted vaginal delivery), (2) offering referral services for obstetrics emergencies with a vehicle or using communication tools, (3) having at least a midwife or a doctor present or on call 24/7, (4) having at least two registered health professionals, including one on duty at the time of the census, and (5) performing resuscitation of newborns, growth monitoring, deworming, infant feed counseling, as well as case management of diarrhea, dehydration and pneumonia, zero otherwise. Only 12% of the 1,274 publicly-supported health facilities present in the census meet these criteria, and 24% of households in my sample live within five kilometers of at least one of them.

Unfortunately, we cannot observe both potential outcomes $Y_{it}(0)$ and $Y_{it}(1)$ for any individual i at time t . In particular, we do not observe what would have been the average outcome of treated individuals in absence of the policy, $\mathbb{E}[Y_{i1}(0)|T_i = 1]$, and have to estimate this counterfactual.

One possibility is to run a before-after analysis by comparing pre-treatment and post-treatment outcomes for the same individuals:

$$\mathbb{E}[Y_{i1}(1)|T_i = 1] - \mathbb{E}[Y_{i0}(1)|T_i = 1]$$

It assumes that there would have been no change in average potential outcome over time in absence of the policy. However, this is very unlikely since maternal and child mortality risks were decreasing even before the policy implementation and, as shown further below, delivery conditions in unaffected areas improved over the study period.

An alternative is to use the average potential outcome of untreated individuals at time $t = 1$ as a counterfactual, that is:

$$\mathbb{E}[Y_{i1}(1)|T_i = 1] - \mathbb{E}[Y_{i1}(0)|T_i = 0]$$

It assumes that treated and untreated individuals only differ by their treatment status. This is unlikely here since the removal of user fees was not randomly assigned across the country but instead was explicitly targeted towards most deprived areas, which are very different from urban areas unaffected by the policy until 2012.

To assess the causal effect of removing user fees in public health facilities on Y , I take advantage of the progressive roll-out of the policy across the country. I observe deliveries and anthropometric measurements occurring before ($t = 0$) and after ($t = 1$) the removal of user fees, both in treated ($T = 1$) and untreated ($T = 0$) areas. It allows me to use a difference-in-differences estimation strategy to identify the causal impact of the policy on Y :

$$\left(\mathbb{E}[Y_{i1}(1)|T_i = 1] - \mathbb{E}[Y_{i0}(1)|T_i = 1] \right) - \left(\mathbb{E}[Y_{i1}(0)|T_i = 0] - \mathbb{E}[Y_{i0}(0)|T_i = 0] \right) \quad (1)$$

Change in $\mathbb{E}[Y_{it}(0)]$ over time in unaffected areas is used to estimate the unobserved counterfactual change for affected areas had user fees not been abolished. This identification

strategy rests on the assumption that:

$$\mathbb{E}[Y_{i1}(0)|T_i = 1] - \mathbb{E}[Y_{i0}(0)|T_i = 1] = \mathbb{E}[Y_{i1}(0)|T_i = 0] - \mathbb{E}[Y_{i0}(0)|T_i = 0]$$

commonly referred to as the parallel trend assumption, which is extensively discussed in Section 4.5.

4.3. Summary statistics

Columns 1-2 in Table 1 show the summary statistics before the policy implementation for children from rural districts (T1) and urban areas (C). Changes in demographic characteristics and outcome variables after the removal are presented in columns 3-4. Column 5 reports p -values obtained when comparing these changes following Equation 1. Columns 6-11 replicate this analysis for rural areas of urban districts (T2) affected one year apart.

Affected areas and the control group are significantly different before the policy change. In particular, children from affected areas have, on average, a mother less educated than their urban counterparts and a higher probability to be born at home without the help of a skilled birth attendant. They also have a worse nutritional status, with a probability of being stunted 35% higher, and are 65% to 71% more susceptible to be severely stunted. Such baseline differences are not a threat to identification, which relies on the parallel trend assumptions.¹⁶

Delivery conditions and child anthropometric indicators changed significantly differently in affected and unaffected areas in the aftermath of the policy. The probability of being assisted by a skilled birth attendant during childbirth and of giving birth in a publicly-supported facility increased significantly faster in affected areas than in control ones. We also observe a much steeper decline in the risk of stunting in affected areas. However, I cannot detect any difference in child mortality risk, both in level before the removal and in changes after it. I show further below that when taking into account year of childbirth- and area-specific effects, demographic characteristics of mothers from affected areas changed in a way that is similar to those from unaffected ones.

¹⁶ Conclusions remain the same when the estimation strategy outlined below is combined with matching (see robustness checks).

Table 1. Extensive summary statistics

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Before		Change after 2006		$\Delta T1 = \Delta C$		Before		Change after 2007	
	T1	C	$\Delta T1$	ΔC	p -value	T2	C	$\Delta T2$	ΔC	p -value
Panel A. Demographic characteristics										
Mother's age at childbirth	26.29	25.63	0.73	0.37	0.085	26.22	25.65	0.84	0.38	0.134
Mother's number of years of education	4.53	7.28	0.74	0.90	0.099	4.59	7.32	0.46	0.95	0.001
Girl	0.50	0.50	-0.01	0.00	0.760	0.51	0.50	0.00	0.00	0.892
Multiple birth	0.04	0.04	-0.01	-0.02	0.028	0.04	0.04	-0.01	-0.02	0.228
Preceding birth interval	36.33	39.83	0.86	4.35	0.000	36.23	40.01	-1.25	4.59	0.000
Panel B. Childbirth conditions										
Assisted by a health professional	0.33	0.80	0.22	0.06	0.000	0.29	0.80	0.17	0.07	0.000
Assisted by a doctor	0.01	0.08	0.02	0.00	0.021	0.01	0.08	0.01	0.01	0.963
Assisted by a nurse or midwife	0.31	0.77	0.21	0.05	0.000	0.27	0.77	0.17	0.06	0.000
Assisted by a clinical officer	0.01	0.01	-0.01	0.00	0.008	0.01	0.01	0.00	0.00	0.155
Assisted by a traditional birth attendant	0.16	0.03	0.06	0.01	0.000	0.21	0.04	0.09	0.01	0.000
Assisted by a relative or no one	0.52	0.17	-0.28	-0.07	0.000	0.50	0.16	-0.26	-0.07	0.000
Delivered at home	0.67	0.20	-0.25	-0.06	0.000	0.70	0.20	-0.20	-0.07	0.000
Delivered in a public health facility	0.33	0.71	0.25	0.13	0.000	0.29	0.72	0.20	0.12	0.000
Delivered in a government-run facility	0.24	0.70	0.28	0.13	0.000	0.26	0.71	0.21	0.13	0.000
Delivered in a mission health facility	0.08	0.01	-0.02	0.00	0.008	0.03	0.01	-0.01	0.00	0.185
Delivered in a private health facility	0.00	0.09	0.00	-0.06	0.000	0.01	0.09	0.00	-0.06	0.000
Panel C. Child mortality										
Death at birth	0.01	0.01	0.00	0.00	0.374	0.01	0.01	0.00	0.00	0.681
Neonatal mortality risk	0.03	0.04	-0.01	-0.01	0.523	0.03	0.04	-0.01	-0.01	0.347
Infant mortality risk	0.09	0.09	-0.04	-0.03	0.660	0.08	0.09	-0.03	-0.03	0.703
N	9,815	3,810	16,199	5,949	22,148	2,549	4,161	3,530	5,949	9,479
Panel D. Child nutritional status										
Stunted	0.54	0.40	-0.10	-0.04	0.000	0.55	0.40	-0.11	-0.04	0.007
Severely stunted	0.28	0.17	-0.08	-0.02	0.000	0.29	0.17	-0.10	-0.03	0.000
Wasted	0.06	0.06	-0.01	0.00	0.291	0.06	0.06	-0.01	0.01	0.303
N	5,788	2,211	13,544	4,842	18,386	1,795	3,029	2,942	4,842	7,784

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Unweighted statistics. The first two columns report summary statistics for live births occurring before April 2006 (Panels A, B, and C) or children measured in a survey wave prior to the removal (Panel D) in rural districts (T1) and control areas (C). Columns 3 and 4 show changes in average characteristics observed in the aftermath of the policy in both groups. Column 5 reports p -values computed from crude differences-in-differences in which I compare changes over time in areas affected from April 2006 versus control areas following Equation 1. Columns 6 to 10 replicate this analysis for areas affected from July 2007 (T2). Control areas correspond to urban areas unaffected by the policy until 2012. In Panel D, the sample is restricted to children alive at survey time. Note that column 2 reports summary statistics for children of the control group born (Panels A, B, and C) or observed (Panel D) up to April 2006, while column 7 reports summary statistics for children of the control group born (Panels A, B, and C) or observed (Panel D) up to July 2007.

4.4. Estimation strategy

Taking advantage of the progressive roll-out of user fee removal across the country, I employ a difference-in-differences design and estimate the average effect of the policy from the following equation:

$$y_{imta} = \alpha + \gamma Exposed_{ta} + X'_{imta}\Gamma + \delta_a + \delta_t + \varepsilon_{imta} \quad (2)$$

where y_{imta} is the outcome of interest for child i of mother m who lives in area a , and t the time period relevant for the outcome being investigated. It will refer to year of childbirth when looking at retrospective childbirth outcomes and child mortality, and to survey year when looking at contemporaneous outcomes, including anthropometric indicators measured at survey time. Areas are the geographic unit at which the policy has been rolled out. Each area corresponds either to a rural district, the rural area of an urban district, or the urban area of an urban district, which gives a total of 79 areas. δ_a denotes area fixed effects, which take into account any time-invariant area-specific factors such as risks of diseases,¹⁷ and δ_t time fixed effects, which control for area-invariant time-specific factors such as macroeconomic conditions common to all areas in the country. The independent variable of interest, $Exposed_{ta}$, is an indicator variable taking the value of one if user fees were removed in area a at time t , zero otherwise. A positive γ would indicate an average increase in the outcome of interest after user fee removal in affected areas relative to unaffected ones.¹⁸ X_{imta} is a set of covariates, including a dummy for high-risk multiple pregnancy, as well as mother's year of birth for childbirth conditions, mother's year of birth and child's sex when looking at child mortality, and child's sex and age dummies¹⁹ for anthropometric outcomes.²⁰

Recent advances in econometric theory show that the two-way fixed effects (TWFE) estimator with staggered treatment adoption may yield to biased estimates in presence of heterogeneous treatment effects (e.g. [de Chaisemartin and D'Haultfœuille 2020, 2021](#); [Goodman-Bacon 2021](#)). [Goodman-Bacon \[2021\]](#) shows that the TWFE difference-in-differences esti-

¹⁷ Due to data limitations, I do not observe the effective area of birth of each child. A mother may have migrated since then or may have given birth in an area different from the one where she currently lives. Hence, her current area of residence might not be the same as the one where she gave birth. I can only partially deal with this issue by restricting my sample to mothers who already lived there before childbirth, leaving the results unchanged (see robustness checks).

¹⁸ Note that for anthropometric outcomes, γ combines the effect of a difference in exposure status (children measured in 2007) and the effect of a difference in length of exposure to the policy (children measured in 2013 and 2014) since the policy has been extended to the entire country from 2012.

¹⁹ Alternatively, controlling for dummies of age in months or a cubic relationship with age in months leaves the results unchanged (Appendix Figure H.1).

²⁰ Results are virtually unchanged when controlling for a full set of maternal covariates which are not included in the main specification due to endogeneity issues (see robustness checks).

mator is a weighted average of all possible two groups-two periods difference-in-differences estimators. In particular, already-treated units act as a control group for not-yet treated units, which is problematic under time-varying treatment effects. The resulting bias then feeds through to γ based on the weight attached to such two groups-two periods comparisons. In our context, the problem arises when rural districts are used as a control group in the two groups-two periods difference-in-differences that estimate the effect of removing user fees in rural areas of urban districts. Based on the Bacon decomposition [Goodman-Bacon, 2021], I find that it accounts for less than 5% of the point estimates obtained with the TWFE estimator, which primarily relies on the comparison of the treated groups (T1 and T2) with the never-treated one (83 to 96% depending on the outcome).

I overcome this issue in two ways. First, I separately estimate the effect in the two treatment groups using only the never-treated (urban areas) as the control group. It has the advantage of allowing for the estimation of phase-specific effects of the policy, and I can check whether the policy had the same effects in both types of treated areas. Second, I use the estimator developed by de Chaisemartin and D’Haultfœuille [2021] which is unbiased in the presence of heterogeneous treatment effects. As expected, conclusions remain unchanged with this alternative estimator.²¹

To take into account serial correlation and to avoid overrejection of the null hypothesis of no effect, robust standard errors are clustered at the area level [Bertrand et al., 2004; Cameron and Miller, 2015] in all specifications. This is an intention-to-treat estimate since some health workers in rural areas may have decided to still charge fees on patients despite the law, and some patients living in urban areas might have received health care in an affected area despite the limitation of the policy to individuals living in the catchment area of affected facilities. Moreover, some individuals supposed to be treated may not have benefited from the policy because of the remoteness of health facilities in rural areas. Hence, compliance with the policy is likely to be imperfect.

4.5. Parallel trends assumption

Change over time in outcomes of interest in urban areas is used to estimate the unobserved counterfactual change for rural areas had user fees not been abolished. The key identifying assumption here, known as the parallel trends assumption, is that in absence of the policy both rural and urban areas would have experienced the same trends in the outcomes of interest. It implies that in absence of the policy, area-specific confounders

²¹ Note that my results are also robust to other estimators proposed by Callaway and Sant’Anna [2020], Borusyak et al. [2021], and Gardner [2021] as shown in Appendix Table D.1.

must be time-invariant and time-specific confounders must be common across treated and untreated areas [Angrist and Pischke, 2009]. It cannot be tested since it would require to observe the average post-treatment outcomes in treated areas in absence of the treatment. One can assess the plausibility of this assumption by checking pre-treatment trends in outcomes between treated and untreated areas, conditional on the covariates included in the estimation. If trends were parallel in pre-treatment periods, then we might expect trends to have remained the same in post-treatment periods had user fees not been removed.

4.5.1. Graphical evidence

First, I present graphical evidence of parallel pre-treatment trends. Figures reported in Appendix C.1 plot the raw and conditional pre-treatment trends in outcomes of interest. Until the removal of user fees, the different outcomes follow similar trends in affected and unaffected areas. After it, the figures show an increase in maternal health care utilization and a decrease in chronic malnutrition in affected areas.

4.5.2. Event-study specification

I also formally test for differential pre-trends between affected and unaffected areas using an event-study design, in which I include leads and lags of exposure to the policy. I estimate the following event-study specification, which allows for year-specific effects of the policy on the outcomes of interest:

$$y_{imta} = \alpha + \sum_{\tau=-L}^{-2} \beta_{\tau} \mathbb{1}_{t=\tau} \times \text{Removed}_a + \sum_{\tau=0}^K \gamma_{\tau} \mathbb{1}_{t=\tau} \times \text{Removed}_a + X'_{imta} \Gamma + \delta_a + \delta_t + \varepsilon_{imta} \quad (3)$$

where L and K are the total number of pre-removal and post-removal periods, respectively. Removed_a is an indicator variable taking the value of one if user fees were removed in area a , zero otherwise. Since user fees were removed in different years in rural districts and rural areas of urban districts, I normalize the year of policy implementation to 0. Hence, $\tau \geq 0$ denotes post-treatment periods and $\tau \leq -1$ pre-treatment periods. As usual in this kind of specification, the omitted one is the last pre-treatment period, that is $\tau = -1$. γ_{τ} now indicates the policy's effect τ years after its implementation, while β_{τ} corresponds to the policy's effect τ years before its implementation, relative to the last pre-treatment period.

This event-study design allows one to assess the effect of user fee removal over time by looking at γ_{τ} , and to formally test the parallel pre-trends between treatment groups. If

trends are parallel before the removal, then the coefficients β_τ should not be significantly different from zero. By doing so, I can assess the timing of the policy's effects and check for diverging trends prior to policy implementation.

Point estimates and 95 percent confidence interval are reported in Figures 3 and 5.²² Compared to unaffected ones, results suggest that affected areas did not exhibit a significantly different pattern prior to user fee removal, whatever the outcome considered.²³

4.5.3. Placebo tests

Finally, I implement a broad set of placebo tests where I compare unexposed children from both types of areas. For this, I drop children born in the aftermath of the policy and use the full set of lags of the real implementation date as starting points of a series of fictitious policies. Then, I run difference-in-differences regressions using the newly defined implementation dates. If affected and unaffected areas were on similar slopes before the removal, point estimates from these regressions should be statistically insignificant and close to zero. This is what I find, as reported in Appendix Figure C.6. Here again, it fails to reject the null of pre-treatment parallel trends between affected and unaffected areas: only 9 point estimates out of 255 are marginally significant at the five percent level.

All together, these results strongly support the identifying assumption and thus the causal interpretation of my results.

*
* *

²² Similar figures for rural districts and rural areas of urban districts separately are presented in Appendix Figures C.2 to C.5.

²³ I do not investigate the effect of the policy on prenatal visits since treated and control districts were already on different slopes before the removal of user fees, which prevents the causal interpretation of the corresponding point estimates. This is not surprising since prenatal visits were made free of charge in 1995 and increased gradually over time. Moreover, attendance was already high before the removal, with 96% of women making at least one prenatal visit and 72% at least four prenatal visits. The results remain unchanged when I control for having done at least four prenatal visits in the estimation and when I control for a linear time trend interacted with the share of pregnancies for which at least four prenatal visits have been done within the area of residence, before policy implementation (Appendix Table H.2). This is not done in the main specification due to the endogenous nature of prenatal visits.

5. Results

5.1. Effect on maternal health care utilization

5.1.1. The probability of giving birth in a publicly-supported health facility increased sharply after the removal

Table 2 reports the average effect of the policy on place of delivery. The result suggests a sharp increase in medical deliveries, which is significant at less than 0.1 percent. The user fee removal led to a rise of 13.9 percentage points in the probability of giving birth in a medical facility, a 43 percent increase relative to the pre-policy mean (Panel A, column 1). This result is confirmed when the potential bias introduced by heterogeneous treatment effects is taken into account (Panel B, column 1). I then estimate the effect separately for rural districts and rural parts of urban districts (Panels C and D). Rural districts exhibit a stronger effect of the policy, but relative to the pre-policy mean the results remain similar. This result echoes the one from [Hangoma et al. \[2018\]](#), who find an increase of overall utilization of care following the removal. However, point estimates from the event-study suggest that this increase did not materialize right after the removal (see Figure 3), a result consistent with [Lépine et al. \[2018\]](#) and [Chama-Chiliba and Koch \[2016\]](#) who respectively find no effect on health care utilization and deliveries in public facilities in the very short-term.

Columns 2 and 3 report the effect in publicly-supported and private facilities. The aggregate effect found in column 1 is exclusively driven by deliveries in publicly-supported health facilities, with a strong and sustained effect over time (see Figure 3), which is reassuring since the policy change only applies to this type of facility. One might be concerned if the removal of user fees only causes mothers to switch from the private sector to the public one and did not reach those delivering at home without a skilled birth attendant. This is not the case here since the overall utilization of health facilities increases, and there is no effect on private facilities.

5.1.2. A higher share of childbirths were assisted by a skilled birth attendant after the removal

Given the high increase in institutional deliveries I find, one can reasonably expect to observe an increase in the share of births assisted by a skilled birth attendant unless health worker absenteeism dramatically increased as a result of the policy. Table 2 reports the results for medical assistance received during childbirth.

Table 2. The effect of user fee removal on childbirth conditions

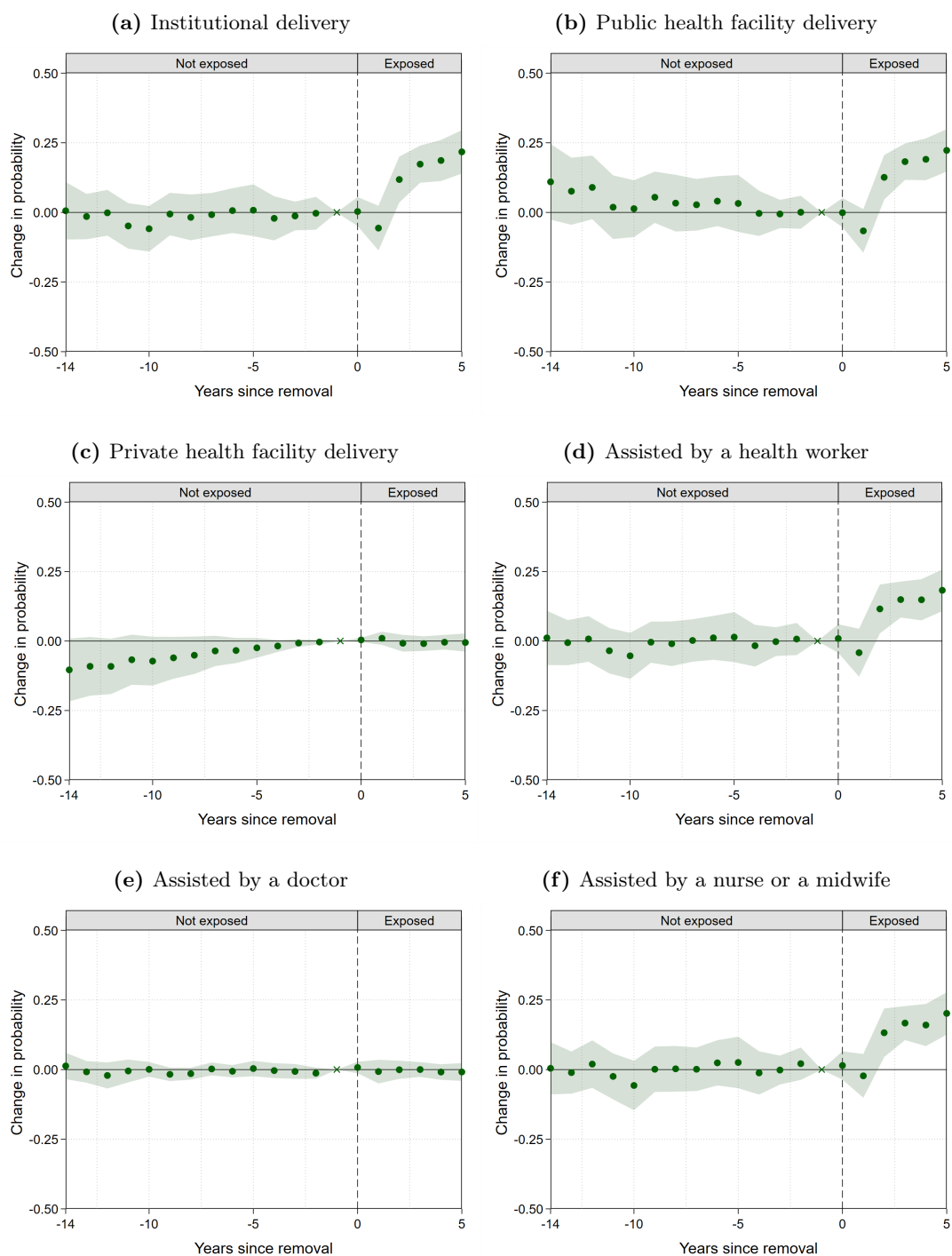
	(1)	(2)	(3)	(4)	(5)	(6)
	Institutional delivery	Type of health facility		Assisted by a		
		Public	Private	Health worker	Doctor	Nurse or Midwife
<i>Panel A. Average effect of user fee removal</i>						
Affected by the policy	0.139*** (0.024)	0.108*** (0.034)	0.031 (0.022)	0.114*** (0.023)	0.004 (0.011)	0.120*** (0.024)
Mean before policy	0.323	0.319	0.004	0.318	0.013	0.300
R ²	0.224	0.202	0.244	0.218	0.045	0.208
N	25,485	25,485	25,485	25,580	25,580	25,580
<i>Panel B. Average effect of user fee removal using de Chaisemartin and D'Haultfœuille [2021] estimator</i>						
Affected by the policy	0.126** (0.053)	0.131*** (0.049)	-0.005 (0.019)	0.110** (0.051)	0.002 (0.019)	0.128** (0.054)
Mean before policy	0.323	0.319	0.004	0.318	0.013	0.300
N	25,485	25,485	25,485	25,580	25,580	25,580
<i>Panel C. Effect in rural districts</i>						
Affected from 2006	0.165*** (0.024)	0.131*** (0.034)	0.034 (0.025)	0.136*** (0.024)	0.005 (0.012)	0.143*** (0.024)
Mean before policy	0.330	0.326	0.004	0.325	0.013	0.306
R ²	0.229	0.205	0.252	0.222	0.048	0.211
N	21,974	21,974	21,974	22,063	22,063	22,063
<i>Panel D. Effect in rural parts of urban districts</i>						
Affected from 2007	0.132*** (0.046)	0.106** (0.051)	0.026 (0.024)	0.104** (0.043)	-0.007 (0.013)	0.111** (0.045)
Mean before policy	0.297	0.291	0.006	0.291	0.015	0.274
R ²	0.292	0.259	0.268	0.295	0.057	0.270
N	9,431	9,431	9,431	9,442	9,442	9,442

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth. The table reports the average (Panels A and B) and phase-specific effect (Panels C and D) of user fee removal on the probability to give birth in a health facility (column 1), in a publicly-supported health facility (column 2), in a private one (column 3), to give birth with a skilled birth attendant (column 4), with a doctor (column 5) and with a nurse or a midwife (column 6). Each coefficient is from a different regression. All regressions control for area and year of childbirth fixed effects, as well as mother's year of birth and a dummy for multiple births.

* $p < .10$; ** $p < .05$; *** $p < .01$

Figure 3. Event study estimates of the effect of user fee removal on childbirth conditions



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification. Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include mother's year of birth and a dummy for multiple pregnancy. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are dummies equal to one if mother gave birth (a) in a health facility, (b) in a public or mission health facility, and (c) in a private health facility, in presence of (d) a health worker, (e) a doctor, and (f) a nurse or a midwife, zero otherwise.

Column 4 indicates an 11.4 percentage points increase in the probability of giving birth with the help of a skilled birth attendant in affected areas. Compared to the pre-policy mean, it represents a 36 percent increase (Panel A). This result remains remarkably stable when the potential bias arising from heterogeneous treatment effects is considered (Panel B). It also suggests a stronger effect in rural districts (42 percent increase, Panel C) than in rural parts of urban districts (36 percent increase, Panel D). The pattern presented in Figure 3 is striking: before policy implementation, there is no differential trend between rural and urban areas, whereas after the removal, the probability of being assisted by a skilled birth attendant increased significantly faster in rural areas than in unaffected ones. In both treatment groups, the effect is however exclusively driven by deliveries with a nurse or a midwife (column 6), while the probability of being assisted by a doctor remains close to zero (column 5). This is consistent with the high concentration of doctors in cities and urban areas.

Finally, I investigate the effect on postnatal check-ups received by the mother after delivery. I find that the removal of user fees has increased the probability for mothers to receive a postnatal check-up within the first 24 hours following childbirth. The results indicate a significant 7.8 percentage points increase in rural districts and a nearly identical effect in rural parts or urban districts, though not significant, perhaps due to smaller sample size (see Appendix Table E). While this result suggests that more women benefited from postnatal check-ups, it should also be interpreted in light of the large increase in institutional deliveries I found (+13.9 percentage points). The large discrepancy in the two effects suggests poor quality of care since many women who delivered in health facilities did not benefit from a medical check-up that could have prevented postpartum complications. This could explain, at least to some extent, why there is no discernible trend break in maternal mortality ratio around the policy (Appendix Figure F.1).

5.1.3. These positive effects decrease with distance from the nearest facility

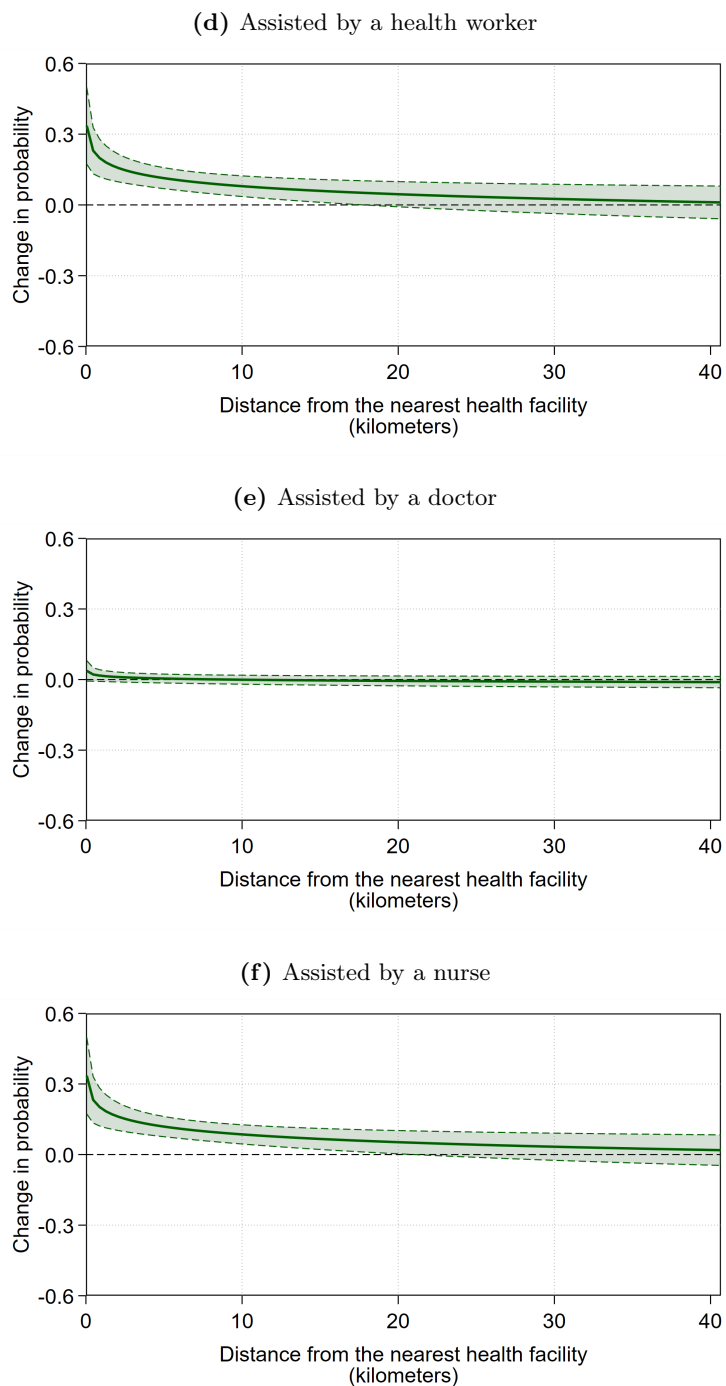
The policy may have had heterogeneous effects with respect to the physical access of households to public health facilities. In particular, it may have benefited more those living near a health facility. This is exactly what I find. To investigate this, I use the log of the straight-line distance between each DHS cluster position and the nearest publicly-supported health facility as a proxy for travel time interacted with exposure to free health care. Figure 4 plots the corresponding marginal effect of the policy on childbirth conditions outcomes. As expected, the positive effect on childbirth conditions decreases as the distance increases.

Figure 4. Marginal effect of exposure to the policy on childbirth conditions depending on distance from the nearest health facility



continued →

Figure 4 (*continued*). Marginal effect of exposure to the policy on childbirth conditions depending on distance from the nearest health facility



Source: Author's calculations from DHS 2007 and 2013.

Notes: The figures plot the marginal effect of the policy change depending on the distance from the nearest health facility. Distance corresponds to the straight-line distance between each DHS cluster from the 2007 and 2013 survey waves and the nearest publicly-supported health facility from the 2005 Health Facility Census. Each figure is from a separate estimation where the distance is log-transformed and interacted with exposure to the policy. Control variables include area and year of childbirth fixed effects, as well as mother's year of birth and a dummy for multiple births. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level.

I find no differential effect of the policy on childbirth conditions with respect to the local availability of a qualified health facility (results not shown available upon request). This is not surprising for at least two reasons. First, parents may not be aware of the effective quality of services offered in all health facilities near their home. Second, even if they are, a spontaneous, non-planned delivery may force them to go to the nearest one, whatever the perceived quality.

5.2. Effect on child health

5.2.1. *Chronic malnutrition decreased after the removal of user fees*

The average effect of the policy on anthropometric indicators is presented in columns 1 to 3 of Table 3. The results clearly indicate a positive effect of free primary health services on child nutritional status with a significant 8 percent reduction in the prevalence of stunting. The effect is even stronger on severe stunting, with a 15 percent reduction relative to the pre-policy period (Panel A).²⁴ This is in line with [Bagnoli \[2019\]](#) and [Friedman and Keats \[2019b\]](#), who respectively find a significant and positive effect of health insurance and free deliveries on child height-for-age z -score in Ghana. These results are encouraging since childhood stunting is a strong marker of recurrent and severe infections with long-lasting effects on health, and is commonly used as a proxy for healthy growth. Conclusion remains the same when using the [de Chaisemartin and D’Haultfoeuille \[2021\]](#) estimator (Panel B).

There is no discernible effect on acute malnutrition. This is not surprising since wasting does not reflect the cumulative effects of poor health conditions but is rather the result of a rapid deterioration in nutritional status over a short period of time, probably independent of a regular access to formal care.

Since analysis of child anthropometric indicators is solely based on survivors’ measurement at survey time, one potential threat to identification for these outcomes is selection due to endogenous mortality. In particular, survivors may be stronger than those who died prematurely. However, such selection effect is unlikely to play a role here as we do not observe any effect of the policy on child mortality outcomes as discussed further below. Nonetheless, I test the robustness of my results by considering selection through survival in two ways.²⁵ First, I use an inverse probability weighting method to weight observations according to predicted survival probabilities at survey time. Second, following [Cosslett](#)

²⁴ Importantly, these results are not driven by a differential seasonality effect in the measurement of anthropometric indicators across treatment groups.

²⁵ I do not implement the standard Heckman two-step procedure since the predictors of the selection equation and the main equations are the same.

[1991], I use a semi-parametric approach by including one indicator variable for each centile of predicted survival probabilities as additional control variables. In both cases, point estimates remain extremely similar (results reported in Appendix Table H.3).

I also investigate the effect of removing user fees on health investment in children through vaccination. In particular, I check whether child's vaccinations against polio, measles, diphtheria, pertussis, tetanus, and tuberculosis were up-to-date at survey time. Results are presented in column 4 of Appendix Table E.1. I find no discernible effect of the policy on child's vaccination.

5.2.2. The decrease in chronic malnutrition only occurs after a certain duration of exposure to the removal of user fees

Duration of exposure to the policy may drive the average treatment effect I found on anthropometric outcomes. One can reasonably expect that children should benefit more if they have been exposed longer to free primary health care. To investigate any heterogeneous effect of the duration of exposure, I change the $Exposed_{ta}$ term in my equation for a continuous measure of exposure based on date of measurement, date of birth and date of user fee removal in area a . This measure ranges from 0 to 59 months of exposure. Results are reported in Table 4. I find that being exposed to the policy for an additional month significantly reduces chronic malnutrition (columns 1 and 3) but has no impact on the risk of being wasted (column 5). However, such effects may require a minimum duration of exposure to manifest. This is exactly what I find (columns 2, 4, and 6). For instance, results suggest that children need to be exposed to the policy for more than 12 months for their risk of being severely stunted to shrink.

5.2.3. Child mortality risk is only affected near health facilities providing essential emergency obstetric care and child health services

The results for child mortality outcomes appear in columns 4 to 6 of Table 3. For neonatal and infant mortality, I dropped children who did not reach the corresponding age at survey time to avoid censoring bias, and by 2012 since the policy was then extended to the control group. All point estimates are precisely estimated and very close to zero, suggesting that, on average, the removal of user fees has had no impact on child mortality, regardless of the definition considered and whatever the distance from the nearest health facility, as shown in Figure 6.

Table 3. The effect of user fee removal on child health

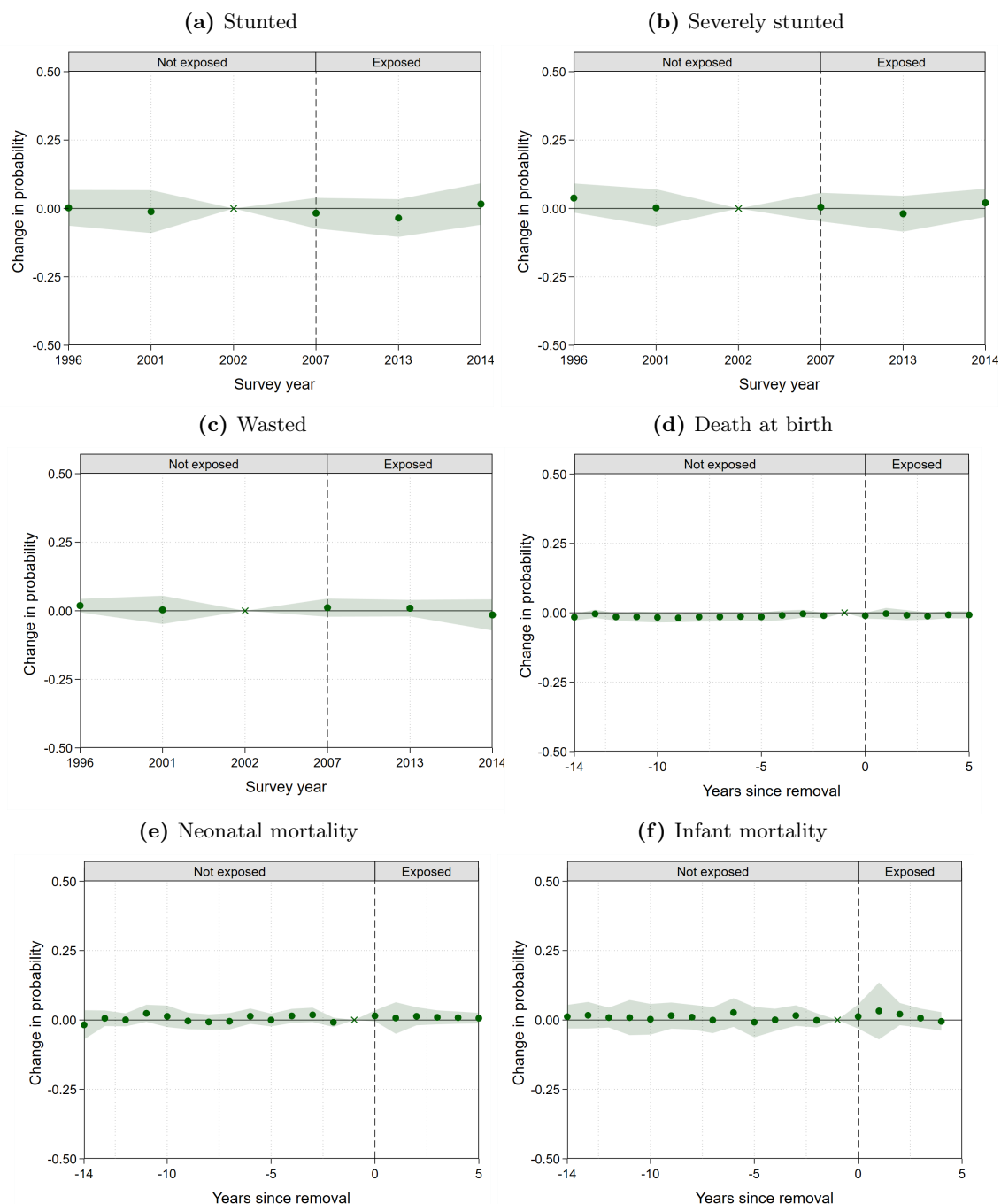
	(1)	(2)	(3)	(4)	(5)	(6)
	Stunted	Severely stunted	Wasted	Death at birth	Neonatal mortality	Infant mortality
<i>Panel A. Average effect of user fee removal</i>						
Affected by the policy	-0.044** (0.021)	-0.043*** (0.015)	-0.004 (0.011)	0.002 (0.003)	0.003 (0.005)	-0.004 (0.008)
Mean before policy	0.545	0.286	0.063	0.009	0.033	0.086
R ²	0.086	0.066	0.022	0.017	0.029	0.036
N	21,106	21,106	21,065	25,678	25,265	19,173
<i>Panel B. Average effect of user fee removal using de Chaisemartin and D'Haultfœuille [2021] estimator</i>						
Affected by the policy	-0.094** (0.042)	-0.028 (0.034)	0.009 (0.033)	-0.002 (0.006)	0.019 (0.020)	0.052 (0.040)
Mean before policy	0.545	0.286	0.063	0.009	0.033	0.086
N	21,106	21,106	21,065	25,678	25,265	19,173
<i>Panel C. Effect in rural districts</i>						
Affected from 2006	-0.055** (0.022)	-0.052*** (0.015)	0.001 (0.013)	0.004 (0.003)	0.004 (0.005)	-0.005 (0.009)
Mean before policy	0.544	0.283	0.063	0.009	0.033	0.087
R ²	0.083	0.063	0.022	0.019	0.030	0.037
N	18,206	18,206	18,159	22,148	21,785	16,486
<i>Panel D. Effect in rural parts of urban districts</i>						
Affected from 2007	-0.073*** (0.022)	-0.070*** (0.020)	-0.008 (0.013)	0.002 (0.003)	0.005 (0.007)	0.000 (0.012)
Mean before policy	0.548	0.294	0.063	0.009	0.033	0.081
R ²	0.088	0.065	0.020	0.018	0.026	0.032
N	7,708	7,708	7,682	9,479	9,344	7,163

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a child. The sample is restricted to children alive at survey time in columns 1 to 3 (anthropometric indicators). The table reports the average (Panels A and B) and phase-specific effect (Panels C and D) of user fee removal. *Stunted* (respectively *Severely stunted*) is a dummy which equals one if the height-for-age ratio is at least two (respectively three) standard deviations below WHO *z*-score, zero otherwise. *Wasted* is a dummy equals to one if the weight-for-height ratio is at least two standard deviations below WHO *z*-score, zero otherwise. In columns 4, 5 and 6, the dependent variable is respectively a dummy which equals one if the child died at birth, within her first 28 days of life and before reach the age of one, zero otherwise. Each coefficient is from a different regression. All regressions control for area and time fixed effects, a dummy for multiple births and child's sex. Columns 1 to 3 also control for child's age dummies, and columns 4 to 6 for mother's year of birth. Time fixed effects correspond to survey years in columns 1 to 3, and to years of childbirth in columns 4 to 6. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.

* $p < .10$; ** $p < .05$; *** $p < .01$

Figure 5. Event study estimates of the effect of user fee removal on child health



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification. Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include a dummy for multiple pregnancy and child's sex. Regressions for anthropometric outcomes also control for child's age dummies, and regressions for mortality outcomes for mother's year of birth. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are dummies equal to one if child is (a) stunted (height for age z -score < -2), (b) severely stunted (height for age z -score < -3), and (c) wasted (weight for height z -score < -2), or died (d) at birth, (e) within her first 28 days of life, and (f) before reaching the age of one year, zero otherwise. Note that for anthropometric outcomes, points estimates for 2013 and 2014 should be interpreted as the effect of a difference in length of exposure to free health care, since the policy was extended throughout the country from 2012. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. Hence, it is not possible to assess the effect on infant mortality for children born in 2011.

Table 4. The effect of the length of exposure to user fee removal on child nutritional status

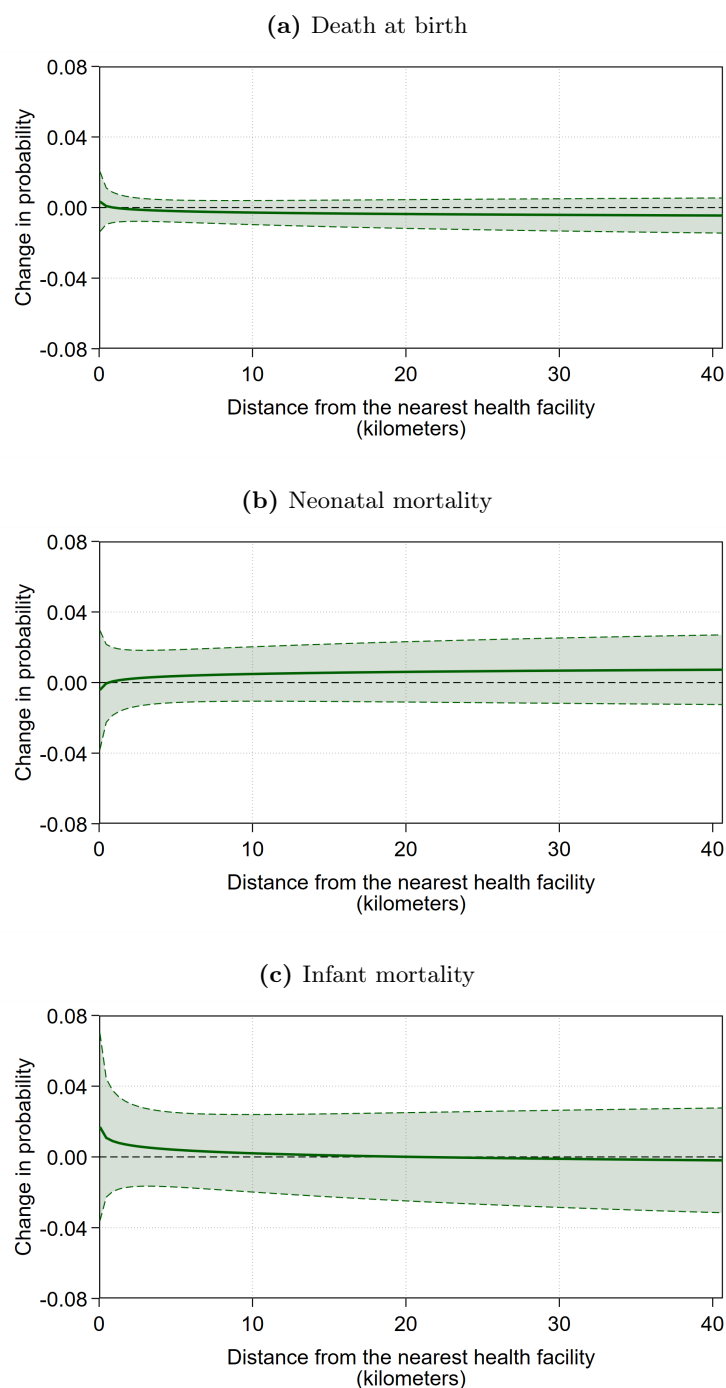
	(1)	(2)	(3)	(4)	(5)	(6)
	— Stunted —		— Severely stunted —		— Wasted —	
Linear duration of exposure (in months)	−0.001*** (0.000)		−0.001*** (0.000)		0.000 (0.000)	
Duration of exposure (in months):						
]0, 12]		−0.011 (0.030)		−0.018 (0.024)		0.011 (0.013)
]12, 24]		−0.036 (0.024)		−0.050*** (0.015)		−0.002 (0.011)
]24, 36]		−0.033 (0.028)		−0.050** (0.021)		−0.008 (0.016)
]36, 48]		−0.080*** (0.026)		−0.066*** (0.019)		−0.001 (0.017)
]48, 59]		−0.070*** (0.025)		−0.057*** (0.017)		−0.001 (0.015)
Mean before policy	0.545	0.545	0.286	0.286	0.063	0.063
R ²	0.086	0.086	0.066	0.066	0.022	0.022
N	21,106	21,106	21,106	21,106	21,065	21,065

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a child. The sample is restricted to children alive at survey time. The table reports the average effect of the length of exposure to the user fee removal on anthropometric indicators. *Stunted* (respectively *Severely stunted*) is a dummy which equals one if the height-for-age ratio is at least two (respectively three) standard deviations below WHO *z*-score, zero otherwise. *Wasted* is a dummy equals to one if the weight-for-height ratio is at least two standard deviations below WHO *z*-score, zero otherwise. All regressions control for area and survey year fixed effects, a dummy for multiple births, as well as child's sex and age dummies.

* $p < .10$; ** $p < .05$; *** $p < .01$

Figure 6. Marginal effect of exposure to the policy on child mortality outcomes depending on distance from the nearest health facility



Source: Author's calculations from DHS 2007 and 2013.

Notes: The figures plot the marginal effect of the policy change depending on the distance from the nearest health facility. Distance corresponds to the straight-line distance between each DHS cluster from the 2007 and 2013 survey waves and the nearest publicly-supported health facility from the 2005 Health Facility Census. Each figure is from a separate estimation where the distance is log-transformed and interacted with exposure to the policy. Control variables include area and year of childbirth fixed effects, as well as mother's year of birth, a dummy for multiple births and child's sex. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. It is not possible to investigate such heterogeneous effects for anthropometric outcomes since clusters from the pre-policy period (surveyed in 1996 and 2001) were not georeferenced, so that all surviving children georeferenced were measured after the policy change. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level.

Next, I examine whether the absence of an effect on child mortality outcomes masks any heterogeneity with respect to the presence of a publicly-supported facility providing emergency obstetric care and child health services locally. Point estimates are reported in Table 5. I find that the policy has led to a decline in newborn mortality risk at birth within affected areas for households living close to a qualified health facility relative to both those that are not (-0.011, significant at the 5% level) and those located in an unaffected area (-0.012, significant at the 5% level). Conclusions remain the same when using a restricted sample around the census date. It suggests that combined with an improved physical access to essential health services for maternal and child care, such as management of obstetric emergencies and resuscitation of newborns, removing user fees can be an effective way to reduce child mortality risk. This result echoes the one from [Bagnoli \[2019\]](#) in Ghana, who observes a positive effect of health insurance only for children living in regions with a high quality of care. This is also consistent with [Godlonton and Okeke \[2016\]](#) who find that the increase in institutional births following a ban on informal health providers in Malawi was accompanied by a reduction in newborn mortality only for households close to a high-quality health facility.

5.3. Selection issues and fertility behaviors

The null effect on child mortality may be explained either by a real absence of effect,²⁶ or by selection effects and fertility decisions induced by the policy change.

5.3.1. Selection into Pregnancy and Composition Effects

One potential threat to identification is that demographic characteristics of mothers may have changed in a different way in affected and unaffected areas after the removal. In case of selection into pregnancy, specific women may react to the policy by having more babies. A related concern is that we can only observe childbirths and children from mothers who survived until survey time. The policy change may have helped high-pregnancy related risks women survive by reducing their risk of maternal death. In that case, affected and unaffected areas may have diverged in post-treatment periods not only in terms of policy implementation but also in terms of characteristics of women giving birth. If such women also tend to have babies with poor health outcomes, my results may underestimate the gains in terms of child health.

²⁶ Due, for example, to an insufficient or a drop in quality of public health services, or because mortality is an indicator too extreme to capture the health improvements brought about by free health care.

Table 5. The effect of user fee removal on child mortality depending on the local availability of a qualified publicly-supported health facility

	(1)	(2)	(3)
	Death at birth	Neonatal mortality	Infant mortality
<i>Panel A. Average effect of user fee removal - Whole sample</i>			
Affected by the policy	-0.001 (0.003)	0.006 (0.008)	0.007 (0.011)
Qualified health facility within 5 km	0.008 (0.006)	0.010 (0.008)	0.008 (0.012)
Affected by the policy × Qualified health facility within 5 km	-0.011** (0.005)	-0.020** (0.009)	-0.021 (0.016)
Mean before policy	0.011	0.030	0.062
R ²	0.022	0.031	0.033
N	14,267	13,969	10,350
<i>Panel B. Average effect of user fee removal - Sample: ± 4 years around 2005 facility census</i>			
Affected by the policy	0.001 (0.004)	0.010 (0.009)	0.014 (0.012)
Qualified health facility within 5 km	0.009 (0.007)	0.011 (0.009)	0.006 (0.013)
Affected by the policy × Qualified health facility within 5 km	-0.011* (0.007)	-0.027** (0.012)	-0.022 (0.014)
Mean before policy	0.011	0.030	0.062
R ²	0.025	0.035	0.035
N	9,019	8,944	7,716
<i>Panel C. Average effect of user fee removal - Sample: ± 3 years around 2005 facility census</i>			
Affected by the policy	0.002 (0.005)	0.006 (0.011)	0.024 (0.019)
Qualified health facility within 5 km	0.009 (0.008)	0.003 (0.011)	0.003 (0.015)
Affected by the policy × Qualified health facility within 5 km	-0.014* (0.008)	-0.034* (0.018)	-0.044 (0.037)
Mean before policy	0.011	0.030	0.062
R ²	0.029	0.040	0.037
N	6,415	6,340	5,112

Source: Author's calculations from DHS 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth. The table reports the average effect of user fee removal on child mortality outcomes estimated on the whole sample (Panel A), and alternatively for children born ± 4 years (Panel B) or ± 3 years around the 2005 Health Facility Census (Panel C). All regressions control for area and time fixed effects, as well as mother's year of birth, a dummy for multiple births, child's sex and the log of the straight-line distance between each DHS cluster from the 2007 and 2013 survey waves and the nearest publicly-supported health facility. A health facility is considered *qualified* if it provides a set of essential emergency obstetric and child health services, based on the 2005 Health Facility Census. See footnote 15 for a full description of this indicator. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. It is not possible to investigate such heterogeneous effects for anthropometric outcomes since clusters from the pre-policy period (surveyed in 1996 and 2001) were not georeferenced, so that all surviving children georeferenced were measured after the policy change.

* $p < .10$; ** $p < .05$; *** $p < .01$

In Appendix Table G.1, Panel A, I test the absence of compositional changes in affected areas relative to unaffected ones by estimating difference-in-differences regressions with maternal characteristics as dependent variables. Results suggest no composition effect in women giving birth, which strengthens the causal interpretation of my results. Then, I replicate this analysis separately for births in a publicly-supported facility (Panel B) and home births (Panel C) to check if the average characteristics of women giving birth in each type of delivery place changed differently in affected and unaffected areas after the removal. The average education level of mothers giving birth in a publicly-supported health facility decreased in affected areas relative to unaffected areas, and women who persist in giving birth at home despite the policy change become relatively older in affected areas than in unaffected ones.

5.3.2. Selection into Medical Delivery

The policy may have failed to reach households with the higher maternal and child mortality risks, for which returns to formal health services are potentially high. For example, there might be a selection effect into medical delivery within affected areas in case of imperfect information concerning the policy. In particular, more educated women may have a better access to information and a higher capacity to ensure the removal of user fees. If such women also have *ex ante* relatively low-mortality risk babies, the probability of delivering in a health facility may increase without any effect on child mortality.

I explore this possibility in two ways. In Panel A of Appendix Table G.2, I interact exposure to the policy with mother's education. I find that more educated mothers did not respond more to the policy than others. Then, I interact exposure to the policy with an indicator for whether mothers have already experienced an infant death before childbirth and can thus be considered at risk.²⁷ Risky mothers benefited from the policy as much as the non-risky ones, which suggests no advantageous selection within affected areas (Appendix Table G.2, Panel B).

In addition, I find no heterogeneous effect according to household's material wealth except for child nutritional status, for which the policy has essentially benefited the poorest (Appendix Figure G.3). The same conclusion applies when focusing on rural districts only. However, within rural areas of urban districts, the removal of user fees has essentially benefited the poorest, including in terms of access to delivery services.

²⁷ In my sample, 24% of non-first born children have at least one elder sibling who died before reaching the age of one.

5.3.3. *Selection into Live Birth*

The policy might have led to selection into live birth if improvement in delivery conditions helped fragile babies survive childbirth. If these babies would not have survived in absence of the policy, then the probability of observing weaker, high-child mortality risk babies increases in affected areas relative to unaffected ones. In such a case, my sample of exposed children would be negatively selected, potentially leading to underestimate the gains in child health.

I test this assumption in two ways. First, using information from the reproductive calendar collected in the 2007 and 2013 DHS survey waves, I create a new database where each observation is now a pregnancy occurring during the last five years preceding the survey, whatever its final outcome, and not necessarily a live birth. I find no effect of the policy on the risk of stillbirth (Appendix Table G.4, column 1). Second, I check this assumption by looking at the gender composition of live births after the policy implementation. Male fetuses are commonly known to be biologically weaker and more susceptible to disease and premature death than female ones due to sex differences in genetic makeup. This is particularly true in Sub-Saharan Africa, even when controlling for the preconception environment [Pongou, 2013]. A recent meta-analysis finds a risk of stillbirth 10 percent higher for male fetuses than for females, a pattern consistent across countries of different income groups [Mondal et al., 2014]. Hence, if the policy has helped fragile babies survive, one should observe an increase in the proportion of male births in affected areas relative to unaffected ones. However, I find no evidence of such an effect (Appendix Table G.4, column 3).

5.3.4. *Fertility*

Couples may have changed their fertility decisions in response to the removal. By lowering the direct cost of having a child, the policy change may have induced parents to have more children with shorter birth intervals in a way that increases child mortality risk [Molitoris et al., 2019]. Such fertility decisions are likely to be endogenous. In particular, it may be influenced by unobserved characteristics at the household level, including parents' preferences, and motivated by a replacement effect in case of child death [Bhalotra and van Soest, 2008; Hossain et al., 2007].

To take it into account, I restrict my sample to first-born children and conclusions remain unchanged (Appendix Table G.5).²⁸ I then explore the effect on birth spacing and find

²⁸ Point estimates are even higher than for the full sample, which is not surprising since parents may be

that the policy has not resulted in riskier birth intervals (columns 1 and 2, Appendix Table G.7).²⁹ As an alternative test for a fertility effect, I transform my cross-sectional individual data into a panel at the area \times birth date \times survey year level and find no aggregate effect of the policy change on the number of reported births per 1,000 surveyed mothers (column 3, Appendix Table G.7).

5.4. Robustness checks

Contamination effects.— One concern is that some individuals living in control areas might have benefited from the policy if they seek care in a treated one. In such a case, point estimates will likely represent lower bounds of the true effects. Using data from the 1998 Living Conditions Monitoring Survey, Lépine et al. [2018] have identified three urban districts (Kasama, Mazabuka, and Mongu) in which a significant part of the population (12% to 25%) declared seeking care in a rural district. People living in such districts might have benefited from the policy while they should not. Conclusions remain unchanged when these three districts are excluded from the analysis (Appendix Figure H.5). In the same way, those living in control enumeration areas spatially close to a treated one could have benefited from it. I show that conclusions are robust to the exclusion of control enumeration areas located less than five kilometers away from an affected one (Appendix Figure H.6).

Migration.— Due to data limitation, the effective area of birth of each child is unknown. Of the 25,678 births occurring during the last five years preceding the survey, 85% occurred in the same locality as the place of residence.³⁰ It is not possible to track where the remaining 15% of births occurred: in another district or in another place within the same district. This figure overestimates the share of births for which treatment status might be incorrectly assigned, as place of birth and place of residence can be different but in the same area or in different areas but with the same treatment status. Overall, it suggests that migration should not drive my results. This is exactly what I find. I show that dropping mothers who have migrated since childbirth leaves the results unchanged (Appendix Figure H.5).

Other robustness checks.— Further robustness checks are performed and extensively dis-

more concerned with their first birth and cannot rely on their own past experience as parents when choosing where to give birth, a result consistent with Sialubanje et al. [2015]. Point estimates by rank of birth plotted in Appendix Figure G.6 suggest that the positive effects of user fee removal fade away with birth rank. However, this gradient is less pronounced once mother's education level and wealth are taken into account. This is consistent with the fact that children with a high birth rank are reported by mothers on average less educated than the other ones, and are observed in the poorest households.

²⁹ The World Health Organization recommends a birth interval of at least 24 months after a live birth to prevent maternal, perinatal, and infant disorders [WHO, 2007].

³⁰ For sake of comparison, I find a very similar figure with the national census data from 1990, 2000 and 2010 (86.9% of children were born in their current district of residence).

cussed in Appendix H. Most importantly, the results are qualitatively unchanged if I include additional control variables and district-specific linear time trends. Point estimates also remain very similar when I use an alternative definition of exposure to the policy based on geographic coordinates. I also test the robustness of my results to combining difference-in-differences with several matching approaches, which leaves the conclusions unchanged (Appendix Table H.7).

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6. Discussion and Concluding Remarks

This chapter offers new evidence on the extent to which the removal of user fees affects demand for curative health services and child health in a resource-limited setting. Exploiting variation in the timing of the abolition of user fees across districts of Zambia, this chapter points to large and sustained positive effects of the policy on maternal health care utilization and delivery conditions.

However, these effects did not materialize immediately after the removal. This might be explained by several factors specific to the reform [Carasso et al., 2010]. First, it was announced suddenly by the incumbent President during a pre-election period. While this suddenness is an advantage in identifying the policy's impacts, it left little time and capacity to precisely design the reform, to anticipate its effects, and finally to provide adequate resources to facilities. Second, and related to this, the implementation rules of the policy were unclear and changed at the last time, causing confusion at the district and facility levels during the first months following the official removal date. In particular, it was unclear where user fees should be abolished. Third, health facilities were initially seriously under-compensated for the loss of user fee revenues, leading to the discontinuation of several health services. The replacement grant was initially based on the projected loss of revenues based on fees collected prior to the removal but was seriously underestimated. Major delays in disbursement of compensation funds were also observed, with facilities receiving it 8 to 12 months after the removal. Last, the important shortage of essential drugs and medical supplies, as well as the inefficient allocation of funds in 2006 in favor of hospitals, which has resulted in a 40% drop in districts' primary health services funding [Carasso et al., 2010], have certainly limited the effectiveness of the reform in its early stages. Hadley [2011] suggests that even when drugs were available, they were not used efficiently. While necessary, compensation for the loss of user fee revenues and the increased

workload should not mask earlier, deeper problems such as health workers shortage and lack of equipment.

This chapter also finds that the removal of user fees drastically reduced child chronic malnutrition but only for those exposed at least 12 months to free health care. There is no discernible impact on child mortality, a result which is not driven by selective fertility nor by a selection effect into live births. A potential explanation for these limited effects on child health is a drop in the quality of health services after the reform. Due to data limitations, it cannot be tested directly, but several pieces of evidence suggest a drop in effective quality after user fees were removed while perceived quality remained stable or even improved [Masiye et al., 2010]. Overall, these results are in line with a broad set of empirical studies looking at the effect of free health care or health insurance, which find an increase in health care utilization but no or limited effect on health, both in low- (Ansah et al., 2009; Powell-Jackson et al., 2014; and Erlangga et al., 2019 for a review) and high-income countries [Card et al., 2004; Chen et al., 2007; Finkelstein and McKnight, 2008], even if evidence is more mixed for the latter.

This chapter suffers from several caveats I wish to stress here. First, some individuals living in unaffected areas may have sought care in an affected one. While I cannot completely rule out this possibility, I show that the results remain the same when districts with potentially significant contamination effects are excluded and when control areas close to treated ones are removed. Moreover, in some facilities, informal payments may have been introduced or increased following the removal of user fees. For these reasons, results presented in this chapter must be interpreted as lower bound estimates of the true effects of the policy in an ideal framework with perfect compliance and enforcement of the policy change. Second, due to data limitations, it is not possible to study the effect of the policy on maternal mortality. However, there is suggestive evidence that there was no compositional change in mothers reporting births after the policy change, and there is no distinguishable trend break in the national maternal mortality ratio after the removal (Appendix Figure F.1). Finally, the absence of effect on child mortality does not imply that free health care is ineffective in improving child health, and so for several reasons: mortality is certainly an extreme indicator of child health conditions and too blunt a measure to reflect health improvements associated with free health care; I find encouraging results for chronic malnutrition, one of the leading causes of child morbidity and mortality; and the removal of user fees may have impacted other important health issues not explored due to data limitations such as medical treatment of malaria.

This chapter contributes to the ongoing debate on the relative effectiveness of demand- and supply-side interventions in improving population health in low-income countries. It suggests that removing user fees is a good way to stimulate individuals' demand for curative health services but is clearly not sufficient *per se* to generate huge gains in individuals' health. If access has improved, returns to formal health services are limited. Health care quality appears as a crucial piece of the puzzle since child mortality risk only decreased in the vicinity of qualified health centers. These conclusions have important policy implications for population health. They call for massive efforts to improve the capacity of such health care systems to provide financially accessible, high-quality health services to all.

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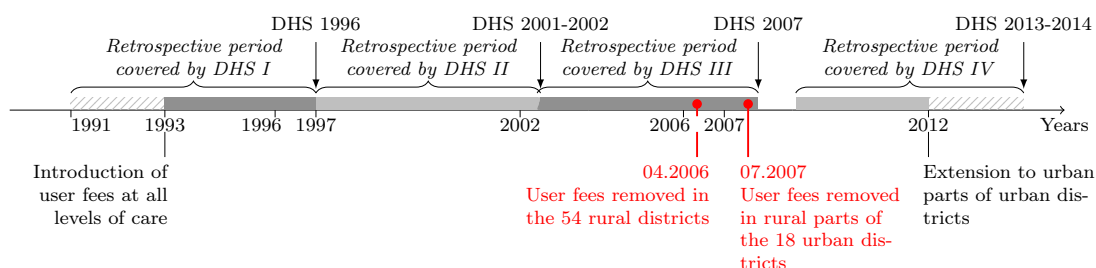
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APPENDICES TO CHAPTER 1

Appendix A. Timeline of the policy and survey waves

Figure A.1. Timeline of the policy



Appendix B. Demographic and Health Surveys

B.1. Sampling frame

The Demographic and Health Surveys sample design is based on a two-stage sampling procedure. First, enumeration areas, known as clusters, are selected from a sampling frame corresponding to a list of enumeration areas from the most recent national population census. The sampling frame is stratified by province and urban/rural areas within each province, and enumeration areas are randomly selected using a probability proportional to size method. Then an exhaustive listing of all the households present in each cluster is carried out. Second, 20 to 30 households per cluster are randomly selected with equal probability. Within sampled households, all women aged 15-49 who were either permanent residents of the household or visitors present on the night before the survey were eligible for survey interview. Sample design and questionnaires are standardized across survey waves, allowing for pooled cross-section analysis.

B.2. Distribution of birth year

Table B.1. Distribution of live births by year of birth and treatment status

	Rural districts	Rural areas of urban districts	Urban areas of urban districts	N
1993	747	98	361	1,206
1994	794	109	368	1,271
1995	815	116	347	1,278
1996	628	95	260	983
1997	627	165	230	1,022
1998	748	229	279	1,256
1999	815	255	274	1,344
2000	884	280	294	1,458
2001	858	230	270	1,358
2002	399	136	178	713
2003	772	186	266	1,224
2004	796	159	283	1,238
2005	739	209	320	1,268
2006	824	210	304	1,338
2007	432	82	141	655
2008	116	24	74	214
2009	1,712	310	582	2,604
2010	1,754	312	568	2,634
2011	1,739	325	550	2,614
N	16,199	3,530	5,949	25,678

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013. *Notes:* Based on my sample, DHS 1996 was collected from 1996-07-18 to 1997-01-06, DHS 2001-2002 from 2001-11-08 to 2002-06-03, DHS 2007 from 2007-04-03 to 2007-10-08, and DHS 2013 from 2013-08-20 to 2014-04-16. Few households are surveyed in the last days of each wave, and by construction there is no data for the last three months of 2007 nor for the first eight months of 2008, which explains the lower sample sizes for these two years. A higher number of households was sampled in 2013 than in the two previous waves. See Appendix A.1 for a timeline of the policy change and periods covered by the different survey waves.

B.3. Sample sizes and anthropometric measurement

Table B.2. Sample sizes

	1996	2001	2007	2013	Whole sample	
<i>Panel A. Child mortality analysis</i>						
Sample size	4,734	6,677	6,201	8,066	25,678	
% of children alive at survey time	0.868	0.872	0.913	0.934	0.901	
<i>Panel B. Childbirth conditions</i>						
Sample size - Place of delivery	4,724	6,634	6,163	7,964	25,485	
↳ % births with missing information	0.002	0.006	0.006	0.013	0.008	
Sample size - Assistance received	4,727	6,665	6,182	8,006	25,580	
↳ % births with missing information	0.001	0.002	0.003	0.007	0.004	
<i>Panel C. Child anthropometric measurement</i>						
<i>Among survivors</i>	% measured	0.940	0.945	0.927	0.913	0.929
	% not present	0.036	0.016	0.009	0.032	0.023
	% refused	0.009	0.003	0.013	0.004	0.007
	% sick or other	0.015	0.036	0.051	0.051	0.041
Sample size after cleaning of height-for-age z -score [†]	3,813	5,375	5,086	6,832	21,106	
Sample size after cleaning weight-for-height z -score [†]	3,801	5,396	5,068	6,800	21,065	

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The table reports the different sample sizes for child mortality (Panel A), childbirth conditions (Panel B) and child nutritional status (Panel C) analysis.

Based on weight and height measures, I compute anthropometric z -scores.

[†] Among children measured, I exclude from the analysis the ones with biologically implausible z -scores values according to the World Health Organization, ie. height-for-age z -score below -6 or above 6 for stunting, and weight-for-height z -score below -5 or above 5 for wasting [WHO, 2019]. They represent 1.8% and 1.9% of measured children, respectively.

B.4. Scrambling procedure and geographic coordinates

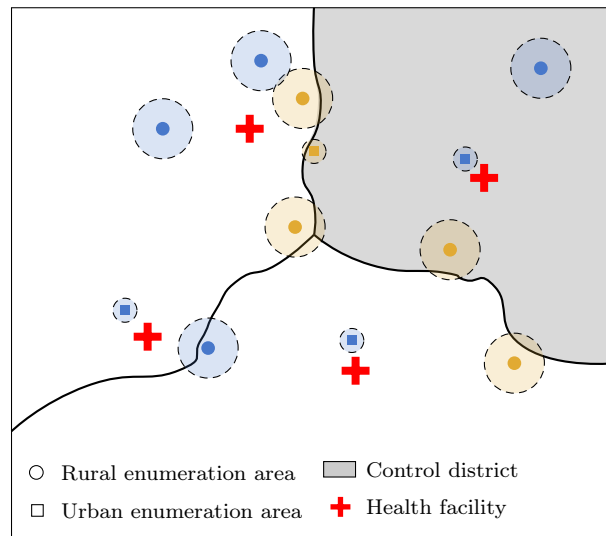
For confidentiality reasons, geographic coordinates have been randomly displaced by the DHS. Urban clusters are randomly displaced within a radius of 2 kilometers around their real location, creating a positional error ranging from a minimum of zero and a maximum of two kilometers. For rural clusters, the maximum displacement increases to five kilometers and up to ten kilometers for a further 1% of them. See [DHS methodology for geographic data](#)³¹ for additional information.

This may create measurement errors and an attenuation bias since households may have been assigned to the wrong district and potentially to the wrong treatment status. This is a problem only for those enumeration areas located near the boundary of a district which

³¹ <https://dhsprogram.com/What-We-Do/GPS-Data-Collection.cfm>

has not the same treatment status as the assigned one. In Figure B.3, I show the case where an urban cluster and four rural ones (in yellow) are potentially assigned to the wrong treatment status as their (unknown) exact location can be either in a treated or a control area.

Figure B.3. Random displacement of enumeration area and treatment status



Notes: Large circles represent a radius of five kilometers around rural clusters and small circles a radius of two kilometers around urban clusters.

B.5. Pre-treatment differences in childbirth conditions and child health by wealth level

Table B.4. Pre-treatment differences in childbirth conditions and child health outcomes between the poorest 50% and the richest 50%

	(1)		(2)		(3)		(4)		(5)		(6)		(7)		(8)		(9)		(10)		(11)		(12)	
	National level		Rural districts		T1		Rural areas of urban districts		T2		Urban areas of urban districts		C		Urban areas of urban districts		T2		Urban areas of urban districts		C		Urban areas of urban districts	
	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%	Poorest 50%	Richest 50%
Panel A. Childbirth conditions																								
Assisted by a health professional	0.24	0.63	0.000	0.23	0.42	0.000	0.22	0.36	0.000	0.22	0.36	0.000	0.69	0.91	0.000	0.69	0.91	0.000	0.69	0.91	0.000	0.69	0.91	0.000
Assisted by a doctor	0.01	0.05	0.000	0.01	0.02	0.000	0.00	0.03	0.000	0.00	0.03	0.000	0.05	0.11	0.000	0.05	0.11	0.000	0.05	0.11	0.000	0.05	0.11	0.000
Assisted by a nurse or midwife	0.23	0.60	0.000	0.22	0.40	0.000	0.21	0.34	0.000	0.21	0.34	0.000	0.67	0.87	0.000	0.67	0.87	0.000	0.67	0.87	0.000	0.67	0.87	0.000
Institutional delivery	0.25	0.63	0.000	0.24	0.43	0.000	0.23	0.36	0.000	0.23	0.36	0.000	0.69	0.91	0.000	0.69	0.91	0.000	0.69	0.91	0.000	0.69	0.91	0.000
Delivered in a public health facility	0.24	0.58	0.000	0.24	0.42	0.000	0.23	0.36	0.000	0.23	0.36	0.000	0.69	0.73	0.002	0.69	0.73	0.002	0.69	0.73	0.002	0.69	0.73	0.002
Delivered in a private health facility	0.00	0.05	0.000	0.00	0.01	0.000	0.00	0.01	0.095	0.00	0.01	0.095	0.00	0.18	0.000	0.00	0.18	0.000	0.00	0.18	0.000	0.00	0.18	0.000
Panel B. Child mortality																								
Death at birth	0.01	0.01	0.894	0.01	0.01	0.335	0.01	0.01	0.165	0.01	0.01	0.165	0.01	0.01	0.786	0.01	0.01	0.165	0.01	0.01	0.786	0.01	0.01	0.786
Neonatal mortality risk	0.03	0.03	0.942	0.04	0.03	0.084	0.04	0.03	0.337	0.04	0.03	0.337	0.04	0.03	0.360	0.04	0.03	0.337	0.04	0.03	0.360	0.04	0.03	0.360
Infant mortality risk	0.09	0.09	0.957	0.09	0.09	0.849	0.08	0.08	0.725	0.08	0.08	0.725	0.10	0.08	0.020	0.10	0.08	0.725	0.10	0.08	0.020	0.10	0.08	0.020
N	7,995	7,946	15,941	4,959	4,856	9,815	1,295	1,254	2,549	1,295	1,254	2,549	1,925	1,885	3,810	1,925	1,885	3,810	1,925	1,885	3,810	1,925	1,885	3,810
Panel C. Child nutritional status																								
Stunted	0.57	0.46	0.000	0.57	0.52	0.000	0.59	0.51	0.001	0.59	0.51	0.001	0.44	0.36	0.000	0.44	0.36	0.001	0.44	0.36	0.000	0.44	0.36	0.000
Severely stunted	0.31	0.22	0.000	0.31	0.26	0.000	0.32	0.27	0.013	0.32	0.27	0.013	0.19	0.15	0.008	0.19	0.15	0.013	0.19	0.15	0.008	0.19	0.15	0.008
Wasted	0.06	0.06	0.244	0.07	0.06	0.027	0.06	0.07	0.485	0.06	0.07	0.485	0.07	0.06	0.417	0.07	0.06	0.485	0.07	0.06	0.417	0.07	0.06	0.417
N	4,691	4,611	9,302	2,933	2,855	5,788	882	913	1,795	882	913	1,795	1,074	1,137	2,211	1,074	1,137	2,211	1,074	1,137	2,211	1,074	1,137	2,211

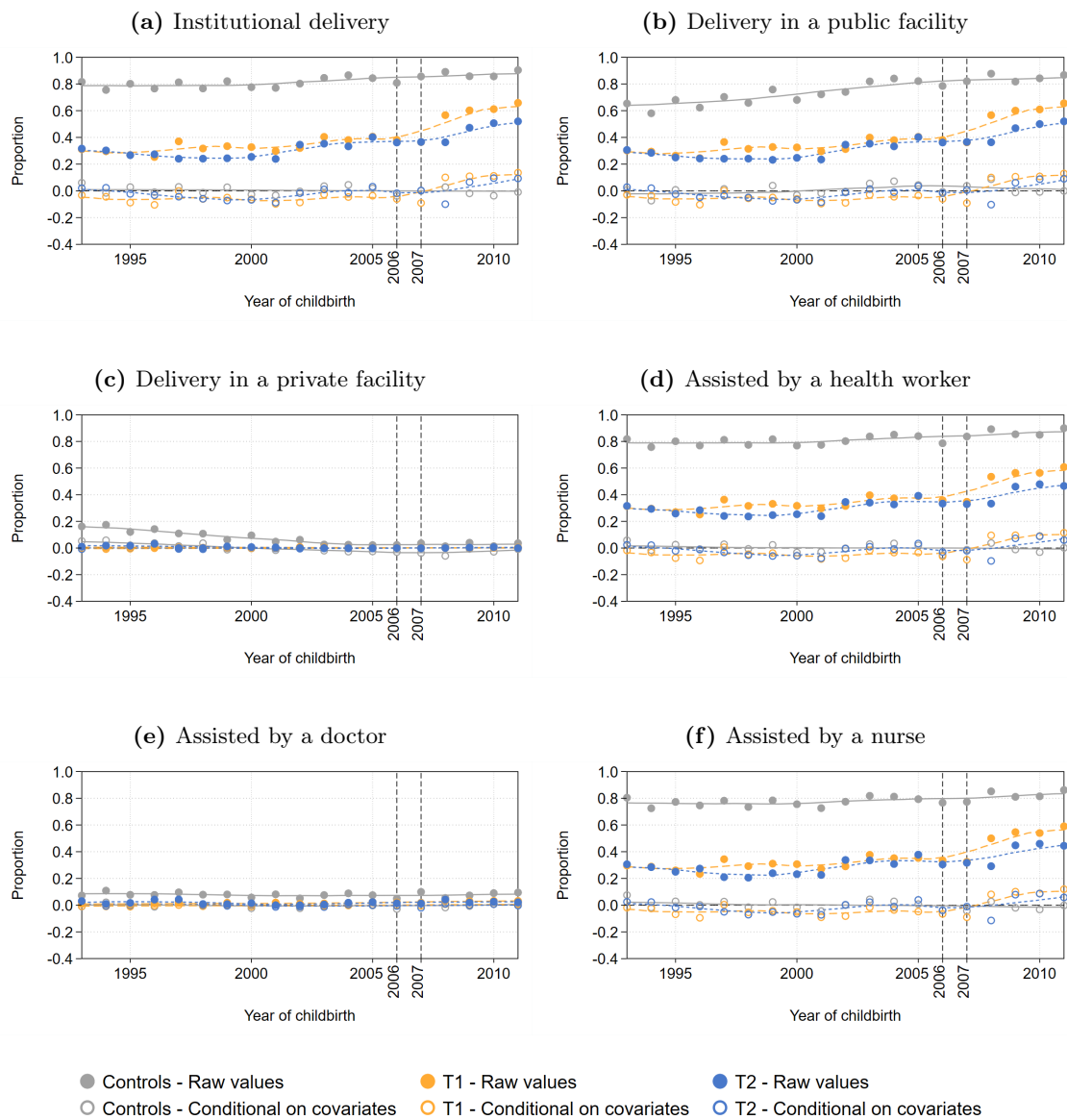
Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Unweighted statistics. The table reports summary statistics for live births occurring before the removal of user fees (Panels A and B) or children measured in a survey wave prior to the removal (Panel D), disaggregated between the poorest 50% and the richest 50%. This analysis is carried out at the national level (columns 1-2), and within each treatment groups (T1, T2 and C). Columns 3, 6, 9 and 12 report *p*-values associated with the comparison between the poor and the rich. In Panel D, the sample is restricted to children alive at survey time.

Appendix C. Parallel trends and event-study analysis

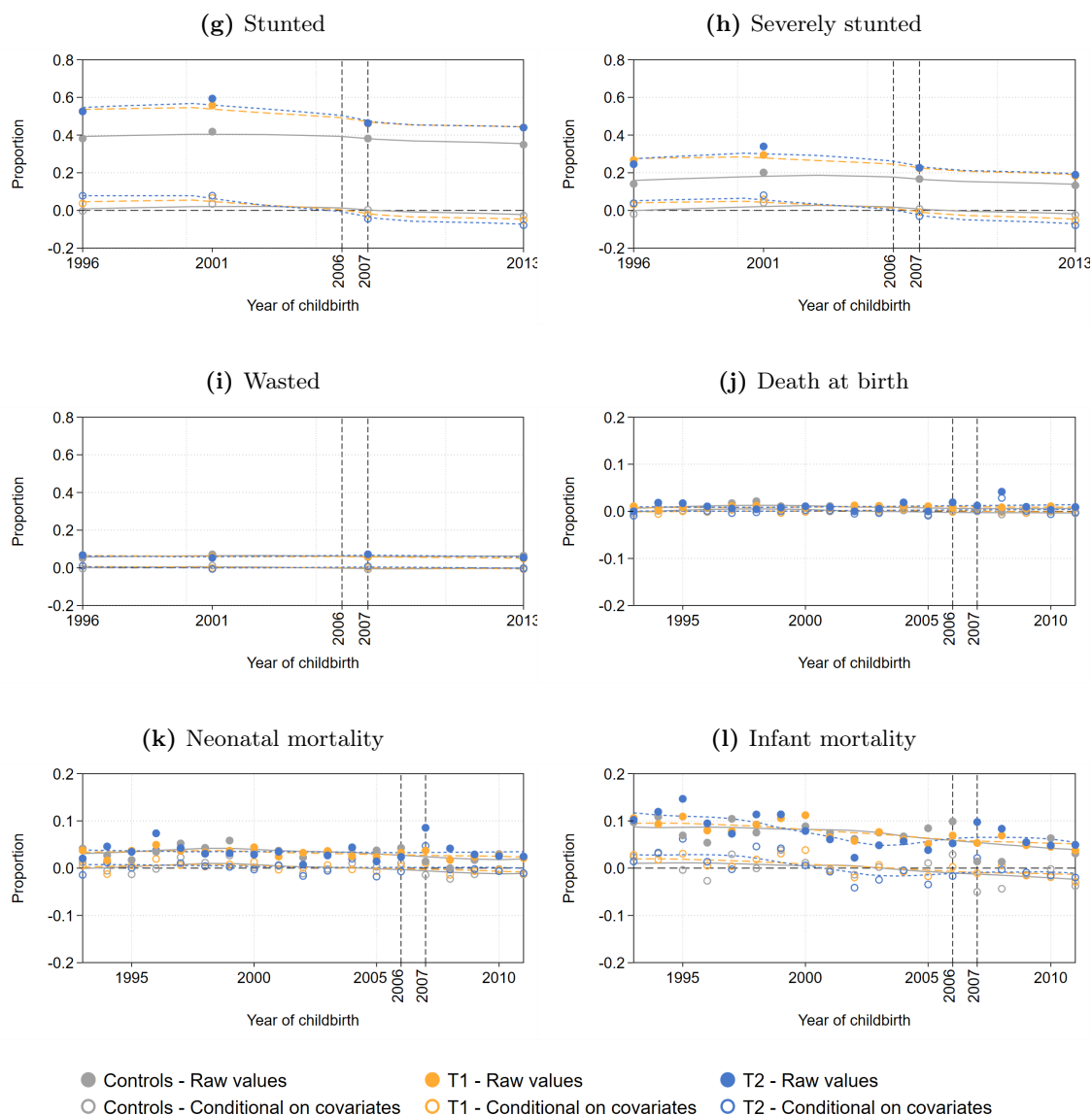
C.1. Trends in outcomes of interest

Figure C.1. Trends in condition of childbirth and child mortality



continued →

Figure C.1 (*continued*). Trends in condition of childbirth and child health outcomes



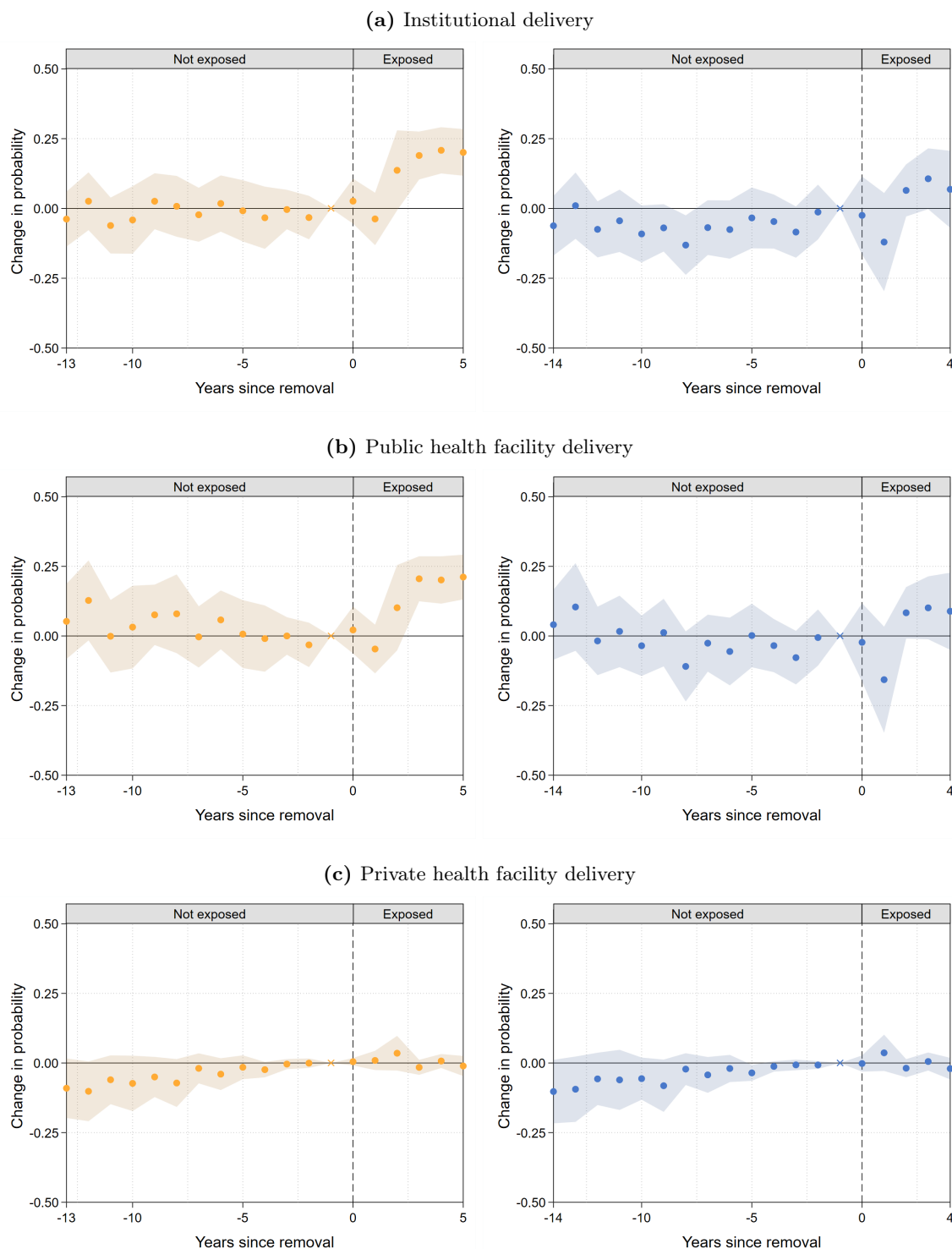
Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figures plot the raw trends of each outcome for affected (T1 for rural districts, T2 for rural areas of urban districts) and unaffected areas (controls). It also reports the residual trends after controlling for all covariates and area fixed effects, as in the main specification. The vertical dashed lines indicate the starting date of the policy in rural districts (April 2006) and in rural areas of urban districts (July 2007). For anthropometric indicators, the vertical dashed line indicates the survey year from which children measured in affected areas were exposed to the policy.

C.2. Event-study estimates

Figures C.2 to C.5 plot point estimates from the event study specification and 95 percent confidence intervals for the outcomes of interest for rural districts and rural areas of urban districts separately. It provides strong evidence of the absence of differential trends between affected and unaffected areas prior to the removal, which supports the identifying assumption and the causal interpretation of my results.

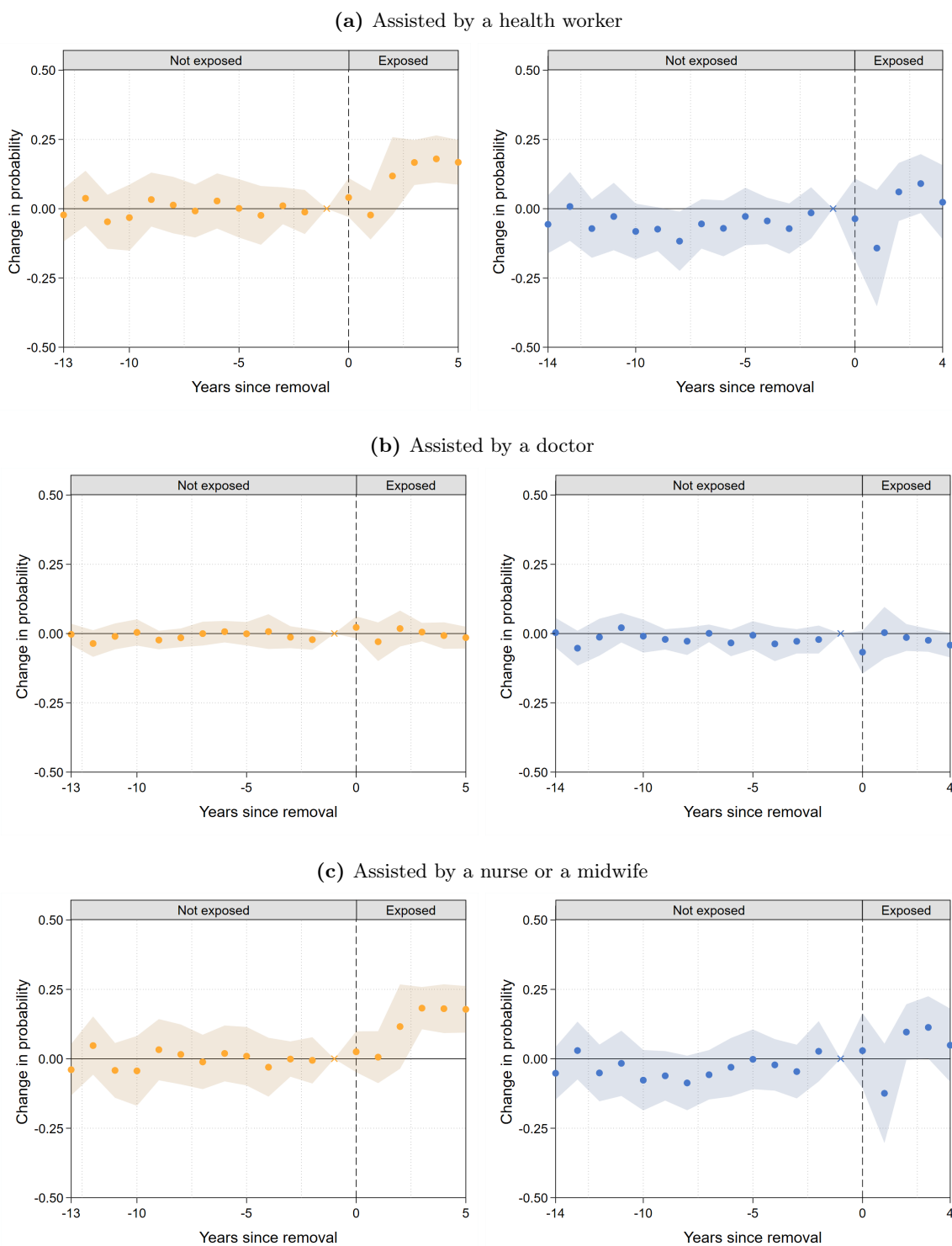
Figure C.2. Event study estimates of the effect of user fee removal on place of delivery



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include mother's year of birth and a dummy for multiple pregnancy. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are a dummy equals to one if mother gave birth (a) in a health facility, (b) in a public or mission health facility, and (c) in a private health facility, zero otherwise.

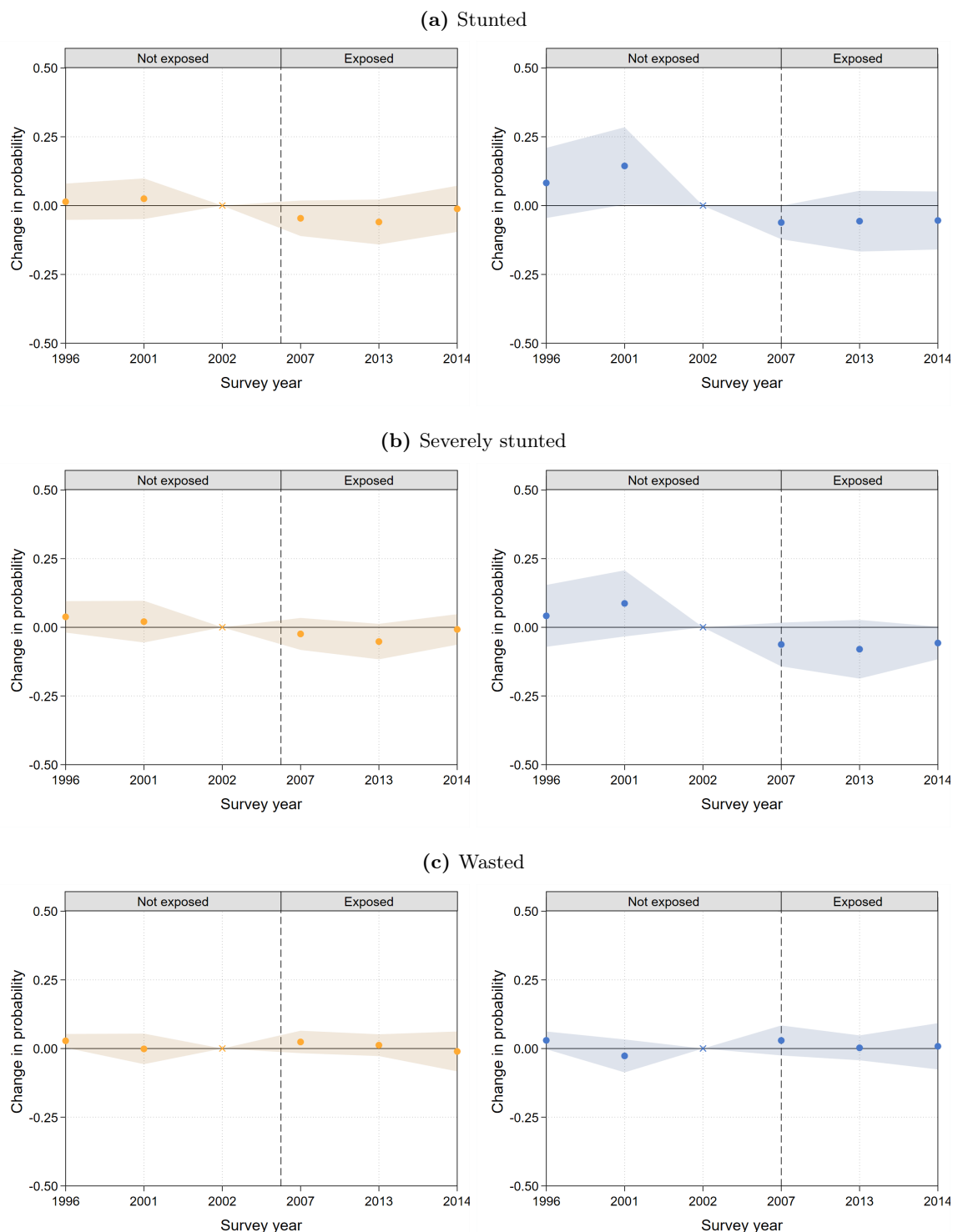
Figure C.3. Event study estimates of the effect of user fee removal on assistance received during childbirth



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include mother's year of birth and a dummy for multiple pregnancy. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are a dummy equals to one if mother gave birth in presence of (a) a health worker, (b) a doctor, and (c) a nurse or a midwife, zero otherwise.

Figure C.4. Event study estimates of the effect of user fee removal on nutritional status

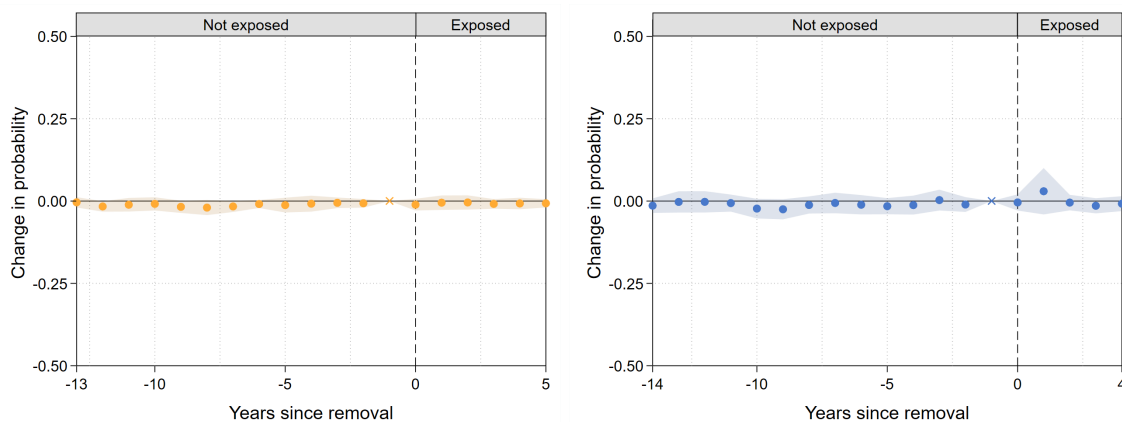


Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

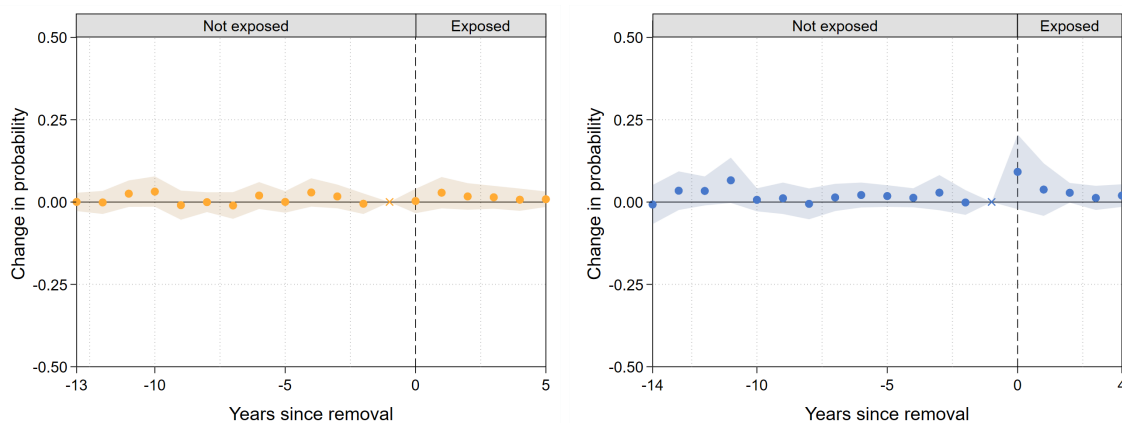
Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). In addition to area and survey year fixed effects, the covariates include a dummy for multiple pregnancy, child's age dummies and child's sex. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy (the survey year 2002). Outcomes of interest are a dummy equals to one if child is (a) stunted (height for age z -score < -2), (b) severely stunted (height for age z -score < -3), and (c) wasted (weight for height z -score < -2), zero otherwise.

Figure C.5. Event study estimates of the effect of user fee removal on child mortality

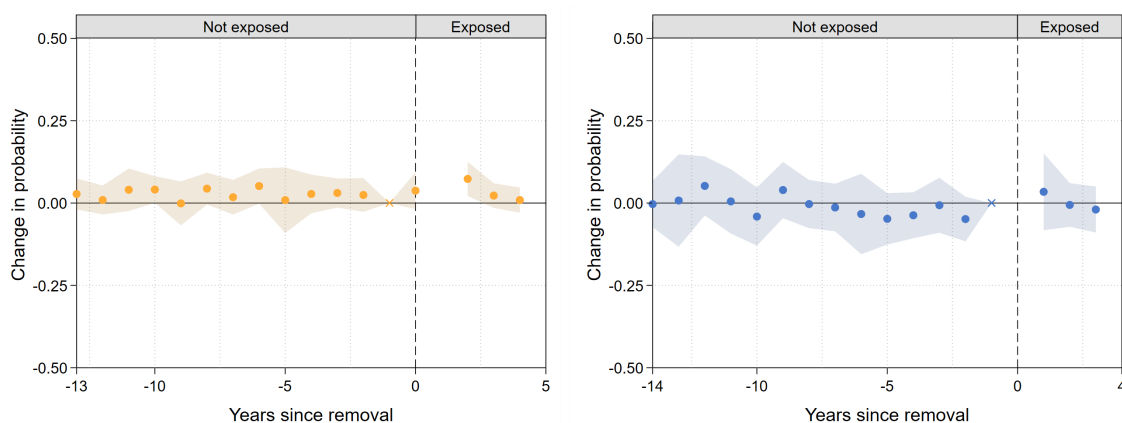
(a) Death at birth



(b) Neonatal mortality



(c) Infant mortality



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

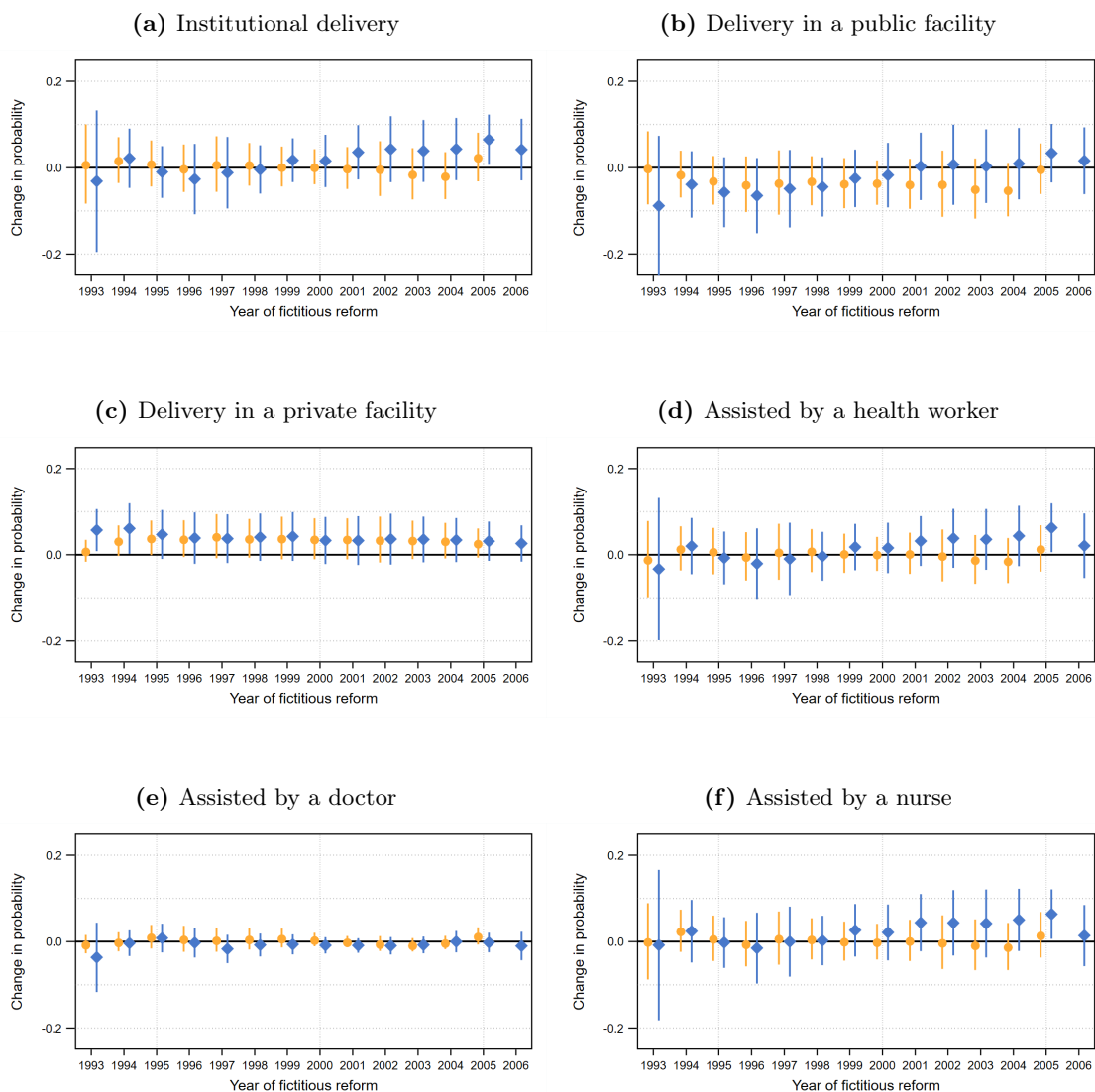
Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include mother's year of birth, a dummy for multiple pregnancy and child's sex. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are a dummy equals to one if child died (a) at birth, (b) within her first 28 days of life, and (c) before reaching the age of one year, zero otherwise. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. Hence, it is not possible to assess the effect on infant mortality for children born in 2007 and 2011.

C.3. Placebo tests

I conduct a broad set of placebo tests to check for the existence of differential trends between affected and unaffected areas before the removal by comparing unexposed children from affected and unaffected areas. Children born after the beginning of the policy are dropped. I create fictitious removal policies for each year prior to the real implementation date. Affected and unaffected areas remain the same. For each fictitious policy, I estimate a difference-in-differences regression. The independent variable of interest is an interaction of a new indicator variable for post-fictitious removal observations and an indicator taking the value of one if user fees were removed in area a , zero otherwise.

One should observe no effect of these fictitious policies on the different outcomes of interest in absence of differences in trends before the reform. Appendix Figure C.6 plots the point estimates and 95 percent confidence intervals from these regressions. Only 9 point estimates out of 255 are marginally significant at the 5% level, which strongly supports the identifying assumption.

Figure C.6. Placebo tests

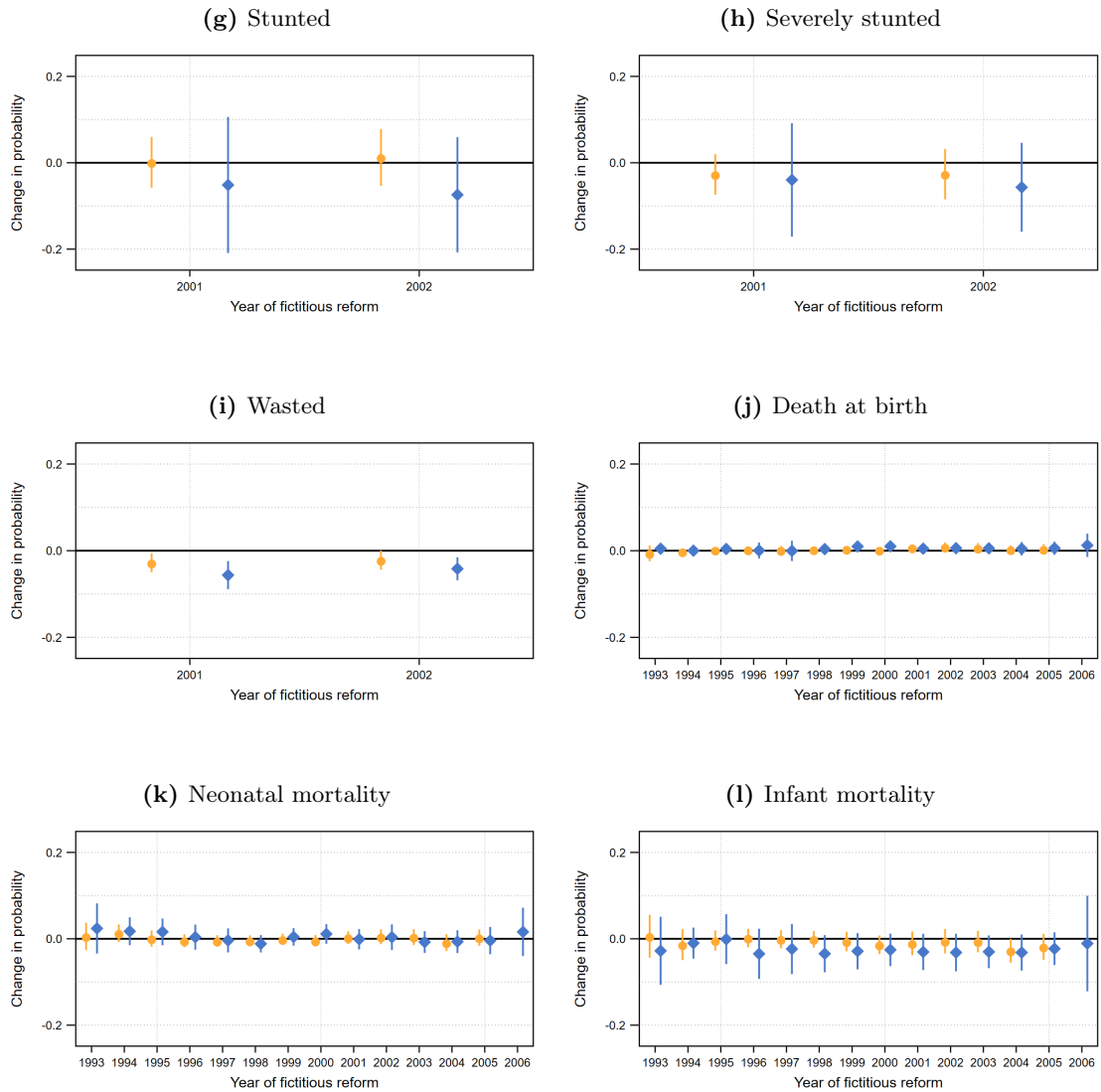


continued →

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Each point corresponds to a separate difference-in-differences estimation for fictitious years of implementation of the policy in rural districts (yellow dots) and rural areas of urban districts (blue diamonds). The sample is restricted to non-exposed children: childbirths and anthropometric measurements occurring after the real removal of user fees are dropped. Control variables include area and time fixed effects, as well as mother's year of birth and a dummy for multiple births. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

Figure C.6 (*continued*). Placebo tests



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Each point corresponds to a separate difference-in-differences estimation for fictitious years of implementation of the policy in rural districts (yellow dots) and rural areas of urban districts (blue diamonds). The sample is restricted to non-exposed children: childbirths and anthropometric measurements occurring after the real removal of user fees are dropped. Control variables include area and time fixed effects, child's sex and a dummy for multiple births. Additional controls include mother's year of birth for mortality outcomes, and child's age dummies for anthropometric outcomes. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

Appendix D. Alternative estimators

Table D.1. Alternative estimators for difference-in-differences with staggered implementation of the policy

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Institutional delivery	Public facility	Private facility	Health worker	Doctor	Nurse or midwife	Stunted	Severely stunted	Wasted	Death at birth	Neonatal mortality	Infant mortality
Panel A. Callaway and Sant'Anna [2020]												
Affected	0.132*** (0.026)	0.141*** (0.026)	-0.009 (0.008)	0.057** (0.026)	-0.019 (0.014)	0.074*** (0.025)	-0.056* (0.032)	-0.048* (0.027)	-0.027* (0.016)	-0.008** (0.004)	-0.007 (0.010)	-0.005 (0.014)
by the policy	[0.081; 0.183]	[0.091; 0.192]	[-0.025; 0.006]	[0.006; 0.108]	[-0.046; 0.007]	[0.024; 0.123]	[-0.118; 0.007]	[-0.101; 0.005]	[-0.059; 0.004]	[-0.017; -0.000]	[-0.027; 0.014]	[-0.033; 0.022]
Panel B. Borusyak et al. [2021]												
Affected	0.156*** (0.021)	0.122*** (0.029)	0.034* (0.020)	0.127*** (0.021)	0.001 (0.010)	0.139*** (0.022)	-0.060*** (0.014)	-0.056*** (0.011)	-0.001 (0.009)	0.002 (0.003)	0.007* (0.004)	0.002 (0.007)
by the policy	[0.114; 0.198]	[0.066; 0.178]	[-0.006; 0.074]	[0.086; 0.168]	[-0.017; 0.020]	[0.095; 0.182]	[-0.087; -0.033]	[-0.077; -0.036]	[-0.019; 0.016]	[-0.003; 0.007]	[-0.001; 0.015]	[-0.011; 0.015]
Panel C. Gardner [2021]												
Affected	0.165*** (0.024)	0.133*** (0.033)	0.031 (0.024)	0.134*** (0.023)	0.002 (0.012)	0.144*** (0.025)	-0.038** (0.018)	-0.039*** (0.013)	-0.009 (0.011)	0.003 (0.003)	0.004 (0.005)	0.002 (0.008)
by the policy	[0.118; 0.211]	[0.069; 0.197]	[-0.016; 0.079]	[0.088; 0.180]	[-0.021; 0.025]	[0.095; 0.193]	[-0.074; -0.003]	[-0.063; -0.014]	[-0.030; 0.012]	[-0.002; 0.008]	[-0.005; 0.013]	[-0.013; 0.017]
N	25,485	25,485	25,485	25,580	25,580	25,580	21,106	21,106	21,065	25,678	25,265	19,173

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.
Notes: Robust standard errors clustered at the area level reported in parentheses. 95% confidence intervals in brackets. The unit of observation is a childbirth (columns 1 to 6) or a child (columns 7 to 12). The table reports the point estimates obtained with different estimators. Each coefficient is from a different regression. All regressions control for area and time fixed effects, and a dummy for multiple births. Columns 1 to 6 also control for mother's year of birth, columns 7 to 9 for child's sex and age, and columns 10 to 12 for mother's year of birth and child's sex. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.
 * $p < .10$; ** $p < .05$; *** $p < .01$

Appendix E. Postnatal check-ups and child vaccination

Table E.1. The effect of user fee removal on postnatal check-up

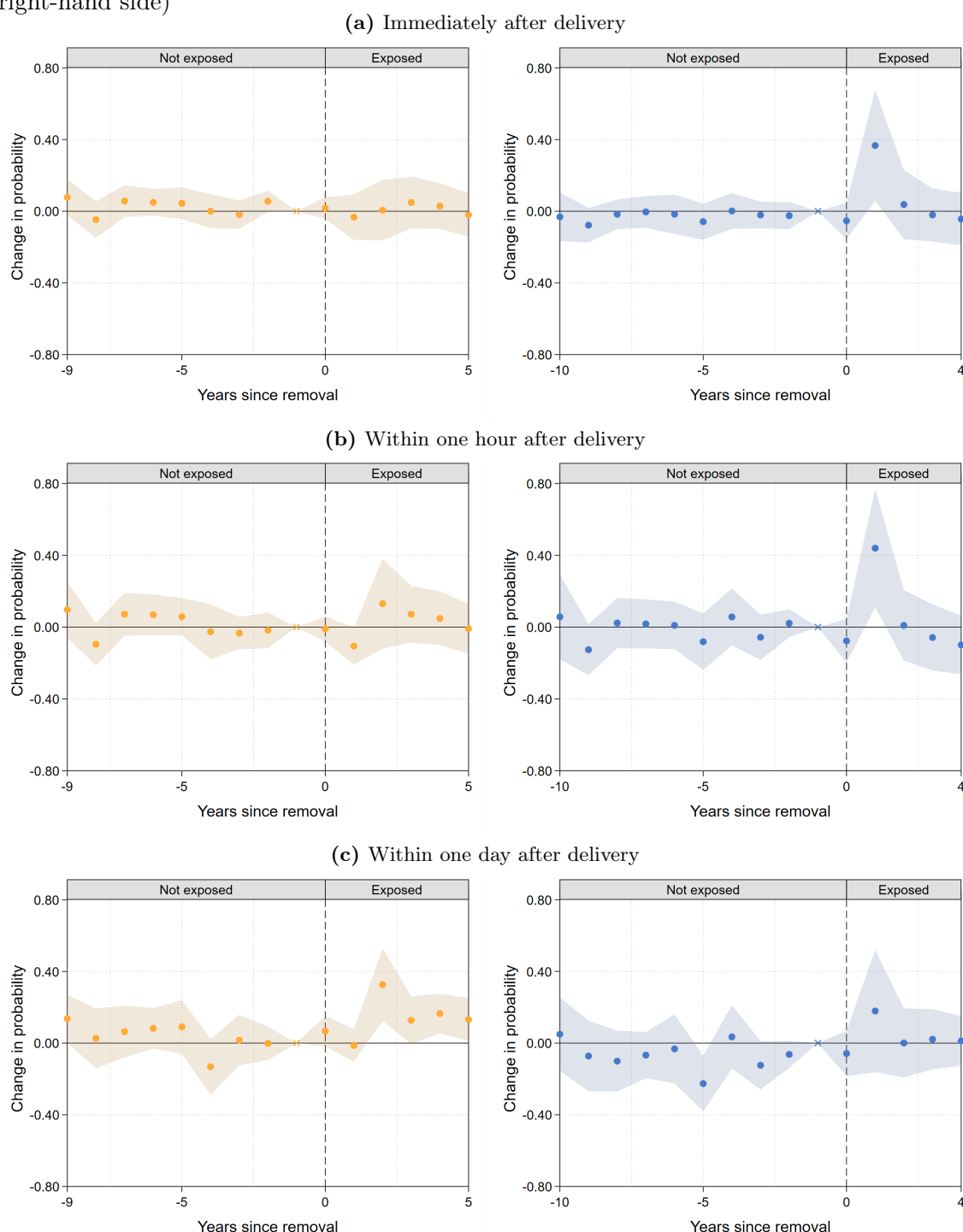
	(1)	(2)	(3)	(4)
	Mother's health was checked			Child
	immediately	within one hour	within one day	up-to-date vaccinations
	after delivery			
<i>Panel A. Average effect of user fee removal</i>				
Affected by the policy	-0.011 (0.028)	-0.013 (0.029)	0.061* (0.035)	-0.009 (0.017)
Mean before policy	0.087	0.137	0.271	0.481
R ²	0.076	0.106	0.180	0.311
N	12,346	12,346	12,346	23,075
<i>Panel B. Average effect of user fee removal using de Chaisemartin and D'Haultfœuille [2021] estimator</i>				
Affected by the policy	0.029 (0.035)	0.009 (0.044)	0.101* (0.059)	0.008 (0.031)
Mean before policy	0.087	0.137	0.271	0.481
N	12,346	12,346	12,346	23,075
<i>Panel C. Effect in rural districts</i>				
Affected from 2006	-0.014 (0.031)	-0.003 (0.032)	0.078* (0.039)	-0.002 (0.018)
Mean before policy	0.098	0.143	0.284	0.485
R ²	0.072	0.106	0.176	0.308
N	10,573	10,573	10,573	19,910
<i>Panel D. Effect in rural parts of urban districts</i>				
Affected from 2007	0.008 (0.059)	-0.044 (0.061)	0.080 (0.070)	-0.059 (0.045)
Mean before policy	0.057	0.120	0.231	0.471
R ²	0.092	0.113	0.216	0.349
N	4,922	4,922	4,922	8,500

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth. The table reports the average (Panels A and B) and phase-specific effect (Panels C and D) of user fee removal on postnatal care received by mothers immediately (column 1), within one hour (column 2) or within one day (column 3) after delivery, and the probability that child's vaccination is up to date at survey time (column 4). Information about the timing of postnatal care is only available for the last birth of each mother, and is not present in DHS 1996. Each coefficient is from a different regression. All regressions control for area and time fixed effects, and a dummy for multiple births. Columns 1 to 3 also control for mother's year of birth, and column 4 for child's sex and age dummies. Time fixed effects correspond to years of childbirth in columns 1 to 3, and to survey years in column 4.

* $p < .10$; ** $p < .05$; *** $p < .01$

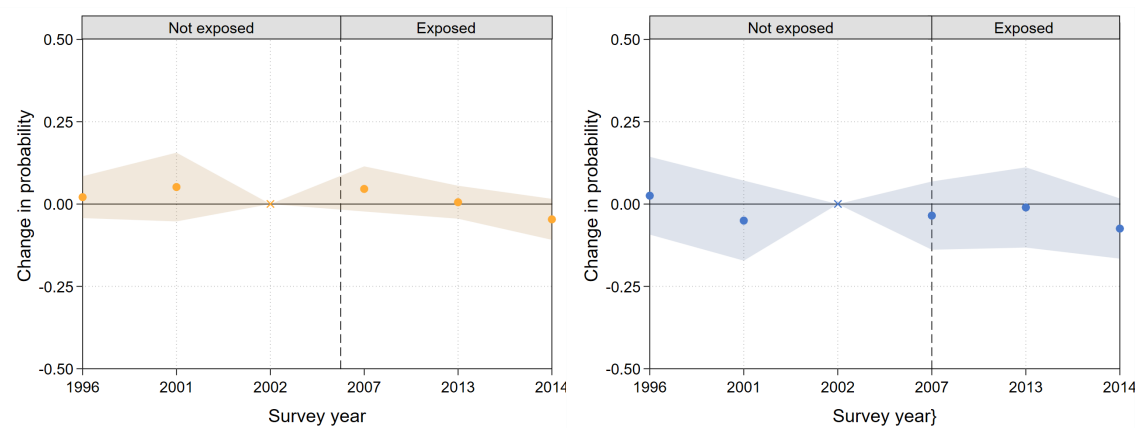
Figure E.2. Event study estimates of the effect of user fee removal on postnatal check-up for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side)



Source: Author's calculations from DHS 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). Year of implementation is normalized to zero. In addition to area and year of childbirth fixed effects, the covariates include mother's year of birth and a dummy for multiple pregnancy. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. Outcomes of interest are a dummy equals to one if mother's health was checked (a) immediately, (b) within one hour, and (c) within one day after delivery, zero otherwise. This information is not available in DHS 1996 and is only reported for the last birth of each mother.

Figure E.3. Event study estimates of the effect of user fee removal on the probability that child’s vaccinations are up to date at survey time



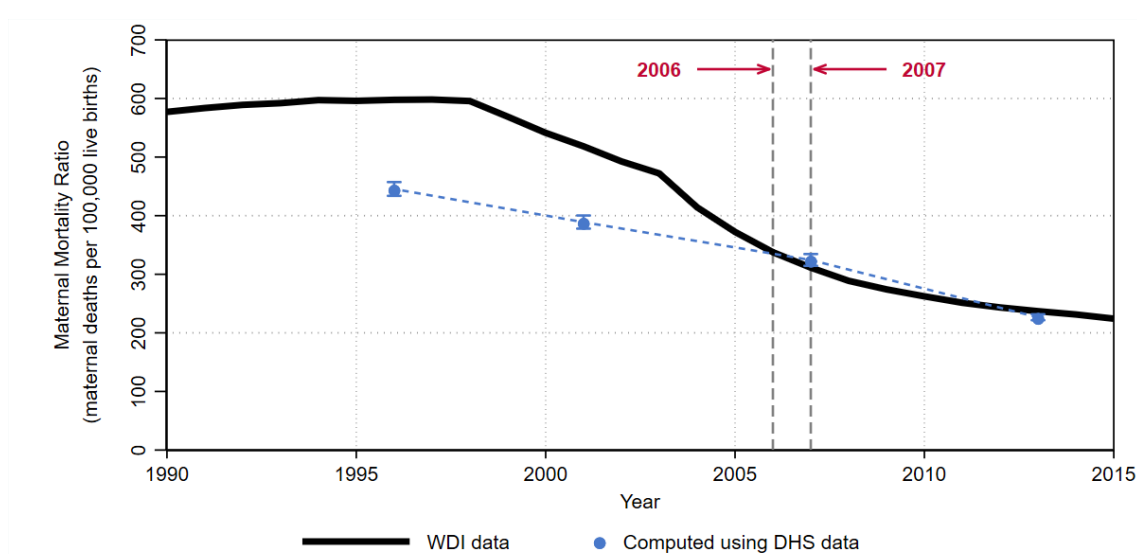
Source: Author’s calculations from DHS 2001, 2007 and 2013.

Notes: These figures show the coefficients for interaction terms between time dummies and treatment status obtained from an event-study specification for areas affected from April 2006 (left-hand side) and those affected from July 2007 (right-hand side). In addition to area and survey year fixed effects, the covariates include a dummy for multiple pregnancy, as well as child’s sex and age dummies. Shaded areas represent 95% confidence intervals with robust standard errors clustered at the area level. The omitted category is the last pre-treatment time dummy. The outcome of interest is a dummy equals to one if child’s vaccinations are up to date for polio, measles, Diphtheria-Pertussis-Tetanus (DPT) and the Bacillus Calmette–Guérin (BCG) vaccine against tuberculosis, depending on child’s age and the immunization schedule, and zero otherwise.

Appendix F. Evolution of the aggregate maternal mortality ratio

Due to data limitations, it is not possible to study the effect of the policy on maternal mortality. The Demographic and Health Surveys collect information on maternal mortality but only for the siblings of surveyed women. However, it does not gather information on where the women's siblings lived, making the assignment to treatment impossible. Instead, Appendix Figure F.1 plots the estimated national maternal mortality ratio from the World Development Indicators [World Bank, 2022] over the 1990-2015 period. It also reports the national maternal mortality ratio computed with the DHS data.

Figure F.1. Evolution of Maternal Mortality Ratio in Zambia since 1990



Source: World Development Indicators [World Bank, 2022] and author's calculations from DHS 1996, 2001, 2007 and 2013

Notes: The figure shows the maternal mortality ratio estimates from the World Development Indicators and the raw values obtained from the DHS.

Appendix G. Compositional changes, selection effects and fertility

G.1. Selection into pregnancy and compositional changes in mothers giving birth

Table G.1. The average effect of user fee removal on mothers' characteristics for different samples of births

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Years of education	At least one child born before first cohabitation	Never in union	Age at childbirth	Already experienced infant death	Polygamous	Health Index
Panel A. Average effect of user fee removal on characteristics of women giving birth Affected by the policy							
	-0.227*	-0.001	0.000	0.265	-0.010	0.005	0.030
	(0.130)	(0.006)	(0.010)	(0.228)	(0.014)	(0.011)	(0.065)
Mean before policy implementation	4.538	0.028	0.053	26.275	0.304	0.180	-0.397
R ²	0.181	0.022	0.047	0.012	0.033	0.067	0.529
N	25,653	24,037	25,677	25,678	22,279	21,154	25,678
Panel B. Average effect of user fee removal on characteristics of women giving birth in a publicly-supported health facility Affected by the policy							
	-0.529***	0.003	0.001	-0.167	0.005	0.011	-0.015
	(0.173)	(0.009)	(0.014)	(0.245)	(0.014)	(0.012)	(0.065)
Mean before policy implementation	5.797	0.038	0.082	25.767	0.262	0.141	-0.147
R ²	0.134	0.024	0.046	0.016	0.032	0.065	0.467
N	12,512	11,405	12,527	12,527	10,084	9,925	12,527
Panel C. Average effect of user fee removal on characteristics of women giving birth at home Affected by the policy							
	-0.089	-0.002	0.001	1.034***	0.009	0.000	0.020
	(0.164)	(0.009)	(0.014)	(0.368)	(0.024)	(0.026)	(0.050)
Mean before policy implementation	3.941	0.024	0.039	26.505	0.322	0.197	-0.521
R ²	0.117	0.026	0.058	0.019	0.034	0.061	0.427
N	12,467	11,976	12,474	12,475	11,587	10,624	12,475

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.
Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth. The table reports the average effect of user fee removal on different maternal characteristics for the full sample of births (Panel A), births in a publicly-supported health facility (Panel B) and home births (Panel C). These characteristics are: the number of years of education of the mother, a dummy which equals one if she had at least one child born before cohabitation (zero otherwise), a dummy which equals one if she has never been in union (zero otherwise), her age at childbirth, a dummy which equals one if she already experienced an infant death before the removal of user fees (zero otherwise), a dummy which equals one if she belongs to a polygamous household (zero otherwise), and an index of material wealth computed by the DHS. Each coefficient is from a different regression. All regressions control for area and year of childbirth fixed effects. In column 5, the sample is restricted to children whose mother had already experienced a childbirth before policy implementation. In column 6, the sample is restricted to mothers in union or living with a man at survey time.
* $p < .10$; ** $p < .05$; *** $p < .01$

G.2. Selection into medical deliveries and heterogeneous treatment effects

Table G.2. Heterogeneous effects of the policy according to mother's education and past experience of infant deaths

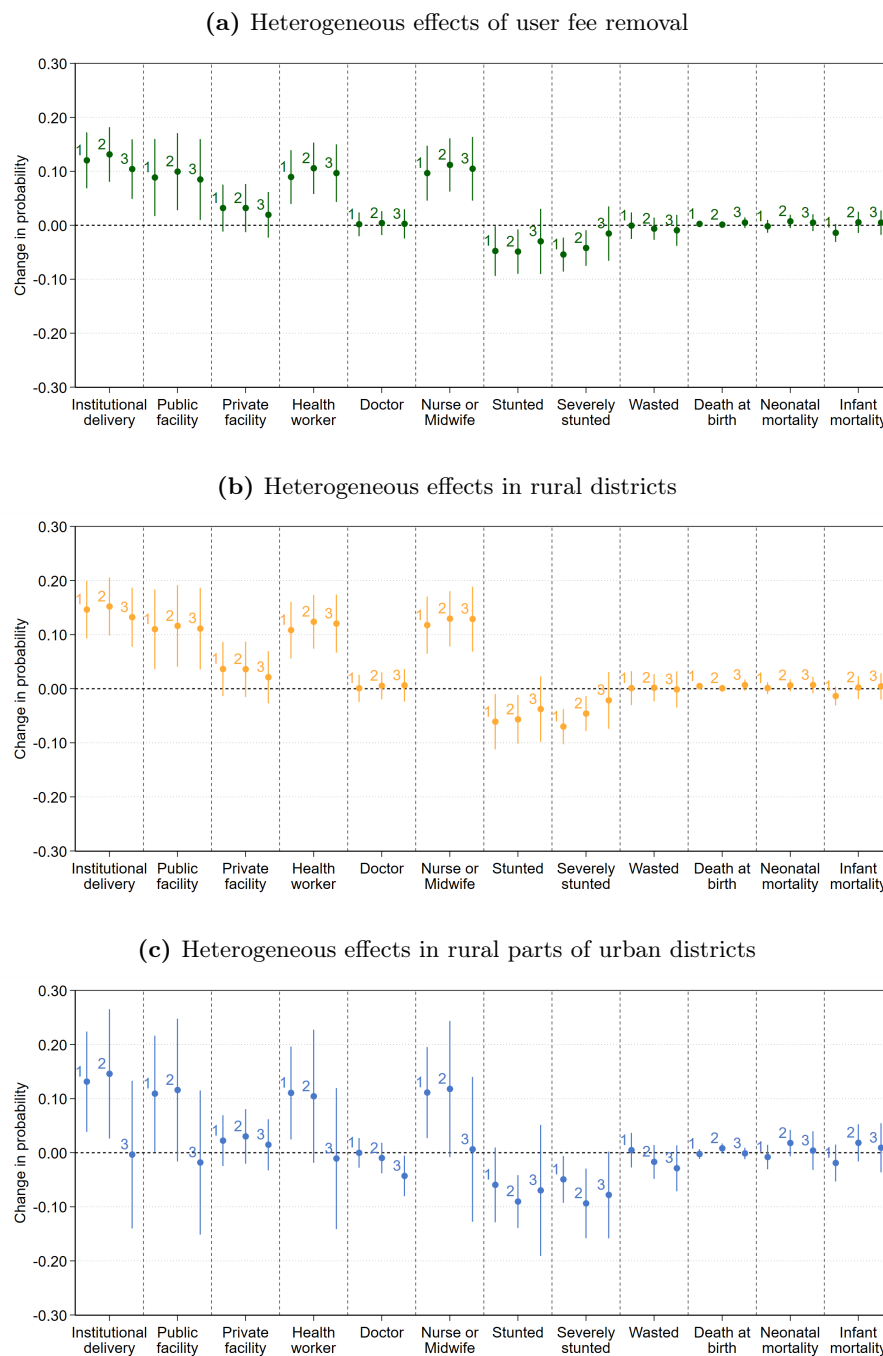
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Institutional delivery	Public facility	Private facility	Health worker	Doctor	Nurse or midwife	Stunted	Severely stunted	Wasted	Death at birth	Neonatal mortality	Infant mortality
<i>Panel A. Heterogeneous effects according to mother's number of years of education</i>												
Affected by the policy	0.148*** (0.030)	0.107*** (0.035)	0.040* (0.024)	0.116*** (0.028)	0.015 (0.014)	0.113*** (0.027)	-0.068** (0.026)	-0.056*** (0.019)	-0.003 (0.013)	0.004 (0.003)	0.004 (0.006)	-0.012 (0.010)
Years of education	0.037*** (0.003)	0.034*** (0.003)	0.002*** (0.001)	0.037*** (0.003)	0.006*** (0.001)	0.034*** (0.003)	-0.013*** (0.002)	-0.009*** (0.001)	-0.001* (0.001)	0.000 (0.000)	0.000 (0.001)	-0.002** (0.001)
Affected by the policy × Years of education	0.000 (0.004)	0.002 (0.004)	-0.002** (0.001)	0.001 (0.004)	-0.002 (0.001)	0.003 (0.004)	0.004* (0.002)	0.002 (0.002)	0.000 (0.001)	0.000 (0.000)	0.000 (0.001)	0.002 (0.001)
Mean before policy	0.323	0.318	0.004	0.318	0.013	0.299	0.545	0.286	0.063	0.009	0.033	0.086
R ²	0.277	0.250	0.246	0.272	0.053	0.255	0.091	0.070	0.022	0.017	0.029	0.036
N	25,462	25,462	25,462	25,556	25,556	25,556	21,092	21,092	21,050	25,653	25,240	19,156
<i>Panel B. Heterogeneous effects according to mother's past experience of infant deaths</i>												
Affected by the policy	0.113*** (0.026)	0.085** (0.035)	0.028 (0.021)	0.090*** (0.025)	0.013 (0.011)	0.095*** (0.025)	-0.047** (0.023)	-0.041** (0.016)	-0.006 (0.011)	0.003 (0.003)	0.008 (0.007)	0.004 (0.012)
Already experienced infant death	-0.040*** (0.010)	-0.030*** (0.010)	-0.010** (0.004)	-0.039*** (0.010)	0.002 (0.003)	-0.036*** (0.010)	-0.005 (0.013)	-0.015* (0.009)	-0.008 (0.005)	0.007*** (0.002)	0.018*** (0.005)	0.032*** (0.007)
Affected by the policy × Already experienced infant death	0.002 (0.017)	-0.005 (0.017)	0.007** (0.003)	-0.002 (0.016)	-0.004 (0.006)	-0.005 (0.015)	0.012 (0.019)	0.019 (0.014)	-0.004 (0.009)	0.004 (0.004)	-0.003 (0.007)	0.001 (0.011)
Mean before policy	0.302	0.297	0.005	0.297	0.010	0.281	0.537	0.279	0.062	0.008	0.029	0.081
R ²	0.218	0.194	0.278	0.213	0.048	0.205	0.085	0.065	0.022	0.021	0.038	0.040
N	20,129	20,129	20,129	20,206	20,206	20,206	17,034	17,034	16,990	20,287	19,945	15,116

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth (columns 1 to 6) or a child (columns 7 to 12). The table reports the heterogeneous effects of the policy according to mother's number of years of education (Panel A) and past experience of infant deaths (Panel B) on the different maternal health care utilization (columns 1 to 6) and child health (columns 7 to 12) outcomes. Each coefficient is from a different regression. All regressions control for area and time fixed effects, and a dummy for multiple births. Columns 1 to 6 also control for mother's year of birth, columns 7 to 9 for child's sex and age dummies, and columns 10 to 12 for mother's year of birth and child's sex. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.

* $p < .10$; ** $p < .05$; *** $p < .01$

Figure G.3. Heterogeneous effects of the policy according to tercile of material wealth



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figures plot the total effect of the removal of user fees on the corresponding outcome for each tercile of material wealth specified. 1 corresponds to the poorest third of households, 2 to the middle third, and 3 to the richest third. All regressions control for area and year of childbirth fixed effects, as well as a dummy for multiple birth and terciles of material wealth. Regressions for delivery conditions and child mortality outcomes also control for mother's year of birth. Child's sex and age dummies are included as additional control variables when looking at child nutritional outcomes, and child's sex is controlled for when looking at child mortality outcomes. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

G.3. Selection into live birth

Table G.4. The effect of user fee removal on stillbirths, miscarriages/abortions and live births composition

	(1)	(2)	(3)
	Stillbirth	Miscarriage/Abortion	Male birth
<i>Panel A. Average effect of user fee removal</i>			
Affected by the policy	0.001 (0.005)	-0.001 (0.009)	0.003 (0.011)
Mean before policy	0.012	0.033	0.496
R ²	0.011	0.022	0.006
N	14,337	14,913	25,678
<i>Panel B. Average effect of user fee removal using de Chaisemartin and D'Haultfœuille [2021] estimator</i>			
Affected by the policy	0.009 (0.017)	-0.018 (0.022)	0.026 (0.038)
Mean before policy	0.012	0.033	0.496
N	14,337	14,913	25,678
<i>Panel C. Effect in rural districts</i>			
Affected from 2006	0.004 (0.005)	-0.004 (0.010)	0.008 (0.012)
Mean before policy	0.011	0.034	0.497
R ²	0.012	0.022	0.005
N	12,956	12,956	22,148
<i>Panel D. Effect in rural parts of urban districts</i>			
Affected from 2007	-0.004 (0.006)	0.011 (0.013)	0.007 (0.016)
Mean before policy	0.012	0.031	0.490
R ²	0.020	0.035	0.012
N	5,411	5,411	9,479

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a pregnancy (columns 1 and 2) or a live birth (column 3). The table reports the average (Panels A and B) and phase-specific effect (Panels C and D) of user fee removal on the probability of stillbirth (column 1), of miscarriage or abortion (column 2) and on the sex ratio for live births (column 3). Due to data limitations, miscarriages and abortions cannot be distinguished. Each coefficient is from a different regression. All regressions control for area and year of childbirth fixed effects, as well as mother's year of birth. Column 3 also controls for a dummy for multiple births, which is not possible in columns 1 and 2. The sample used in columns 1 and 2 corresponds to all pregnancies that occurred during the last five years preceding the 2007 and 2013 survey waves, whatever their final outcome. This information is not available for the 1996 and 2001 survey waves.

* $p < .10$; ** $p < .05$; *** $p < .01$

G.4. Fertility and heterogeneous effects according to rank of birth

Table G.5. The effect of user fee removal when the sample is restricted to first born

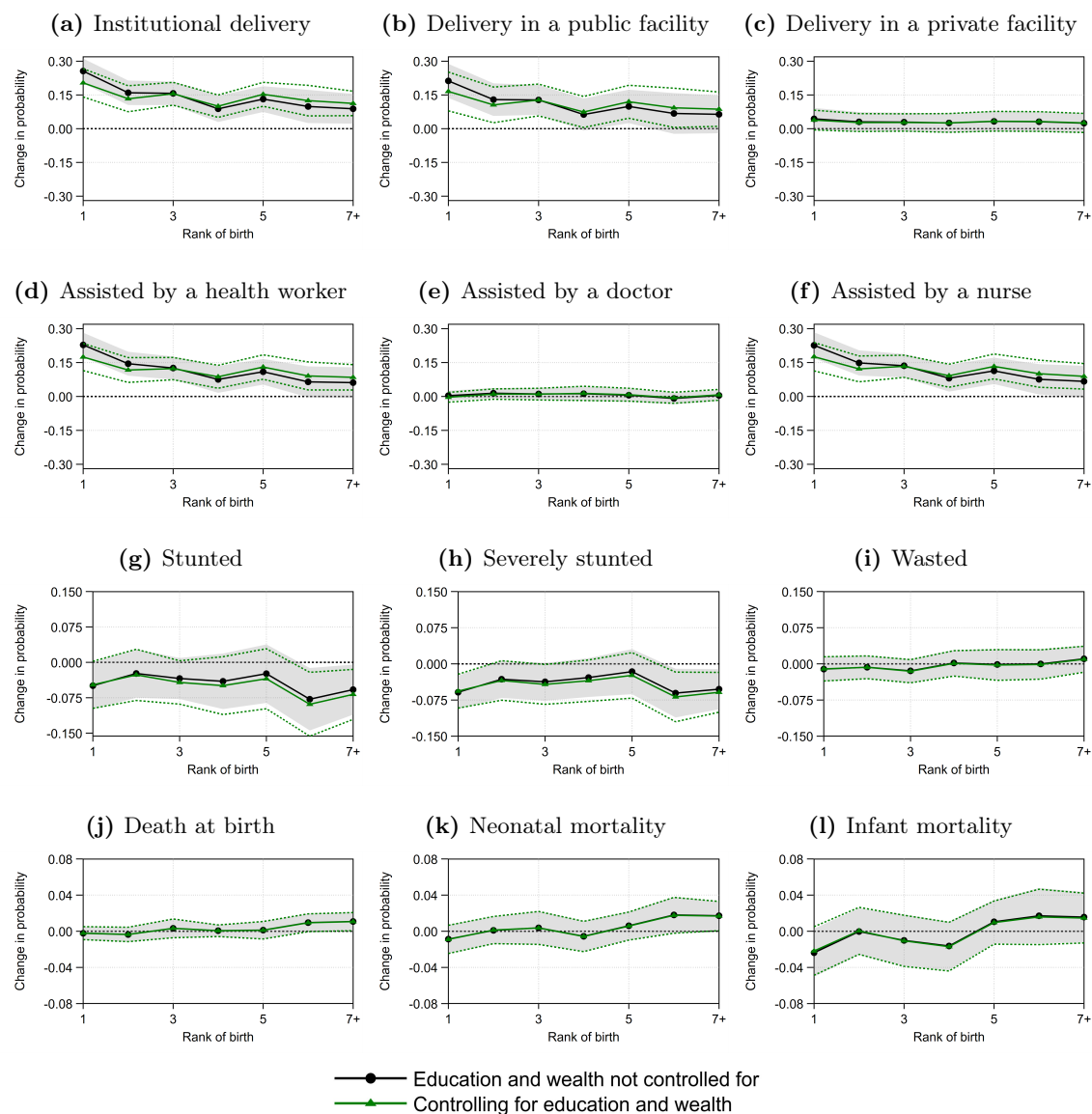
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Institutional delivery	Public facility	Private facility	Health worker	Doctor	Nurse or midwife	Stunted	Severely stunted	Wasted	Death at birth	Neonatal mortality	Infant mortality
Panel A. Average effect of user fee removal												
Affected by the policy	0.240*** (0.034)	0.211*** (0.041)	0.029 (0.021)	0.214*** (0.034)	-0.006 (0.019)	0.220*** (0.037)	-0.045 (0.039)	-0.068** (0.031)	-0.005 (0.018)	-0.006 (0.006)	-0.016 (0.011)	-0.029 (0.017)
Mean before policy	0.411	0.407	0.004	0.405	0.027	0.375	0.581	0.316	0.066	0.012	0.048	0.109
R ²	0.256	0.235	0.165	0.244	0.079	0.221	0.108	0.095	0.045	0.040	0.039	0.065
N	5,356	5,356	5,356	5,374	5,374	5,374	4,072	4,072	4,075	5,391	5,320	4,057
Panel B. Effect in rural districts												
Affected from 2006	0.235*** (0.036)	0.203*** (0.044)	0.032 (0.023)	0.204*** (0.037)	-0.009 (0.021)	0.215*** (0.040)	-0.074** (0.036)	-0.096*** (0.028)	0.009 (0.020)	-0.003 (0.006)	-0.016 (0.012)	-0.028 (0.017)
Mean before policy	0.421	0.418	0.003	0.417	0.028	0.388	0.586	0.313	0.065	0.012	0.051	0.109
R ²	0.251	0.229	0.174	0.238	0.080	0.214	0.107	0.092	0.041	0.045	0.042	0.066
N	4,706	4,706	4,706	4,723	4,723	4,723	3,585	3,585	3,580	4,738	4,675	3,546
Panel C. Effect in rural parts of urban districts												
Affected from 2007	0.296*** (0.068)	0.275*** (0.075)	0.021 (0.022)	0.270*** (0.065)	0.007 (0.026)	0.252*** (0.066)	-0.067 (0.073)	-0.069 (0.052)	-0.020 (0.023)	-0.010 (0.008)	-0.004 (0.017)	-0.058** (0.027)
Mean before policy	0.371	0.363	0.008	0.357	0.025	0.326	0.564	0.327	0.071	0.010	0.035	0.106
R ²	0.311	0.275	0.203	0.310	0.106	0.267	0.100	0.084	0.051	0.068	0.048	0.067
N	2,258	2,258	2,258	2,262	2,262	2,262	1,713	1,713	1,708	2,268	2,240	1,699

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth (columns 1 to 6) or a child (columns 7 to 12). The sample is restricted to first born children. The table reports the average (Panel A) and phase-specific effect (Panels B and C) of user fee removal on the different maternal health care utilization (columns 1 to 6) and child health (columns 7 to 12) outcomes. Each coefficient is from a different regression. All regressions control for area and time fixed effects, and a dummy for multiple births. Columns 1 to 6 also control for mother's year of birth, columns 7 to 9 for child's sex and age dummies, and columns 10 to 12 for mother's year of birth and child's sex. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.

* $p < .10$; ** $p < .05$; *** $p < .01$

Figure G.6. Heterogeneous effects according to child's rank of birth



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figures plot the point estimates from interaction terms between a dummy for exposure to user fee removal and a set of dummies for child's rank of birth. Each figure is from a separate estimation. Control variables include area and time fixed effects, and a dummy for multiple births. Additional controls include mother's year of birth for childbirth conditions, mother's year of birth and child's sex for mortality outcomes, and child's sex and age dummies for anthropometric outcomes. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. Green triangles represent the point estimates obtained with the same set of covariates, as well as mother education level and household material wealth. Shaded areas (resp. green dotted lines) represent 95% confidence intervals for point estimates obtained with the initial set of covariates (resp. point estimates obtained with the additional covariates) with robust standard errors clustered at the area level.

Table G.7. The effect of user fee removal on fertility behaviors

	(1)	(2)	(3)
	Preceding birth interval	Preceding birth interval < 24 months	Number of reported births per month per 1,000 surveyed mothers
<i>Panel A. Average effect of user fee removal</i>			
Affected by the policy	-2.602*** (0.701)	0.005 (0.013)	1.437 (2.085)
Mean before policy	36.313	0.169	30.025
R ²	0.098	0.033	0.354
N	20,286	20,286	9,907
<i>Panel B. Effect in rural districts</i>			
Affected from 2006	-2.728*** (0.728)	0.008 (0.013)	2.825 (1.904)
Mean before policy	36.334	0.170	28.825
R ²	0.099	0.035	0.286
N	17,409	17,409	8,423
<i>Panel C. Effect in rural parts of urban districts</i>			
Affected from 2007	-4.779*** (1.173)	0.015 (0.020)	0.817 (5.574)
Mean before policy	36.233	0.166	36.707
R ²	0.127	0.043	0.435
N	7,211	7,211	3,703

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

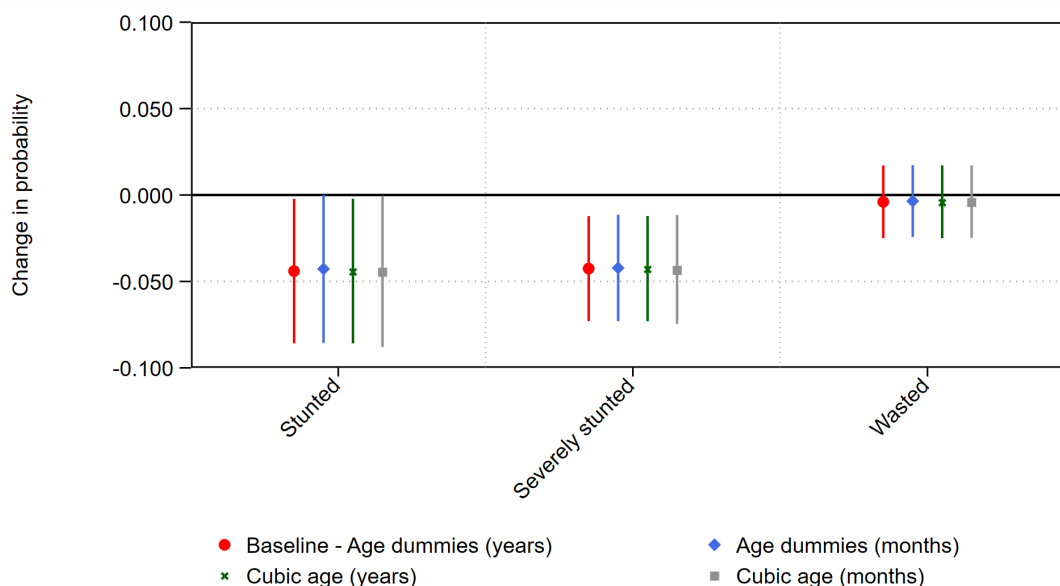
Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a birth (columns 1 and 2) or an area × birth date × survey year cell (column 3). The table reports the average (Panel A) and phase-specific effect (Panels B and C) of user fee removal on preceding birth interval in months (column 1), a dummy for preceding birth interval being less than 24 months (column 2) and number of reported births per month per 1,000 mothers surveyed (column 3). All regressions control for area and year of childbirth fixed effects. Columns 1 and 2 also control for a dummy for multiple births and column 3 for birth month fixed effects.

* $p < .10$; ** $p < .05$; *** $p < .01$

Appendix H. Sensitivity analysis

H.1. Alternative specification of age for anthropometric outcomes

Figure H.1. Anthropometric results for alternative specification of child age



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figure shows the point estimates obtained for each anthropometric indicators based on alternative ways to control for child's age. Each point corresponds to a separate difference-in-differences estimation. The red dots plot the baseline results when age dummies (in years) are controlled for. Blue diamonds show the results when controlling for dummies of age expressed in months. Green crosses correspond to the results obtained when controlling for a cubic relationship with child's age in years. Gray squares plot point estimates when controlling for a cubic relationship with child's age in months. In all specifications, other control variables include area and time fixed effects, a dummy for multiple births, as well as child's sex. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

H.2. Accounting for patterns in prenatal visits

Table H.2. Results when accounting for prenatal visits

	(1)	(2)	(3)	(4)	(5)	(6)
	Institutional delivery	Type of health facility		Assisted by a		
		Public	Private	Health worker	Doctor	Nurse or Midwife
<i>Controlling for having done at least 4 prenatal visits[†]</i>						
Panel A. Average effect of user fee removal						
Affected by the policy	0.132*** (0.024)	0.101*** (0.034)	0.031 (0.022)	0.109*** (0.024)	0.011 (0.012)	0.114*** (0.025)
N	16,954	16,954	16,954	17,026	17,026	17,026
Panel B. Effect in rural districts						
Affected from 2006	0.162*** (0.023)	0.126*** (0.034)	0.036 (0.026)	0.135*** (0.024)	0.012 (0.013)	0.140*** (0.025)
N	14,787	14,787	14,787	14,853	14,853	14,853
Panel C. Effect in rural parts of urban districts						
Affected from 2007	0.126** (0.051)	0.105* (0.055)	0.020 (0.024)	0.094** (0.044)	-0.001 (0.015)	0.098** (0.045)
N	6,624	6,624	6,624	6,634	6,634	6,634
<i>Controlling for a linear time trend interacted with the share of pregnancies that have completed at least 4 prenatal visits within each area before policy implementation</i>						
Panel D. Average effect of user fee removal						
Affected by the policy	0.108*** (0.025)	0.092*** (0.032)	0.016 (0.018)	0.086*** (0.024)	-0.002 (0.010)	0.093*** (0.024)
N	25,485	25,485	25,485	25,580	25,580	25,580
Panel E. Effect in rural districts						
Affected from 2006	0.135*** (0.029)	0.119*** (0.034)	0.016 (0.020)	0.106*** (0.027)	-0.001 (0.011)	0.114*** (0.027)
N	21,974	21,974	21,974	22,063	22,063	22,063
Panel F. Effect in rural parts of urban districts						
Affected from 2007	0.102** (0.046)	0.105** (0.050)	-0.004 (0.021)	0.079* (0.044)	-0.013 (0.012)	0.080* (0.046)
N	9,431	9,431	9,431	9,442	9,442	9,442

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a childbirth. The table reports the average effect of user fee removal on the probability to give birth in a health facility (column 1), in a publicly supported health facility (column 2), in a private one (column 3), to give birth with a skilled birth attendant (column 4), with a doctor (column 5) and with a nurse or a midwife (column 6). Each coefficient is from a different regression. All regressions control for area and year of childbirth fixed effects, as well as mother's year of birth and a dummy for multiple births.

* $p < .10$; ** $p < .05$; *** $p < .01$

[†] This information was collected for all births that occurred during the last five years preceding the survey in DHS 1996, and only for the last birth in DHS 2001, 2007 and 2013.

H.3. Correction for selective mortality

Table H.3. The effect of user fee removal on anthropometric indicators when taking into account potential selective mortality

	(1) Child is alive at survey time	(2) — Stunted —	(3) —	(4) — Severely stunted —	(5) —	(6) — Wasted —	(7) —
<i>Panel A. Average effect of user fee removal</i>							
Affected by the policy	-0.020 (0.105)	-0.044** (0.021)	-0.045** (0.022)	-0.044*** (0.016)	-0.041** (0.016)	-0.005 (0.011)	-0.002 (0.011)
Mean before policy	0.881	0.545	0.545	0.286	0.286	0.063	0.063
R ²	0.056	0.087	0.092	0.068	0.072	0.023	0.027
N	25,651	21,080	21,080	21,080	21,080	21,039	21,039
<i>Panel B. Effect in rural districts</i>							
Affected from 2006	-0.021 (0.107)	-0.055** (0.023)	-0.059** (0.023)	-0.054*** (0.015)	-0.053*** (0.016)	0.000 (0.013)	0.002 (0.013)
Mean before policy	0.881	0.544	0.544	0.283	0.283	0.063	0.063
R ²	0.056	0.084	0.091	0.066	0.071	0.023	0.028
N	22,138	18,199	18,199	18,199	18,199	18,152	18,152
<i>Panel C. Effect in rural parts of urban districts</i>							
Affected from 2007	-0.096 (0.197)	-0.074*** (0.022)	-0.070*** (0.023)	-0.072*** (0.021)	-0.071*** (0.021)	-0.007 (0.013)	-0.005 (0.013)
Mean before policy	0.886	0.549	0.549	0.295	0.295	0.063	0.063
R ²	0.058	0.090	0.099	0.067	0.076	0.022	0.036
N	9,429	7,661	7,661	7,661	7,661	7,636	7,636
Model	Logit	OLS	OLS	OLS	OLS	OLS	OLS
Inverse Probability Weighting	No	Yes	No	Yes	No	Yes	No
Semi-parametric approach	No	No	Yes	No	Yes	No	Yes

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a child (column 1) or a surviving child (columns 2 to 7). The table reports the average (Panel A) and phase-specific effect (Panels B and C) of user fee removal on the probability of being alive at survey time (column 1), stunted (columns 2 and 3), severely stunted (columns 4 and 5) and wasted (columns 6 and 7). All regressions control for area and time fixed effects, a dummy for multiple births and a dummy for girls. Column 1 also controls for mother's year of birth, and columns 2 to 7 for child's age dummies. The inverse probability weighting method consists in weighting observations according to the predicted survival probabilities at survey time obtained from column 1. The semi-parametric approach follows [Cosslett \[1991\]](#) by including one indicator variable for each centile of predicted survival probabilities obtained from column 1 as additional control variables.

* $p < .10$; ** $p < .05$; *** $p < .01$

H.4. Recall bias and age heaping

There are two possible sources of measurement error due to recall bias. First, mothers may under-report births and child deaths. I argue that recall bias can reasonably be considered low in this setting since a child's birth and death are milestones in a woman's life, and the recall period of five years is relatively short. Another reason is that the retrospective birth history questionnaire of the DHS is very precise, and interviewers are asked to check the accuracy of reported births with respect to the rest of the survey. Second, mothers may have rounded up child's age at death, leading to a mismeasurement in child mortality outcomes. To check the sensitivity of my results to age-heaping, I use an expanded definition of neonatal and infant mortality: neonatal mortality is now defined as the probability of dying within the first month of life instead of 28 days, and infant mortality now includes children who are reported to be dead at the age of one. Results barely changes with these new definitions (Appendix Table H.4).

Table H.4. The effect of user fee removal on child mortality when allowing for age-heaping

	(1)	(2)
	Died within first month of life	Died within first year of life
<i>Panel A. Average effect of user fee removal</i>		
Affected by the policy	0.005 (0.005)	-0.005 (0.009)
Mean before policy	0.039	0.096
R ²	0.032	0.036
N	25,265	19,173
<i>Panel B. Effect in rural districts</i>		
Affected from 2006	0.007 (0.005)	-0.005 (0.009)
Mean before policy	0.039	0.096
R ²	0.033	0.037
N	21,785	16,486
<i>Panel C. Effect in rural parts of urban districts</i>		
Affected from 2007	0.007 (0.007)	-0.010 (0.014)
Mean before policy	0.037	0.096
R ²	0.025	0.033
N	9,344	7,163

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. The unit of observation is a child. The table reports the average (Panel A) and phase-specific effect (Panels B and C) of user fee removal on the probability for a child to die within her first month of life (column 1) and within her first year of life (column 2). Each coefficient is from a different regression. All regressions control for area and year of childbirth fixed effects, as well as mother's year of birth, a dummy for multiple births and child's sex. Children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.

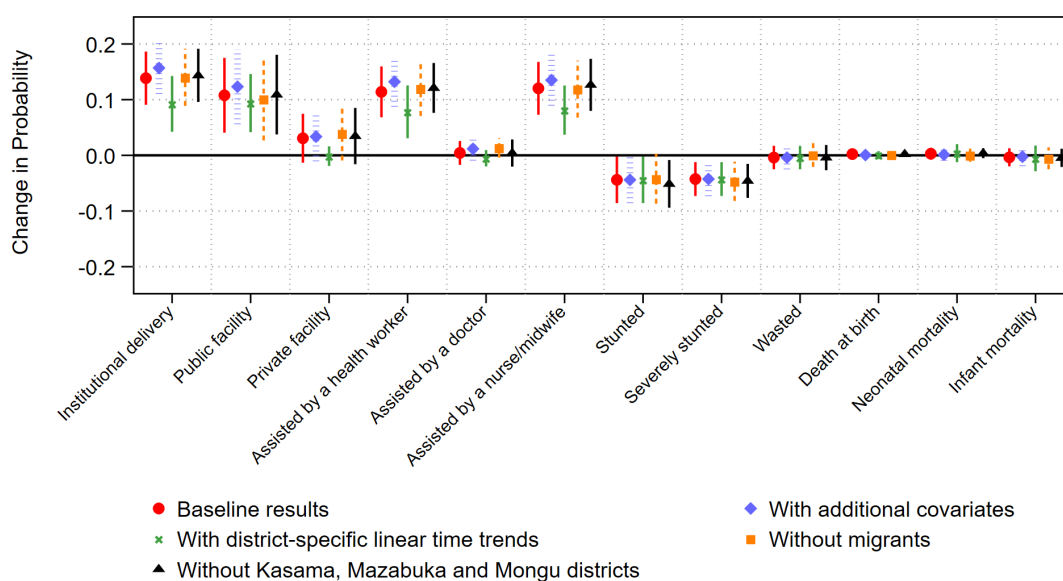
* $p < .10$; ** $p < .05$; *** $p < .01$

H.5. Additional control variables and treatment assignment

Additional covariates - Results remain similar when I include a full set of maternal covariates as additional control variables. I also present results when controlling for district-specific linear time trends. While these trends may pick up at least part of the effect, it gives some insight into the robustness of the results. As expected, point estimates are smaller in magnitude, but conclusions remain similar (Appendix Figure H.5).

Sensitivity to treatment assignment - I now check the sensitivity of my results to a finer assignment to treatment within urban districts by applying the eligibility criteria provided by the government. This refinement is not possible for the 1996 and 2001 survey waves since GPS coordinates are not available. I now consider as affected from July 1st, 2007, those individuals whose nearest health facility is located more than 15 kilometers away from the administrative center of the district and 20 kilometers away for districts located along a railway. For this, I use the GPS coordinates of all publicly-supported health facilities collected during the 2005 Health Facility Census. Results barely change when using this new definition of treatment areas (Appendix Figure H.6). Due to the scrambling procedure applied by DHS on GPS coordinates for confidentiality reasons, households surveyed in 2007 and 2013 may have been assigned to the wrong treatment area (see Appendix B.4). This is a problem only for those enumeration areas located near the boundaries (2 kilometers or less for urban enumeration areas and 5 kilometers or less for rural enumeration areas) of a district which has not the same treatment status as the actual one. Ninety-eight enumerations areas are concerned. The results are not significantly different when I excluded the corresponding 1,298 births from the analysis (see Appendix Figure H.6).

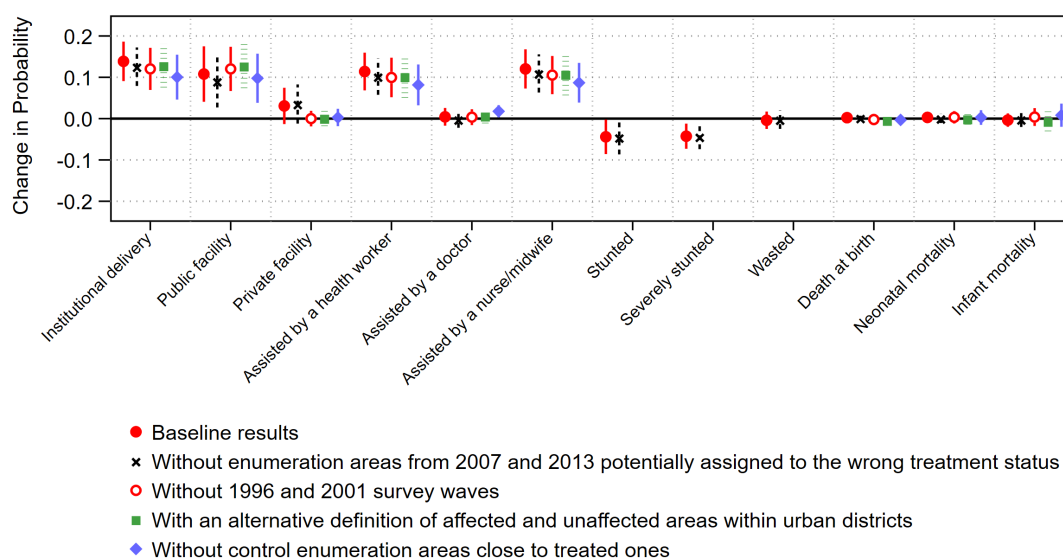
Figure H.5. Robustness checks: alternative specifications and samples



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figure shows the different point estimates obtained for each outcome from alternative specifications and when removing migrants from the analysis. Each point corresponds to a separate difference-in-differences estimation. The red dots plot the baseline results presented in Tables 2 and 3. Blue diamonds correspond to a specification with additional covariates, namely mother's year of birth by child's year of birth fixed effects, mother's number of years of education, religion and a set of dummies for rank of birth. Green crosses correspond to point estimates when controlling for district-specific linear time trends. Orange squares show the effect of user fee removal when excluding children whose family have migrated since their birth. Black triangles plot point estimates when excluding the three urban districts identified by Lépine et al. [2018] as having a significant part of their population that declare seeking care in a rural district. In all specifications, control variables include area and time fixed effects, as well as a dummy for multiple births. Additional controls include mother's year of birth for childbirth conditions, mother's year of birth and child's sex for mortality outcomes, and child's sex and age dummies for anthropometric outcomes. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

Figure H.6. Robustness checks: alternative samples



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figure shows the different point estimates obtained for each outcome for different samples. Each point corresponds to a separate difference-in-differences estimation. The red dots plot the baseline results presented in Tables 2 and 3. Black crosses show the results when the DHS 2007 and 2013 samples are restricted to under-5 children living in a rural enumeration area located more than 5 kilometers away from the boundaries of a district with another treatment status (more than 2 kilometers away for children living in an urban enumeration area). For the rest of the results presented in this figure, GPS coordinates from DHS 2007 and 2013 are used. Hence, DHS 1996 and 2001 are left apart and it is no longer possible to assess the effect of the policy on anthropometric indicators since there is no more pre-policy period for these outcomes measured at survey time. Red circles correspond to the results obtained without DHS 1996 and 2001. These are the benchmark for the last two sets of results presented here. Green squares correspond to point estimates when using an alternative criteria to classify enumeration areas within urban districts as affected or not. Individuals affected from 2007 are those living in an urban district and whose nearest health facility is located more than 15 kilometers away from the administrative center of the district and 20 kilometers away for districts located along the line of rail. Unaffected ones are those living within these radiuses. Blue diamonds plot point estimates when control enumeration areas located less than 5 kilometers away from an affected enumeration area are dropped. In all specifications, control variables include area and time fixed effects, as well as a dummy for multiple births. Additional controls include mother's year of birth for childbirth conditions, mother's year of birth and child's sex for mortality outcomes, and child's sex and age dummies for anthropometric outcomes. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group. The lines represent 95% confidence intervals with robust standard errors clustered at the area level.

H.6. Robustness to matching

Table H.7. The average effect of user fee removal when difference-in-differences is combined with matching

	(1) Institutional delivery	(2) Public facility	(3) Private facility	(4) Health worker	(5) Doctor	(6) Nurse or midwife	(7) Stunted	(8) Severely stunted	(9) Wasted	(10) Death at birth	(11) Neonatal mortality	(12) Infant mortality
Panel A. Propensity score nearest neighbor matching												
Affected	0.169*** (0.050)	0.160*** (0.050)	0.009 (0.007)	0.144*** (0.051)	0.027** (0.012)	0.190*** (0.045)	-0.116** (0.055)	-0.105** (0.051)	-0.011 (0.037)	0.006 (0.009)	0.020 (0.024)	0.012 (0.050)
by the policy	[0.070; 0.268]	[0.061; 0.259]	[-0.004; 0.023]	[0.043; 0.245]	[0.003; 0.051]	[0.100; 0.279]	[-0.225; -0.007]	[-0.207; -0.002]	[-0.086; 0.063]	[-0.012; 0.025]	[-0.028; 0.067]	[-0.087; 0.112]
N	16,586	16,586	16,586	16,658	16,658	16,658	13,954	13,954	13,937	16,726	16,443	12,499
Panel B. Propensity score kernel matching												
Affected	0.160*** (0.057)	0.150** (0.058)	0.010 (0.008)	0.134** (0.057)	0.027*** (0.010)	0.149*** (0.052)	-0.085* (0.047)	-0.069* (0.037)	0.021 (0.032)	0.009 (0.006)	0.013 (0.015)	0.002 (0.038)
by the policy	[0.047; 0.273]	[0.034; 0.265]	[-0.006; 0.025]	[0.020; 0.247]	[0.007; 0.048]	[0.046; 0.252]	[-0.178; 0.009]	[-0.142; 0.004]	[-0.043; 0.084]	[-0.003; 0.022]	[-0.017; 0.043]	[-0.073; 0.076]
N	19,359	19,359	19,359	19,430	19,430	19,430	16,280	16,280	16,241	19,509	19,190	14,619
Panel C. Mahalanobis distance matching												
Affected	0.209*** (0.043)	0.204*** (0.043)	0.005 (0.005)	0.178*** (0.043)	0.014 (0.009)	0.197*** (0.043)	-0.095* (0.054)	-0.054 (0.044)	0.038 (0.041)	0.009 (0.007)	0.013 (0.014)	0.014 (0.047)
by the policy	[0.123; 0.295]	[0.118; 0.290]	[-0.005; 0.016]	[0.092; 0.263]	[-0.003; 0.032]	[0.111; 0.282]	[-0.203; 0.014]	[-0.142; 0.034]	[-0.044; 0.120]	[-0.005; 0.024]	[-0.015; 0.042]	[-0.079; 0.108]
N	18,390	18,390	18,390	18,469	18,469	18,469	15,455	15,455	15,432	18,547	18,239	13,834
Panel D. Entropy balancing												
Affected	0.155*** (0.057)	0.152*** (0.057)	0.001 (0.004)	0.127** (0.057)	0.023*** (0.009)	0.160*** (0.053)	-0.124** (0.055)	-0.099** (0.046)	0.036 (0.043)	0.017* (0.009)	0.027 (0.020)	0.023 (0.047)
by the policy	[0.040; 0.266]	[0.038; 0.266]	[-0.006; 0.008]	[0.013; 0.240]	[0.006; 0.040]	[0.054; 0.265]	[-0.233; -0.015]	[-0.190; -0.007]	[-0.049; 0.121]	[-0.001; 0.034]	[-0.012; 0.066]	[-0.070; 0.116]
N	20,938	20,938	20,938	21,017	21,017	21,017	17,595	17,595	17,553	21,102	20,759	15,769

Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: Robust standard errors clustered at the area level reported in parentheses. 95% confidence intervals in brackets. The unit of observation is a childbirth (columns 1 to 6) or a child (columns 7 to 12). The table reports the point estimates obtained with different matching methods. Matching is done on both household (material wealth index, sex and age of household head, whether the household moved since the child's birth, whether the household is polygamous or not), maternal (education level, age at first sexual intercourse, age at first cohabitation) and child characteristics (sex of the child, whether the child is from a multiple birth, and birth rank). Each coefficient is from a different regression. All regressions control for area and time fixed effects, and a dummy for multiple births. Columns 1 to 6 also control for mother's year of birth, columns 7 to 9 for child's sex and age, and columns 10 to 12 for mother's year of birth and child's sex. For neonatal and infant mortality, children who did not reach the corresponding age at survey time are dropped to avoid censoring bias, and those who did not reach this age by 2012 are also excluded since the policy was then extended to the control group.

* $p < .10$; ** $p < .05$; *** $p < .01$

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Chapter 2

Mothers and Fathers: Education, Coresidence and Child Health

co-written with Élodie Djemai and Anne-Laure Samson

A B S T R A C T

Using four waves of the Demographic and Health Surveys from Zimbabwe, we exploit the exogenous variation generated by the 1980 Education Reform to identify the respective causal effect of mother's and father's education on health investments and child health. Based on a simultaneous-equation model, we consider possible selection into coresidence between parents and children, endogeneity of education levels, and parental education sorting. Our results suggest that father's education significantly affects perinatal and child health investments, and matters more than mother's. Results are robust to several robustness checks. While we observe selection into coresidence with the child, it does not affect the causal effect of education on health investments and child health. Parental educational sorting is also shown to be important. Our findings suggest that not considering both parents' education simultaneously may produce misleading conclusions. This chapter has important policy implications as the results suggest that child health policies targeting and involving fathers could have sizeable effects on their own and in complement to existing policies exclusively focused on mothers and mothers-to-be.



Location of Zimbabwe

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1. Introduction

The factors leading to better health are as important to economists as to other researchers in social sciences and policy-makers. The lack of resources at both the governmental and individual levels has long been highlighted as the main barrier to improving health in developing countries. Poor people in low-income countries face a variety of health-related risks, with young children accounting for most of the global disease burden. Of the 55.4 million deaths in 2019, 9.3% were of children under the age of five. In Africa, this figure reached 34.8%.¹ Over 46% of all deaths in low-income countries in 2019 were caused by so-called Group I conditions, which include communicable, maternal, neonatal, and nutritional disorders. By way of contrast, only 6.6% of deaths in high-income countries were due to these causes [World Health Organization, 2020].² These conditions caused 57% of all deaths in Sub-Saharan Africa in 2019. As such, most deaths could be avoided by adopting preventive actions [Banerjee and Duflo, 2011] such as vaccination, water filtering, breastfeeding, and the use of bed nets. Education plays a key role here via its induced increased demand for prevention.

Since the model of health demand in Grossman [1972], the education-health relationship has appeared in a wide body of theoretical and empirical research. On average, the more-educated have better health and live longer than the less-educated (e.g. Lleras-Muney, 2005). Education not only affects the adults' own health, but parental education also impacts the health of their children.

There are many channels through which education might affect health. The first is wealth. The educated are likely to have better labor opportunities and higher wages so that they can more likely afford the cost of prevention, treatment, and private health insurance, have better access to health care and health centers, and have less painful jobs. Second, the educated are more likely to understand the prevention messages they receive than their less-educated counterparts. Third, they have greater incentives to invest in preventive behaviors as, given the wage differential, the gap in terms of the future loss from illness is higher for the educated than for the less-educated. Last, education teaches discipline, compliance with rules and exams, exertion of effort, and accepting constraints, as noted in Basu [2002]. As such, it might help educated people to adopt costly preventive behaviors. Most of these mechanisms also apply when explaining why parental education might improve child health.

¹ Authors' calculation from World Health Organization [2020].

² The gap was even larger in 2000: 65.4% in low-income countries v.s. 6.6% in high-income countries.

Using the four waves of the Demographic and Health Surveys collected in Zimbabwe³ from 1994 to 2010, we examine health investments and the health outcomes of children aged 0-59 months born between 1990 and 2011. We compare child health investments and child health outcomes from households with educated mothers and fathers with those in which parents are less educated.

The major problem in this comparison is the endogeneity of education, from the correlation between the unobservable characteristics leading to education and those leading to health investments. Two examples of these unobserved characteristics are ability and time preference. Education and health are two indicators of human capital. As such, investing in education and investing in health both imply costly investments today for an uncertain future benefit. In addition, if educated parents are in better health than less-educated parents, this affects the child's health via the intergenerational transmission of health (e.g. [Bhalotra and Rawlings 2011](#)).

A number of contributions have exploited exogenous variation in education to identify the causal relationship between education and outcomes such as employment, fertility, and health. Recent articles have explored the relationship between education and health in developing countries, as major reforms to the latter's school systems took place between 1970 and 2000. Using information on reforms allows us to estimate the causal effect of education on health outcomes in a quasi-experimental setting, as it provides exogenous variation in enrolment in primary or secondary school, the number of years of completed schooling or the likelihood of dropping out of school in instrumental-variable or regression-discontinuity approaches. Examples of these reforms are compulsory school enrollment [[Agüero and Bharadwaj, 2014](#); [Grépin and Bharadwaj, 2015](#); [Güneş, 2015](#); [Silles, 2011](#)], the rise of the school-leaving age [[Albouy and Lequien, 2009](#); [Kemptner et al., 2011](#)], the supply of schools [[Bhalotra and Clarke, 2014](#); [Breierova and Duflo, 2004](#); [Silles, 2009](#)], the provision of trained teachers [[Shrestha and Shrestha, 2020](#)], the implementation of Universal Primary Education policies [[Behrman, 2015](#); [Delesalle, 2021](#); [Osili and Long, 2008](#)] and changes in school fees [[Chicoine, 2021](#); [Hahn et al., 2018](#); [Masuda and Yamauchi, 2020](#); [Oyelere, 2010](#); [Silles, 2009, 2011](#); [Zenebe Gebre, 2020](#)]. We here exploit the exogenous increase in education produced by the 1980 reform to estimate the causal effect of mother's and father's education on child health in Zimbabwe. The 1980 Education Reform is a nationwide reform that

³ Zimbabwe is a low-income country of 16 million inhabitants located in Southern Africa with a Gross Domestic Product (GDP) per capita of US\$ 1,362.5 (constant 2015 US\$) in 2021 [[World Bank, 2022](#)]. The under-5 mortality rate was 86 per 1,000 live births in 2010, and life expectancy at birth was 61 in 1985, 44 in 2002, and 51 in 2010 [[World Bank, 2022](#)]. The large fall at the end of the 1990s reflects high HIV prevalence. The HIV prevalence rate in the Demographic and Health Surveys was 21.1% for women aged 15-49 and 14.5% for men in 2005 (vs. 17.7 and 12.3 respectively in 2010).

mostly consists in compulsory primary school enrollment, the removal of primary school fees, and automatic admission to secondary school. It affects all school-aged children and most importantly children born after 1965 as they were aged 15 or less at the time of the reform and were exogenously more likely to pursue in secondary school compared to those born earlier.

Coresidence between parents and children might also bias the estimates, as it might not be random in the population and covers a non-negligible share of children: only 52.7% of our survey children aged 0-59 months live with both parents. It is well-established in the literature that children growing up in single-parent households acquire less human capital, whether the parents divorced or one died (see [Adda et al., 2011](#); [Fitzsimons and Mesnard, 2014](#)). Living with both parents, compared to living with only one or neither, is not random and might affect child health. We treat this as a selection issue, as the education of the parent is not observed if he or she does not live in the same household as the observed child. The selection equations, one for each parent, are identified using exogenous variations in community practices (e.g. the share of mothers who give birth before being married). Our analysis of selection into coresidence provides new insights into the current literature on the education-health relationship that has to date neglected this dimension. [Emran et al. \[2018\]](#) document this source of bias, calling it a truncation bias due to coresidency in the estimations of intergenerational mobility.

We also contribute to the literature on the respective role of mothers and fathers on child outcomes. The role of father's education has been overlooked in the current literature, with only relatively few contributions [[Alderman and Headey, 2017](#); [Apouey and Geoffard, 2016](#); [Breierova and Duflo, 2004](#); [Case and Paxson, 2001](#); [Chou et al., 2010](#); [De Neve and Subramanian, 2017](#); [Lindeboom et al., 2009](#); [Averett et al., 2005](#)]. This could reflect the common wisdom that mothers matter more than fathers in raising children. Another purely-empirical reason is that mothers are more likely than fathers to live with their children in many countries, leading to empirical challenges when trying to evaluate the role of fathers. [Case and Paxson \[2001\]](#) study the role of father's and mother's education and coresidence in child health in the US, but without modeling selection into coresidence or marital sorting.

Our work here also takes into account the marital education sorting of parents as an additional source of bias in the estimates, with the size of the bias being *a priori* even larger in articles that examine the effect of each parent's education in separate models. If the correlation between education levels is high, the estimate of the effect of mother's

education on child's health without controlling for father's education may instead pick up the effect of father's education. This source of bias is acknowledged, even though not resolved, in [Carneiro et al. \[2013\]](#) who estimate the effect of maternal education on child outcomes. Using data from Nepal and investigating the role of female education on child welfare, [Fafchamps and Shilpi \[2014\]](#) conclude that "at least part of the predictive power of mother education on child welfare is driven by marriage market effects and higher father's education." The bias may also come from unobservable characteristics (such as ability and time preference) that drive (un)educated people to match together. Marital sorting based on education has been documented in developed and developing countries (e.g. [Azam and Djemai, 2019](#); [Chiappori et al., 2009](#); [Hahn et al., 2018](#); [Van Bavel and Klesment, 2017](#); [Garfinkel et al., 2002](#)).

The father's contribution is modeled in three ways in recent work. First, the effect of the average mother's and father's education is estimated in [Breierova and Duflo \[2004\]](#). However, this does not allow us to consider differences between parents nor to use exposure to the reform as an instrumental variable, as men are usually older than their spouses. Second, two separate models are estimated, one controlling for mother's education and the other for father's education, as in [Apouey and Geoffard \[2016\]](#), [Chou et al. \[2010\]](#) and [De Neve and Subramanian \[2017\]](#). From our viewpoint, this is debatable for two reasons: in the case of educational marital sorting, part of the effect of mother's education may reflect that of the father's, and there is no discussion about coresidence, even though the sample sizes vary from one estimation to the other. If one parent is absent because of divorce or death, the parent who is living with the child might compensate for the absence, and all the more so when (s)he is more educated and, as such, has more room to adjust. Some papers have explored the role of the absence of one parent on the formation of human capital and suggest that human capital is greatly affected. One example is [Adda et al. \[2011\]](#), who evaluate the long-term consequences of parental death and find that mothers and fathers have differential effects on child cognitive and non-cognitive skills. The third approach is to estimate the effect of both mother's and father's education in the same equation, as in [Lindeboom et al. \[2009\]](#) and [Alderman and Headey \[2017\]](#). In the latter, maternal and paternal education are referred to even for non-biological parents, whereas the effect might be different, given work on child fostering and step-mothers (e.g. [Case and Paxson, 2001](#)). In this chapter, we focus on the role of biological mothers and fathers, and estimate their respective effects in a single equation.

[Grépin and Bharadwaj \[2015\]](#), [De Neve and Subramanian \[2017\]](#) and [Makate and Makate](#)

[2018] are closest to our analysis, as they consider the 1980 Education Reform in Zimbabwe to estimate the causal effect of parental education on child health. Grépin and Bharadwaj [2015] focus on the effect of maternal education on child mortality and Makate and Makate [2018] on dietary practices and nutrition, while we here estimate the effect of both father's and mother's education on child's current health, health investments, prenatal care, and childbirth conditions. De Neve and Subramanian [2017] estimate the effect of father's and mother's education on child malnutrition, as we do, but their estimation strategy differs from ours in several respects: they estimate the respective effects in separate regressions, the outcomes are different, they do not take marital sorting into account nor selection into coresidence. De Neve and Subramanian [2017] find a negative correlation between nutrition outcomes (namely being stunted, wasted, or underweight) and mother's duration of schooling, as well as with father's duration of schooling, but no evidence of a causal effect of parental education when schooling is instrumented by exposure to the reform.

Our results mostly confirm the existing evidence: there is a high correlation between mother's education and child health, and between father's education and child health. We instrument education by the exposure to the 1980 reform that affects those born in 1965 or later. The instrumented effect of father's education on prenatal care, birth conditions, and vaccination continues to be positive; however, that of mother's education becomes zero. This conclusion still holds when we exclude parents born in 1965 and 1966, who are only partially exposed to the reform, and when we restrict the sample to parents born in years close to 1965. The conclusion also holds when selection into coresidence is taken into account. While the inverse Mills ratios being significant in most cases shows that the selection has an effect on child's health, including the Mills ratios does not affect the size and significance of the 2SLS coefficients of parental education on child's health. We also provide some supporting evidence that while wealth and fertility might be mediators, education directly affects child health. Lastly, our finding shows that if father's education is not included in the equation, the instrumented effect of mother's education on birth outcomes and sleeping under a bed net is significant. This suggests that the effect of father's education is confounded in the effect of mother's education when not considered simultaneously due to assortative matching of the parents.

The remainder of this chapter is organized as follows. Section 2 presents the data. Section 3 describes the reform and its impact on parents' education. The estimation strategy is presented in Section 4. The empirical results are described in Section 5, and the robustness checks and extensions appear in Section 6. Last, Section 7 concludes.

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2. Data

2.1. Sample

We use household-level data collected by the Demographic and Health Surveys in Zimbabwe. This survey is nationally representative of households and was collected in 1994, 1999, 2005 and 2010.⁴ The sampling is in two stages and independent in each survey round. First, enumeration areas are selected based on the most recent available census. Second, a complete listing of the households living in the selected enumeration areas (also called communities here) is established in order to randomly select the sampled households, and in the latter every women aged 15-49, whether permanent residents or visitors (who slept in the household the night before the survey) are eligible for interview. We here use the data files from the household roster, the female questionnaire, and to a lesser extent the male questionnaire.

The household roster includes the complete list of household members and, for each member, his age and highest level of education. For children, the identification codes of the mother and father are reported if they live in the same household. As such, we have different types of households and family composition. We observe children who are not living with their parents (e.g. foster children) and children living with either one or both parents. By construction, if a sampled mother is not living with one of her children, this child is not a household member and is not present for the collection of anthropometric measures.

The analysis focuses on children aged between 0 and 59 months old. Mothers are asked specific questions about all the children they had over the five years preceding the survey as part of the female questionnaire. These questions cover prenatal care, delivery conditions and preventive behaviors such as vaccination. We also have anthropometric measures for children in this age group. The four rounds of survey data cover 19,702 children aged 0-59 months.⁵, hence we observe young children born over the period from 1990 to 2011. However, to observe the level of education of both parents, we need to restrict the sample to children who currently live with both parents (N=10,381 children). We will discuss

⁴ Two additional waves are available: 1988 and 2015. First, we cannot use the 1988 survey wave in our strategy design since it is impossible to link the household children to their fathers. Second, we do not use the 2015 survey wave because as parents are younger, the number of parents who have not been exposed to the reform is very small. Only 0.2% of the children in this wave were born to unexposed mothers, and 5.5% to unexposed fathers.

⁵ In the rest of the chapter, we use the term children to refer to children of this specific age group, and mothers and fathers for parents of children of this age group.

later the potential selection bias arising from the coresidence in Section 4.3. Mothers from this sample are born between 1916 and 1995, and fathers between 1898 and 1993. To circumvent survival bias of the parents, we removed the oldest parents from this sample, i.e. all parents born before 1950.⁶ Our analytical sample is composed of 9,365 children aged 0-59 months.

Note that it might be the case that couples are observed more than once if they have more than one child aged 0-59 months at the time of the survey. One may argue that the duplicates are more likely to concern younger parents and/or less educated parents on average. Such types of couples would as a result stand for a greater weight in the regression. In our analytical sample, 77% of the couples have only one observed child and 23% have more than one child 0-59 months old. In other words, we have a total of 9,365 children and a total of 7,497 mothers. 5,744 have only one observed child 0-59 months old. Our core empirical results hold when we weight the observations by the square root of the number of observations for each couple.⁷

2.2. Data description

The summary statistics for the entire sample appear in column 1 of Table 1. Over the entire sample, 50% of the children are girls, the average age is 2 and 24% live in urban areas. For 98% of the 0-59 months children living in sampled households the mother is still alive, and for 95% the father is alive. Coresidence with the mother is 31 percentage points more likely than coresidence with the father: 85% of children live in the same household as their mother, and 54% in the same household as their father.

The summary statistics for the outcome variables appear in Panel B. These can be grouped into three categories: *(i)* outcomes related to prenatal care and birth, namely a dummy for the mother having had at least four prenatal visits during her pregnancy, being born in a health facility and their mother having been assisted by medical staff at birth; *(ii)* malnutrition with a dummy for being stunted (too short for their age) and a dummy for being wasted (too thin for their height)⁸; and *(iii)* prevention (vaccination and sleeping under a mosquito bed net).⁹

⁶ Only 100 children have mothers born before 1950 and 1,004 have fathers born before 1950. We show as a robustness check further below that our results remain unchanged when parents born before 1950 are kept in the analysis.

⁷ Results reported in Appendix Table C.1.

⁸ Children are stunted if their height-for-age z -score is more than two standard deviations below the reference value. Children are wasted if their weight-for-height z -score is more than two standard deviations below the reference value.

⁹ The use of bed nets is not asked in the 1994 and 1999 survey waves. We do not analyze breastfeeding as 98% of children were breastfed. We are unable to estimate the effect of parental education on child mortality because it is impossible to link deceased children to their fathers, as the information to do so

69% of sampled children were born in a health facility, and 68% of them had a birth assisted by a skilled medical attendant. In 71% of cases, their mother attended at least four prenatal visits. 33% of young children are stunted and 6% are wasted. The average number of injections from the recommended immunization package (BCG, Diphtheria-Pertussis-Tetanus, measles, polio) received by a child aged 0-59 months is 5.73 (out of 8), and 9% of sampled children slept under a bed net the night before the survey.

Mother and father education appear in Panel C of Table 1. Our unit of observation is the child even for the statistics of the parents. The average number of years of schooling is 7.6 for mothers and 8.3 for fathers. 69% (73%) of mothers (fathers) completed primary school, and 52% (59%) attended at least one year of secondary school.

Other control variables appear in Panel D. Mothers' average age is 28.3 and that for fathers 36.2. This age difference corresponds to the usual age-difference figure found in existing work (e.g. [d'Albis et al., 2012](#)). Variables used to estimate the probability of the children to live with his father or mother are also reported. The average proportion of respondents who are separated, divorced or widowed in the community is 15%, the proportion of women who had their first child born before marriage is 20%, and the average proportion of polygamous households is 14%.

Note that the descriptive statistics for the entire sample (column 1), for the sub-sample of children living with both parents (column 2) and for the sub-sample of children who live with both parents and who have parents born in 1950 or later (column 3) are very similar. This suggests that our analytical sample is not highly selected. In Panel C, the outcomes are equal in column 2 and column 3 suggesting that the restriction of the sample based on parents born after 1950 is not an issue.

is only available for alive children living in the sampled households.

Table 1. Summary statistics on the main variables

	(1)	(2)	(3)
	All	Sample of children living with both parents	Sample of children living with both parents born in 1950 or later
<i>Panel A. Child characteristics</i>			
Girl	0.50	0.50	0.50
Age	2.00	1.88	1.84
Urban	0.24	0.30	0.32
Rich	0.33	0.39	0.41
Polygamous household	0.14	0.12	0.10
Mother alive	0.98	1.00	1.00
Father alive	0.95	1.00	1.00
Mother present	0.85	1.00	1.00
Father present	0.54	1.00	1.00
1994	0.21	0.21	0.17
1999	0.19	0.19	0.18
2005	0.30	0.30	0.32
2010	0.30	0.30	0.33
<i>Panel B. Outcomes</i>			
At least 4 prenatal visits [#]	0.71	0.71	0.71
Health Facility Birth [#]	0.69	0.67	0.68
Birth assisted by medical staff [#]	0.68	0.67	0.68
Stunted	0.33	0.32	0.32
Wasted	0.06	0.06	0.06
Number of injections received by child	5.73	5.71	5.71
Slept under net last night	0.09	0.12	0.12
<i>Panel C. Parental Education</i>			
Years of education (mother) [#]	7.57	7.48	7.82
Complete primary at least (mother) [#]	0.69	0.67	0.71
Secondary school at least (mother) [#]	0.52	0.51	0.54
Years of education (father) [†]	8.27	8.26	8.64
Complete primary at least (father) [†]	0.73	0.73	0.77
Secondary school at least (father) [†]	0.59	0.59	0.63
<i>Panel D. Other Characteristics</i>			
Mother's age [#]	28.30	28.67	27.78
Father's age [†]	36.16	36.15	33.89
Separated (% in cluster)	0.15	0.14	0.14
First child born before marriage (% in cluster)	0.20	0.17	0.17
Polygamous (% in cluster)	0.14	0.14	0.14
N	19,702	10,374	9,365

Source: Authors' calculations from the Demographic and Health Surveys 1994, 1999, 2005 and 2010

Notes: Unweighted statistics. The data covers children aged 0-59 months at survey time, with a maximum of 19,702 observations (for some outcomes, the number of observations slightly varies due to data availability). The sub-sample in column 2 corresponds to 0-59 months children living with both parents, and the sub-sample in column 3 to 0-59 months children living with both parents, born in 1950 or later.

[#] Observed only if the mother is currently living with her child

[†] Observed only if the father is currently living with his child

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3. The Reform and its Impact on Education

3.1. The 1980 Education Reform

Before 1980 when the United Kingdom officially recognized the Independence of Zimbabwe, there were enormous inequalities in education between Whites and Blacks. For Whites, who represented 3.5% of the population, education was free and compulsory until the age of 15 and admission to secondary school was automatic after the pupils passed their primary school final exam [Dorsey, 1989]. However, education was neither free nor compulsory for Blacks, who faced considerable selection at each grade. As a result, in the 1970s, only 4% of Black pupils were in secondary school: the analogous figure was 43% for White pupils [Dorsey, 1989]. There was also inequality between boys and girls. In 1975, the girl/boy ratio was 85% in primary school and 71% in secondary school (World Bank [2022], as reported in Appendix Table A.1).

The first Black majority government – led by the Zimbabwe African National Union (ZANU) party – came to power with Independence in 1980. Education was one of its top priorities and the new Constitution declared education as a fundamental human right (Education Act 2004). From 1980 on, the Government launched a vast reform to raise school attendance and the education of every child [Colclough et al., 1990]. This expansion was universal as it concerned both girls and boys and covered the whole country. The main policy changes took place in 1980 and can be summarized as follows:

- Primary education became free and compulsory for all pupils. Given the official duration of primary education, all children would leave school with at least 7 years of education.
- Admission to secondary school became automatic for all pupils, whatever their performance in the primary-school final exam. Secondary education remained paying.
- Age-restrictions were removed to allow older children to re-enter school.
- The government changed the school zoning system that gave Whites access to the best schools; it also introduced double-session schooling in almost all urban schools and some rural ones.

The reform took place in 1980, and was accompanied by an increase in the supply of schools

and teachers in the following years. The Government reconstructed all schools that had been destroyed during the war and built new primary and secondary schools, in particular in marginalized areas and disadvantaged urban centers [Kanyongo, 2005]. New teachers were recruited. The World Development Indicators statistics [World Bank, 2022] show that there has been a huge jump in the number of primary-school teachers between 1980 and 1985 and an even larger jump in secondary-school teachers (from 3,782 in 1980 to 19,507 in 1985). This implies that the pupils to teachers ratio remained quite stable at around 40 in primary schools and 28 in secondary schools. Lastly, government expenditure on education rose sharply around the time of the reform, from 2.5% of GDP in 1980 to 12.5% in 1990.

3.2. Parental education and exposure to the reform

The reform has affected children of primary-school and secondary-school age. It aimed to increase access to primary education and facilitate entry to secondary school, theoretically at age 13 as primary education lasts seven years.¹⁰ The exposure to the reform has been used in previous analyses as it provides exogenous variation in educational attainment [Agüero and Bharadwaj, 2014; Croke et al., 2016; De Neve and Subramanian, 2017; Grépin and Bharadwaj, 2015; Makate and Makate, 2018]. In all these analyses, the cohorts born in 1967 or later are defined as exposed as they were 13 or younger in 1980. The cohorts born in the period 1963-1966 are considered slightly differently across the articles (excluded, control or partially exposed).¹¹ Consequently, we define as being exposed to the Education Reform all individuals who were 15 or younger in 1980, in other words all individuals born in or after 1965. This definition includes the individuals who were 13 or younger in 1980 (i.e. fully exposed) and those who were 14 and 15 in 1980 as the policy allowed over-age individuals to re-enter school (they are thus partially exposed). We will discuss the robustness of our results to the inclusion of the latter in the exposed group as a robustness check.

In our sample, 89% (70%) of the children have mothers (fathers) who were exposed to the reform (Table 2, Panel A). Mothers exposed to the reform have an average of 8.2 years of education, versus 4.8 years for those not exposed (columns 2 and 3). 76% of

¹⁰ Up to 1986, children started primary school at age 7 [World Bank, 2022].

¹¹ Agüero and Bharadwaj [2014] and Grépin and Bharadwaj [2015] restrict their sample to the individuals who were aged 9-20 in 1980. Agüero and Bharadwaj [2014] define the treatment variable taking on the value of 1 for individuals whose age in 1980 is less than or equal to 15, and 0 otherwise. Grépin and Bharadwaj [2015] consider women who were aged 13 and younger in 1980 to have been fully exposed to the policy, women aged 14 and 15 in 1980 to have been partially exposed, and women aged 16 or older in 1980 the control group. Croke et al. [2016] and De Neve and Subramanian [2017] do not exclude the partially exposed. The only difference between their two definitions is that those born in 1963 appear in the "partially exposed" group in Croke et al. [2016] and in the control group in De Neve and Subramanian [2017].

the exposed mothers (37% of non-exposed mothers) completed primary school, and 59% attended secondary school (18% of the non-exposed). On average, fathers exposed to the reform had 9.4 years of education versus 6.9 years for those not exposed. 85% of the fathers exposed to the reform (column 4) completed primary school versus 59% of those not exposed (column 5), and 74% attended secondary school versus 38% of the non-exposed. The impact of the reform is then about three to four additional years of education for both fathers and mothers. Fathers had much more education than mothers before the reform, and this gender difference remains after the reform.

Table 2. Summary statistics - Parents' exposure to the reform and education levels

	(1)	(2)		(3)		(4)		(5)	
	Analytical sample	<i>Mother</i>		<i>Father</i>		<i>Mother</i>		<i>Father</i>	
		Exposed	Not Exposed	Exposed	Not Exposed	Exposed	Not Exposed	Exposed	Not Exposed
Panel A. Exposure to the 1980 Education Reform									
Mother exposed	0.89	1.00	0.00	1.00	0.63	1.00	0.63	1.00	0.63
Father exposed	0.70	0.79	0.01	1.00	0.00	1.00	0.00	1.00	0.00
Panel B. Education									
Years of education (mother)	7.82	8.19	4.82	8.46	6.28	8.46	6.28	8.46	6.28
Complete primary at least (mother)	0.71	0.76	0.37	0.78	0.55	0.78	0.55	0.78	0.55
Attended secondary school (mother)	0.54	0.59	0.18	0.62	0.36	0.62	0.36	0.62	0.36
Years of education (father)	8.64	8.94	6.20	9.36	6.92	9.36	6.92	9.36	6.92
Complete primary at least (father)	0.77	0.81	0.51	0.85	0.59	0.85	0.59	0.85	0.59
Attended secondary school (father)	0.63	0.68	0.25	0.74	0.38	0.74	0.38	0.74	0.38
N	9,365	8,335	1,030	6,599	2,766	6,599	2,766	6,599	2,766

Source: Authors' calculations from the Demographic and Health Surveys 1994, 1999, 2005 and 2010

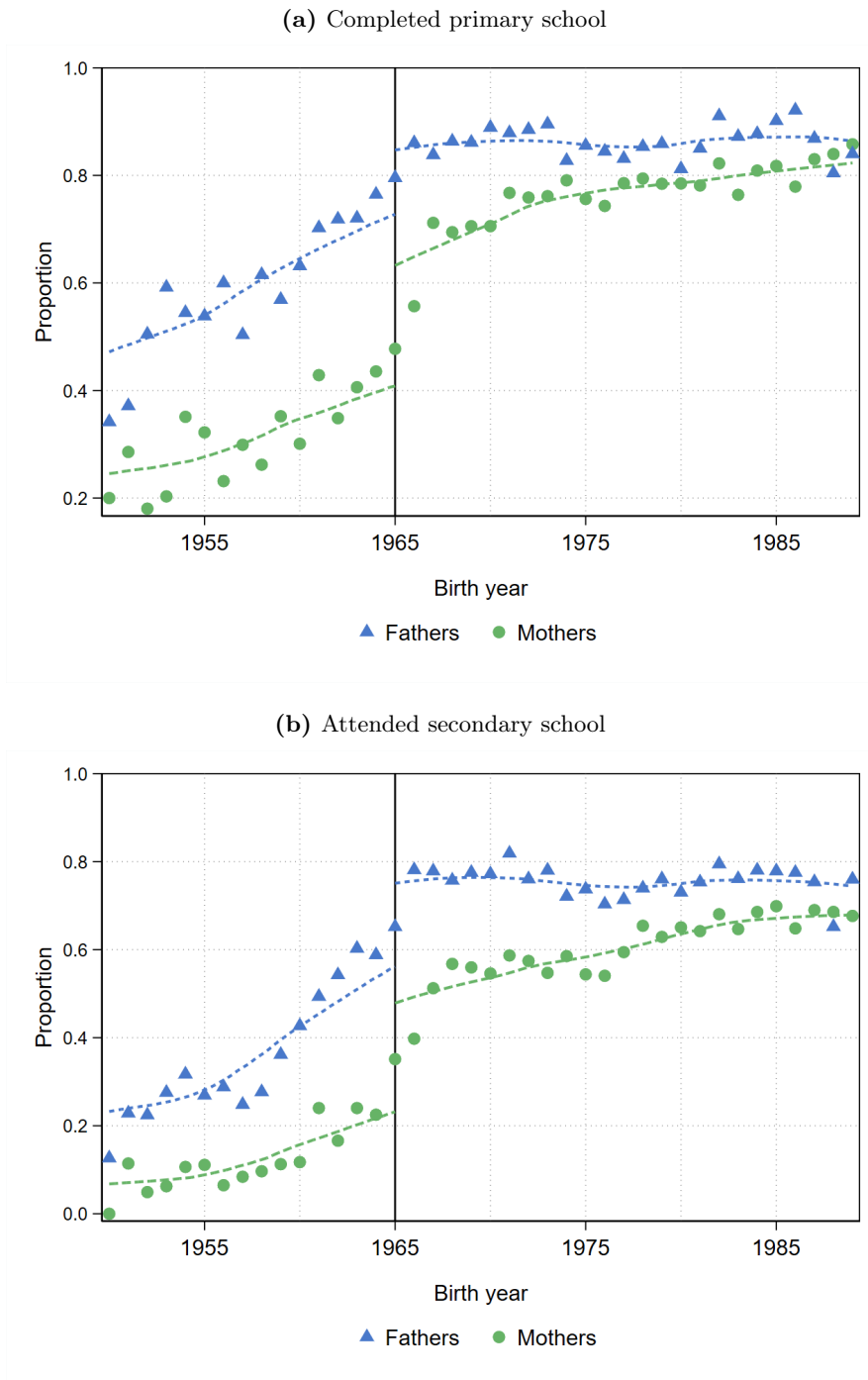
Notes: Unweighted statistics. The data covers children aged 0-59 months at survey time. The analytical sample (column 1) corresponds to 0-59 months children living with both parents born in 1950 or later. Exposed mothers and fathers are those born in or after 1965.

Figure 1 depicts the proportion of mothers and fathers in each birth cohort who completed primary school (Figure 1(a)), who attended secondary school for at least one year (Figure 1(b)), and their respective average number of years of education (Figure 1(c)). The vertical line corresponds to the 1965 cohort, that is the first cohort exposed to the Education Reform.

Two main features emerge from these figures. First, as shown in Figures 1(a) and 1(b), the reform resulted in an expansion of pupils completing primary education, and attending secondary school. The number of years of education in Figure 1(c) is also larger for those born in or after 1965 compared to those born before 1965. Second, school attainment started to rise even for cohorts born before 1966 for all the three measures of education

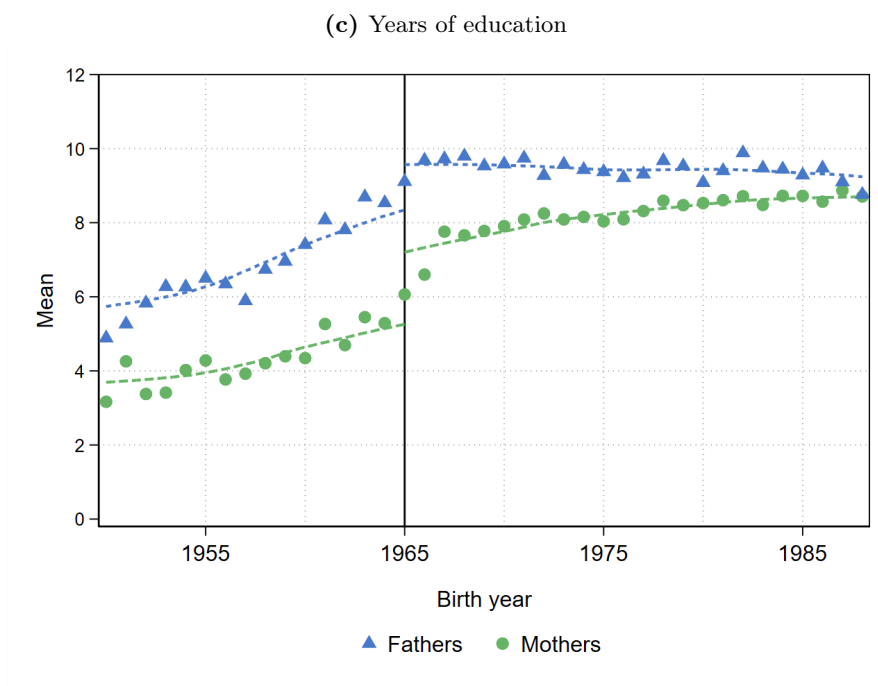
(Figures 1(a)-(c)). Although schooling was not compulsory for them, they might have benefited from age restrictions to school enrollment being lifted: these cohorts were affected via easier school access after 1980.

Figure 1. Mother's and father's education by birth year



continued →

Figure 1 (*continued*). Mother's and father's education by birth year



Source: Authors, based on Demographic and Health Surveys 1994, 1999, 2005 and 2010

Notes: The figure represents the average educational attainment of fathers and mothers depending on their year of birth, in terms of (a) probability of having completed primary school, (b) probability of having attended secondary school, and (c) number of years of education completed. The vertical lines represent the first cohort exposed to the 1980 Education Reform. The unit of observation corresponds to a mother (resp. a father) born in or after 1950.

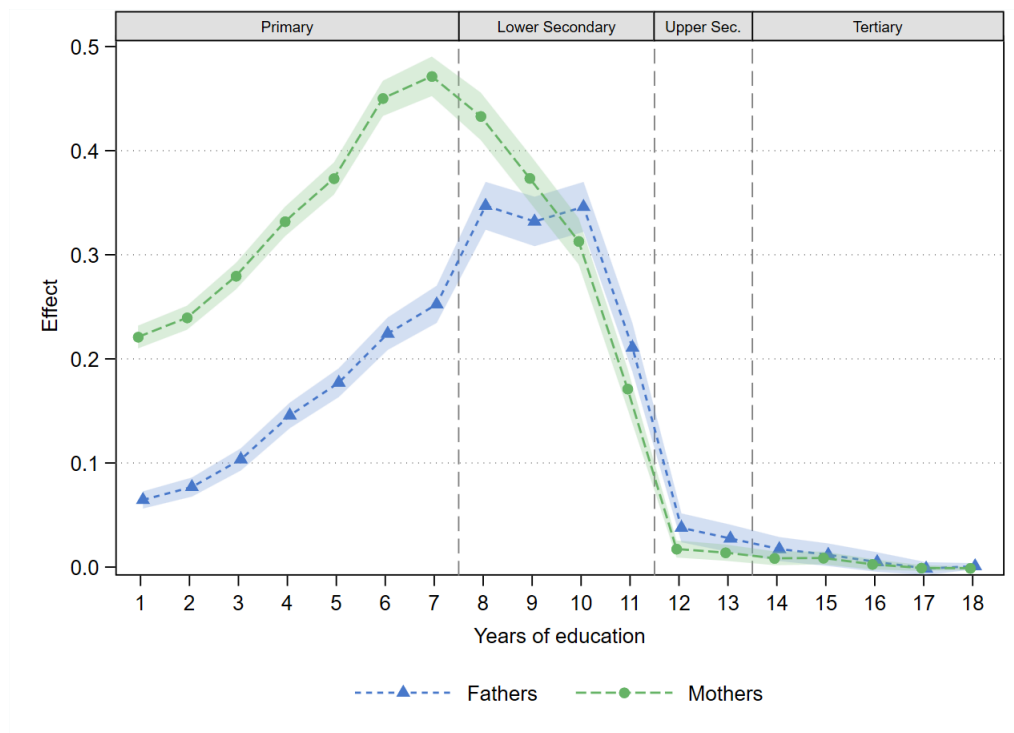
Overall, the increase in education thus applied to both sexes, even though it is more pronounced for mothers. Fathers benefited from an easier access to secondary school as they were already more likely to be enrolled in primary school than mothers.

We estimate the effect of being exposed to the reform on each additional year of education. In Zimbabwe, primary education lasts seven years and secondary education six years (including two years of lower secondary education and four years of upper secondary education), so that pupils completing both cycles have 13 years of education. Figure 2 displays the effect of exposure to the 1980 reform on the probability to reach at least a certain number of years of education, by gender from 1 to 18 years.¹² For both mothers and fathers, the reform had a considerable impact on attendance to primary and lower secondary school while the impact of the reform on the probability to reach upper secondary and tertiary school is very small and fails to be significantly different from 0 for reaching at least 16-18 years of education. Exposure to the reform increased attendance to all primary and lower secondary school levels. For mothers, the highest effects are observed on the probability to

¹² Coefficients come from the estimation of several linear probability models, separately by gender.

reach at least 6 (+45 percentage points), 7 (+47 percentage points) and 8 (+43 percentage points) years of education, i.e. the end of primary school and the beginning of secondary education. For fathers, the reform has the highest estimated effects on the probability to reach at least 8, 9 and 10 years of education (+35, +33, and +35 percentage points), i.e. lower secondary education.

Figure 2. Reform exposure and educational attainment



Source: Authors, based on Demographic and Health Surveys 1994, 1999, 2005 and 2010

Notes: The figure represents the effect of exposure to the 1980 Education Reform on the probability to have reached at least the corresponding number of years of education and 95 percent confidence intervals. Each coefficient is estimated from a separate linear probability model. The unit of observation corresponds to a mother (resp. a father) born in or after 1950.

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4. Econometric Specification

We estimate the joint impact of father's and mother's education on a number of child health investments and child health outcomes. Given the way in which the Demographic and Health Surveys are collected (as described above), this is only possible when the child lives with both parents. Our econometric strategy therefore tackles three econometric issues: *(i)* the endogeneity of father's and mother's education; *(ii)* marital education sorting (i.e. homogamy); and *(iii)* selection into coresidence, as the sample of children who live with both parents may be non random.

4.1. The endogeneity of education

In the child's outcome equation, father's and mother's education are likely endogenous, leading to inconsistent estimates of the impact of education on health outcomes. Unobservable parental characteristics (such as time preference, ability and intrinsic motivation) may similarly influence their investment both in their own human capital (education) and in the health of their children. In addition, parental education is correlated with parental health status, and healthy parents are more likely to have healthy children. Not controlling for parent's own health status can then lead to a second source of endogeneity bias.

As shown in Figure 1, free and compulsory primary education as well as easier access to secondary education brought about an exogenous rise in educational attainment. Fathers and mothers born in or after 1965 (i.e. who were 15 or younger in 1980, or were not yet born) were young enough to benefit from the 1980 Education Reform, which substantially enlarged their schooling opportunities, as opposed to those born before. The exogenous variation in education levels due to the age-specific nature of the reform can be exploited using two different frameworks to tackle the endogeneity of parents' education.

One could use the regression discontinuity (RD) approach in a fuzzy design, as in [Grépin and Bharadwaj \[2015\]](#), the only difference being that we have two running variables that partially determine mother and father education levels: both the mother's and father's birth year. Using the discontinuity in the probability to attend secondary school and in the average number of years of education due to the education reform for those born in 1965, observed in Figure 1, it would amount to implement a double regression discontinuity approach (as in [Stancanelli and Van Soest, 2012](#) or [Müller and Shaikh, 2018](#) who study retirement decisions within couples). However, this approach is not suitable in our case. Indeed, the RD approach provides a local estimate of the effect of education on outcomes,

around the 1965 birth year threshold. The sample used for the estimates is restricted to observations that are close to this cut-off. However, by construction, the size of our sample is already small, and restricting it to parents born for example 10 years around the reform (which seems the largest credible bandwidth to implement a RD approach) would lead to a sample containing only 3,645 observations, which is too small to provide precise estimates. More importantly, the double RD approach imposes the use of the same bandwidth for fathers and mothers, meaning that we should run the estimates on mothers and fathers both born between, for example, 1955 and 1975. This would create a huge selection bias: this would lead to keep only couples of the very similar ages. Indeed, contrary to OECD countries where the age gap between spouses is quite low and such identical bandwidth can be used (as in [Lindeboom et al., 2009](#) in the United Kingdom for example), the age gap is much larger in Zimbabwe (7 years on average).

We therefore prefer to rely on the 2SLS procedure, with two first-stage regressions: one each for mother's and father's education using the individual exposure to the reform as the instrumental variable. Both first-stage equations are defined as follows:

$$\begin{cases} Educ_{iht}^M = \beta_0^M + \beta_1^M T_{iht}^M + \beta_2^M f(B^M - 1965) + X'_{iht} \theta^M + \delta_t + \varepsilon_{iht}^M & (1) \\ Educ_{iht}^F = \beta_0^F + \beta_1^F T_{iht}^F + \beta_2^F g(B^F - 1965) + X'_{iht} \theta^F + \delta_t + \varepsilon_{iht}^F & (2) \end{cases}$$

where i refers to the child ($i = 1, \dots, N$; N denotes the size of the analysis sample),¹³ h the household and t the survey year. M denotes child i 's mother and F the father.

We consider two alternative dependent variables as in the previous articles that studied the impact of the 1980 Education reform. $Educ^M$ and $Educ^F$ refer either to the number of years of education reported by child i 's mother and father respectively, or to dummies indicating whether the mother and father attended secondary school. The continuous variable is our preferred measure of education: indeed, the reform had an impact on the attendance to both primary and secondary school, hence this variable reflects the full effect of the reform.¹⁴

T_{iht}^M (T_{iht}^F) equals one if the mother (father) was born in or after 1965, i.e. was 15 or younger in 1980, and zero otherwise. The direct impacts of the reform are given by β_1^M

¹³ We find the same results if we estimate these equations on the initial sample, i.e. the sample not restricted to having both mothers and fathers currently living with the observed child. The coefficients are stable in size and significance. The F-statistics are even greater than those obtained over the restricted sample. These results are shown in Appendix Table C.2.

¹⁴ The continuous education variable is strictly positive for almost all parents in the sample: only 3% of fathers and 5% of mothers have no education. This small share of zero values justifies our use of ordinary least squares (OLS) regressions in the first stage.

for the mother and β_1^F for the father. $f(B^M - 1965)$ (in Equation 1) and $g(B^F - 1965)$ (in Equation 2), where B is the parent's birth year, denote linear trends before and after the 1965 birth year threshold. As in Agüero and Bharadwaj [2014], the linear trends are included to model the different trends in education for the cohorts born before and after 1965, as suggested in Figure 1. More precisely, $f(B^M - 1965)$ is defined as $\beta_{2,1}^M(B^M - 1965)\mathbb{1}_{B^M \geq 1965} + \beta_{2,2}^M(B^M - 1965)\mathbb{1}_{B^M < 1965}$ and similarly for the father with $g(B^F - 1965)$. We will consider two versions of our first-stage regressions. The first one does not include these linear trends and the only excluded instrument is T_{iht}^M (or T_{iht}^F for the father). In the second one, the two trends for the mother will be added to the mother's first-stage (Equation 1) and the two trends for the father will be added to the father's first-stage (Equation 2). The evolution shown in Figure 1 is rather smooth for both mothers and fathers, and both before and after the birth year 1965 except for the fathers' attendance to secondary school (in Figure 1(b)). We explored the linear spline specification that would best fit the latter evolution of education across birth cohorts. Such specification does not improve our first-stage regressions (coefficients are often non significant, and Kleibergen-Paap F-statistics are lower) and yields to similar 2SLS results.¹⁵

X_{iht} is a vector of control variables that will be included in the second stage equations (such as child sex and age) and that have to be included in the first-stages for identification purposes. We also control for survey year fixed effects, δ_t . Given our econometric strategy, other variables also need to be included in these first-stage regressions: these will be described in Section 4.4, where the final model is set out.

For the instrumental variable to be valid, it has to be correlated with the observed level of education and not correlated with the error term of the second-stage equation (Equation 9 below). The first correlation is discussed when presenting the results from the first-stage estimations in Section 5.1. The second requirement is the exclusion restriction: the instrument should have no effect on the outcome other than through the first-stage channel. The exclusion restriction here is that having a parent being born a year of birth before or after 1965 does not affect child health through any other mechanisms than parental education. We may worry if the difference between those born before or after 1965 is related to any structural change due to Independence, and in particular to the health reform that was implemented in 1980. Although there is no impact evaluation of the health reform, it has been documented by Bassett et al. [1997] and Hecht et al. [1993]. The main progresses of the health care sector occurred between 1980 and 1985 in rural areas, and

¹⁵ Results reported in Appendix Tables C.3 and C.4.

then stagnated, mostly because the share of budget devoted to health by the government decreased [Bassett et al., 1997].¹⁶ Overall, it leads to a strong increase in immunization coverage and contraception use, average life expectancy increased and infant mortality declined [Hecht et al., 1993].

The health-care reform likely improved individuals' health, whatever their age. The parents in our sample, who were born before 1980 or soon after, were affected by this reform via an increased supply of health care and a better immunization program [Grépin and Bharadwaj, 2015]. Parents have been exposed to these improvements during a short period of time (between 1980 and 1985) and at different ages (ie. in 1985, parents exposed to the education reform were between 1 and 20 years old while the non-exposed were between 21 and 35 years old). Therefore, exposed parents may have benefited more from the improved health care system since their childhood, while non-exposed parents are more likely to have benefited from the increased access of the health care system during their fertile ages, for the births of their first children (not in our sample), but also for the subsequent ones, born between 1990 and 2011, from our sample. Both exposed and non-exposed parents may have benefited from this health care reform, at different times in their life cycle. This implies that the health reform did not benefit specifically to those born before or after 1965. This is confirmed by our data. If we estimate the effect of being exposed to the 1980 Education Reform on mother's height and on her probability of being underweight at survey time in a reduced-form model (Equation 1), we do not find any significant effect.

4.2. Assortative mating

Marital educational sorting may be an issue in our model. In the analytical sample, 86% of children whose mothers have completed primary school have a father who also completed primary school, and 82% of children whose mothers have attended secondary school have a father who also attended secondary school. There is consequently substantial correlation between mother's and father's years of education: 0.64.

Note however that assortative mating does not appear with the exposure to the education reform as the levels are already high for the parents born before 1965. Indeed 65% of the children have parents with the same level of education (both having no education, primary or at least some secondary) when parents are both unexposed, while this proportion raises

¹⁶ Before independence, the health care system was urban-centered and focused on curative rather than preventive care. The government adopted a policy of "health-for-all" in order to increase geographical and financial access to care [Bassett et al., 1997]. More precisely, it increased the number of rural health centers, midwives and nurses were trained and deployed in previously undeserved rural areas, rural water and sanitation were improved and low-income Zimbabwean were entitled to free health services [Hecht et al., 1993].

to 72% of children when both of them are exposed. If we estimate Equations 1 and 2 with a dummy for assortative mating as the dependent variable in a reduced-form approach, the effect of being exposed to the reform is only significant for fathers. This suggests that educated men are more likely to marry educated women as there is now more educated women in the marriage market.

If women and men with similar education tend to live with or marry each other, the unobservable characteristics that explain mothers' education (such as intrinsic motivation, time preference) may well be correlated with unobservables that explain fathers' education.

In our final model, mother's and father's education are therefore estimated simultaneously, taking into account the correlation between the residuals of both equations (ε_{iht}^M and ε_{iht}^F). We find a positive and very-significant correlation (0.56 for the number of years of education and 0.41 for secondary-school attendance) between these residuals: fathers and mothers with similar intrinsic incentives or aspirations towards human-capital investment tend to live and have children with each other.

4.3. Selection into coresidence

Of the 19,702 sampled children aged 0-59 months, 52.7% live with both parents, 13.5% with neither, 1.3% with their father only and 32.5% with their mother only. More details on the different possible cases are presented in Appendix B. The percentage of children living with both parents is fairly stable over the successive survey rounds: 52.8% in 1994, 52% in 1999, 53% in 2005 and 52.7% in 2010. This low percentage of children living with both parents is also found in other countries, although Zimbabwe has one of the lowest percentages in Sub-Saharan Africa according to Pilon and Vignikin [2006].¹⁷

Appendix Table B.2 shows the difference between children who coreside with both parents and children who do not coreside with both parents. The figures suggest that except for the number of injections, the difference in terms of child health outcomes is significant but is small in magnitude. There is no difference in child's sex and a slight difference in age (those in the excluded sample are 0.3 years older than those in the analytical sample). The characteristics of the household in which the child lives significantly and strongly differ: the children who coreside are much more likely to live in urban areas (32%) and in rich households (41%) compared to those who do not live with both parents (18% in urban areas and 26% in rich households). One might extrapolate then that the levels of

¹⁷ Pilon and Vignikin [2006] document large disparities in Sub-Saharan Africa: in Namibia, only 26% of children below the age of 15 live with both parents, 33% in South Africa, 50% in Zimbabwe and Rwanda, 65% in Benin, 71% in Ethiopia and 78% in Burkina Faso.

education might also differ across both groups, as education is associated with urbanisation and wealth. It is likely that the (unobserved) level of education of the parents who do not coreside with their children is lower than the level of education of those who coreside with their children.

Our estimations may suffer from selection bias due to the coresidence restriction, for which we need to correct. The unit of analysis here is all children 0-59 months living in sampled households, and selection bias is addressed via Heckman [1976]’s two-step procedure.¹⁸ We estimate two Probit selection equations, one each for the mother and father. Let $Coreidence_{iht}^M$ ($Coreidence_{iht}^F$) be a dummy for child i living with her mother M (father F) and zero otherwise. We have:

$$Coreidence_{iht}^M = \begin{cases} 1 & \text{if } Coreidence_{iht}^{*M} > 0 \\ 0 & \text{otherwise} \end{cases} \quad \text{and} \quad Coreidence_{iht}^F = \begin{cases} 1 & \text{if } Coreidence_{iht}^{*F} > 0 \\ 0 & \text{otherwise} \end{cases}$$

where $Coreidence_{iht}^{*M}$ and $Coreidence_{iht}^{*F}$ are latent variables defined as follows:

$$\begin{cases} Coreidence_{iht}^{*M} = \alpha_0^M + Z'_{iht}\alpha_1^M + X'_{iht}\phi^M + \theta_t + \mu_{iht}^M & (3) \\ Coreidence_{iht}^{*F} = \alpha_0^F + Z'_{iht}\alpha_1^F + X'_{iht}\phi^F + \theta_t + \mu_{iht}^F & (4) \end{cases}$$

As before, i indexes the child ($i = 1, \dots, N_T$, where N_T denotes the size of the initial sample), h the household and t the year of the survey. The models include the child characteristics (X'_{iht}) from the outcome equation as described below, and survey year fixed effects, θ_t .

The estimation of these selection equations requires exclusion restrictions, i.e. variables that influence coresidence but have no direct effect on the outcome. We use community-level variables denoted by Z_{iht} : the proportion of sampled women who gave birth to their first child before getting married, the proportion of respondents who are currently divorced, separated or widowed, and the proportion of polygamous unions in each community.¹⁹ These community-level variables are proxy variables for the social norms that

¹⁸ We estimate a selection equation to explain why children are or are not currently living with each parent. Only 2% of mothers and 5% of fathers of sampled children are dead. Fathers/mothers who do not live with their child are therefore mainly parents who have somehow decided not to live together: divorcees, migrants who quit the household and those who have entrusted their child to somebody else’s care. We hypothesise that all of these potential (unobserved) reasons can be summarized by one single selection equation, a hypothesis that is of course debatable. However we do not impose these reasons to be the same for mothers and fathers. And we see that the proportion of children who live with their mother only is much higher than the proportion of children who live with their father only. We estimate separately the two probabilities.

¹⁹ If we add the female and male national AIDS-related mortality rates (from UNAIDS data) averaged between the child birth year and the survey year, these two variables are not significant in the selection equation for mothers and in that for fathers. Second-stage results, however, are robust to this inclusion.

prevail in the community regarding household living arrangements. There are 1,262 different communities in the entire sample, each of which is large enough to be distinct from the individual considered so that the norms influence the parents' behaviors and not the other way round.²⁰

These variables are likely to satisfy the exogeneity assumption. To illustrate, consider the proportion of women who gave birth before getting married. If a woman gives birth before marriage, it is likely that she does not live with the father of the child until she gets married with the father. The pregnancy before marriage might be more or less prevalent across communities depending on the acceptance of the communities for such a practice, or depending on the consequences the communities put under the mother and the child in such circumstances. The social norms in the community might influence the individual probability of having a child outside marriage and in turn the probability of coresidence with one's child. The social norms regarding living arrangements have no direct impact on child health except through its effect on the living arrangement of his own family. Same arguments prevail for the other community-level variables as exclusion restrictions for the selection into coresidence equations.

4.4. Final specification

Our final specification aims to identify the causal effect of parental education on a number of child-health outcomes. We address education endogeneity via the policy reform that allowed some parents to enroll and stay longer in school when they were school-aged. We do so via two-stage least squares (2SLS) estimation. Selection into coresidence is taken into account using Heckman [1976]'s two-step procedure, and marital homogamy using correlated error terms between fathers' and mothers' education equations. We use the procedure described in Wooldridge [2010] to estimate a full model that takes all these issues into account in the five-equation model described below.

These variables must account for the incidence of the HIV/AIDS epidemic in Zimbabwe over the period. That said, the orphanhood due to AIDS-related death is unlikely to drive the selection in coresidence as the proportion of orphans is small in the initial sample. In addition, the exclusion restriction might be violated if a high number of AIDS-related deaths switch financial, material and human resources away from the delivery and child care units of health facilities.

²⁰ The number of observations used to compute each proportion slightly differ depending on the sample we need. To compute the proportion of women who gave birth before getting married, the sample is all women who had at least one child. On average the proportion is computed over a sample of 14 women per cluster (median of 13, minimum 2 and maximum 47). To compute the proportion of separated respondents, the sample is composed of all individuals that have ever been in union, and comprised on average 25.7 respondents per cluster, with a median value of 24, a minimum of 4 and a maximum of 62. The proportion of polygamous unions is computed over the sample of married women and distinguishes whether they are married to a monogamous or polygamous husband. The sample used is of 13.9 observations per cluster on average (median 13, minimum 2, maximum 47).

$$\left\{ \begin{array}{l} \text{Coresidence}_{iht}^{*M} = \alpha_0^M + Z'_{iht}\alpha_1^M + X'_{iht}\phi^M + \theta_t + \mu_{iht}^M \quad (5) \\ \text{Coresidence}_{iht}^{*F} = \alpha_0^F + Z'_{iht}\alpha_1^F + X'_{iht}\phi^F + \theta_t + \mu_{iht}^F \quad (6) \\ \text{Educ}_{iht}^M = \beta_0^M + \beta_1^M T_{iht}^M + \beta_2^M f(B^M - 1965) + \beta_{3,M}^M \lambda_{iht}^M + \beta_{4,F}^M \lambda_{iht}^F + X'_{iht}\theta^M \\ \quad + \delta_t + \varepsilon_{iht}^M \quad (7) \\ \text{Educ}_{iht}^F = \beta_0^F + \beta_1^F T_{iht}^F + \beta_2^F g(B^F - 1965) + \beta_{3,M}^F \lambda_{iht}^M + \beta_{4,F}^F \lambda_{iht}^F + X'_{iht}\theta^F \\ \quad + \delta_t + \varepsilon_{iht}^F \quad (8) \\ H_{iht} = \gamma_0 + \gamma_1^M \text{Educ}_{iht}^M + \gamma_1^F \text{Educ}_{iht}^F + \gamma_2^M \lambda_{iht}^M + \gamma_2^F \lambda_{iht}^F + X'_{iht}\Gamma + \eta_t + \xi_{iht} \quad (9) \end{array} \right.$$

In Equation 9, H_{iht} is a child health investment or a child health outcome, and Educ_{iht}^M and Educ_{iht}^F are variables for mother's and father's level of education respectively.²¹ Child health is measured using the different outcomes, presented in Section 2.2.

The outcome equation includes some exogenous variables that also appear in the selection and first-stage equations: X_{iht} includes child characteristics (age and sex) and we control for survey year fixed effects. In the main models, we do not include variables such as household wealth quintiles, urban location, or dummy variables for province of residence as they are very likely to be endogenous. However we consider them in the section that discusses the mechanisms either as the outcome variables or as additional control variables.

As described in Wooldridge [2010], we test and correct for any selection bias by adding the inverse Mills ratios from the Probit estimation of Equations 5 and 6 to both the first-stage and outcome equations (respectively Equations 7, 8, and 9). The two Inverse Mills ratios are λ^M and λ^F , and a test for selection bias is $\gamma_2^M = 0$ and $\gamma_2^F = 0$ in Equation 9. The sign of γ_2^M (resp. γ_2^F) captures the sign of the correlation between μ_{iht}^M (resp. μ_{iht}^F) and ξ_{iht} . In addition, the sample selection bias, if any, would show that selection into coresidence has a direct impact on child' health, it does not necessarily imply that the productivity of each factor in the production function of health is impacted by the selection. In particular, the estimates of γ_1^M and γ_1^F may be constant with and without correcting for selection problem.

²¹ We are unable to include an interaction between mother's and father's education, to test for complementarity between the two. There is no child who are born from an unexposed mother and an exposed father.

Equations 5 and 6 are estimated separately via Probits, and Equations 7 to 6 are estimated simultaneously using linear-probability models. This joint estimation allows us to take into account any correlation between the error terms: ε_{iht}^M and ε_{iht}^F may be correlated due to assortative matching; ε_{iht}^M and ξ_{iht} as well as ε_{iht}^F and ξ_{iht} may also be correlated if mothers (fathers) have unobserved characteristics that influence both their choice of education and their ability to improve their child's health. We do not consider any correlation between μ_{iht}^M and ε_{iht}^M , μ_{iht}^F and ε_{iht}^F or μ_{iht}^M , μ_{iht}^F and ξ_{iht} , as these error terms refer to samples of different sizes. Note that standard errors are clustered at the enumeration area level in all equations, because proportions computed at the enumeration area level are included in the set of right-hand side variables in Equations 5 and 6. Lastly, selection Equations 5 and 6 have to be estimated using the whole initial sample. In our baseline analysis, Equations 7, 8 and 9 are estimated on the analytical sample.

*
* *

5. Results

5.1. First-stage results

The estimation results of both first-stage Equations 1 and 2 are presented in Table 3.²² To improve readability, coefficients of X_{iht} are not reported in this table.²³ Panel A and B differ according to the education variable considered: the number of years of education in Panel A or the dummy for having attended secondary school in Panel B. In both panels, columns 1 and 2 refer to the mothers' first-stage regression and columns 3 and 4 to the fathers'. In columns 1 and 3, we do not control for linear trends for the birth years before and after 1965: we impose $\beta_2^M = 0$ in Equation 1 and $\beta_2^F = 0$ in Equation 2. In columns 2 and 4, the additional pre- and post-1965 linear trends are added.

Exposure to the reform has a tremendous impact on educational attainment. In Panel A, the average number of school years is 2.95 years higher for mothers exposed to the

²² In practice, as the first-stage and outcome equations are estimated simultaneously, we have as many first-stage regressions as outcomes. Given that the sample size varies slightly between outcomes, depending on the number of missing values, the results from the first-stage estimations may also vary. However, this turns out not to be the case: the results are very similar across outcomes and sample sizes. In this section, and in the chapter in general, we only report and comment on the first-stage regressions for the analytical sample.

²³ The final specification, described in Section 4.4, also includes the inverse Mills ratio, that corrects for possible selection bias in the outcome equation. These ratios also have to be included as a right-hand side variable in the first-stage equations; in Table 3, there is no correction for selection, but the coefficients on the instrumental variables are stable in size and significance when including this correction (see the Benchmark Panel of Appendix Table D.1).

reform compared to the non-exposed (column 1), with a corresponding figure of 2.35 years for fathers (column 3). The reform therefore had a huge impact on parents' education levels and a greater effect on mothers than fathers (and significantly so at the 1% level).²⁴ Adding pre- and post-1965 linear trends decreases the impact of exposure to the reform, with a much smaller coefficient in column 2 (column 4) than in column 1 (column 3). The coefficients are however still significant, and larger for mothers (+2.14 years for mothers, +0.92 years for fathers).

Table 3. First-stage equations

	(1)	(2)	(3)	(4)
	Mother's education		Father's education	
	<i>Years of education</i>			
Panel A.				
Exposed	2.955*** (0.168)	2.143*** (0.285)	2.351*** (0.121)	0.923*** (0.195)
Pre-reform trend		0.147*** (0.046)		0.249*** (0.021)
Post-reform trend		0.019** (0.008)		-0.029*** (0.008)
N	9,337	9,337	9,288	9,288
Mean of dep.	7.82	7.82	8.64	8.64
Kleibergen-Paap F-Statistic	310.31	116.25	374.84	191.28
Effective F-Statistic [Montiel Olea and Pflueger, 2013]	310.31	126.86	374.84	210.37
	Mother's education		Father's education	
	<i>Attended secondary school</i>			
Panel B.				
Exposed	0.340*** (0.019)	0.277*** (0.032)	0.334*** (0.016)	0.140*** (0.025)
Pre-reform trend		0.011** (0.005)		0.033*** (0.003)
Post-reform trend		0.002 (0.001)		-0.003** (0.001)
N	9,343	9,343	9,315	9,315
Mean of dep.	0.54	0.54	0.63	0.63
Kleibergen-Paap F-Statistic	322.06	117.27	442.45	224.49
Effective F-Statistic [Montiel Olea and Pflueger, 2013]	322.06	111.34	442.45	225.14

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between columns (1)-(2) and (3)-(4) due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for control variables of the second stage equation (child's sex and age). The Kleibergen-Paap and Montiel Olea and Pflueger [2013] F-statistics of excluded instruments are obtained from the estimation of Equations 1 and 2. There is no correction for selection into coresidence.

* $p < .10$; ** $p < .05$; *** $p < .01$

²⁴ The pre-reform level of education differs between mothers and fathers: see the descriptive statistics in Table 2 and Figure 1.

Our first-stage regressions are convincing. In columns 1 and 3, the Kleibergen-Paap F-statistics on excluded instruments (exposure to the reform) indicate that our instruments are not weak ($F=310.3$ for mothers; $F=374.8$ for fathers). The Kleibergen-Paap F-statistics decrease when the pre-reform and post-reform trends are added in the set of instrumental variables in columns 2 and 4, though are still large enough (116.3 for mothers and 191.3 for fathers). These conclusions remain unchanged when using the [Montiel Olea and Pflueger \[2013\]](#) effective F-statistic.

The same pattern is observed in Panel B, for attendance to secondary school. Exposure to the reform increases attendance to secondary school by 34 percentage points for mothers (column 1) and 33 percentage points for fathers (column 3). The inclusion of linear trends both decreases the impact of the reform (+28 percentage points for mothers in column 2, +14 percentage points for fathers in column 4), as well as the Kleibergen-Paap F-statistic for the excluded instruments.

Even though the Kleibergen-Paap F-statistics for the excluded instruments decrease when trends are included in the set of instrumental variables, we rely on these first-stage regressions in the rest of the chapter as it fits better the graphical relationship from the figure above, and allows controlling for the exposure duration²⁵.

5.2. The selection-equation estimation results

The selection-equation estimation results appear in columns 1 and 2 of Table 4. We find that the greater the proportion of sampled women who gave birth to their first child before getting married in each community and the higher the proportion of sampled respondents who are currently divorced, separated or widowed in each community, the lower the probability that the child lives with her mother (in column 1). Same results are found for coresiding with her father (in column 2). The size of the marginal effects being larger for coresiding with the father suggests that social norms regarding pregnancy before marriage and broken unions reduces even more the probability that the child lives with her father than that with her mother. We find that the proportion of polygamous households in each community increases the probability of living with their mother and decreases the probability of living with their father.

²⁵ Note that the second-stage estimates are unchanged when the dummy variable for being exposed to the reform is used as the unique instrumental variable.

Table 4. Selection equations for mothers and fathers

	(1)	(2)
	Mother present	Father present
Separated (% in cluster)	−0.220*** (0.036)	−0.682*** (0.065)
First child born before marriage (% in cluster)	−0.135*** (0.017)	−0.405*** (0.035)
Polygamous (% in cluster)	0.042* (0.023)	−0.113*** (0.042)
N	19,694	19,689
Mean of dep.	0.85	0.54
Correctly specified	56.09	59.18

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for control variables of the second stage equation (child's sex and age). The selection equations are estimated for all children present in sampled households. The table reports marginal effects.

* $p < .10$; ** $p < .05$; *** $p < .01$

5.3. Second-stage results

We use the analytical sample to jointly estimate the effect of father's and mother's education on several outcomes. Results are presented in Tables 5 and 6 where education is measured by the number of years of education and by attendance to secondary school respectively. In both tables, three estimates are shown: the OLS estimates (Panel A), the 2SLS estimates (Panel B) and the 2SLS estimates correcting for selection (Panel C). The results in Panel C come from our preferred specification that deals with all the estimation issues discussed above; this corresponds to the estimation of the final specification presented in Section 4.4. However, Panel A and B help us to understand whether and how much our results change when correcting for the endogeneity of education and selection.

In Tables 5 and 6, the OLS estimates (Panel A) show a highly significant correlation between education and health investments as well as all child health outcomes, both for the mother and the father. More education of the mother and the father is associated with more prenatal care and improved conditions of birth (i.e. a greater probability of the mother having attended at least four prenatal visits, of being born in a health facility and having a birth assisted by medical staff). Father's education, and to a lower extent, mother's education, are also associated with a better nutritional status (a lower probability to be stunted and wasted). Finally, in the last two columns, mother's education is associated with greater prevention behaviors (more injections of the vaccines, and a higher probability to

sleep under net for any education measure). Father's education is also positively correlated with prevention behaviors although not significantly when considering immunization and secondary school.

However, when the endogeneity of education is taken into account (Panel B), the effect of mother's education is not significant anymore, whatever the measure of education and the health investment or child health outcome considered. The education effect on health turning insignificant when instrumented is also found in [Jürges et al. \[2013\]](#) for example. On the contrary, the 2SLS point estimates of the effect of father's education remain positive and statistically significant on the three perinatal care outcomes when education is measured by having attended secondary school and in one perinatal care outcome when years of education are used. This difference suggests that attending secondary education impacts more than each additional year of education. Table 6 reveals that having a father who have attended secondary school increases the probability to have at least 4 prenatal visits by 14.4 percentage points, the probability of being born in a health facility by 15.8 percentage points and having a birth assisted by medical staff by 14.5 percentage points. Father's education also impacts the number of immunizations in column 6, an effect significantly different from zero at the 10 percent level for both education measures. Father's education has no more impact on nutritional status and on the likelihood that the child slept under bed net.

If we compare the size of the coefficients of father's education on prenatal and birth care in Panel A (OLS coefficients) and Panel B (2SLS coefficients), in most cases, the 2SLS coefficient is more positive than the OLS coefficient, suggesting that the effect is larger if the education levels are randomly distributed in the population. Only when estimating the effect of the number of years of education on birth in health facility and birth assisted by medical staff (columns 2 and 3 of Table 5), the size of the coefficients does not differ across the two models. When measuring the effect of attending secondary school on these two outcomes (columns 2 and 3 of Table 6), the instrumented coefficient is larger and more positive than the naive coefficient but not significantly so. The difference between OLS and 2SLS estimates of education on having at least four prenatal visits is significantly different from zero, and this is true for both measures of education (column 1 of Tables 5 and 6).

Table 5. The impact of mother's and father's education (years of education)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
Panel A. OLS							
Years of education ^M	0.015*** (0.002)	0.039*** (0.002)	0.039*** (0.002)	-0.009*** (0.002)	-0.001 (0.001)	0.050*** (0.014)	0.010*** (0.002)
Years of education ^F	0.007*** (0.002)	0.021*** (0.002)	0.022*** (0.002)	-0.005** (0.002)	-0.002* (0.001)	0.024* (0.014)	0.003* (0.002)
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Panel B. 2SLS							
Years of education ^M	-0.007 (0.010)	0.011 (0.011)	0.015 (0.011)	0.009 (0.010)	0.001 (0.005)	-0.070 (0.066)	0.008 (0.009)
Years of education ^F	0.018** (0.009)	0.010 (0.009)	0.008 (0.009)	0.000 (0.008)	0.005 (0.004)	0.101* (0.060)	0.001 (0.008)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.555***	0.555***	0.555***	0.555***	0.555***	0.555***	0.553***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Panel C. 2SLS with correction for selection into coresidence							
Years of education ^M	-0.007 (0.010)	0.012 (0.011)	0.016 (0.011)	0.009 (0.010)	0.001 (0.005)	-0.075 (0.065)	0.006 (0.009)
Years of education ^F	0.018** (0.009)	0.009 (0.009)	0.007 (0.009)	0.000 (0.008)	0.005 (0.005)	0.114* (0.060)	0.002 (0.008)
Inverse Mills Ratio ^M	0.373 (0.300)	1.637*** (0.334)	1.653*** (0.340)	-0.788*** (0.269)	-0.123 (0.131)	4.168** (1.835)	-0.340 (0.234)
Inverse Mills Ratio ^F	-0.116 (0.097)	-0.379*** (0.113)	-0.386*** (0.114)	0.204** (0.086)	0.034 (0.048)	0.501 (0.608)	0.042 (0.088)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.551***	0.551***	0.551***	0.551***	0.551***	0.551***	0.550***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age. Panel C also controls for the two inverse Mills ratios obtained from the two selection equations. $\rho_{(\varepsilon^M),(\varepsilon^F)}$ denotes the correlation between the residuals of the two first-stage equations.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table 6. The impact of mother's and father's education (attended secondary school)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
Panel A. OLS							
Attended secondary school ^M	0.096*** (0.014)	0.236*** (0.015)	0.243*** (0.014)	-0.048*** (0.013)	-0.004 (0.006)	0.413*** (0.086)	0.042*** (0.011)
Attended secondary school ^F	0.035** (0.014)	0.133*** (0.015)	0.137*** (0.015)	-0.030** (0.013)	-0.012* (0.007)	0.044 (0.089)	0.024** (0.011)
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Panel B. 2SLS							
Attended secondary school ^M	-0.064 (0.092)	0.047 (0.090)	0.082 (0.091)	0.088 (0.086)	0.012 (0.046)	-0.453 (0.592)	0.101 (0.082)
Attended secondary school ^F	0.144*** (0.054)	0.158*** (0.057)	0.145** (0.058)	-0.020 (0.051)	0.024 (0.028)	0.646* (0.376)	0.028 (0.048)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.400***	0.400***	0.400***	0.400***	0.400***	0.400***	0.399***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Panel C. 2SLS with correction for selection into coresidence							
Attended secondary school ^M	-0.070 (0.093)	0.047 (0.092)	0.082 (0.092)	0.090 (0.087)	0.013 (0.047)	-0.531 (0.580)	0.084 (0.084)
Attended secondary school ^F	0.144*** (0.055)	0.152*** (0.058)	0.139** (0.058)	-0.018 (0.052)	0.024 (0.028)	0.725* (0.375)	0.033 (0.049)
Inverse Mills Ratio ^M	0.393 (0.317)	1.618*** (0.335)	1.603*** (0.342)	-0.807*** (0.287)	-0.151 (0.145)	4.634** (1.953)	-0.476* (0.261)
Inverse Mills Ratio ^F	-0.110 (0.100)	-0.352*** (0.110)	-0.347*** (0.111)	0.204** (0.088)	0.043 (0.051)	0.344 (0.625)	0.082 (0.091)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.396***	0.396***	0.396***	0.396***	0.396***	0.396***	0.396***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age. Panel C also controls for the two inverse Mills ratios obtained from the two selection equations. $\rho_{(\varepsilon^M),(\varepsilon^F)}$ denotes the correlation between the residuals of the two first-stage equations.

* $p < .10$; ** $p < .05$; *** $p < .01$

Overall, our findings suggest that when endogeneity is controlled for, inequalities in child nutritional status are no longer due to differences in mother's education and father's education, while father's education significantly improves prenatal care and the conditions of birth.²⁶

Panel B of Tables 5 and 6 also reports the correlation coefficients between the residuals of both first-stage equations computed when estimating the system of equations. As already mentioned, we find a positive and very significant correlation: unobserved factors leading to father's and mother's education are strongly correlated (0.55 for the number of years of education, 0.40 for secondary-school attendance). Our results show a strong assortative mating among parents, that appears both in the correlation between observables (the correlation between mother's and father's years of education is 0.64) and between unobservables.

All these conclusions are maintained when selection into coresidence is taken into account (Panel C, Tables 5 and 6). In these panels, two inverse Mills ratios are included in order to control and correct for any selection bias. The 2SLS point estimates in Panel C are very close to those obtained in Panel B. Therefore, even when the inverse Mills ratios are significant, selection bias in the causal effect of parental education on child's health due to coresidence appears to be limited. It means that the causal effect of parents' education on child health investments and child health outcomes would be the same in both samples, i.e. in the sample of children who live with both parents, and in the sample of children who live with only one of them or neither.

In addition, our results show that the mother specific inverse Mills ratio is significant for most outcomes and the sign implies better health outcomes, meaning that unobserved factors that make mothers to live with their child tend to be associated with better conditions of birth, better nutritional status, and greater prevention through vaccination. Father specific inverse Mills ratio are less significant, but when they are, they have a less intuitive sign: fathers' unobserved characteristics that make them live with their child would be associated with lower health investment.²⁷ Several interpretations can be brought. One potential explanation may be related to migration behavior. If the father is not present because he is a migrant, then he will not coreside with the child at survey time but could

²⁶ Our sample is not restricted to children living in a nuclear family (i.e. with their mother and father and no other adult member): education levels of the other household members (grandparents, uncles and aunts, etc.) could also impact children health outcomes and alter the impact of parents' education on children health outcomes. Conclusions remain unchanged when we restrict the sample to nuclear families (Appendix Table C.5).

²⁷ This opposite sign is not due to a high correlation between fathers and mothers Mills ratio: the same sign is found when mothers' Mills ratio are not included in the equation.

send financial resources that would positively contribute to child’s health. Another related explanation could be that fathers who coreside have a worse health status than those who do not: given the intergenerational transmission of health care use and health status, this could lead to worse children health outcomes. However, we cannot investigate these channels in more details, as we do not observe characteristics of non-coresident parents. This is also one of the reasons that lead to a selection equation with excluded instruments defined at the community level.

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6. Discussion

6.1. Robustness checks

We check the robustness of the causal effects of parental education to alternative specifications of the impact of the reform on educational outcomes. The first-stage estimates appear in Appendix Table D.1, and we present the second-stage point estimates in Table 7 (controlling for selection into coresidence). We here focus on prenatal care and birth conditions as they constitute the main variables for which we observe a causal effect of parental education.

First, we consider the case of the partially exposed who are the parents born in 1965 and 1966. Robustness 1 excludes these observations from the sample as in [Agüero and Bharadwaj \[2014\]](#) and [Grépin and Bharadwaj \[2015\]](#) while Robustness 2 considers them as not exposed to the reform. In both cases, the point estimates are unchanged²⁸: mother’s education does not significantly affect prenatal care and birth outcomes while father’s education mostly significantly improves them. The size of the effect of father’s attending secondary school is stable: attending secondary school increases the probability of having four prenatal visits and the probability of being assisted by medical staff by 15 and 11 percentage points respectively.

Second, in Robustness 3, we include the parents born before 1950 to check whether this restriction was driving our results. We conclude that our core results are conservative in that they are lower-bounds of these coefficients. The size of the coefficients increases in columns 5 and 6, and the coefficients in columns 2 and 3 turn significant.

²⁸ First-stage estimates are very close to those obtained in Table 3 (see Appendix Table D.1).

Third, Robustness 4 to 6 are used to assess the plausibility of our exclusion restriction. We start by restricting the sample to children whose parents are born in years surrounding 1965. In the main specification, we do not restrict the sample based on the parents' years of birth, except that we dropped the children whose parents are born before 1950 to avoid survival bias. This implies that parents are born over a very large period of time.²⁹ Restricting the sample to children of parents born around the pivotal year (1965) allows considering parents that are rather homogeneous (as in a regression discontinuity approach) as they are likely to have faced the same economic, social and political environment at similar ages, and to be equally affected by the health reform. We use different bandwidths in Robustness 4 and Robustness 5. Note that the bandwidths are 7 years larger for fathers than for mothers to take into account the age difference between the parents in our sample. The more restricted bandwidth appears in Robustness 4 where the sample includes children whose mothers are born 5 years before to 5 years after the pivotal year, and whose fathers are born up to 12 years before and 12 years after 1965. The core results are robust for both bandwidths: the causal effect of father's secondary education is still positive and significant and that of mothers not significant. We find similar results as in Table 5 for the causal effect of the number of years of education that is significant on prenatal care.

Then, to better control for the economic and social development of Zimbabwe over time we add some control variables, defined at the child's year of birth³⁰: the GDP per capita, life expectancy at birth, the urbanization rate and the under-five mortality rate obtained from the World Development Indicators [World Bank, 2022]. Even though these additional variables are significant, they do not change the estimates of education (see Robustness 6).

Lastly, the exclusion restriction imposes that having a parent born before or after 1965 does not affect child health through any other mechanism than parental education. If it was violated, we should observe better prenatal care and birth conditions, even for children of uneducated parents born after 1965. We therefore concentrate on a sub-sample of exposed and non-exposed mothers and fathers, who did not complete their primary education. The estimation of a reduced-form equation on this restricted sample (see Table 8) shows that the exclusion restriction is likely to be valid: prenatal outcomes do not significantly differ between children from exposed and non-exposed parents. This is an indication that having parents born after 1965 does not affect children health outcomes *per se*, at least when parents are not educated.

²⁹ Mothers (fathers) are born up to 31 (28) years after 1965.

³⁰ Ideally, we would like to include variables reflecting the economic and social context faced by each parent around 1980, depending on her age at this date. Such information is, of course, not available. Moreover, these variables would be collinear with the birth cohort.

Table 7. Robustness checks

	(1)	(2)	(3)	(4)	(5)	(6)
	<i>Years of education</i>			<i>Attended secondary school</i>		
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff
Benchmark. Main results						
Education ^M	-0.007 (0.010)	0.012 (0.011)	0.016 (0.011)	-0.070 (0.093)	0.047 (0.092)	0.082 (0.092)
Education ^F	0.018** (0.009)	0.009 (0.009)	0.007 (0.009)	0.144*** (0.055)	0.152*** (0.058)	0.139** (0.058)
N	6,194	7,798	7,838	6,214	7,821	7,862
Robustness 1. Without parents born in 1965 or 1966						
Education ^M	-0.006 (0.011)	0.011 (0.011)	0.015 (0.011)	-0.071 (0.100)	0.041 (0.098)	0.078 (0.099)
Education ^F	0.019** (0.009)	0.006 (0.010)	0.004 (0.010)	0.151*** (0.058)	0.131** (0.062)	0.113* (0.062)
N	5,693	7,179	7,212	5,711	7,200	7,234
Robustness 2. With 1965 and 1966 considered as not exposed						
Education ^M	0.000 (0.010)	0.015 (0.011)	0.019* (0.011)	0.000 (0.100)	0.072 (0.101)	0.113 (0.101)
Education ^F	0.015* (0.009)	0.007 (0.009)	0.004 (0.009)	0.120** (0.057)	0.130** (0.059)	0.114* (0.059)
N	6,194	7,798	7,838	6,214	7,821	7,862
Robustness 3. With parents born before 1950						
Education ^M	0.007 (0.008)	0.013 (0.008)	0.014* (0.008)	0.034 (0.071)	0.097 (0.071)	0.106 (0.071)
Education ^F	0.012* (0.006)	0.021*** (0.007)	0.024*** (0.007)	0.127*** (0.046)	0.224*** (0.050)	0.238*** (0.049)
N	6,688	8,396	8,454	6,713	8,426	8,485
Robustness 4. Bandwidth mother=+5 years and Bandwidth father=+-12 years						
Education ^M	-0.003 (0.018)	-0.003 (0.017)	0.004 (0.017)	-0.050 (0.131)	-0.042 (0.127)	0.013 (0.125)
Education ^F	0.014 (0.018)	0.028 (0.018)	0.027 (0.018)	0.100 (0.109)	0.241** (0.114)	0.233** (0.113)
N	1,118	1,381	1,398	1,126	1,389	1,407
Robustness 5. Bandwidth mother=+8 years and Bandwidth father=+-15 years						
Education ^M	-0.009 (0.013)	0.002 (0.012)	0.010 (0.012)	-0.104 (0.099)	-0.012 (0.099)	0.059 (0.100)
Education ^F	0.026** (0.013)	0.020 (0.013)	0.014 (0.013)	0.196*** (0.071)	0.194*** (0.073)	0.157** (0.075)
N	1,986	2,465	2,490	1,998	2,477	2,503
Robustness 6. Controlling for economic and social indicators						
Education ^M	-0.008 (0.010)	0.012 (0.011)	0.016 (0.011)	-0.072 (0.092)	0.047 (0.091)	0.082 (0.092)
Education ^F	0.017* (0.009)	0.009 (0.009)	0.007 (0.009)	0.137** (0.055)	0.153*** (0.057)	0.140** (0.057)
N	6,194	7,798	7,838	6,214	7,821	7,862

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: 2SLS estimates. The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability, and between lines due to sample restrictions mentioned in the table. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, for child's sex and age, as well as for the two inverse Mills ratios obtained from the two selection equations, as in Panel C of Tables 5 and 6 (Robustness 6 adds macro-level variables).

* $p < .10$; ** $p < .05$; *** $p < .01$

Table 8. Robustness checks on non-educated parents

	(1)	(2)	(3)
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff
Mother exposed	−0.013 (0.077)	−0.105 (0.079)	−0.066 (0.079)
Father exposed	0.066 (0.070)	0.040 (0.077)	−0.031 (0.078)
N	778	1,008	1,018
Mean of dep.	0.66	0.47	0.46

Source: Authors' calculations from the Demographic and Health Surveys.
Notes: 2SLS estimates. The table reports point estimates obtained from the sample of children from the analytical sample whose parents have not completed primary school. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, for child's sex and age, linear trends for parent's year of birth before and after 1965, as well as for the two inverse Mills ratios obtained from the two selection equations.
 * $p < .10$; ** $p < .05$; *** $p < .01$

6.2. The impact of mother's education only

We complement our analysis by looking at the impact of mother's education on child-health outcomes, as this has been the focus of the literature on parental education and child health and we aim to know whether the absence of mother's education effect in our previous findings are due to controlling for father's education. As such, the role of the father in terms of his living with his children and his level of education here is not taken into account. We estimate the effect of mother's education in the sample of children who live with both parents as in the core analysis.

Tables 9 and 10 show the results from the OLS model in Panel A, the 2SLS model in Panel B and the 2SLS model that corrects for selection in coresidence in Panel C, when we estimate Equations 5, 7, and 9 without including any information on the father.

In the OLS specification in Panel A, the education coefficient has the expected sign, as more education is associated with increased health investments and better health, whatever the education variable used (Tables 9 and 10). All of the estimated coefficients are significant, except that on wasting in Table 10. These OLS coefficients are very similar to those found in Tables 5 and 6.

Table 9. The impact of mother's education (years of education)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. OLS</i>							
Years of education ^M	0.020*** (0.002)	0.052*** (0.002)	0.052*** (0.002)	-0.012*** (0.002)	-0.002** (0.001)	0.065*** (0.012)	0.012*** (0.002)
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
<i>Panel B. 2SLS</i>							
Years of education ^M	0.008 (0.008)	0.024*** (0.008)	0.027*** (0.008)	0.006 (0.007)	0.003 (0.004)	0.016 (0.044)	0.011* (0.006)
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
<i>Panel C. 2SLS with correction for selection into coresidence</i>							
Years of education ^M	0.008 (0.008)	0.025*** (0.008)	0.028*** (0.008)	0.006 (0.007)	0.003 (0.004)	0.015 (0.043)	0.009 (0.006)
Inverse Mills Ratio ^M	0.011 (0.144)	0.663*** (0.171)	0.666*** (0.173)	-0.277** (0.134)	-0.048 (0.056)	4.987*** (0.874)	-0.261** (0.116)
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Mean of dep.	0.72	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age. Panel C also controls for the mother-specific inverse Mills ratio obtained from the selection equation for child's coresidence with the mother.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table 10. The impact of mother's education (attended secondary school)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. OLS</i>							
Attended secondary school ^M	0.110*** (0.012)	0.291*** (0.013)	0.299*** (0.013)	-0.061*** (0.012)	-0.009 (0.006)	0.431*** (0.080)	0.051*** (0.010)
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
<i>Panel B. 2SLS</i>							
Attended secondary school ^M	0.077 (0.067)	0.241*** (0.067)	0.263*** (0.068)	0.048 (0.063)	0.026 (0.033)	0.198 (0.386)	0.127** (0.057)
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
<i>Panel C. 2SLS with correction for selection into coresidence</i>							
Attended secondary school ^M	0.074 (0.067)	0.248*** (0.067)	0.269*** (0.068)	0.047 (0.063)	0.026 (0.034)	0.174 (0.374)	0.111* (0.058)
Inverse Mills Ratio ^M	0.005 (0.146)	0.612*** (0.167)	0.615*** (0.169)	-0.279** (0.137)	-0.057 (0.058)	4.940*** (0.875)	-0.306** (0.121)
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Mean of dep.	0.72	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age. Panel C also controls for the mother-specific inverse Mills ratio obtained from the selection equation for child's coresidence with the mother.

* $p < .10$; ** $p < .05$; *** $p < .01$

The 2SLS estimates reported in Panel B are less significant. The effect of mother's education remains statistically significant on the probabilities of being born in a health facility, having a birth assisted by medical staff and sleeping under bed net, for both measures of education. The same results are found in Panel C except that the causal effect of mother's years of education on the probability of sleeping under bed net is slightly reduced and hence loses its significance.

Regarding child nutrition outcomes, the findings in columns 4 and 5 in Panels A and B of Tables 9 and 10 can be compared to those in [De Neve and Subramanian \[2017\]](#). Our results are in line with theirs: the OLS estimates of the effect of maternal schooling on the probabilities of being stunted and wasted are negative and mostly significant, while the 2SLS estimates are insignificant in all cases.

Overall, these results along with those in Tables 5 and 6 suggest that if the child lives with both parents, not controlling for father's education overestimates the effect of mother's education, as the latter captures part of the effect of father's education (especially if they have similar education). This confirms related evidence from [Fafchamps and Shilpi \[2014\]](#) and [Behrman and Rosenzweig \[2002\]](#). Indeed, our results echo those from [Behrman and Rosenzweig \[2002\]](#) for child education, who no longer find an effect of mother's education on children's schooling when father's education and endogeneity bias are considered. They also find that in contrast, father's education has a significantly positive effect on the educational attainment of the next generation.

6.3. Mechanisms

We now consider potential mechanisms through which parental education might influence child health. Parental education may affect many outcomes, which themselves determine child health: these include wealth, access to health services, urbanisation, parental choice regarding family size, and modern contraception use.

We evaluate the role of these different mediators in the effect of parental education on child health by estimating the causal effect of father and mother's education on a sequence of outcome variables and replicating the models that appear in Panel B of Tables 5 and 6. Note that controlling for coresidence is less justified here because these outcome variables are not child-specific. They are observed at the mother level, the father level or at the household level.

Table 11 first considers as mediators the ideal number of children reported by the mother

and the father, as well as the mother’s age at first birth and use of modern contraception. The last row gives the mean of each variable: mothers (fathers) expect to have 4.1 (4.7) children on average, the average age of the mothers at first birth is 19.3, and 69% of them use modern contraception. The second set of mediators is related to living conditions: 34% of the households live in urban areas and 43% are in the two richest quintiles.

Columns 1-4 in Table 11 consider the role of fertility preferences and attitudes. Father’s education level has a significant causal effect on the ideal number of children reported by both mothers (column 1) and fathers (column 2).³¹ Mother’s age at first birth increases with her attendance to secondary school. Mother’s education significantly influences her use of modern contraception at the time of the survey, in column 4: attending secondary school increases it by about 34 percentage points.

Table 11. Mechanisms

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Fertility</i>				<i>Household characteristics</i>		
	Mother’s ideal number of children	Father’s ideal number of children	Mother’s age at first birth	Mother’s use of modern contraception	Urban	Rich	Wealth Index
<i>Panel A. Years of education</i>							
Years of education ^M	-0.086 (0.079)	0.154 (0.120)	0.438 (0.595)	0.039** (0.017)	0.008 (0.008)	0.020** (0.008)	0.005 (0.017)
Years of education ^F	-0.127** (0.055)	-0.321*** (0.107)	-0.057 (0.287)	0.013 (0.015)	0.013* (0.007)	0.010 (0.008)	0.013 (0.016)
N	6,742	4,623	6,816	6,823	7,415	7,415	7,415
Mean of dep.	4.09	4.68	19.29	0.69	0.34	0.43	0.12
<i>Panel B. Attended secondary school</i>							
Attended secondary school ^M	0.064 (0.844)	1.448 (0.998)	11.182** (4.368)	0.337** (0.135)	0.021 (0.068)	0.123 (0.076)	-0.104 (0.170)
Attended secondary school ^F	-1.399*** (0.368)	-2.420*** (0.634)	-0.254 (0.622)	0.115 (0.090)	0.163*** (0.048)	0.157*** (0.052)	0.310*** (0.107)
N	6,763	4,629	6,837	6,844	7,439	7,439	7,439
Mean of dep.	4.09	4.68	19.29	0.69	0.34	0.43	0.12

Source: Authors’ calculations from the Demographic and Health Surveys.

Notes: 2SLS estimates. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects. Additional covariates include parents’ age categories for fertility behaviors. The unit of observation corresponds to a mother (columns 1, 3 and 4) or a father (column 2) born in or after 1950 with a child in the analytical sample, and to the corresponding households in columns 5 to 7. The sample is restricted to couples with at least one child in the analytical sample, keeping one line per couple.

* $p < .10$; ** $p < .05$; *** $p < .01$

³¹ Note that the sample is reduced in column 2 because observing the father’s ideal number of children requires the father to be sampled to answer the male questionnaire. The DHS sampling design is such that not all sampled households are eligible for the male questionnaire in the surveys collected in 1994 and 1999.

Columns 5-7 in Table 11 show that father's attending secondary school increases the probability of living in urban area, the probability that the household belongs to the two richest quintiles by 16 percentage points, and the wealth index of the household. This suggests that part of the effects of father's education on prenatal care and birth conditions comes from the fact that the children are living in richer households who can afford these health services and in urban households who have a better access to health care services. Father's education reduces both types of barriers to access to health care: geographical and monetary. On the contrary, mother education has nearly no effect on these variables.

A complementary approach is to add the mechanisms as additional control variables. We add controls for household's urban status, material wealth and province of residence when estimating Equations 5-9.³² As shown in Appendix Tables E.1 to E.3, conclusion remain unchanged once such covariates are controlled for, either separately (Panels A to C) or simultaneously (Panel D). These results suggest that father's education not only affects child health indirectly through better living conditions, but also has a direct positive effect on perinatal conditions, and on vaccination when controlling for urban status and wealth as the effects of father's education are still significant. Possible reasons behind this persistence might be information and allocation of resources within household. First, it might be that education increases fathers' awareness of the importance of prenatal care and birth condition for the child's health. Given that mothers are the ones giving birth, they might already be more knowledgeable on childbearing-related healthcare, no matter their level of education. Thus, the effect might be salient for fathers and not for mothers. Previous papers have shown that education increases the effect of sensitization messages on health behaviors and outcomes (see [de Walque 2007](#) for example). In our setting, as survey year dummies are included as fixed effects, our findings suggest that for a given access to sensitization campaigns through mass media for instance, educated fathers might respond more to the messages received/heard. Second, education might influence the way the resources are allocated within the household for a given wealth level, and educated fathers might allocate more resources to child human capital accumulation.

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* *

³² We cannot control for the fertility behaviors as we do not observe these variables when the parent does not live with the child

7. Concluding Remarks

Our main results regarding parental education and child health are as follows. Father's education consistently and significantly improves prenatal care, birth conditions and vaccination. On the contrary, mother's education has no causal impact on these outcomes if father's education is controlled for in the equation. Differences observed between education measured as either attending secondary school or years of education suggest that attendance to secondary school makes a strong difference in child's health outcomes. Moreover, once endogeneity is controlled for, child nutrition and prevention through the use of bed net, are not influenced by parental education. Last, unobserved characteristics of both the father and the mother that make them live with their child are strong determinants of children health outcomes. However, if it exists, the selection bias that may arise from selection into coresidence is found to be of a limited amount: the causal effect of parental education on health investments and child health outcomes would be of similar magnitude in the sample of children who live with both parents and in the sample who live with only one of them or neither.

Overall, our results underline the predominance of father's education in determining health investment behaviors in the household. The model with mother's education only yields over-estimates of its impact on child health: mother's education matters less when father's education is controlled for. As such, the results in the existing literature without father's education may have overestimated the impact of mother's education when both mothers and fathers coreside with the child. This comes about due to the assortative matching in our sample: men and women with similar observed education levels and similar intrinsic motivations or aspirations towards investment in human capital tend to live and have children together. The analysis of potential mechanisms that may drive our results show that father's education may reduce both geographical and financial barriers to access to care and then improve prenatal care and birth conditions. Nonetheless, the effect of father's education persists even when controlling for these mechanisms.

Last, our results show that father's education plays an important role in determining prenatal care and birth conditions. These outcomes are of particular importance as they are the main tools to reduce child and maternal mortality. In 2020, 47% of children that do not reach their fifth year die within their first month of life, up from 40% in 1990, with about one third dying within the first 24 hours. As education has risen over recent decades, we may predict that hopefully child and maternal health will drastically increase in line,

and that the considerable burden of disease and death borne by children and pregnant women will fall. This chapter has important policy implications as the results suggest that child health policies targeting and involving fathers could have sizeable effects on their own and in complement to existing policies exclusively focused on mothers and mothers-to-be.

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APPENDICES TO CHAPTER 2

Appendix A. Education system indicators

Table A.1. Education indicators in Zimbabwe over the 1975-2000 period

	1975	1980	1985	1990	1995	2000
Number of teachers - Primary	21,202	28,118	56,067	59,154	63,475	66,440
Number of teachers - Secondary	3,383	3,782	19,507	24,547	27,458	34,163
Teachers /1000 inhab. - Primary	3.44	3.86	6.33	5.65	5.45	5.31
Teachers /1000 inhab. - Secondary	0.55	0.53	2.25	2.41	2.42	2.79
Pupil/teacher ratio - Primary	40.69	43.92	39.50	35.78	39.11	37.03
Pupil/teacher ratio - Secondary	19.38	19.76	27.81	26.93	25.90	24.71
Girl/boy ratio - Primary	85.22	-	94.62	99.12	97.37	97
Girl/boy ratio - Secondary	71.26	-	68.46	88.00	83.66	88.00
Government funding (% of GDP)	-	2.5	7.4	12.5	-	-
Official entrance age - Primary	7	7	7	6	6	6
Official entrance age - Secondary	14	14	14	13	13	13

Source: World Development Indicators [World Bank, 2022].

Appendix B. Selection into coresidence

Our ability to observe the dependent and independent variables of interest depends on the five types of setting in the data, as summarized in Appendix Table B.1.

Table B.1. Selection issues

	(1)	(2) Presence in the sampled household			(3) Education		(4) Type of health data	
	N	Child in the hh	Mother in the hh	Father in the hh	Mother's education	Father's education	Current status	Birth info
Type 1	1,695	No	Yes	NA	7.9	NA	No	Yes
Type 2	10,381	Yes	Yes	Yes	7.5	8.3	Yes	Yes
Type 3	2,659	Yes	No	No	NA	NA	Yes	No
Type 4	248	Yes	No	Yes	NA	8.4	Yes	No
Type 5	6,402	Yes	Yes	No	7.7	NA	Yes	Yes

Source: Authors, based on the the Demographic and Health Surveys 1994, 1999, 2005 and 2010

The analytical sample used to estimate the effect of mother's and father's education on child-health outcomes is restricted to sampled children who live with both parents (household composition of Type 2). If the three are listed as household members, we can match the children to their parents using their IDs, and the educational attainment of each parent is observed. In order to observe current health outcomes, we need either the mother to live in the household (as the birth history is asked of each mother) or the child to live in

the household (and thus be present when the anthropometric measures are taken). Both types of outcomes are observed when the child and mother live in the same household.

In the four other cases, we do not have all of the necessary information (child-health outcomes and father's and mother's education). In Type 1, the child is not a household member, while the mother is (and maybe the father too). The mother declares the child in the birth history, but the child does not appear in the survey either because he/she is dead or is fostered in another household. As these children are not listed in the household roster, we cannot match them to their fathers, so that father's education is unobserved. There are 1,695 children of this type who will not appear in our analysis, including in the selection equations.

Children of Types 3, 4, and 5 currently live in the sampled households but are left out of the analysis sample as they do not live with both parents. Type-3 children live with neither parent, Type-4 children with their father but not their mother, and Type-5 children with their mother only. We have missing data on the birth-outcome variables for Types 3 and 4 children as the mother is not in the sampled household and so does not reply to the questionnaire recording that information. Current child weight and height is observed when the child is present in the household, that is for children of Types 2 to 5.

There are no great differences in school attainment across types. When mother's education is observed, this varies from 7.5 to 7.9 years (see column 5 of Appendix Table [B.1](#)), and for fathers from 8.3 to 8.4 (column 6).

Table B.2. Descriptive statistics - Child's characteristics and health outcomes by coresidence status

	(1) Coresidence Parents born in 1950 or later	(2) No Coresidence	(3) Difference (1)-(2)
<i>Panel A. Child characteristics</i>			
Girl	0.50	0.50	-0.003
Age	1.84	2.14	-0.309***
Urban	0.32	0.18	0.147***
Rich	0.41	0.26	0.147***
<i>Panel B. Outcomes</i>			
At least 4 prenatal visits	0.71	0.70	0.018**
Health Facility Birth	0.68	0.71	-0.028***
Birth assisted by medical staff	0.68	0.70	-0.023***
Stunted	0.32	0.34	-0.026***
Wasted	0.06	0.07	-0.009**
Number of injections received by child	5.71	5.76	-0.050
Slept under net last night	0.12	0.06	0.058***
N	9,365	9,321	18,686

Source: Authors' calculations from the Demographic and Health Surveys 1994, 1999, 2005 and 2010

Notes: Unweighted statistics. The data covers children aged 0-59 months at survey time. The sub-samples correspond to 0-59 months children living with both parents (born in 1950 or later) in column 1, and to those living with only one parent or neither in column 2. Column 3 reports differences between columns 1 and 2.

* $p < .10$; ** $p < .05$; *** $p < .01$

Appendix C. Additional results

C.1. Results obtained with weighted observations

Table C.1. The impact of mother's and father's education with observations weighted by the square root of the total number of observations for each couple

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. Years of education</i>							
Years of education ^M	-0.005 (0.011)	0.012 (0.012)	0.015 (0.012)	0.007 (0.010)	0.000 (0.006)	-0.086 (0.071)	0.001 (0.009)
Years of education ^F	0.017* (0.009)	0.010 (0.010)	0.008 (0.010)	0.002 (0.009)	0.005 (0.005)	0.136** (0.068)	0.003 (0.008)
Inverse Mills Ratio ^M	0.384 (0.308)	1.694*** (0.347)	1.734*** (0.356)	-0.756*** (0.277)	-0.119 (0.136)	5.243*** (1.943)	-0.313 (0.242)
Inverse Mills Ratio ^F	-0.115 (0.100)	-0.403*** (0.117)	-0.415*** (0.120)	0.203** (0.088)	0.037 (0.050)	0.292 (0.652)	0.028 (0.089)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.550***	0.550***	0.550***	0.550***	0.550***	0.550***	0.549***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12
<i>Panel B. Attended secondary school</i>							
Attended secondary school ^M	-0.045 (0.096)	0.036 (0.100)	0.066 (0.100)	0.074 (0.089)	0.009 (0.048)	-0.601 (0.638)	0.061 (0.095)
Attended secondary school ^F	0.134** (0.056)	0.161*** (0.061)	0.148** (0.062)	-0.009 (0.053)	0.023 (0.029)	0.847** (0.420)	0.035 (0.048)
Inverse Mills Ratio ^M	0.378 (0.326)	1.697*** (0.349)	1.706*** (0.357)	-0.767*** (0.296)	-0.145 (0.149)	5.743*** (2.084)	-0.460 (0.282)
Inverse Mills Ratio ^F	-0.102 (0.103)	-0.381*** (0.114)	-0.381*** (0.116)	0.200** (0.090)	0.045 (0.053)	0.114 (0.673)	0.070 (0.095)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.390***	0.390***	0.390***	0.390***	0.390***	0.390***	0.389***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample when each observations are weighted by the square root of the total number of observations for each couple. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age.

* $p < .10$; ** $p < .05$; *** $p < .01$

C.2. First stage estimates without restriction on co-residence status

Table C.2. First-stage equations when the sample is not restricted to children living with both parents

	(1)	(2)	(3)	(4)
	Mother's education		Father's education	
	<i>Years of education</i>			
Panel A.				
Exposed	3.206*** (0.111)	2.172*** (0.180)	2.375*** (0.121)	0.946*** (0.192)
Pre-reform trend		0.142*** (0.023)		0.251*** (0.021)
Post-reform trend		0.035*** (0.006)		-0.032*** (0.008)
N	16,582	16,582	9,512	9,512
Mean of dep.	7.62	7.62	8.64	8.64
Adjusted R ²	0.18	0.18	0.12	0.15
Kleibergen-Paap F-Statistic	830.36	316.41	386.04	194.01
Effective F-Statistic [Montiel Olea and Pflueger, 2013]	830.36	347.56	386.04	217.36
	Mother's education		Father's education	
	<i>Attended secondary school</i>			
Panel B.				
Exposed	0.371*** (0.013)	0.265*** (0.020)	0.337*** (0.016)	0.141*** (0.025)
Pre-reform trend		0.013*** (0.002)		0.033*** (0.003)
Post-reform trend		0.005*** (0.001)		-0.003*** (0.001)
N	16,593	16,593	9,540	9,540
Mean of dep.	0.52	0.52	0.63	0.63
Adjusted R ²	0.11	0.12	0.12	0.15
Kleibergen-Paap F-Statistic	775.29	287.25	461.36	233.77
Effective F-Statistic [Montiel Olea and Pflueger, 2013]	775.29	292.93	461.36	235.76

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between columns (1)-(2) and (3)-(4) due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for control variables of the second stage equation (child's sex and age). The Kleibergen-Paap and Montiel Olea and Pflueger [2013] F-statistics of excluded instruments are obtained from the estimation of Equations 1 and 2. There is no correction for selection into co-residence.

* $p < .10$; ** $p < .05$; *** $p < .01$

C.3. Alternative specification of the trends based on linear splines

Table C.3. First-stage equations based on a linear spline specification

	(1)	(2)	(3)	(4)
	Mother's education		Father's education	
	Years of education	Attended secondary school	Years of education	Attended secondary school
Exposed	1.708*** (0.331)	0.264*** (0.038)	0.683*** (0.233)	0.109*** (0.031)
LS ₁	0.095*** (0.021)	0.003 (0.003)	-0.012 (0.018)	-0.005** (0.003)
LS ₂	-0.015 (0.010)	0.001 (0.002)	-0.045*** (0.014)	-0.001 (0.002)
LS ₃	0.142** (0.055)	0.012** (0.006)	0.286*** (0.032)	0.043*** (0.004)
LS ₄	0.221 (0.241)	-0.001 (0.022)	0.132* (0.069)	0.000 (0.008)
N	9,337	9,343	9,288	9,315
Mean of dep.	7.82	0.54	8.64	0.63
Adjusted R ²	0.13	0.08	0.15	0.15
Kleibergen-Paap F-Statistic	75.02	70.17	116.68	133.95
Effective F-Statistic [Montiel Olea and Pflueger, 2013]	85.25	69.39	135.50	145.07

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between columns due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for control variables of the second stage equation (child's sex and age). The Kleibergen-Paap and Montiel Olea and Pflueger [2013] F-statistics of excluded instruments are obtained from the estimation of Equations 1 and 2. There is no correction for selection into co-residence. The table presents results from an alternative specification using linear splines, where the variables are defined as follows. For the mother, $LS_1 = \mathbb{1}_{B^M \geq 1965} [\mathbb{1}_{B^M < 1965} (B^M - 1965) + 10 \times \mathbb{1}_{B^M \geq 1965}]$; $LS_2 = \mathbb{1}_{B^M - 1965 \geq 10} (B^M - 1965 - 10)$; $LS_3 = \mathbb{1}_{B^M < 1965} [\mathbb{1}_{B^M - 1965 \geq -10} (B^M - 1965) - 10 \times \mathbb{1}_{B^M - 1965 < -10}]$; $LS_4 = \mathbb{1}_{B^M - 1965 < -10} (B^M - 1965 + 10)$. Variables are defined identically for the father.
* $p < .10$; ** $p < .05$; *** $p < .01$

Table C.4. The impact of mother's and father's education using a linear spline specification

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
Panel A. Years of education							
Years of education ^M	-0.002 (0.010)	0.013 (0.011)	0.014 (0.011)	0.007 (0.010)	0.003 (0.005)	-0.067 (0.064)	0.009 (0.008)
Years of education ^F	0.015* (0.009)	0.012 (0.009)	0.011 (0.009)	0.000 (0.008)	0.003 (0.004)	0.111* (0.059)	0.001 (0.007)
Inverse Mills Ratio ^M	-0.132 (0.177)	0.067 (0.204)	0.066 (0.203)	-0.314* (0.172)	0.025 (0.072)	6.358*** (1.051)	-0.206* (0.123)
Inverse Mills Ratio ^F	0.118 (0.079)	0.332*** (0.100)	0.332*** (0.102)	0.085 (0.080)	-0.048 (0.039)	-0.999* (0.517)	-0.044 (0.070)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.557***	0.557***	0.557***	0.557***	0.557***	0.557***	0.556***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12
Panel B. Attended secondary school							
Attended secondary school ^M	-0.045 (0.095)	0.056 (0.092)	0.083 (0.093)	0.075 (0.087)	0.021 (0.047)	-0.568 (0.579)	0.087 (0.083)
Attended secondary school ^F	0.121** (0.057)	0.154*** (0.059)	0.147** (0.058)	-0.010 (0.052)	0.019 (0.028)	0.756** (0.372)	0.034 (0.048)
Inverse Mills Ratio ^M	-0.109 (0.175)	0.154 (0.202)	0.150 (0.199)	-0.329* (0.169)	0.018 (0.070)	6.336*** (1.040)	-0.198 (0.121)
Inverse Mills Ratio ^F	0.128 (0.081)	0.302*** (0.100)	0.296*** (0.101)	0.081 (0.081)	-0.044 (0.039)	-0.907* (0.515)	-0.058 (0.071)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.400***	0.400***	0.400***	0.400***	0.400***	0.400***	0.399***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,740
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age.

* $p < .10$; ** $p < .05$; *** $p < .01$

C.4. Analysis restricted to nuclear families

Table C.5. The impact of mother's and father's education among nuclear families

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. Years of education</i>							
Years of education ^M	-0.020 (0.014)	-0.012 (0.015)	-0.009 (0.015)	0.006 (0.014)	0.006 (0.007)	-0.032 (0.080)	0.007 (0.013)
Years of education ^F	0.035** (0.014)	0.039*** (0.013)	0.035** (0.014)	0.012 (0.013)	0.000 (0.007)	0.177* (0.091)	0.002 (0.011)
Inverse Mills Ratio ^M	0.274 (0.197)	0.644*** (0.215)	0.616*** (0.214)	-0.277 (0.197)	0.021 (0.090)	5.281*** (1.180)	-0.059 (0.172)
Inverse Mills Ratio ^F	0.173 (0.150)	0.254 (0.156)	0.201 (0.156)	0.045 (0.133)	-0.130** (0.058)	0.384 (0.818)	-0.262* (0.138)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.521***	0.521***	0.521***	0.521***	0.521***	0.521***	0.519***
N	3,694	4,718	4,748	4,320	4,288	4,732	3,430
Mean of dep.	0.70	0.66	0.65	0.34	0.06	5.64	0.13
<i>Panel B. Attended secondary school</i>							
Attended secondary school ^M	-0.130 (0.112)	-0.036 (0.127)	-0.003 (0.126)	0.011 (0.112)	0.063 (0.058)	-0.861 (0.696)	0.049 (0.096)
Attended secondary school ^F	0.172** (0.073)	0.231*** (0.079)	0.202** (0.080)	0.022 (0.068)	-0.031 (0.037)	1.431*** (0.494)	0.081 (0.054)
Inverse Mills Ratio ^M	0.556 (0.390)	2.034*** (0.413)	2.016*** (0.417)	-0.477 (0.349)	-0.028 (0.182)	4.859* (2.517)	-0.451 (0.318)
Inverse Mills Ratio ^F	-0.113 (0.125)	-0.448*** (0.133)	-0.457*** (0.133)	0.105 (0.107)	-0.029 (0.059)	0.273 (0.794)	0.114 (0.110)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.368***	0.368***	0.369***	0.368***	0.368***	0.368***	0.366***
N	3,710	4,737	4,767	4,336	4,304	4,751	3,448
Mean of dep.	0.70	0.66	0.65	0.34	0.06	5.65	0.13

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: The table reports point estimates obtained from the analytical sample restricted to nuclear families. Sample size varies between outcomes due to data availability. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, as well as for child's sex and age.

* $p < .10$; ** $p < .05$; *** $p < .01$

Appendix D. Robustness checks for the first stages estimates

Table D.1. First stages equations - Robustness checks

	(1) Mother Education		(3) Father Education	
	Years of education	Attended secondary school	Years of education	Attended secondary school
Benchmark – Main results with correction for selection into coresidence				
Exposed	2.093*** (0.287)	0.271*** (0.032)	0.920*** (0.195)	0.139*** (0.025)
Pre-reform trend	0.148*** (0.046)	0.011** (0.005)	0.249*** (0.021)	0.033*** (0.003)
Post-reform trend	0.024*** (0.008)	0.002* (0.001)	−0.030*** (0.008)	−0.003*** (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	120.00	120.09	191.80	224.59
Robustness 1. Without parents born in 1965 or 1966				
Exposed	2.231*** (0.293)	0.274*** (0.033)	1.111*** (0.211)	0.158*** (0.028)
Pre-reform trend	0.146*** (0.046)	0.011** (0.005)	0.247*** (0.022)	0.033*** (0.003)
Post-reform trend	0.012 (0.008)	0.001 (0.001)	−0.043*** (0.009)	−0.005*** (0.001)
N	8,527	8,533	8,484	8,508
Kleibergen-Paap F-Statistic	114.79	109.74	182.70	210.50
Robustness 2. With 1965 and 1966 considered as not exposed				
Exposed	1.672*** (0.215)	0.177*** (0.028)	0.791*** (0.159)	0.109*** (0.022)
Pre-reform trend	0.242*** (0.039)	0.028*** (0.004)	0.275*** (0.017)	0.037*** (0.002)
Post-reform trend	0.013* (0.008)	0.002 (0.001)	−0.039*** (0.009)	−0.004*** (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	128.19	118.83	197.12	224.54
Robustness 3. With parents born before 1950				
Exposed	2.154*** (0.214)	0.266*** (0.024)	1.556*** (0.155)	0.251*** (0.020)
Pre-reform trend	0.151*** (0.021)	0.010*** (0.002)	0.145*** (0.008)	0.015*** (0.001)
Post-reform trend	0.034*** (0.007)	0.003*** (0.001)	−0.029*** (0.008)	−0.003*** (0.001)
N	10,337	10,334	10,258	10,296
Kleibergen-Paap F-Statistic	238.20	183.01	385.02	365.91

continued →

Table D.1 (*continued*). First stages equations - Robustness checks

	(1)	(2)	(3)	(4)
	Mother Education		Father Education	
	Years of education	Attended secondary school	Years of education	Attended secondary school
Robustness 4. <i>Bandwidth mother=+-5 years Bandwidth father=+-12 years</i>				
Exposed	1.487*** (0.487)	0.227*** (0.056)	1.089*** (0.348)	0.134*** (0.048)
Pre-reform trend	0.034 (0.143)	0.005 (0.014)	0.262*** (0.038)	0.037*** (0.005)
Post-reform trend	0.263*** (0.074)	0.023** (0.010)	-0.095 (0.067)	-0.010 (0.012)
N	1,941	1,944	1,931	1,939
Kleibergen-Paap F-Statistic	38.60	47.04	51.35	57.27
Robustness 5. <i>Bandwidth mother=+-8 years Bandwidth father=+-15 years</i>				
Exposed	1.488*** (0.375)	0.242*** (0.043)	0.977*** (0.264)	0.134*** (0.035)
Pre-reform trend	0.150** (0.072)	0.017** (0.007)	0.249*** (0.023)	0.034*** (0.003)
Post-reform trend	0.155*** (0.035)	0.008 (0.005)	-0.076* (0.043)	-0.005 (0.006)
N	3,343	3,347	3,316	3,329
Kleibergen-Paap F-Statistic	84.63	90.19	105.82	142.74
Robustness 6. <i>Controlling for economic and social indicators</i>				
Exposed	2.107*** (0.286)	0.271*** (0.032)	0.933*** (0.197)	0.139*** (0.026)
Pre-reform trend	0.148*** (0.046)	0.011** (0.005)	0.247*** (0.021)	0.033*** (0.003)
Post-reform trend	0.024*** (0.008)	0.002* (0.001)	-0.031*** (0.008)	-0.003** (0.001)
N	9,283	9,289	9,233	9,260
Kleibergen-Paap F-Statistic	120.28	121.14	188.12	225.04

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, different linear trends before and after 1965, the two inverse Mills ratios, as well as for control variables of the second stage equation (child's sex and age). Robustness 6 adds macro-level variables. The F-statistic of excluded instruments is obtained from the estimation of Equations 1 and 2.

* $p < .10$; ** $p < .05$; *** $p < .01$

Appendix E. Controlling for additional covariates

Table E.1. First stages equations - Controlling for additional covariates

	(1)	(2)	(3)	(4)
	Mother Education		Father Education	
	Years of education	Attended secondary school	Years of education	Attended secondary school
<i>Panel A. Controlling for urban status</i>				
Exposed	1.920*** (0.258)	0.245*** (0.029)	0.817*** (0.181)	0.127*** (0.024)
Pre-reform trend	0.135*** (0.041)	0.009** (0.004)	0.225*** (0.020)	0.030*** (0.002)
Post-reform trend	0.022*** (0.007)	0.002* (0.001)	-0.027*** (0.008)	-0.003** (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	118.92	113.57	174.94	197.75
<i>Panel B. Controlling for material wealth quintiles</i>				
Exposed	1.857*** (0.243)	0.238*** (0.029)	0.777*** (0.175)	0.124*** (0.023)
Pre-reform trend	0.125*** (0.039)	0.008* (0.004)	0.218*** (0.019)	0.029*** (0.002)
Post-reform trend	0.031*** (0.006)	0.003*** (0.001)	-0.018** (0.007)	-0.002* (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	137.75	114.47	175.83	198.62
<i>Panel C. Controlling for province of residence</i>				
Exposed	1.917*** (0.260)	0.244*** (0.029)	0.840*** (0.183)	0.130*** (0.024)
Pre-reform trend	0.149*** (0.042)	0.011*** (0.004)	0.235*** (0.020)	0.031*** (0.002)
Post-reform trend	0.023*** (0.007)	0.002* (0.001)	-0.027*** (0.008)	-0.003** (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	125.83	115.90	185.74	206.38
<i>Panel D. Controlling for urban status, material wealth quintiles and province of residence</i>				
Exposed	1.802*** (0.240)	0.229*** (0.029)	0.765*** (0.174)	0.123*** (0.023)
Pre-reform trend	0.134*** (0.039)	0.009** (0.004)	0.218*** (0.019)	0.029*** (0.002)
Post-reform trend	0.030*** (0.006)	0.003** (0.001)	-0.018** (0.007)	-0.002** (0.001)
N	9,337	9,343	9,288	9,315
Kleibergen-Paap F-Statistic	138.48	109.60	178.93	195.10

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, the two inverse Mills ratios, as well as for control variables of the second stage equation (child's sex and age, and additional controls mentioned in each panel description). The F-statistic of excluded instruments is obtained from the estimation of Equations 1 and 2.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table E.2. The impact of mother’s and father’s education, controlling for additional covariates (years of education)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. Controlling for urban status</i>							
Years of education ^M	-0.008 (0.011)	0.009 (0.011)	0.013 (0.011)	0.010 (0.010)	0.001 (0.006)	-0.083 (0.067)	0.005 (0.009)
Years of education ^F	0.019** (0.009)	0.007 (0.009)	0.004 (0.009)	0.001 (0.009)	0.005 (0.005)	0.114* (0.061)	-0.001 (0.008)
Inverse Mills Ratio ^M	0.291 (0.220)	0.507** (0.207)	0.493** (0.211)	0.047 (0.190)	-0.138 (0.098)	2.031 (1.302)	-0.255* (0.148)
Inverse Mills Ratio ^F	-0.072 (0.081)	-0.025 (0.077)	-0.021 (0.078)	-0.042 (0.070)	0.042 (0.042)	1.212*** (0.467)	-0.029 (0.067)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.474***	0.474***	0.474***	0.474***	0.474***	0.474***	0.471***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
<i>Panel B. Controlling for material wealth quintiles</i>							
Years of education ^M	-0.009 (0.011)	0.007 (0.011)	0.010 (0.011)	0.009 (0.010)	0.001 (0.006)	-0.095 (0.067)	0.005 (0.009)
Years of education ^F	0.020** (0.009)	0.010 (0.009)	0.008 (0.009)	0.001 (0.009)	0.005 (0.005)	0.120* (0.061)	0.000 (0.008)
Inverse Mills Ratio ^M	0.380* (0.206)	0.453** (0.206)	0.452** (0.209)	-0.056 (0.186)	-0.110 (0.096)	2.095 (1.298)	-0.310** (0.149)
Inverse Mills Ratio ^F	-0.105 (0.078)	0.006 (0.075)	0.009 (0.076)	-0.007 (0.070)	0.032 (0.041)	1.236*** (0.479)	0.001 (0.068)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.419***	0.419***	0.419***	0.419***	0.419***	0.419***	0.418***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742

continued →

Table E.2 (*continued*). The impact of mother’s and father’s education, controlling for additional covariates (years of education)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel C. Controlling for province of residence</i>							
Years of education ^M	-0.008 (0.011)	0.012 (0.011)	0.016 (0.011)	0.009 (0.010)	0.001 (0.006)	-0.061 (0.063)	0.006 (0.009)
Years of education ^F	0.020** (0.009)	0.009 (0.009)	0.006 (0.009)	0.001 (0.009)	0.004 (0.005)	0.083 (0.057)	0.003 (0.008)
Inverse Mills Ratio ^M	0.452** (0.203)	0.429** (0.202)	0.406** (0.205)	-0.060 (0.185)	-0.015 (0.097)	1.521 (1.247)	-0.332** (0.136)
Inverse Mills Ratio ^F	-0.062 (0.086)	0.073 (0.090)	0.070 (0.092)	0.040 (0.080)	-0.021 (0.046)	0.693 (0.528)	-0.054 (0.080)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.500***	0.500***	0.500***	0.500***	0.500***	0.500***	0.496***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
<i>Panel D. Controlling for urban status, material wealth quintiles and province of residence</i>							
Years of education ^M	-0.009 (0.011)	0.008 (0.011)	0.011 (0.011)	0.008 (0.010)	0.001 (0.006)	-0.075 (0.064)	0.005 (0.009)
Years of education ^F	0.021** (0.009)	0.010 (0.009)	0.007 (0.009)	0.002 (0.009)	0.004 (0.005)	0.088 (0.058)	0.001 (0.008)
Inverse Mills Ratio ^M	0.239 (0.179)	0.288* (0.168)	0.266 (0.171)	0.043 (0.164)	-0.096 (0.086)	1.581 (1.090)	-0.287** (0.121)
Inverse Mills Ratio ^F	0.001 (0.083)	0.113 (0.078)	0.114 (0.080)	0.007 (0.078)	0.008 (0.044)	0.715 (0.508)	-0.090 (0.077)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.411***	0.411***	0.411***	0.411***	0.411***	0.411***	0.409***
N	6,194	7,798	7,838	7,089	7,041	7,811	5,742
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors’ calculations from the Demographic and Health Surveys.

Notes: Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, for child’s sex and age, for the two inverse Mills ratios, as well as for additional controls mentioned in each panel description.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table E.3. The impact of mother’s and father’s education, controlling for additional covariates (attended secondary school)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel A. Controlling for urban status</i>							
Attended secondary school ^M	-0.088 (0.100)	0.024 (0.097)	0.060 (0.098)	0.102 (0.092)	0.015 (0.051)	-0.621 (0.621)	0.080 (0.089)
Attended secondary school ^F	0.151*** (0.058)	0.118** (0.058)	0.103* (0.058)	-0.008 (0.055)	0.030 (0.030)	0.707* (0.391)	0.005 (0.050)
Inverse Mills Ratio ^M	0.288 (0.223)	0.528** (0.206)	0.503** (0.209)	0.040 (0.192)	-0.164 (0.101)	2.177* (1.317)	-0.282* (0.151)
Inverse Mills Ratio ^F	-0.058 (0.081)	-0.011 (0.076)	-0.002 (0.077)	-0.044 (0.070)	0.051 (0.043)	1.166** (0.467)	-0.024 (0.066)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.312***	0.312***	0.312***	0.312***	0.312***	0.312***	0.311***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
<i>Panel B. Controlling for material wealth quintiles</i>							
Attended secondary school ^M	-0.098 (0.098)	0.004 (0.100)	0.041 (0.100)	0.097 (0.093)	0.015 (0.051)	-0.762 (0.624)	0.065 (0.084)
Attended secondary school ^F	0.152*** (0.058)	0.125** (0.060)	0.110* (0.060)	-0.006 (0.055)	0.029 (0.030)	0.745* (0.393)	0.008 (0.049)
Inverse Mills Ratio ^M	0.385* (0.210)	0.481** (0.206)	0.465** (0.209)	-0.067 (0.188)	-0.132 (0.099)	2.305* (1.319)	-0.325** (0.152)
Inverse Mills Ratio ^F	-0.096 (0.078)	0.014 (0.074)	0.022 (0.075)	-0.009 (0.069)	0.038 (0.041)	1.164** (0.480)	0.003 (0.068)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.274***	0.274***	0.274***	0.274***	0.274***	0.274***	0.275***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755

continued →

Table E.3 (*continued*). The impact of mother's and father's education, controlling for additional covariates (attended secondary school)

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	<i>Prenatal and Birth</i>			<i>Nutrition</i>		<i>Prevention</i>	
	At least 4 prenatal visits	Health Facility Birth	Birth assisted by medical staff	Stunted	Wasted	Number of injections received by child	Slept under net last night
<i>Panel C. Controlling for province of residence</i>							
Attended secondary school ^M	-0.076 (0.098)	-0.024 (0.094)	0.009 (0.096)	0.110 (0.095)	0.014 (0.049)	-0.075 (0.540)	0.144 (0.092)
Attended secondary school ^F	0.170*** (0.059)	0.211*** (0.059)	0.199*** (0.060)	0.015 (0.057)	0.040 (0.030)	0.277 (0.367)	0.002 (0.051)
Inverse Mills Ratio ^M	0.449** (0.208)	0.444** (0.203)	0.410** (0.205)	-0.072 (0.188)	-0.042 (0.101)	1.645 (1.275)	-0.370*** (0.141)
Inverse Mills Ratio ^F	-0.049 (0.087)	0.074 (0.089)	0.074 (0.090)	0.040 (0.079)	-0.012 (0.046)	0.649 (0.530)	-0.053 (0.080)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.339***	0.339***	0.339***	0.339***	0.339***	0.339***	0.336***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
<i>Panel D. Controlling for urban status, material wealth quintiles and province of residence</i>							
Attended secondary school ^M	-0.099 (0.098)	0.012 (0.099)	0.051 (0.099)	0.090 (0.094)	0.014 (0.051)	-0.602 (0.600)	0.069 (0.086)
Attended secondary school ^F	0.162*** (0.058)	0.126** (0.059)	0.107* (0.059)	-0.003 (0.055)	0.025 (0.030)	0.577 (0.377)	0.018 (0.050)
Inverse Mills Ratio ^M	0.232 (0.181)	0.307* (0.169)	0.277 (0.170)	0.036 (0.165)	-0.117 (0.088)	1.652 (1.098)	-0.295** (0.122)
Inverse Mills Ratio ^F	0.017 (0.084)	0.117 (0.078)	0.120 (0.080)	0.005 (0.078)	0.015 (0.045)	0.698 (0.511)	-0.094 (0.077)
$\rho_{(\varepsilon^M),(\varepsilon^F)}$	0.264***	0.264***	0.264***	0.264***	0.264***	0.264***	0.264***
N	6,214	7,821	7,862	7,109	7,061	7,835	5,755
Mean of dep.	0.71	0.68	0.68	0.32	0.06	5.71	0.12

Source: Authors' calculations from the Demographic and Health Surveys.

Notes: * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$. Robust standard errors clustered at the enumeration area level are in parentheses. Each regression controls for survey year fixed effects, for child's sex and age, for the two inverse Mills ratios, as well as for additional controls mentioned in each panel description.

Appendices References

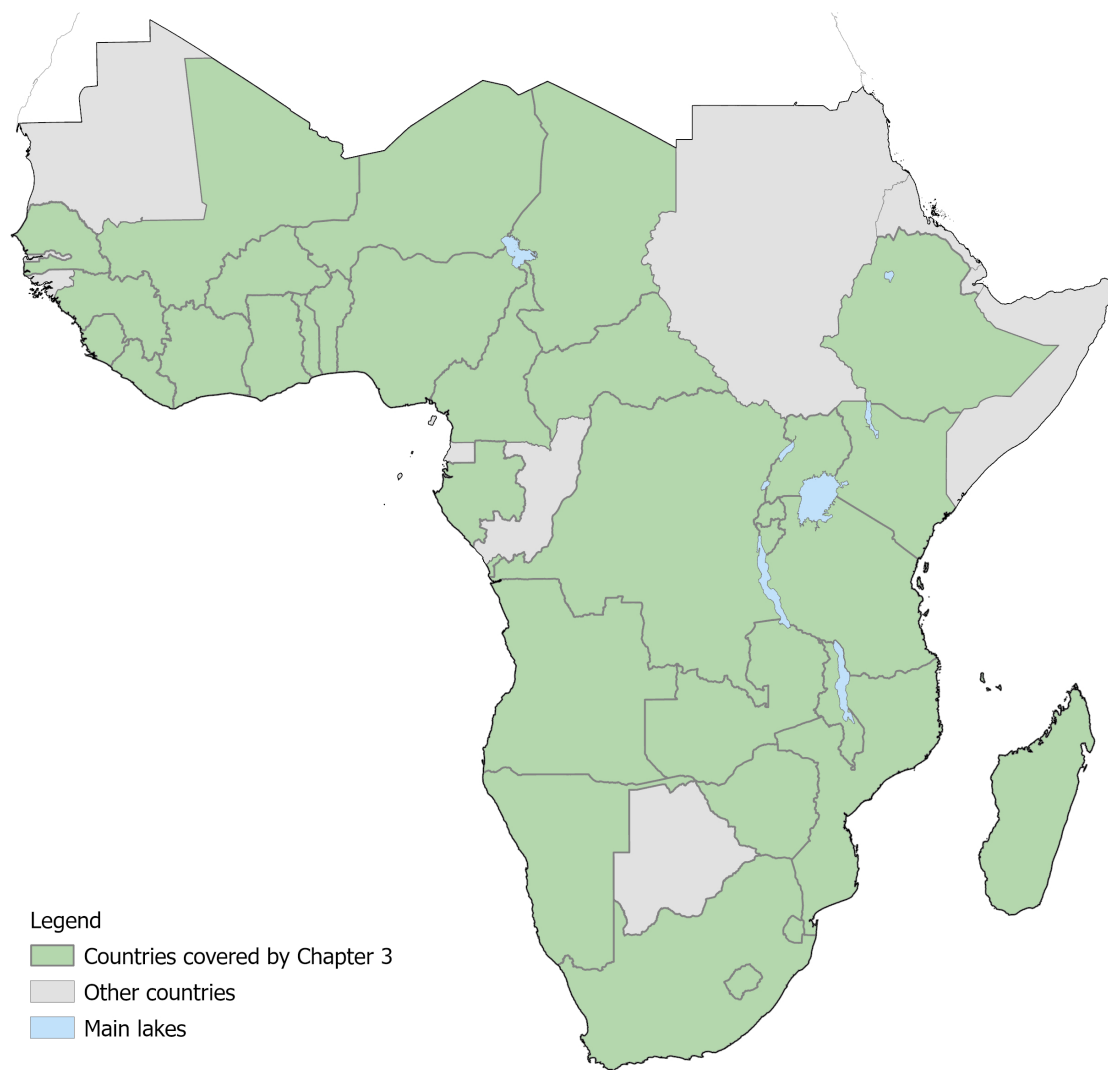
WORLD BANK (2022). World Development Indicators. Available at: <https://data.worldbank.org/indicator>.

Chapter 3

Dams and Health in Sub-Saharan Africa

A B S T R A C T

This chapter provides new evidence on the effect of dams on population health in Sub-Saharan Africa. Based on a representative sample of more than 2.5 million individuals from 34 countries matched to a unique record of dam construction over thirty years, I measure the effect of dam proximity on both child and adult health outcomes. Using river gradient as an exogenous source of variation for dam placement within an instrumental variable set-up, I find conflicting effects: child chronic malnutrition is significantly lower around dams, while malaria prevalence is higher. Overall, I find a 2.1 percentage points increase in post-neonatal mortality in the vicinity of dams. Importantly, the timing of this increase overlaps with a period in which babies' natural immunity against malaria acquired during childbearing fades away. Adults living near a dam also exhibit a higher prevalence of anemia, one of the main symptoms of malaria infection. Several channels are then explored and point to an increase in household material wealth in the vicinity of dams. This effect is potentially driven by labor-market effects since the probability of having a permanent job paid in cash is higher around dams. I also find better access to health care near dams but no effect on fertility behaviors. I finally investigate the role played by dam size and upstream dams. This chapter has important policy implications, which are by no means that dams should be banned, but that complementary policies are needed to mitigate their adverse effect on malaria risk for the local population.



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Acknowledgments specific to Chapter 3

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1. Introduction

Infrastructures are regularly seen as an essential element to bolster economic development. Dams are part of these infrastructures, especially in Sub-Saharan Africa, where safe access to water remains an issue. Dams being seen as a potential source of development, major investments have been made in this sector, with the support of several international organizations such as the World Bank. Either for irrigation, water access, protection from extreme precipitation events, mining waste containment, or hydropower generation, dams have flourished over the continent for over fifty years. In 2015, these infrastructures accounted for 20.5% of electricity production in Sub-Saharan (15.9% in the World) [World Bank, 2022]. They are also widely used for irrigation purposes: the most recent figures date back to 2000, when an estimated 30% to 40% of irrigated lands relied on dams, contributing to 12% of food production worldwide [World Commission on Dams, 2000]. There are approximately 60,000 large dams (height above 15 meters) in the World, and many unreferenced small dams built by communities to ensure a source of water for irrigation and domestic use.

A larger share of the population is expected to depend on dams in the near future, with a growing role of water storage solutions in the face of climate change. Their development in the years to come is a stated objective of the African Union within its Program for the Development of Infrastructures in Africa. While their contribution to hydropower and irrigation is undeniable, non-governmental organizations and newspaper articles regularly point to serious detrimental effects of dam construction on the local population through forced displacement and on the environment. Increased risks of disease near dams have also been reported.¹ Yet, there is no causal evidence of such potential adverse health effects in the vicinity of dams, nor on their contribution to local development over the continent.

The relationship between proximity to a dam and health is ambiguous. On the one hand, dams could have positive effects. Presence of a dam locally provides access to a more sustainable source of water, close to the villages, with a potentially beneficial effect on local agricultural production that may translate into a positive income effect. It might also reduce the opportunity cost of fetching water, often supported by women, allowing them to spend more time in income-generating activities. Such positive income effects may

¹ At the beginning of the twentieth century, the American press was already raising the idea that dams could be partly responsible for the malaria outbreak observed in the country at that time by providing a favorable environment for the proliferation of mosquitoes. Examples of newspaper articles are shown in Appendix Figure A.1. In 2000, the French newspaper *Le Monde* published an article depicting dams as a vector of sanitary disasters in African countries (Appendix Figure A.2).

then translate into higher nutritional intake and a substantial improvement in health status within the local population. On the other hand, dams could negatively affect the surrounding areas. Dams cause an increase in soil salinity with a negative effect on agricultural productivity, potentially leading to a negative income effect. They constitute a potentially contaminated source of water by parasites or fertilizers that can lead to waterborne diseases such as diarrhea or cholera. Dams also create open surface water bodies favorable to the development of infectious diseases such as malaria, one of the leading causes of death in Africa, or dengue. Large dams might also engender forced displacements of the local population, potentially leading to an impoverishment of households due to the loss of their land and social network, as well as large detrimental effects on mental health. Such effects could seriously threaten the health of the local population.

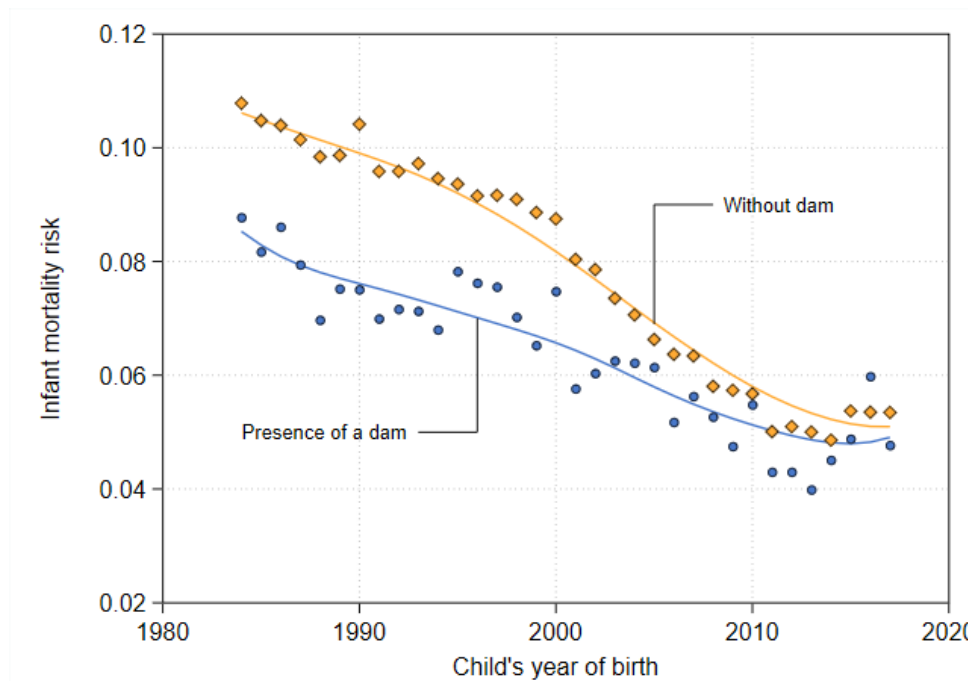
This work contributes to the literature on the local effects of infrastructure access in developing countries. It is among the few papers studying the effect of dams. [Duflo and Pande \[2007\]](#) find heterogeneous effects of large dams on agricultural productivity and rural poverty in India. While agricultural production increases and poverty decreases in districts located downstream from a dam, poverty rises in the district where the dam is located. Following their seminal paper, several papers have looked at the productivity effect of dams in Africa. [Strobl and Strobl \[2011\]](#) find that large dams have a positive effect on cropland productivity in downstream communities but have no effect in their direct vicinity. Focusing on South Africa, [Blanc and Strobl \[2013\]](#) show that this effect depends on the size of the dam: large dams negatively affect cropland productivity within their vicinity while small dams have a positive effect on it. Dam construction might also fuel intrastate conflict, as shown by [Eberle \[2020\]](#).

Two papers try to assess the causal effect of dams on infant mortality risk in developing countries. [Chakravarty \[2011\]](#) uses a two-way fixed effect approach and points to a heterogeneous effect of dams on infant mortality based on a sample of 17 African countries. Relative to those without a dam in their drainage basin, children residing in a basin immediately downstream from a dam experience a lower risk of mortality before reaching the age of one, and those located in a basin further downstream face an increased mortality risk. Infant mortality risk is also increased in the drainage basin where the dam is present. More recently, [Mettetal \[2019\]](#) focuses on dams built for irrigation purposes and finds that their construction has increased infant mortality in former homeland districts of South Africa after Apartheid, an effect driven by water pollution and water scarcity. However, we might wonder whether this result is specific to the context of South Africa,

given its high fertilizer use compared with other Sub-Saharan African countries.² None of these papers investigate other health outcomes, despite the fact that infant mortality may mask conflicting effects of dams on other health indicators such as nutrition and infectious diseases prevalence. Finally, these papers both define exposure to a dam at the district or river basin level, with the risk of underestimating the true local effect of dams, in particular of the small ones which may have very localized effects.

This chapter fills these gaps by using rich microdata and provides new evidence on the effect of dams on population health in Sub-Saharan Africa. Using health measures of over 1.7 million individuals in 34 countries combined with a unique record of dam construction over more than thirty years built for the purpose of this study, this is the first paper to explore the effect of exposure to a dam defined at the individual rather than drainage basin level. Figure 1 below shows the evolution of infant mortality risk computed from nationally representative surveys collected in these countries within 10 kilometers from a dam and in areas located further away.

Figure 1. Evolution of infant mortality over time



Source: Author's computations using birth history from the Demographic and Health Surveys conducted in 34 Sub-Saharan African countries and satellite data.

Notes: The figure plots the evolution of infant mortality risk in Sub-Saharan Africa over time for villages and city blocks located at less (blue dots) or more (yellow diamonds) than 10 kilometers from a dam.

² See Appendix D for maps of fertilizer use in Sub-Saharan African countries in 1980 and 2018.

Two facts emerge from this figure: infant mortality risk is systematically lower in the vicinity of dams over time, but the downward trend is steeper within unexposed areas. However, it is not possible to draw conclusions from these observations as one cannot disentangle at this stage the causal effect of dams from what is coming from their potential endogenous location. To overcome this issue, I use river gradient computed from satellite imagery as an exogenous source of variation to cope with the non-random placement of dams following the approach from [Duflo and Pande \[2007\]](#).

This chapter points to conflicting effects, with a significant reduction of child chronic malnutrition around dams accompanied by a higher risk of malaria transmission. Results also suggest a higher malaria prevalence among adults with an increase of their risk of being severely anemic, one of the main consequences of malaria infection. These findings are consistent with the higher post-neonatal mortality risk found around dams, a period during which babies' natural immunity against malaria acquired during childbearing shrinks.

I then explore several channels through which dams might impact child health. I observe that children have a higher probability of sleeping under a bed net when they live near a dam, suggesting that parents might be aware of their higher exposure to mosquito bites due to the presence of a dam and thus decide to invest in bed nets. However, other child health investment behaviors such as vaccination and iron supplementation are not sensitive to the presence of a dam. Household material wealth increases in the vicinity of dams with important labor-market effects: a higher share of individuals have a permanent job paid in cash rather than seasonal jobs not paid. I find no effect of dams on fertility behaviors but better health care access in their vicinity, potentially driven by positive effects on local development and the loosening of households' financial constraints.

Finally, I propose two extensions to the main analysis by investigating the potential heterogeneous effect of dams according to their size and the role of dams located upstream from the household. Results suggest that both small and large dams have similar effects on child mortality and malaria-related outcomes but that the improvement of child nutritional status is exclusively driven by small dams. However, there is no discernible impact of upstream dams on child health.

This chapter has important policy implications. If dams raise the risk of malaria infections, they should by no means be banned: the results point to substantial economic benefits of having a dam nearby and to a large reduction in child chronic malnutrition. However, their construction should be complemented with other policies to mitigate their adverse

effect on malaria risk for the local population.

The remainder of this chapter is organized as follows. Section 2 presents the data, and Section 3 depicts the empirical strategy. Results, robustness checks and heterogeneity analyses are presented in Section 4. Channels and two extensions to the main model are investigated in Section 5. Section 6 discusses the results and concludes.

*
* *

2. Data

The article combines georeferenced socioeconomic and health data collected from 34 countries with a unique record of dam construction in Africa over more than thirty years. The data covers 920,076 under-five children and 1,652,864 adults surveyed from 1986 to 2020 and contains information on 11,768 dams built until 2020.

2.1. Socioeconomic and health data

The socioeconomic and health data comes from pooling 108 geocoded, nationally representative, Demographic and Health Surveys (DHS) conducted across Sub-Saharan Africa from 1986 and available as of October 2021.³ It gives a dataset of repeated cross-sections covering 34 countries and 548,719 households located in 45,476 different villages or city blocks (enumerations areas, also called DHS clusters), as shown in Figure 2.

All women aged 15-49 who were either permanent residents of sampled households or visitors present on the night before the survey were eligible for survey interview. Men were only interviewed in a randomly-selected sub-sample of households. In households selected for male questionnaire, all men aged 15-59, and in some cases 15-54, who were either permanent residents or visitors were eligible for interview. I restrict the male samples to men aged 15-54 to harmonize the data.⁴ Overall, 1,173,470 women and 479,394 men were interviewed. More details are provided in Appendix B.1.

Women record the outcome of all pregnancies they had in the last five years with detailed information on childbirth conditions, as well as information on the health status

³ Starting in 1986, the georeferencing of DHS survey locations has been progressively extended to become standard practice since the early 2000s (see Appendix Figure B.1). Conclusions remain unchanged when focusing on the 86 DHS surveys conducted in 2000 or after (available upon request).

⁴ Out of 471,677 men aged 15-59 in the original samples, I exclude 15,582 men aged 55-59 (3.3%). Results remain unchanged if we further exclude the 26,351 men aged 50-54 to align with the age range of surveyed women (available upon request).

of their under-five children. I therefore restrict my analysis to these children. I use four kinds of health information: mortality indicators, reported health status regarding several pathologies, anthropometric measures, and biomarkers collected at survey time.

2.1.1. Child health

Child mortality.— A first indicator of child health is given by mortality status. Child mortality remains high in Sub-Saharan Africa: the subcontinent accounts for 48.4% of infant deaths in the World in 2020, and children are 12.5 times more likely to die within their first year of life there than in high-income countries. Premature deaths are measured by deaths during the first week of life as well as neonatal and infant mortality risks, that is the probability for a child to die during the first 28 days of life and before reaching the age of one year, respectively, based on the retrospective birth history reported by women.⁵ To further track the dynamic of child mortality in the vicinity of dams, I also estimate the effect on the risk of dying before 6 months and between 28 days and 12 months of life (post-neonatal mortality), and on monthly survival probabilities within the first four years of life. To avoid censoring bias, for each child mortality outcome, I exclude from the analysis children who did not reach the corresponding age at survey time.

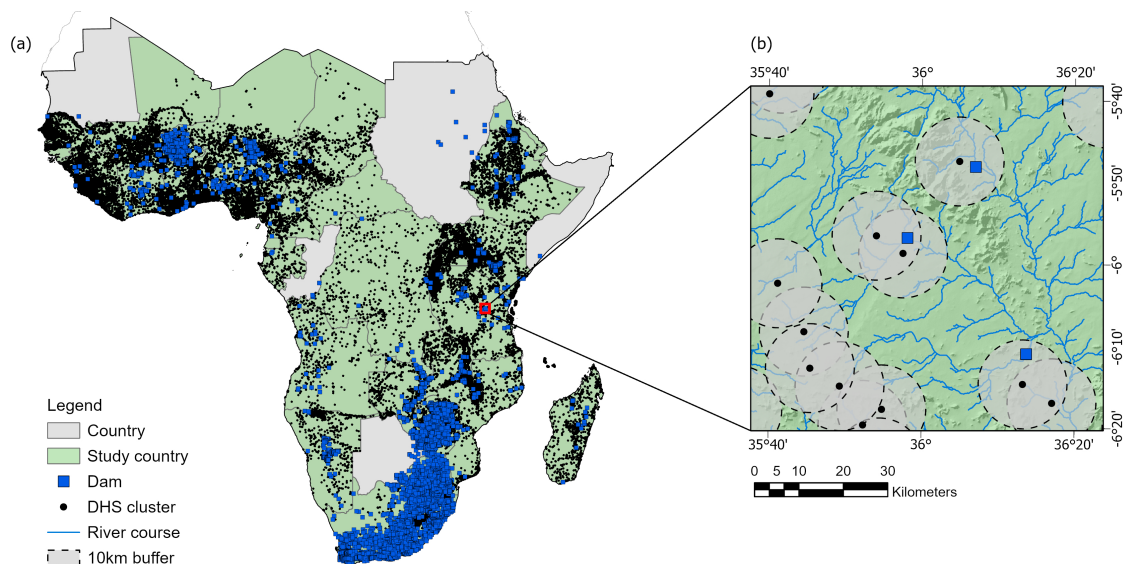
Nutrition.— Nutritional status is both a key marker of a child's health status and an important determinant of it. I use anthropometric measures taken at survey time to construct three nutritional z -scores following WHO standards, namely height-for-age (HAZ), weight-for-age (WAZ), and weight-for-height (WHZ) z -scores. Four indicators of malnutrition are derived from these measures, one each for being stunted (HAZ<-2), severely stunted (HAZ<-3), wasted (WHZ<-2), or severely wasted (WHZ<-3). Stunting and wasting are referred as measures of chronic and acute malnutrition, respectively, and are strong predictors of overall health status and mortality risk among under-five children. Of the original sample of under-five children alive at survey time, 66% were measured.

Women also report whether their child had diarrhea during the last two weeks preceding the survey. Diarrhea is one of the leading causes of under-five deaths in Sub-Saharan Africa, with 356,230 deaths in 2019, representing 13.3% of all under-five deaths [[Global Burden of Disease, 2019](#)]. Diarrhea is usually a symptom of an intestinal infection caused by contaminated food or drinking water. It is also referred as a strong marker of poor sanitation and hygiene.

⁵ As this information comes from the retrospective birth history reported by women, one concern is measurement error due to recall bias. I argue that recall bias can be considered low in this setting since the birth and death of a child are milestones in a woman's life, and the recall period of five years is relatively short.

Malaria.— Malaria is caused by parasites (*Plasmodium*) transmitted through the bites of infected female *Anopheles* mosquitoes and is one of the leading causes of under-five deaths across the continent, accounting for 12.9% of under-five deaths [Global Burden of Disease, 2019]. Sub-Saharan Africa carries the highest share of the global malaria burden: in 2019, 96.7% of all under-five malaria-related deaths were recorded in the subcontinent.⁶ Severe malaria often causes fever and headache. After the infective mosquito bite, parasites infect red blood cells, which burst. It leads to a fall in hemoglobin level that may increase the risk of severe anemia. The DHS offer a wide range of indicators to track the existence of a recent malaria episode. Women report whether their child had fever during the last two weeks preceding the survey. However, *Plasmodium* infections can be asymptomatic and may not be detected by fever-based surveillance, in particular in case of recurrent infections [White et al., 2014]. To overcome this issue, I make use of blood-based biomarkers collected at survey time. Hemoglobin concentration measurement is used to detect abnormally low levels of hemoglobin and severe anemia. I also exploit results from rapid diagnostic tests (RDT) and blood smear tests collected by the DHS to detect the presence of malaria parasites in under-five children. These biomarkers are however not collected in all survey waves.⁷

Figure 2. DHS clusters and dams in Sub-Saharan Africa, with a zoom in on Western Tanzania



Source: Author's computation

Notes: The map shows (a) the location of DHS clusters and dams in Sub-Saharan Africa, and (b) a zoom-in on Tanzania showing the 10 kilometers buffer around each DHS cluster and river courses.

⁶ In 2020, Sub-Saharan Africa was home to 96.3% of malaria cases and 97.6% of malaria deaths in the World following calculations based on WHO [2021].

⁷ Note that the results on child contemporaneous health outcomes are not significantly different when I restrict the sample to children tested for malaria parasites (results not shown available upon request).

2.1.2. *Child health investments*

One channel through which proximity to dams can affect children's health is the improvement of local economic conditions, which can in turn increase investments in children's health. Child health investments are here proxied by on-time vaccination against polio, tuberculosis (BCG), measles, and diphtheria-pertussis-tetanus (DPT). I also use information on whether under-five children slept under a bed net the night before the interview, were given iron supplementation during last week, and were dewormed in the last six months.

2.1.3. *Employment and wealth*

Dam construction may bring new economic opportunities for workers and, more broadly, bolster local economic development. These effects are assessed through employment and wealth variables. Employment variables refer to current working status, type of remuneration, and job seasonality. The DHS do not contain data on income or consumption but provide a material wealth index constructed via a principal-components analysis based on assets ownership and housing characteristics.

2.1.4. *Other variables*

Adult health.— Much less information is collected on adult health status and is not available in all survey waves. The health status of eligible adult household members is hence proxied by their hemoglobin level and anemia status. I also use anthropometry measures to compute the body mass index (BMI)⁸ but this information is unfortunately only collected for mothers of children born in the last five years preceding the survey. Overall, at least one health measure is collected for 791,196 adults in our sample.

Preventive behaviors, fertility, and health care access.— The DHS data contain information on preventive behaviors adopted at the household level and fertility behaviors. I use information on mosquito bed net ownership, which is potentially affected by the presence of a dam nearby. I explore several outcomes related to fertility, namely the number of births, birth spacing, and age at childbirth. Health care access is proxied by health facility delivery.

Child and household covariates.— Further information is used in the empirical analysis, including child's year of birth, sex, and a dummy for multiple births. Similar covariates are used at the adult level.

⁸ I trimmed the 763 observations for which the BMI is higher than 50.

2.2. Dams data

2.2.1. Dams database

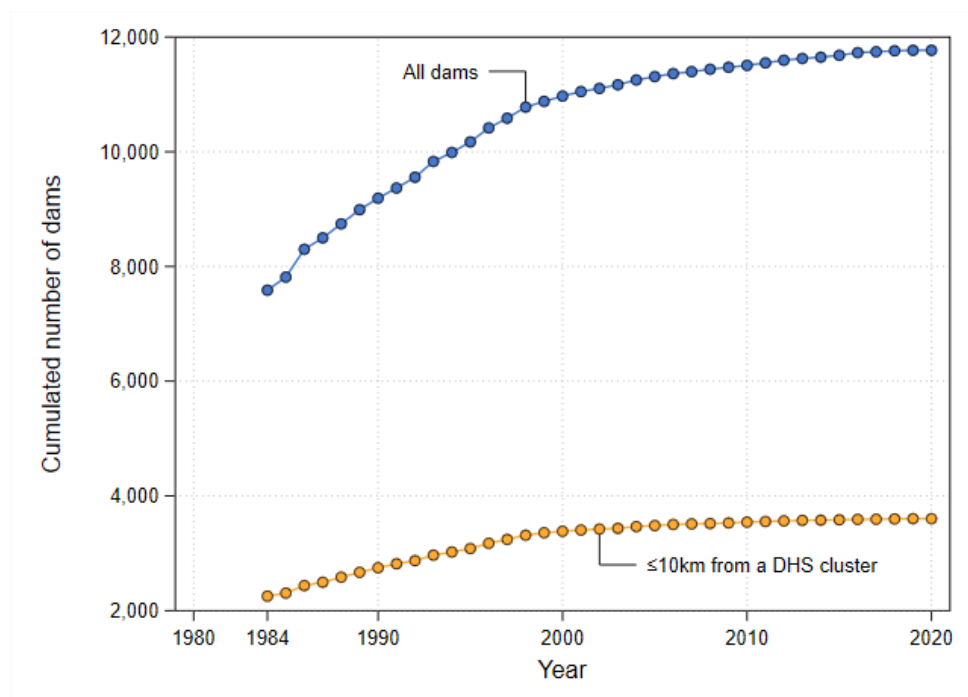
Several organizations maintain records of dam placement across the World. However, these sources are largely incomplete, either because they mainly focus on large dams (height of at least 15m) or because they lack information on dam construction date. This chapter overcomes these issues by creating a unique record of dam location and construction year across Sub-Saharan Africa, whatever their size.

I cleaned and combined data from the four main existing sources of dams location across the continent: the Global Reservoir and Dam Database (GRanD), the GLObal geOreferenced Database of Dams (GOODD), the FAO's African Geo-referenced Dams Database (AQUASTAT), and the South African Register of Large Dams.⁹ When the construction date was missing, I retrieved it based on historical monthly-based satellite imagery from the NASA/USGS Landsat program, starting 1972. However, the construction year cannot be accurately retrieved until 1984 due to the bad quality of satellite imagery beforehand. I complement the data by adding 4,189 unreferenced dams I found based on satellite imagery, for which I extract the exact location and construction year based on historical satellite imagery. More details can be found in Appendix B.2. Overall, it covers 11,768 georeferenced dams with information on construction year. It constitutes a unique database that is to date the most comprehensive source of dams information for Sub-Saharan Africa. Figure 2 maps the spatial distribution of dams in 2020¹⁰ while the evolution of the number of dams over time at the subcontinent level is shown in Figure 3. The total number of dams is multiplied by 1.5 between the mid-1980s and 2020, with a rapid expansion until the 2000s. About 30% of the dams are located within 10 kilometers of a locality sampled by the DHS.

⁹ After cleaning, 392 dams are from the GRanD database, 1,720 from the GOODD database, 196 from the AQUASTAT database and 5,271 from the South African Register of Large Dams (Appendix B.2).

¹⁰ We observe a very high density of dams in South Africa and Zimbabwe. The high concentration of dams in South Africa has both historical and climatic roots. While the history of South African dams dates back to the mid-17th century, large-scale investments were effectively launched from the beginning of the 20th century. The objective was to support growing cities and irrigation farming under semi-arid conditions. In particular, when the ostrich feather market collapsed in 1914, many South African farmers returned to farm crops, increasing the demand for water storage solutions. Recurrent and prolonged droughts have also precipitated the construction of dams. Government subsidies were also introduced to sustain the development of private irrigation schemes during the 1930s and 1940s. The full history of dam construction in South Africa can be found in van Vuuren [2012]. The Zimbabwean dam history is relatively less documented. Dating back to the beginning of the 20th century, dam construction was essentially spurred by the growing agricultural sector to ensure adequate water supply.

Figure 3. Evolution of the number of dams in Sub-Saharan Africa over time



Source: Author's computation based on the Global Reservoir and Dam Database (GRanD), the Global geOreferenced Database of Dams (GOODD), the FAO's African Geo-referenced Dams Database (AQUASTAT), the South African Register of Large Dams and own identification and processing based on historic satellite imagery.

Notes: The figure shows the evolution of the number of dams in Sub-Saharan Africa over time based on the year of construction. Construction year cannot be accurately assigned until 1984 due to the quality of satellite imagery beforehand.

2.2.2. Exposure to dams

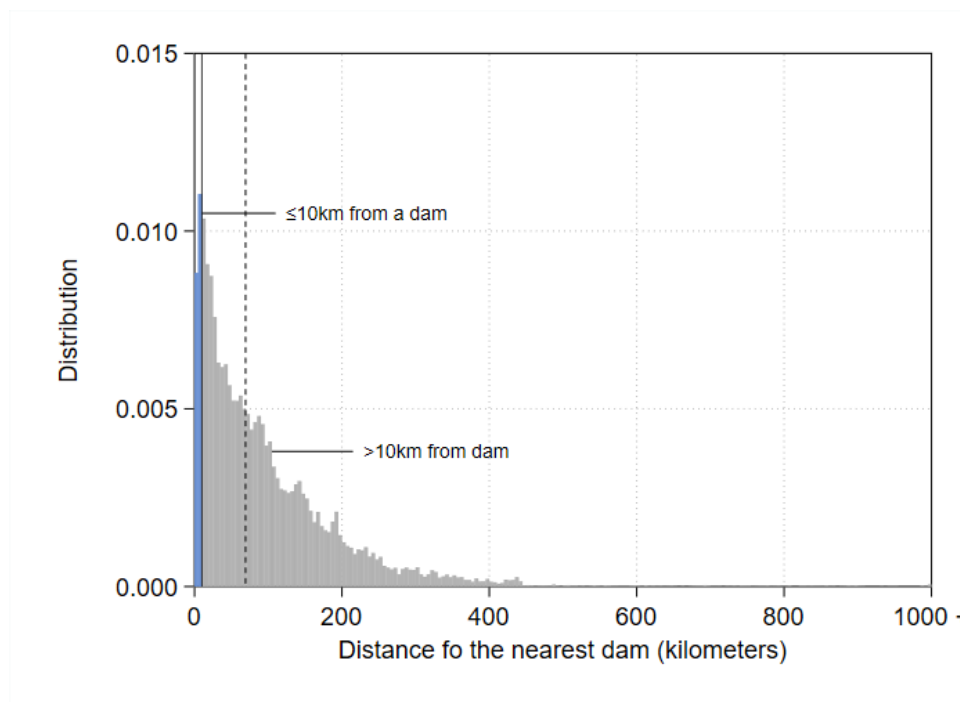
Exposure to dams depends on two dimensions: time and space. One departure from existing studies is the level of treatment assignment. Proximity to dams is here defined at the DHS cluster level, while all existing studies assign treatment at the district or river basin level. Assigning treatment at a too aggregated level can lead to the underestimation of the true local effect of dams, in particular of the small ones which may have very localized effects: individuals far from the dam but living in the same basin as it would be considered treated, although they are actually not affected by it.

To ensure respondents' confidentiality, the DHS randomly displace the geographic coordinates of the clusters. Urban clusters coordinates are displaced up to 2 kilometers from their true location, while rural clusters are displaced up to 5 kilometers and up to 10 kilometers for a further 1% of them. The DHS recommend using buffers larger than 5 kilometers to take into account GPS displacement.¹¹ A household is thus assigned to all dams present

¹¹ More details available here: <https://www.dhsprogram.com/GIS/Geographic-Data-Analysis.cfm> and in Appendix B.1.2 for the DHS scrambling procedure.

within 10 kilometers from it (see Figure 2(b)).¹² Figure 4 plots the distribution of the distance of DHS clusters to the nearest dam. The median distance to the nearest dam is 69 kilometers, and 9.7% of the 45,476 DHS clusters have at least one dam within 10 kilometers. For DHS clusters with at least one dam nearby, the median and average number of dams within 10 kilometers are 1 and 2.8, respectively.

Figure 4. Distribution of the distance to the nearest dam at the DHS cluster level



Source: Author's computations from DHS and satellite data.

Notes: The figure plots the distribution of the distance to the nearest dam at the DHS cluster level, in kilometers. Blue bars correspond to distances below or equal to 10 kilometers, for which clusters are considered near a dam. Gray bars correspond to distances above 10 kilometers. Dashed line represents the median of the distribution.

The time dimension will either refer to child's year of birth for mortality outcomes or to survey date for contemporaneous outcomes, including health measurements at survey time and employment variables. This dimension relies on the timing of DHS relative to dam construction. An individual is thus considered exposed if there is at least one dam within 10 kilometers from household location by survey time (by child birth, for child mortality outcomes).

Time variation is minimal: only 90 DHS clusters located at less than 10 kilometers from a dam were surveyed before its construction,¹³ and 59 within five years after it. As a result,

¹² I show as a robustness check that conclusions remain unchanged when using alternative radii around DHS clusters position to determine exposure to a dam (see Section 4.3.1).

¹³ It only represents 2% of DHS clusters located at less than 10 kilometers from a dam and 0.2% of all DHS clusters present in my sample.

variation in exposure to a dam within my sample almost exclusively comes from the spatial dimension.¹⁴

2.3. Geographic, hydrologic and topographic data

I extract topographic data from the digital elevation model (DEM) of NASA's Shuttle Radar Topography Mission (SRTM) at 3 arc-second resolution ($\approx 90\text{m}$ at the equator). Raw raster files contain elevation data in meters for each $90\text{m} \times 90\text{m}$ cell covering Africa, which I used to compute the average terrain elevation and slope within 10 kilometers around each DHS cluster.

I then derived the hydrologic network for the whole continent based on 3 arc-second SRTM data hydrologically conditioned by [Lehner et al. \[2008\]](#). It allows me to determine all river courses (drainage lines) position throughout the continent using advanced geographic information system (GIS) techniques and to calculate the total length of rivers around each DHS cluster. Based on SRTM data, I then compute the slope of the drainage lines.

The river network is finally used to delineate the watershed of each DHS cluster, that is the upstream area of land draining all the rivers and rainfall to the 10 kilometers buffer surrounding the cluster position. More details can be found in [Appendix B.3](#).

2.4. Summary statistics

Column 1 in [Table 1](#) shows the summary statistics for the whole sample of children, then disaggregated according to dam proximity (columns 2 and 3). Column 4 reports p -values obtained when comparing children near a dam and those far from it.

Material poverty appears to be lower in the vicinity of a dam than farther away, as children from DHS clusters located less than 10 kilometers from it have a 40% lower risk of belonging to the lowest two material wealth quintiles. There is also a significant difference in terms of topographic environment, with a higher elevation and a gentler gradient of both terrain and drainage lines around villages and city blocks located near a dam.

¹⁴ For this reason, I cannot use an identification strategy that relies on time variation such as a difference-in-differences approach or the comparison of already treated with not-yet treated localities.

Table 1. Summary statistics

	(1) Whole sample	(2) ≤ 10km from a dam	(3) > 10 km from dam	(4) (2) = (3) <i>p</i> -value
<i>Panel A. Child's characteristics</i>				
Girl	0.494	0.494	0.494	0.864
Age (years)	1.977	1.970	1.978	0.226
Lowest two quintiles of material wealth	0.465	0.288	0.479	0.000
<i>Panel B. Topographic characteristics</i>				
Elevation	660.478	813.822	647.985	0.000
Terrain gradient (%)	6.457	5.820	6.509	0.000
Drainage lines length (km)	78.542	78.072	78.580	0.000
Drainage lines gradient (%)	0.831	0.762	0.837	0.000
<i>Panel C. Dam exposure</i>				
Distance to nearest dam (km)	104.607	5.597	112.673	0.000
Exposed to a dam at birth	0.073	0.963	0.000	0.000
Number of dams at birth	0.180	2.389	0.000	0.000
Exposed to a dam by survey time	0.073	0.970	0.000	0.000
Number of dams by survey time	0.182	2.413	0.000	0.000
<i>Panel D. Mortality outcomes</i>				
Died within one week of life	0.026	0.024	0.027	0.000
Neonatal mortality	0.032	0.029	0.032	0.000
Died before 6 months of life	0.048	0.044	0.048	0.000
Infant mortality	0.063	0.058	0.063	0.000
Post-neonatal mortality	0.030	0.027	0.030	0.000
N	920,076	69,312	850,764	920,076
<i>Panel E. Nutritional status of child alive at survey time</i>				
Stunted	0.358	0.331	0.361	0.000
Severely stunted	0.164	0.143	0.166	0.000
Wasted	0.096	0.094	0.096	0.335
Severely wasted	0.035	0.038	0.034	0.000
Diarrhea episode during last two weeks	0.163	0.151	0.164	0.000
N	811,524	61,453	750,071	811,524
<i>Panel F. Malaria-related outcomes of child alive at survey time</i>				
Fever episode during last two weeks	0.244	0.220	0.246	0.000
Hemoglobin level [†]	10.261	10.431	10.247	0.000
Anemic [†]	0.641	0.598	0.644	0.000
Severe anemia [†]	0.036	0.029	0.037	0.000
Positive blood smear test [†]	0.182	0.244	0.179	0.000
N	796,879	61,131	735,748	796,879

Source: Author's calculations from DHS and satellite data.

Notes: Unweighted statistics. The first column reports summary statistics for the whole sample of reported live births (Panels A to D) and children alive at survey time (Panels E and F). The following two columns report the same statistics when the sample is restricted to households with (column 2) or without (column 3) a dam at less than 10 kilometers. Column 4 reports the *p*-values from *t*-tests on the equality of means reported in columns 2 and 3.

[†] Biomarkers collected for a sub-sample of children

The average distance to the nearest dam is 5.6 kilometers for communities located near a dam and 112.7 kilometers for the other ones. Among children located near a dam, 96% were born after dam construction and were thus exposed to it at birth. The figure for exposure at survey time is very similar (97%) since the sample is restricted to under-five children, and only 59 DHS clusters have been exposed to a dam for the first time within the last five years preceding the survey.

The probability for a child to die within her first week of life is 2.6% and the infant mortality rate 6.3%. Child mortality risks are of the same magnitude in both groups.

Average nutritional outcomes suggest a lower risk of chronic malnutrition measured by stunting prevalence around dams. A lower share of children had a reported fever episode during the last two weeks preceding the survey near dams. Hemoglobin levels are fairly similar across the two groups, with a slightly lower risk of anemia in the vicinity of dams. However, among children tested for malaria, the positivity rate is 36% higher in the vicinity of a dam than in areas located more than 10 kilometers from it, suggesting a potential adverse effect of dams on the local population.

*
* *

3. Identification Strategy

The objective of this article is to assess the local health effects of dams in Sub-Saharan Africa. One potential challenge in identifying a causal relationship between dams and health is the potentially non-random placement of dams.

3.1. Naive approach

Let Dam_{jtsbc} be a dummy variable for the presence of a dam within 10 kilometers from cluster j at time t surveyed in year s in basin b and country c .¹⁵ Time either refers to the year of birth for child mortality outcomes, or to the year of interview for contemporaneous ones. I could estimate the effect of the presence of a dam on outcome y_{ijtsbc} for individual i with the following model:

$$y_{ijtsbc} = \beta_0 + \beta_1 Dam_{jtsbc} + \mathbf{C}_{ij}\boldsymbol{\omega}_c + \mathbf{G}_{jt}\boldsymbol{\omega}_g + \eta_c + \eta_b + \eta_s + \eta_{cs} + \epsilon_{ijtsbc} \quad (1)$$

¹⁵ Note that drainage basins are not bounded by administrative boundaries and may span several countries.

where \mathbf{C}_{ij} is a set of individual characteristics, namely gender and year of birth, as well as a dummy for multiple births in the analysis of child mortality outcomes. \mathbf{G}_{jt} is a vector of geographic controls linked to local development consisting of population density, elevation, latitude and longitude (and their interaction), terrain slope, monthly precipitation and temperature, land suitability for food and cash crops, and total river length within 10 kilometers from cluster j , at time t .¹⁶ η_c and η_b denote country and drainage basin fixed effects, respectively, which absorb any time-invariant, country- and region-specific factors such as risks of disease, historical factors, and nature of the hydrological network. η_s denotes survey year fixed effects, which I interact with country fixed effects (η_{cs}). I additionally control for survey month and drainage basin \times survey month fixed effects in the analysis of contemporaneous outcomes to absorb any basin-specific seasonal effect. I will alternatively estimate the impact of the total number of dams at time t near cluster j .

Standard errors.— Standard errors are clustered at the DHS cluster level. Given the large number of health outcomes being investigated, I also present p -values adjusted for multiple hypothesis testing following Romano and Wolf [2005, 2016] in Appendix Table E.1. Reassuringly, inference based on Romano-Wolf p -values closely matches the one based on cluster-adjusted robust standard errors.

3.2. The non-random placement of dams

Despite the fact that a large set of potential confounding factors are already controlled for, some unobservables influencing dam placement may also affect individual health and economic outcomes. Specific localities may receive more dams for a bunch of reasons, including strategic and political ones, that are difficult to completely control for in Equation 1. For example, areas with higher crop production capacities or with growing industries may attract more dams for irrigation and hydropower purposes: in this case, the presence of a dam will be positively correlated with the error term ϵ_{ijtsbc} . Conversely, governments may favor the most deprived areas with low agricultural productivity and scarce access to water to bolster economic development. If such areas also lack adequate health infrastructures due, for example, to their remoteness, the presence of a dam will be negatively correlated with ϵ_{ijtsbc} . Such non-random placement of dams will lead the ordinary least squares (OLS) estimates of the parameter β_1 to be upward or downward biased relative to the true causal effect of dams.

I address the endogenous nature of dam placement through an instrumental variables (IV)

¹⁶ I control for total precipitation and average temperature during month of birth for child mortality outcomes, and during survey month for the other ones.

approach. Following [Duflo and Pande \[2007\]](#), the idea is to exploit river gradient as an exogenous source of variation in dams suitability. This identification strategy rests on two assumptions. First, DHS clusters with favorable river gradients for dam construction are more likely to have a dam nearby. Second, river gradient does not have a direct effect on health and economic outcomes other than through the presence of a dam, conditional on the covariates that are controlled for.

3.2.1. *The non-monotonic relationship between river gradient and its suitability for dams*

The suitability of a site for dam construction depends on topographic factors, including the gradient at which a river flows. The dam engineering literature defines a non-monotonic relationship between river gradient and its suitability for dam construction. A moderate river gradient is considered suitable for gravity-based irrigation dams, while a very steep river gradient is considered suitable for large hydroelectric dams. Flat river gradients are considered unsuitable as they will not carry sufficient water to the dam. Moderately steep river gradients are also unsuitable: they will carry too much water for the irrigation dam wall to resist but not enough to spin the turbines of a hydroelectric dam. [Duflo and Pande \[2007\]](#) uncover such a non-monotonic relationship in the Indian context: moderate (1.5 to 3 percent) and very steep (6 percent and above) river gradients are positively related to dam construction, while steep river gradients (3 to 6 percent) are negatively related to it, relative to flat ones (0 to 1.5 percent). Following their seminal paper, [Strobl and Strobl \[2011\]](#); [Blanc and Strobl \[2013\]](#); [Lipscomb et al. \[2013\]](#); [Mettetal \[2019\]](#) and [Eberle \[2020\]](#) have also used river gradient as an exogenous source of variation to predict dam placement.

In order to identify the causal impact of dams on local development, I exploit this non-monotonic relationship between a river gradient and its suitability for dam construction to instrument for the presence of a dam near a DHS cluster. I estimate Equation 1 by two-stage least squares (2SLS) where Dam_{jtsbc} is instrumented as follows:

$$Dam_{jtsbc} = \gamma_0 + \sum_{k=2}^5 \gamma_k RG_{jbc}^k + \mathbf{C}_{ij}\phi_c + \mathbf{G}_{jt}\phi_g + \eta_c + \eta_b + \eta_s + \eta_{cs} + \varepsilon_{jtsbc} \quad (2)$$

RG_{jbc}^k is the share of rivers within 10 kilometers from cluster j belonging to the k -th category of river gradient out of 5: flat (<0.5%), gentle (0.5 to 1.5%), moderate (1.5 to 3%), steep (3 to 6%) and very steep (6% and above) gradients. I adapt the slope categories from [Duflo and Pande \[2007\]](#) by separating flat and gentle gradients to better capture the many small dams built across Africa, including *sand dams* used by rural communities to

collect seasonal water for livestock, irrigation, and domestic use.

First-stage results.— I investigate this relationship in Table 2 where first-stage estimates are reported. The proportion of flat rivers within 10 kilometers corresponds to the omitted category. Results are in line with the engineering literature and echo those from [Duflo and Pande \[2007\]](#). As observed in columns 1 and 3, gentle and moderate river gradients, as well as very steep ones, are more likely to be associated with dam construction than steep rivers.¹⁷ This relationship persists when exploiting *within-country* and *within-basin* variation (columns 2 and 4).

These conclusions are consistent across both the under-five children (columns 1 to 4) and adults (columns 5 and 6) samples. Importantly, the Kleibergen-Paap F-statistic on the excluded instrument reaches values greater than 60, well above the rule of thumb of 10, suggesting that river gradient is a strong instrument.

I also report the effective F-statistic derived from the [Montiel Olea and Pflueger \[2013\]](#) weak instruments test, which is robust to heteroskedasticity and clustering and tests the null hypothesis that the two-stage least squares bias exceeds a fraction τ of the ordinary least squares bias. Effective F-statistics are well above the corresponding critical values for $\tau = 10\%$ and $\tau = 5\%$ for a significance level of 5%, suggesting that river gradient is not a weak instrument for dam placement.

Spatial Randomization Test.— To ensure these effects are not spurious, especially given the large sample size, I undertake a spatial randomization test. I estimate placebo distribution for each γ_k coefficient and for the Kleibergen-Paap F-statistic. Dams' locations are simultaneously and randomly displaced over the continent keeping the share of DHS clusters located less than 10 kilometers from a dam constant and equals to the one in the initial dataset. Dams' construction years are also randomized. Figure 5 plots the corresponding distributions based on 2,000 iterations. The p -values correspond to the probability of the point estimates and F-statistic shown in column 2 of Table 2 being drawn from these distributions. It confirms that the non-monotonic relationship observed in Table 2 is not driven by spurious correlation triggered by misspecification of the model.

¹⁷ Second-stage results remain the same when gentle and moderate river gradients are aggregated into a single category (results available upon request).

Table 2. First stage estimates

	(1)	(2)	(3)	(4)	(5)	(6)
	<i>Under-5 children</i>				<i>Adults</i>	
	Exposed		Exposed by survey time		Exposed by survey time	
<i>River Gradient</i>						
]0.5%; 1.5%]	0.128*** (0.011)	0.040*** (0.011)	0.124*** (0.011)	0.041*** (0.011)	0.144*** (0.012)	0.029** (0.012)
]1.5%; 3%]	0.094*** (0.021)	0.036 (0.022)	0.093*** (0.021)	0.039* (0.022)	0.133*** (0.023)	0.074*** (0.024)
]3%; 6%]	-0.117*** (0.022)	-0.302*** (0.023)	-0.127*** (0.022)	-0.299*** (0.023)	-0.191*** (0.025)	-0.364*** (0.025)
> 6%	0.163*** (0.021)	0.043* (0.022)	0.164*** (0.021)	0.052** (0.023)	0.207*** (0.022)	0.071*** (0.024)
Mean of dependent variable	0.073	0.073	0.073	0.073	0.092	0.092
Kleibergen-Paap F-statistic	68.473	72.904	66.099	68.212	84.547	74.543
Kleibergen-Paap p -value	0.000	0.000	0.000	0.000	0.000	0.000
R ²	0.119	0.293	0.120	0.293	0.159	0.342
N	919,508	919,508	919,508	919,508	1,651,464	1,651,464
Country FE		✓		✓		✓
Basin FE		✓		✓		✓
Country × Time FE		✓		✓		✓
<i>Montiel Olea and Pflueger [2013] test for weak instruments</i>						
Effective F-statistic	72.179	57.804	72.156	58.341	81.362	60.811
Critical value for $\tau = 5\%$	23.297	22.990	23.227	22.911	22.886	22.956
Critical value for $\tau = 10\%$	14.009	13.849	13.965	13.802	13.759	13.851

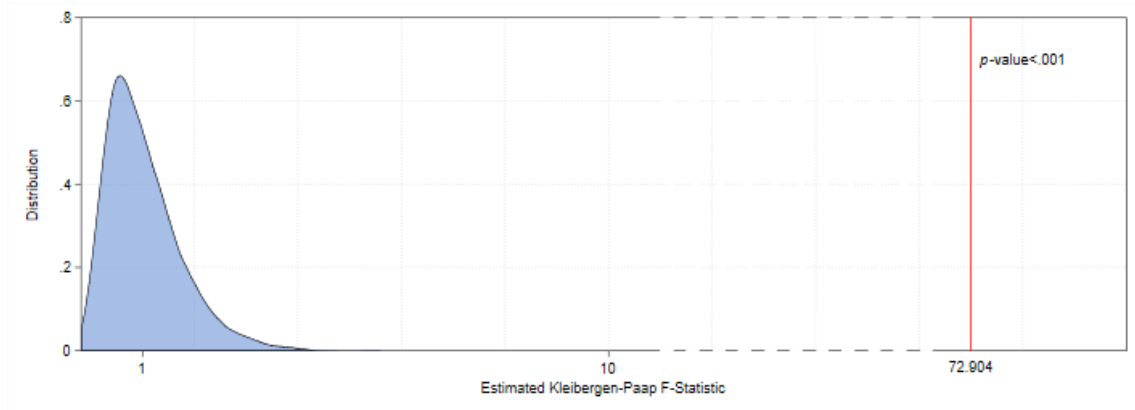
Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child (columns 1 to 4) or an adult (columns 5 and 6). The table reports the point estimates of river gradient shares RC_{jbc}^k on a dummy for the presence of a dam (columns 1, 2, and 5) or the number of dams (columns 3, 4, and 6) within 10 kilometers from DHS cluster j , following Equation 2. All regressions control for land gradient, elevation, population density, land suitability for crops, rivers length, rainfall and temperature within the 10 kilometers buffer. Population density, rainfall and temperature data are computed either for month of birth (columns 1 and 3) or survey month (columns 2 and 4 to 6). Other covariates include child's sex and year of birth, as well as cluster j 's geographic coordinates. Country, drainage basin, survey year and country × survey year fixed effects are included. τ corresponds to the fraction of the worst case bias used in the null hypothesis from the Montiel Olea and Pflueger [2013] weak instruments test for a significance level of 5%.

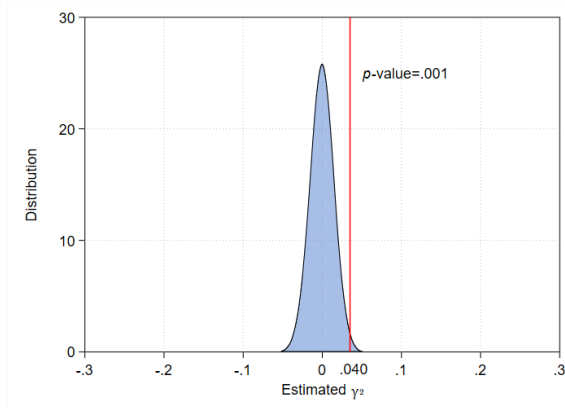
* $p < .10$; ** $p < .05$; *** $p < .01$

Figure 5. Spatial Randomization Test

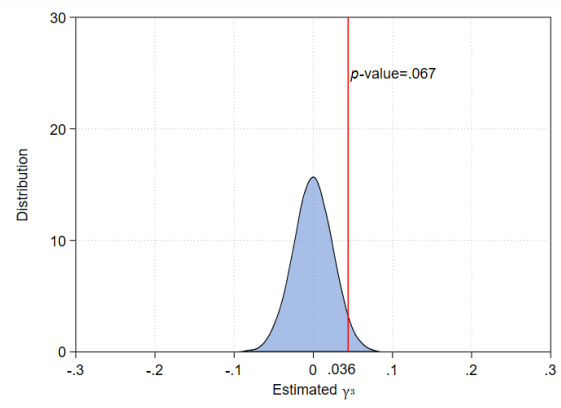
(a) Kleibergen-Paap F-statistic



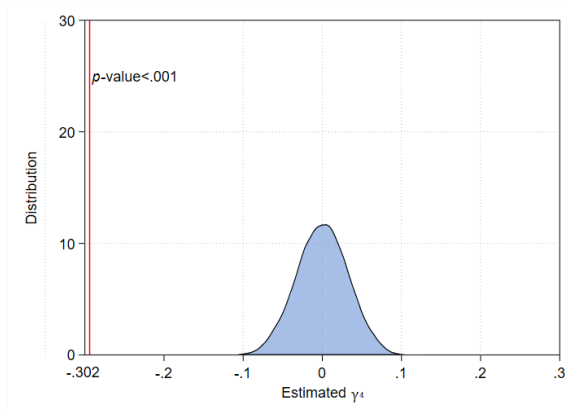
(b) Coefficient on]0.5%; 1.5%] gradient share



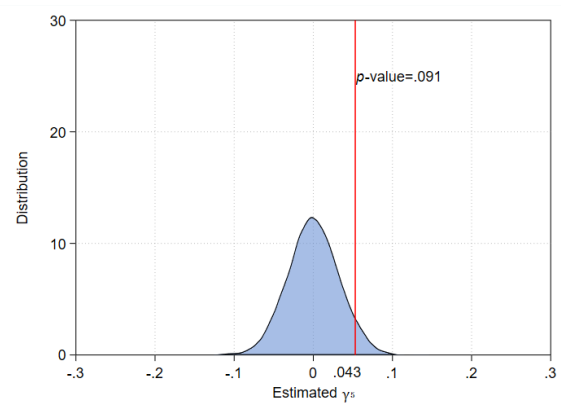
(c) Coefficient on]1.5%; 3%] gradient share



(d) Coefficient on]3%; 6%] gradient share



(e) Coefficient on > 6% gradient share



Source: Author's calculations from DHS and satellite data.

Notes: These figures plot the distribution of (a) Kleibergen-Paap F-statistic, and (b-e) the four γ_k coefficients from Equation 2 from 2,000 iterations. In each iteration, dams' locations were simultaneously and randomly displaced over the continent, keeping the share of DHS clusters located less than 10 kilometers from a dam constant. Dams' construction years were also randomly assigned. The reported p -values correspond to the probability of the point estimates and F-statistic shown in column 2 of Table 2 (red lines) being drawn from these distributions. Control variables and fixed effects are the same as in column 2 of Table 2.

Local Average Treatment Effect.— The parameter β_1^{2SLS} now captures the local average treatment effect (LATE) of exposure to dams built because of favorable local river gradient and which would not have been built otherwise, on the outcome y_{ijtsbc} . This effect may differ from the average treatment effect of dams β_1 as I do not identify the impact of dams built for other reasons, such as economic or political factors. However, what is interesting is that it measures the effect of *optimal* dams placed in the most suitable areas according to topographic and technological criteria. From policy makers' perspective, it makes the 2SLS estimates more informative than the OLS ones.

3.2.2. *Exclusion restriction and falsification exercise*

The identification strategy relies on the assumption that absent dam construction, river gradient has no effect on health outcomes. While this exclusion restriction seems reasonable, further precautions are needed as river gradient might be correlated with other topographic factors that can directly influence health and economic opportunities through the delivery of other types of infrastructures. In particular, land gradient has been shown to be a strong determinant of electricity grid location [Dinkelman, 2011], cellular coverage [Batzilis et al., 2010], as well as roads [Djemai, 2018] and sewerage systems [Bancalari, 2020] placement. Another threat to identification is the spatial distribution of population which might be correlated with river gradient. Flat rivers may be preferred for domestic use and agriculture on the riverbank.

To alleviate these concerns, I first control for land gradient, elevation, land suitability for crops, and population density around each cluster. By doing so, I make sure that my results are not driven by these characteristics.

Second, I undertake the following falsification exercise. Since presence of a dam should be the only channel through which river gradient affects health conditions of the local population, there should be no effect of river gradient on health in areas where no dam has been built. I check this by restricting my sample to DHS clusters located in river basins without dams and at least 20 kilometers from the nearest dam to prevent any spillover effect. I then run the following reduced-form model :

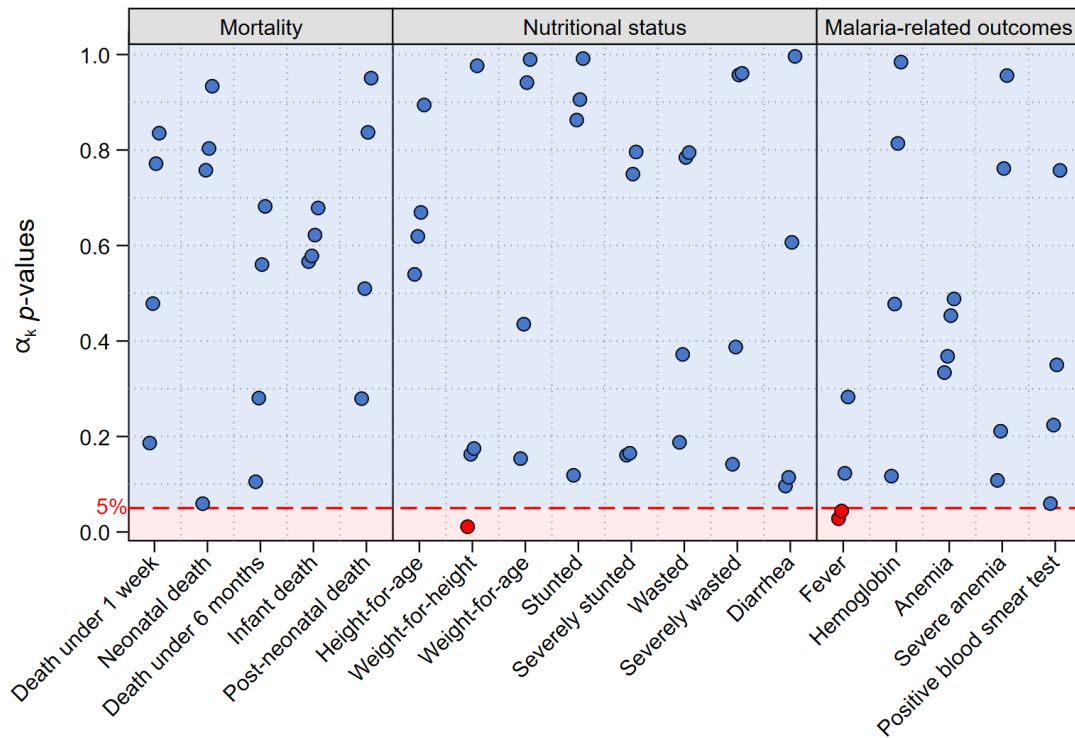
$$y_{ijtsbc} = \alpha_0 + \sum_{k=2}^5 \alpha_k RG_{jbc}^k + \mathbf{C}_{ij}\varphi_c + \mathbf{G}_{jt}\varphi_g + \eta_c + \eta_b + \eta_s + \eta_{cs} + \xi_{ijtsbc} \quad (3)$$

with the same notation as described above.

Significant coefficients α_k obtained from Equation 3 on this restricted sample will invali-

date the exclusion restriction. In contrast, non-significant α_k will support my identifying assumption. Figure 6 plots the p -values of each α_k t -test. Out of 64 coefficients on river gradient, only three are significant at the five percent level. It accounts for 4.7% of the point estimates, just below the 5% risk of type-I error inherent to hypothesis testing.

Figure 6. Falsification exercise



Source: Author's calculations from DHS and satellite data.

Notes: The figure plots the α_k 's p -values obtained for each outcome of interest based on Equation 3 with robust standard errors clustered at the enumeration area level. The sample is restricted to DHS clusters located in river basins without dams and at least 20 kilometers from the nearest dam to prevent any spillover effect. All regressions control for land gradient, elevation, population density, land suitability for crops, rivers length, rainfall and temperature within the 10 kilometers buffer. Population density, rainfall and temperature data are computed either for month of birth (child mortality outcomes) or survey month (contemporaneous outcomes measured at survey time). Other covariates include child's sex and year of birth, as well as cluster j 's geographic coordinates. Country, drainage basin, survey year and country \times survey year fixed effects are included. Survey month and basin \times survey month fixed effects are added for nutrition- and malaria-related outcomes measured at survey time.

These results suggest that in the absence of a dam, river gradient does not affect my outcomes of interest, which strongly supports the validity of the exclusion restriction and thus the causal interpretation of my results.

*
* *

4. Results

4.1. Child health

4.1.1. *Presence of a dam near a community has no effect on neonatal mortality but increases the risk of a post-neonatal death*

Table 3 reports the results from Equation 1 for child mortality outcomes. Whatever the indicator considered, the presence of a dam within 10 kilometers at birth is negatively correlated with child mortality, with a correlation of -0.003 for the probability for a child to die before reaching the age of one (Panel A, column 4). Despite the large set of covariates that are controlled for, exposure to dams might still be endogenous, causing the OLS point estimates to be biased.

Table 3. Effect of being born near a dam on child mortality risk

	(1)	(2)	(3)	(4)	(5)
	Died within one week of life	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality
<i>Panel A. OLS</i>					
Exposed at birth	-0.002** (0.001)	-0.002** (0.001)	-0.003** (0.001)	-0.003** (0.001)	-0.002** (0.001)
R ²	0.017	0.019	0.022	0.025	0.011
<i>Panel B. 2SLS</i>					
Exposed at birth	0.009 (0.012)	0.003 (0.013)	0.015 (0.017)	0.034* (0.020)	0.021* (0.012)
Kleibergen-Paap F-statistic	72.902	72.428	74.090	74.198	72.576
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Reduced-Form <i>p</i> -value	0.391	0.705	0.674	0.303	0.137
Mean of dependent variable	0.026	0.032	0.048	0.063	0.030
N	919,508	910,886	825,691	729,423	881,938

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a birth occurring during the last 59 months preceding the survey. The table reports the point estimates of exposure to a dam at birth from Equation 1 on child mortality, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, as well as population density, rainfall and temperature at birth within the 10 kilometers buffer. Other covariates include a dummy for multiple birth, child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year and country × survey year fixed effects are included. Children who did not reach the corresponding age at survey time are excluded to avoid censoring bias. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

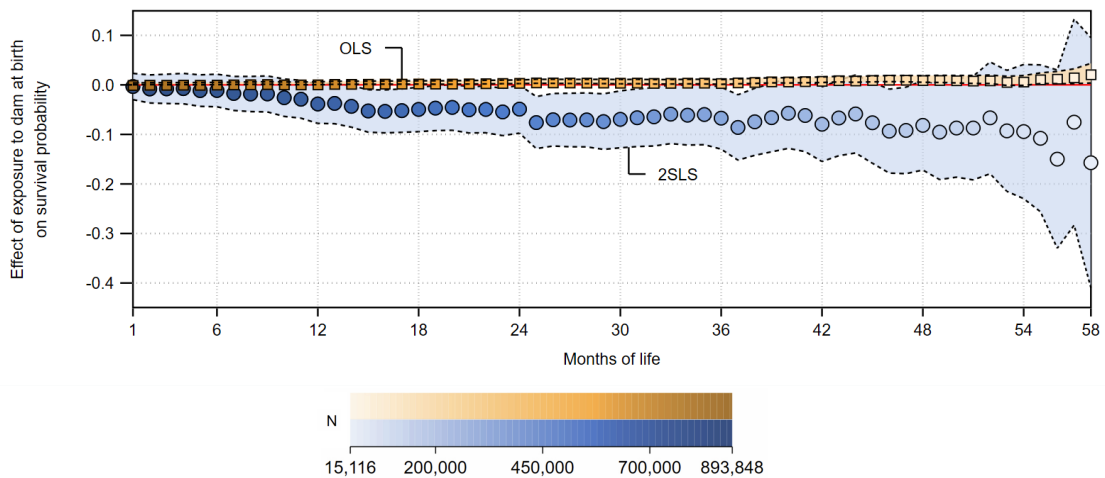
p*<.10; *p*<.05; ****p*<.01

When dam placement is instrumented by river gradient around the DHS clusters (Panel B), I find no discernible effect of proximity to a dam on the probability for a child to die

during her first week of life (column 1), nor before reaching the age of 28 days (column 2). However, column 5 indicates a 2.1 percentage points increase in post-neonatal mortality risk, that is the probability of dying after 28 days but before one year of life, resulting in a significantly higher risk of infant mortality in the vicinity of dams (column 4). These conclusions are not influenced by age at death heaping, as shown in Appendix Table F.1.

To investigate the exact timing of this effect, I estimate the impact of being born near a dam on monthly survival probabilities within the first four years of life. Figure 7 plots the corresponding point estimates and 95% confidence intervals. Until six months of life, dam exposure has no effect on a child’s survival chances, confirming the result from Panel B of Table 3. After six months, survival probability near dams starts to decrease and then stabilizes after 24 months of life. Note that this decline in survival chances near dams intervenes when young babies’ natural immunity against malaria acquired through their mothers during childbearing fades away. This is consistent with a potential effect of dam proximity on malaria risk.

Figure 7. Effect of exposure to dam at birth on survival probability



Source: Author’s calculations from DHS and satellite data.

Notes: This figure plots the point estimates and 95% confidence intervals of exposure to a dam at birth from Equation 1 on the probability to survive until a given month of life (from one month to 58 months) from OLS (yellow) or 2SLS (blue), the presence of dam being instrumented following Equation 2. The unit of observation is a birth occurring during the last 59 months preceding the survey. Each point is from a separate regression. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3. Children who did not reach the corresponding age at survey time are excluded to avoid censoring bias, which prevents from estimating the effect on the probability to survive until 59 months of life. Color intensity of the dots indicate the sample size on which the corresponding estimation is based.

The negative bias in OLS estimates confirms the non-random placement of dams. A potential explanation for this difference resides in the local nature of IV estimates, as

it measures the effect of proximity to a dam built because of a suitable river gradient. IV estimates exclude variation coming from dams built for other motivations, including political reasons such as ethnic favoritism or because of the initial level of development of the area, which is not the case for the average treatment effect measured by OLS. Overall, it suggests that part of the dams present in my data were built around localities with initially lower risk of child mortality.¹⁸

4.1.2. Children living near a dam have a better nutritional status than those living far from it

The effect of living near a dam on child anthropometric indicators is reported in Table 4. Child nutritional status is positively correlated with proximity to a dam (Panel A), with a point of estimate of 0.053 for the height-for-age z -score. Chronic malnutrition, captured by the risk of being stunted or severely stunted, is also smaller around dams.

IV estimates confirm that child nutritional status is causally determined by the presence of a dam nearby (Panel B). For instance, chronic malnutrition is 23.4 percentage points lower within 10 kilometers from a dam built because of a favorable river gradient (column 4) compared with areas located further away from it, and severe chronic malnutrition is 14 percentage points lower (column 5).

I find no association between the presence of a dam and wasting, which captures acute malnutrition (Panel A, column 6), a result confirmed by the IV estimate. Wasting is often referred as the result of a rapid deterioration of child's nutritional status over a short period of time, for example as a result of a diarrhea episode. One could have expected to observe an increase in acute malnutrition near dams if the water they contain is exploited for domestic use without further precaution. It seems that this is not the case here since there is no effect of the proximity to a dam on the reported cases of diarrhea (column 8).

Overall, these results point to large and positive effects of dam infrastructures on child nutritional status, with a decrease in chronic malnutrition that could compensate for their potential negative effects on other health outcomes.

¹⁸ Unfortunately, due to the almost nonexistent time variation in exposure to a dam outlined above, I cannot explore pre-treatment characteristics of DHS clusters exposed to a dam.

Table 4. Effect of living near a dam on child nutritional status

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Height-for-Age z-score	Weight-for-Height z-score	Weight-for-Age z-score	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Panel A. OLS								
Exposed by survey time	0.053*** (0.015)	0.004 (0.012)	0.048*** (0.012)	-0.017*** (0.004)	-0.012*** (0.003)	-0.001 (0.002)	-0.001 (0.001)	-0.004 (0.002)
R ²	0.109	0.085	0.108	0.085	0.065	0.054	0.036	0.048
Panel B. 2SLS								
Exposed by survey time	0.791*** (0.237)	-0.211 (0.184)	0.378** (0.184)	-0.234*** (0.063)	-0.140*** (0.050)	0.042 (0.032)	0.003 (0.018)	-0.013 (0.039)
Kleibergen-Paap F-statistic	53.374	53.352	52.102	53.374	53.374	53.352	53.352	60.914
Kleibergen-Paap p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Reduced-Form p-value	0.000	0.115	0.012	0.000	0.000	0.372	0.642	0.011
Mean of dependent variable	-1.395	-0.253	-0.996	0.358	0.164	0.096	0.035	0.163
N	550,980	543,046	560,336	550,980	550,980	543,046	543,046	804,442

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child alive at survey time. The table reports the point estimates of exposure to a dam by survey time from Equation 1 on child nutritional status, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster j 's geographic coordinates. Country, drainage basin, survey year, country \times survey year, survey month and basin \times survey month fixed effects are included. Reduced-form p -values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.
* $p < .10$; ** $p < .05$; *** $p < .01$

4.1.3. Malaria risk increases for children living in the direct vicinity of a dam

The results for malaria-related outcomes appear in Table 5. OLS estimates suggest no correlation between exposure to a dam and declared fever cases during the last two weeks prior to the survey, nor with anemia. However, for the subsample of children tested for malaria, I find a negative association between proximity to a dam and presence of malaria parasites in blood, suggesting a lower risk of infection in the vicinity of dams.

Here again, these estimates probably suffer from an endogeneity bias due to the non-random placement of dams. IV estimates convey a completely opposite message. Results from Panel B point to a large effect of dam exposure on malaria-related outcomes. Living within 10 kilometers of a dam decreases hemoglobin level by 1.089 g/dL, a 10.6 percent reduction relative to the average child hemoglobin concentration in my sample. This huge effect feeds through to a rise in anemia prevalence by 24.5 percentage points and a 5 percentage points increase in the probability of being severely anemic. Compared to the proportion of children with severe anemia, it represents a 139 percent increase.

Table 5. Effect of living near a dam on risk of malaria for children

	(1)	(2)	(3)	(4)	(5)
	Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test
<i>Panel A. OLS</i>					
Exposed by survey time	-0.004 (0.003)	0.013 (0.019)	0.002 (0.005)	-0.003 (0.002)	-0.047*** (0.009)
R ²	0.070	0.172	0.129	0.040	0.303
<i>Panel B. 2SLS</i>					
Exposed by survey time	0.074 (0.051)	-1.089*** (0.313)	0.245*** (0.084)	0.050** (0.023)	0.247*** (0.093)
Kleibergen-Paap F-statistic	60.938	38.130	38.130	38.130	16.609
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.000	0.000	0.000	0.001	0.000
Mean of dependent variable	0.244	10.261	0.641	0.036	0.182
N	795,714	258,036	258,036	258,036	102,499

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child alive at survey time. The table reports the point estimates of exposure to a dam by survey time from Equation 1 on child malaria-related outcomes, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year, country × survey year, survey month and basin × survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

p*<.10; *p*<.05; ****p*<.01

Malaria infections being one of the main causes of anemia due to the burst of infected red blood cells, these results are strongly suggestive of a rise in malaria risk around dams. This is exactly what column 5 indicates: living in the direct vicinity of a dam leads to a significant 24.7 percentage point increase in the probability of having malaria parasites in blood, according to microscopy.

There is no discernible effect on declared fever cases during the last two weeks preceding the survey. This result might be due to the fact that individuals regularly exposed to mosquito bites may develop asymptomatic forms of malaria, in which cases mothers will not declare a feverish child despite malaria infection detected by the blood smear test.

4.2. Adult health

Next, I examine whether adults living in the vicinity of a dam also exhibit a higher risk of malaria infection. Adults are not tested for presence of malaria parasites in blood in the DHS. I thus rely on hemoglobin level and anemia status as proxies for malaria exposure since other causes of anemia should not spatially differ according to dam proximity.¹⁹ Results are shown in Table 6. I find a positive association between exposure to a dam nearby and body mass index but no correlation with hemoglobin level. When the non-random placement of dams is considered, living near a dam significantly decreases adult hemoglobin level by 1.057g/dL (column 2), an 8.3 percent reduction relative to the average adult hemoglobin concentration in my sample. I also find risks of anemia and severe anemia in adulthood respectively 29.4 and 2.4 percentage points higher around dams (columns 3 and 4). These results are consistent with the higher vulnerability to malaria uncovered among under-five children (Table 5). However, dams have no causal effect on body mass index among women (column 1).

¹⁹ Among the subsample of 101,965 under-five children for whom hemoglobin level and blood smear test have been collected, I find that children tested positive for malaria have a hemoglobin concentration 1.12g/dL lower than those tested negative, as well as a probability of being anemic and severely anemic 21.9 and 6.6 percentage points higher, respectively.

Table 6. Effect of living near a dam on adult health

	(1)	(2)	(3)	(4)
	Body Mass Index	Hemoglobin level	Anemia	Severe anemia
<i>Panel A. OLS</i>				
Exposed by survey time	0.223*** (0.038)	-0.022 (0.015)	0.005 (0.003)	0.001 (0.001)
R ²	0.207	0.291	0.106	0.014
<i>Panel B. 2SLS</i>				
Exposed by survey time	-0.694 (0.569)	-1.057*** (0.251)	0.294*** (0.055)	0.024*** (0.009)
Kleibergen-Paap F-statistic	55.451	34.660	34.660	34.660
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.000	0.000	0.000	0.000
Mean of dependent variable	22.843	12.707	0.351	0.014
N	441,588	521,264	521,264	521,264

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is an adult, except in column 1 where the analysis is done among mothers of children born in the five years preceding the survey due to data limitation, and not currently pregnant. The table reports the point estimates of exposure to a dam by survey time from Equation 1 on adult health outcomes, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include adult's sex (except in column 1, where the analysis is only done for women due to data limitation) and year of birth, as well as cluster j 's geographic coordinates. Column 1 also controls for the interval between the last birth and survey time in months. Country, drainage basin, survey year, country \times survey year, survey month and basin \times survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

* $p < .10$; ** $p < .05$; *** $p < .01$

4.3. Robustness and heterogeneity

4.3.1. Robustness checks

Continuous definition of exposure to dams.— Alternatively, I use a continuous definition of exposure to dams instead of a binary exposure variable. For this, I compute the number of dams present within 10 kilometers from each DHS cluster at birth for child mortality outcomes and at survey time for contemporaneous ones based on construction years. Conclusions based on Appendix Tables F.2 to F.4 are unchanged. I also find that an additional dam within a 10 kilometers radius around the child's locality increases the probability of reporting a fever episode during the last two weeks prior to the interview by 5.4 percentage points (column 1, Appendix Table F.4).

Selective mortality.— Nutritional and malaria-related measures are by definition only collected for children alive at survey time. As we observe a significantly higher infant mortality

risk around dams, the effect of dam exposure on child contemporaneous health outcomes might be plagued by selection due to endogenous mortality. In particular, if dams are disproportionately harmful to *ex ante* weaker children, then survivors might be stronger than those who died prematurely. In that case, I will overestimate the positive effects I found on child nutritional status and underestimate the negative effect on malaria-related outcomes.

To check this possibility, I consider selection through survival in two ways. First, I weight observations according to their predicted survival probability at survey time following the inverse probability weighting approach. Second, I use the semi-parametric approach from [Cosslett \[1991\]](#) by adding centiles of predicted survival probabilities as additional covariates in Equation 1. However, it is not possible to implement the standard Heckman two-step procedure since the predictors of the selection equation will be the same as in the main model. Results and 95% confidence intervals are shown in Appendix Figure F.6. Whatever the method used, point estimates are not significantly different from the baseline results. It suggests that the two competing effects I find on child contemporaneous health outcomes are not driven by selective mortality.

Correction for spatial correlation of error terms.— Given the geospatial nature of the data, and in particular that several clusters may depend on the same dam, I check the robustness of inference when allowing for spatial correlation in error terms within a 100 kilometers radius around each cluster j following the correction proposed by [Conley \[1999\]](#) with a linear decay (Bartlett kernel). Corresponding confidence intervals are shown in Appendix Figure E.2 and confirm our main conclusions despite larger standard errors.

Additional robustness checks.— Results are virtually unchanged when controlling for a full set of maternal and households covariates, including mother’s years of education and age at birth, material wealth index, sex and age of household head as well as child’s rank of birth (Appendix Figure F.8) and when dropping one DHS country at a time (Appendix Figures F.9 to F.11). I also investigate how these effects vary with the distance used to define exposure to a dam. Due to the scrambling procedure applied by the DHS to maintain the confidentiality of the respondents, using distance thresholds below 10 kilometers will introduce noise in the estimation. Conversely, using larger distance thresholds leads to the risk of diluting point estimates since individuals considered as exposed may in reality be too far from the dam to benefit or suffer from it. Point estimates are reported in Appendix Figures F.12 to F.14 for distance thresholds ranging from 5 to 20 kilometers. As expected, the effect of dam exposure is progressively diluted as the distance threshold

increases. The increase in child mortality appears to be concentrated in the direct vicinity of dams. The positive effect of living near a dam on nutritional health tends to decline but remains significant at the 1% level for all the distances considered. However, the increase in the prevalence of malaria-related symptoms and positive blood smear test around dams and its significance progressively vanish as distance increases, potentially highlighting the highly-concentrated nature of the detrimental effects of dams.

4.3.2. *Heterogeneous effects*

I next explore potential heterogeneous effects of dams. To do so, I implement a control function (CF) approach in which I interact exposure to a dam with variables for which heterogeneity in treatment effect is suspected, denoted H_{ijtsbc} .²⁰ This approach relies on similar identification conditions as 2SLS, coincides with 2SLS in linear models but is more efficient than 2SLS in models with non-linear functions of the endogenous variable [Wooldridge, 2015]. This approach proceeds in two steps. The first one coincides with the first stage of 2SLS described by Equation 2 where exposure to a dam is regressed on excluded instruments along with covariates from the second stage. The second step then mimics Equation 1 but controlling for the first-stage estimated residuals $\widehat{\varepsilon}_{jtsbc}$ (the control function) instead of replacing exposure to a dam by prediction from the first-stage:

$$y_{ijtsbc} = \beta_0 + \beta_1 Dam_{jtsbc} + \mathbf{C}_{ij}\boldsymbol{\omega}_c + \mathbf{G}_{jt}\boldsymbol{\omega}_g + \eta_c + \eta_b + \eta_s + \eta_{cs} + \pi\widehat{\varepsilon}_{jtsbc} + \epsilon_{ijtsbc} \quad (4)$$

with $\beta_1^{CF} = \beta_1^{2SLS}$ in the linear model, as shown in Appendix Table G.1. A simple test of exogeneity of exposure to a dam then consists in testing the null hypothesis that $\pi = 0$ as $\widehat{\varepsilon}_{jtsbc}$ absorbs unobservables related to dam exposure. In addition to clustering at the enumeration area level, standard errors are bootstrapped with 500 replications to adjust for the two-step nature of the control function approach. An interesting feature of this approach is that one only needs to add one control function even when the model is non-linear in the endogenous regressor so that we can perform heterogeneity analysis through

²⁰ A first alternative is to split the sample according to the variable for which heterogeneity in treatment effect is suspected, run separate 2SLS regressions and then compare the different point estimates obtained. However, to be valid, such an approach requires the sample, instruments, and covariates to be balanced according to H_{ijtsbc} , otherwise the true difference in point estimates will be blurred by the heterogeneous effects of instruments and covariates. As a result, we do not employ this approach here. A second alternative is to interact exposure to a dam with H_{ijtsbc} in the second stage and to employ the interaction between the instruments and H_{ijtsbc} as additional instruments in the first stages. The main drawback of this approach is that it can lead to weak instruments if the effect of the instruments in the first stages is not heterogeneous with respect to H_{ijtsbc} , which "can be quite inefficient relative to the more parsimonious control function approach" (Wooldridge 2015, p. 429).

the following equation, controlling for H_{ijtsbc} in the first stage:

$$y_{ijtsbc} = \beta_0 + \beta_1 Dam_{jtsbc} + \beta_2 Dam_{jtsbc} \times H_{ijtsbc} + \beta_3 H_{ijtsbc} + \mathbf{C}_{ij}\boldsymbol{\omega}_c + \mathbf{G}_{jt}\boldsymbol{\omega}_g + \eta_c + \eta_b + \eta_t + \eta_{ct} + \widehat{\pi\varepsilon_{jtsbc}} + \epsilon_{ijtsbc} \quad (5)$$

where β_2 stands for any heterogeneity in effect of dam exposure with respect to H_{ijtsbc} .

I investigate potential heterogeneous effects of dams depending on the different Sub-Saharan African regions, whether the child lives in an agricultural household or not as well as child's sex and rank of birth. The effect of dams might vary between the main Sub-Saharan African regions as the geographical distribution of dams is essentially concentrated in Western and Southern Africa. Results reported in Appendix Figure G.5 suggest that this is not the case. Then we might expect the effect of dams to be different according to the main activity of the household. In particular, agricultural households might be the ones that benefit most from dam construction due to improved irrigation capacity but could also be the most affected by increased soil salinity. I define as agricultural each household in which at least one member surveyed by the DHS works in the agricultural sector. Results reported in Appendix Table G.2 suggest no heterogeneity in the effect on child mortality and nutritional status. However, the increase in malaria risk near dams is significantly higher for children from agricultural households than others, an effect potentially driven by higher proximity to irrigated land. Finally, there is no *a priori* reason why dams *per se* would impact children differently by gender or birth order. However, the decisions households make in response to presence of a dam nearby may vary across children, which is what I explore now. Results are reported in Appendix Table G.3 and Appendix Figure G.4. Overall, I observe no heterogeneity with respect to child's sex and rank of birth: the effects are equally shared between siblings, suggesting that parents are not arbitrating between their children because of the dam.

4.4. Size of the IV-OLS gap

Comparison of IV point estimates with OLS ones reveals sizeable differences that are usually interpreted as evidence against the validity of the instrument without further precaution. Such an interpretation could be misleading as it rests on the assumption that the OLS point estimate is informative of the true effect β_1 we try to uncover. Considering OLS point estimate to gauge the validity of the instrument hence implies to consider $\widehat{\beta_1^{OLS}}$ as close enough to β_1 to be a good benchmark. In that case, it assumes a low endogeneity of the explanatory variable, questioning the necessity to draw on an instrumental variable

approach. However, in some context, we can reasonably expect the endogeneity to be high due to omitted variables.

Four main reasons may explain the large discrepancy between IV and OLS point estimates: the instrument might be weak, the exclusion restriction might be violated, the OLS point estimates might be severely biased due to a large set of unobserved confounders and the compliers might be more sensitive to the endogenous explanatory variable than the rest of the population.

I here discuss several pieces of evidence to assess the plausibility of these different reasons. As shown in Section 3.2.1, river gradient is not a weak instrument as it is highly correlated with dam construction and the Montiel Olea and Pflueger [2013] test strongly rejects the null hypothesis of weak instrument. While it is not possible to test the validity of the exclusion restriction, this assumption seems reasonable given the large set of covariates that are controlled for and the falsification exercise conducted in Section 3.2.2, suggesting that the exclusion restriction is not violated. As discussed above, another potential explanation is that the effect of dam proximity on child health might be plagued by severe endogeneity, making the OLS point estimate unreliable for gauging the magnitude of the corresponding IV point estimate. Ciacci [2021] suggests to derive the amount of selection on unobservables proportional to selection on control variables δ that would be necessary for $\widehat{\beta}_1^{OLS}$ to reach $\widehat{\beta}_1^{2SLS}$ following Oster [2019]. A low value of $|\delta|$ is in favor of the IV estimate as it suggests that even a small selection on unobservables relative to observables can drive the OLS estimate to the same level as IV. Conversely, a large value of $|\delta|$ suggests that the IV estimate can only be reached under an implausibly high selection on unobservables compared to observables. I here extend this approach by extracting 100 equally-spaced values within the $\widehat{\beta}_1^{2SLS}$ 95% confidence interval and compute the corresponding δ for each draw. I then plot the corresponding distribution of $\widehat{\delta}$ for each outcome, which gives an idea of the selection on unobservables that will make the OLS estimate falls within the $\widehat{\beta}_1^{2SLS}$ 95% confidence interval. Results are shown in Appendix Figures H.1 to H.3. Of the 1,800 $\widehat{\delta}$ computed, 93.4% lie within $]-1; 1[$, suggesting that even a selection on unobservables lower than selection on observables is enough for the OLS estimate to reach values within the IV confidence interval. This result has two main implications. First, it suggests that the discrepancy between OLS and 2SLS estimates observed in Tables 3 to 5 is primarily driven by OLS estimates being skewed by a high selection on unobservables. This is confirmed by the relatively large magnitude of the estimated parameter $\widehat{\pi}$ from Equation 4 and the rejection of the null that $\pi = 0$ for most of the outcomes investigated (Appendix Table

G.1). Second, it supports the validity of river gradient as an instrument to address the non-random location of dams.

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* *

5. Channels and Extensions

5.1. Channels

I explore several pathways through which presence of a dam might influence individuals' health, including health investment behaviors, labor market, migration and fertility effects triggered by dam construction.

5.1.1. The increase in malaria cases occurs despite a higher possession and utilization of mosquito bed nets around dams

Table 7 reports the effect of dam exposure by survey time on child health investments. There is no discernible effect of living near a dam on child up-to-date vaccination (column 1) nor on the probability of taking iron supplementation during the last week preceding the survey (column 4). Vaccination of children is influenced by both demand- and supply-side constraints. While potential income effects triggered by dams might, to some extent, lift the financial barrier to vaccination for households, vaccine stockouts and other logistical constraints may prevent such health investment.

Exposure to a dam reduces the probability of deworming over the last six months, an effect potentially driven by the better nutritional status of children living near a dam. Presence of a dam nearby has a strong positive effect on both ownership and utilization of mosquito bed nets (columns 2 and 3). This is potentially driven by the fact that parents may realize that the risk of mosquito bite is higher in the vicinity of dams and thus invest in nets. Nonetheless, it is not sufficient to avoid malaria transmission, given the increased probability of being tested positive for malaria.

Table 7. Effect of living near a dam on health investments

	(1)	(2)	(3)	(4)	(5)
	Up-to-date vaccinations	Bed net ownership	Bed net use	Iron supplementation	Deworming
<i>Panel A. OLS</i>					
Exposed by survey time	0.001 (0.003)	0.011** (0.005)	0.012** (0.005)	0.007 (0.005)	0.007 (0.005)
R ²	0.258	0.277	0.254	0.113	0.239
<i>Panel B. 2SLS</i>					
Exposed by survey time	0.063 (0.053)	0.302*** (0.076)	0.339*** (0.081)	-0.174 (0.118)	-0.127* (0.071)
Kleibergen-Paap F-statistic	60.642	49.831	46.358	18.491	42.331
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.002	0.000	0.000	0.003	0.000
Mean of dependent variable	0.342	0.628	0.414	0.130	0.420
N	731,066	455,139	672,633	492,465	561,822

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child, except in column 2 where the analysis is done at the household level. The table reports the point estimates of exposure to a dam by survey time from Equation 1 on wealth and child health investments outcomes, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster j 's geographic coordinates. Country, drainage basin, survey year, country \times survey year, survey month and basin \times survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

* $p < .10$; ** $p < .05$; *** $p < .01$

5.1.2. *Jobs are less seasonal and more often paid in cash, leading households to be better off in the vicinity of dams*

Next, I check whether there exists a positive wealth effect of dams on surrounding communities that may explain the increased nutritional status of young children. I investigate this point by looking at both the material wealth index and employment variables. Results are shown in Table 8. Working status is positively correlated with the presence of a dam (Panel A, column 1), but I find no causal effect on it (Panel B). However, the local labor market is significantly modified around dams in terms of the type of employment and remuneration. Jobs tend to be less seasonal (column 5) and more often paid in cash (column 2), the share of job not paid being significantly lower than in labor markets far from dams (column 4). Wealth is also positively associated with the presence of a dam nearby, a conclusion that still holds when dam exposure is instrumented (Panel B). On average, the material wealth index is 18% higher in the vicinity of dams. Households thus tend to be better off around dams, which might explain the positive effect found on nutritional status and bed net ownership.

Table 8. Effect of living near a dam on wealth and labor market opportunities

	(1)	(2)	(3)	(4)	(5)	(6)
	Currently working or worked in the past year	Paid in cash	Paid exclusively in-kind	Not paid	Seasonal, occasional or temporary job	Log- wealth index
Panel A. OLS						
Exposed by survey time	0.011*** (0.003)	0.038*** (0.004)	-0.006** (0.002)	-0.033*** (0.005)	-0.032*** (0.004)	0.015*** (0.002)
R ²	0.204	0.202	0.086	0.187	0.166	0.231
Panel B. 2SLS						
Exposed by survey time	-0.026 (0.045)	0.332*** (0.069)	-0.045 (0.036)	-0.287*** (0.067)	-0.467*** (0.069)	0.180*** (0.019)
Kleibergen-Paap F-statistic	63.282	59.821	59.821	59.821	60.423	69.533
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.752	0.000	0.000	0.000	0.000	0.000
Mean of dependent variable	0.697	0.676	0.052	0.272	0.414	3.003
N	1,538,027	997,807	997,807	997,807	1,031,612	1,620,023

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is an adult. The table reports the point estimates of exposure to a dam by survey time from Equation 1 on wealth and child health investments outcomes, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include adult's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year, country × survey year, survey month and basin × survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero. * $p < .10$; ** $p < .05$; *** $p < .01$

5.1.3. Migration behaviors

Dams may bolster economic migration if individuals decide to move near dams to benefit from the enhanced labor market conditions observed in their vicinity. The data reveals that migration patterns are in fact different near dams. In communities located close to a dam, 66.3% of surveyed mothers said they have migrated to their current place of residence,²¹ which is 8.1 percentage points more than in communities without a dam. If migration *per se* is not a threat to identification, selective inward and outward migration spurred by dams are potential concerns.

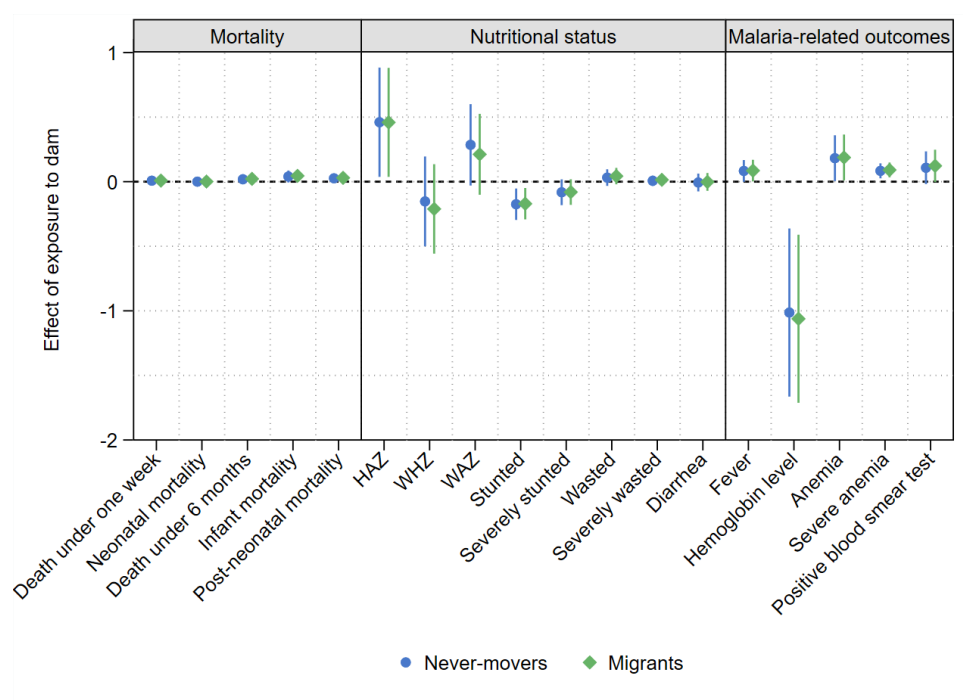
Population composition effects due to migration might affect my results. Inward migration is a potential issue. For example, specific households may decide to move near a dam to benefit from the enhanced labor market conditions in its vicinity. In terms of child health, selective inward migration of households with children more at risk of dying prematurely or more likely to be infected by malaria might also drive my results. For child nutritional status, it might be the case that children from migrant households have, on average, a worse nutritional status that may lead to the underestimation of the true effect of dams on chronic malnutrition.

To check this possibility, I exploit the limited information on migration available in the DHS, which was not collected during DHS Phase VI surveys (end of the 2000s-early 2010s). Of the 351,167 households for which this information is available, 63.3% were living for at least ten years in their current place of residence, and 43.3% have never moved. I estimate Equation 5 to check whether the net effect of dams on child health differs according to migration status (never movers vs. inward migrants).

Figure 8 plots the corresponding point estimates and 95% confidence intervals. Results are not significantly different between migrants and never movers. The pattern is similar to what I find in Table 3: there is essentially no effect of dams on child mortality very early in life, but a positive effect during the post-neonatal period that drives the increase in infant mortality risk. Overall, it suggests that inward migration is not the mechanism at play. It confirms the intuition from [Mettetal \[2019\]](#) in the context of South Africa.

²¹ We might expect this figure to vary with the size of the dam if large dams generate larger effects than smaller ones, but I find that the share of migrants in the community does not meaningfully differ according to the size of the dam (66.2% to 67.4%). Section 5.2 further explores the role of dam size.

Figure 8. Heterogeneous effect according to migration status



Source: Author's calculations from DHS and satellite data.

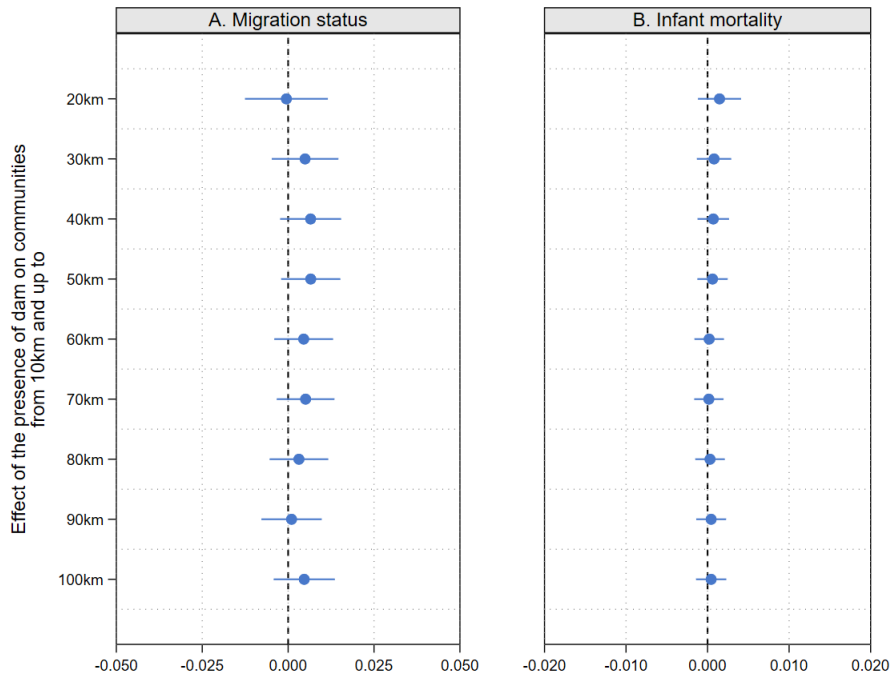
Notes: This figure plots the point estimates along with 95% confidence intervals of the effect of exposure to a dam for never movers (circles) and migrants (diamonds) following Equation 5. Bootstrapped standard errors (500 replications) are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3, 4 or 5 depending on the outcome.

Selective outward migration is also a potential concern. For example, households particularly aware of the increasing risk of malaria infection near open surface stagnant water may decide to move away from the dam to reduce their exposure to mosquito bites. More generally, households particularly cautious about their child's health and health investments may adopt such behavior to avoid the expected adverse effects of dams. If this is the case, then one might observe a higher risk of child mortality around dams only due to a composition effect.

It is not possible to completely rule out this concern since the DHS do not collect information on place of origin, but I provide suggestive evidence that it was not the case. To investigate this point, I exclude from my sample households located less than 10 kilometers from a dam. Then, I check whether there is an abnormally high share of migrants and low infant mortality risk in communities located from 10 kilometers and up to 100 kilometers from a dam after its construction compared with communities further away. In that case, it would suggest that dams trigger selective outward migration. Point estimates are reported in Figure 9. I find no evidence of selective outward migration to surrounding

areas: the share of migrants and infant mortality risk are not significantly different within communities located around dams but not in their direct vicinity than within communities located further away.

Figure 9. Migration rate and infant mortality risk in dams' surrounding areas



Source: Author's calculations from DHS and satellite data.

Notes: These figures plot the point estimates along with 95% confidence intervals of the effect of living between 10 kilometers and up to a given distance (y-axis) from a dam on (A) migration status and (B) infant mortality risk. Individuals residing at 10 kilometers or less from a dam are excluded. Each point is from a separate regression. Robust standard errors are clustered at the enumeration area level. Control variables include land gradient, elevation, land suitability for crops, rivers length, cluster j 's geographic coordinates, as well as child's sex and year of birth. Population density, rainfall and temperature within the 10 kilometers buffer (A) at survey time or (B) at birth are also controlled for, as well as a dummy for multiple birth in the estimation of the effect on infant mortality risk. Country, drainage basin, survey year and country \times survey year fixed effects are included. For infant mortality, children who did not reach the age of one at survey time are excluded to avoid censoring bias.

5.1.4. Fertility behaviors and health care access

The fertility effect of dams is unclear and must be empirically assessed. Fertility behaviors might differ around dams as they can be both a response to and a consequence of dam proximity. On the one hand, the higher infant mortality risk observed near dams may spur fertility through a replacement effect of deceased children. On the other hand, the enhanced local economic conditions are likely to have both a substitution and an income effect: they may exacerbate the opportunity cost of parenting but also bring additional resources that couples may decide to devote to having more children.

Table 9. Effect of dams on fertility behaviors and health care access

	(1)	(2)	(3)	(4)	(5)
	Preceding birth interval	Preceding birth interval < 24 months	Mother's age at child birth	Number of reported births per year per 100 surveyed mothers	Institutional delivery
<i>Panel A. OLS</i>					
Exposed	1.154*** (0.161)	-0.007*** (0.002)	-0.232*** (0.047)	-0.495*** (0.095)	0.036*** (0.005)
R ²	0.058	0.026	0.017	0.073	0.276
<i>Panel B. 2SLS</i>					
Exposed	-1.249 (2.594)	0.025 (0.030)	-0.781 (0.678)	2.051 (1.350)	0.270*** (0.075)
Kleibergen-Paap F-statistic	67.485	72.866	72.881	82.405	72.421
Kleibergen-Paap p-value	0.000	0.000	0.000	0.000	0.000
Reduced-Form p-value	0.000	0.039	0.000	0.009	0.000
Mean of dependent variable	38.815	0.153	26.614	20.486	0.557
N	725,169	919,475	919,515	230,960	881,826

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a birth (columns 1 to 3 and 5) or an enumeration area \times birth year cell (column 4). Column 1 is further restricted to non-first born children as there is no preceding birth interval for them. Preceding birth interval is considered as being above 24 months for first born children in column 2. The table reports the point estimates of exposure to a dam from Equation 1 on fertility outcomes and institutional delivery, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density, as well as rainfall and temperature at birth within the 10 kilometers buffer and cluster j 's geographic coordinates. Country, drainage basin, survey year, birth year and country \times survey year fixed effects are included. Column 5 additionally controls for a dummy for multiple birth. Reduced-form p -values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

* $p < .10$; ** $p < .05$; *** $p < .01$

In columns 1 to 3 of Table 9, I explore how exposure to dam affects preceding birth interval and mother's age at birth. I then transform my cross-sectional birth-level data into a panel at the enumeration area \times year of birth level to investigate the effect of dams on the number of reported births per year per 100 surveyed mothers (column 4). OLS estimates suggest that women tend to be younger at childbirth and to have fewer children with longer birth intervals near dams (Panel A). However, I find no causal effect of proximity to a dam on fertility outcomes (Panel B), suggesting that the health impacts of dams are not driven by fertility behaviors.

Finally, dams might increase health care access through improved local economic conditions and by relaxing households' financial constraints. This is what I find, as shown in column 5 of Table 9. The probability of giving birth in a health facility is significantly higher around dams.²² Nevertheless, the contribution of health care access to child health improvement could be small as returns to formal health services might be limited [Renard, 2022].

5.2. The role of dam size

The effect of dams on child health might depend on their size. Soil salinity and mosquito density may be higher near large dams, but small dams may accumulate insufficient amounts of water to support the agricultural sector throughout the year. Blanc and Strobl [2013] have shown that large dams have a negative effect on cropland productivity within their vicinity, while small dams have a positive impact on it: their effect on nutritional status might thus be heterogeneous.

5.2.1. Empirical approach

Estimation of dam size – Unfortunately, information on dam size is not available in the data. To overcome this issue, I construct a proxy for dam size using mean annual runoff in the dam's watershed as a measure of the total quantity of water draining into the dam reservoir. To do so, I exploit total monthly runoff data provided by NASA since 2000 using satellite and ground-based observational data from the Global Land Data Assimilation System (GLDAS). More information can be found in Appendix I. I then compute the mean annual runoff accumulation to the dam in cubic meters. Appendix Figure I.2 plots the corresponding distribution. To investigate the respective effect of small and large dams,

²² Alternatively, I estimate the effect of dam exposure on a mother's probability of reporting a visit to a health facility during the last 12 months preceding the survey and find a 30 percentage point increase in doing so (result not shown). However, the interpretation of this effect is tricky as we cannot disentangle an increase in preventive and regular consultations from a potential rise in curative consultations driven by a higher prevalence of specific diseases around dams. For this reason, I prefer to use institutional deliveries as a proxy for health care access in the area.

I then create an indicator for large dams based on the distribution of this proxy, using the 80th, 85th and 90th percentile as potential thresholds to ensure the results are not driven by the choice of the threshold.

Overall, 3.1% of under-children are only exposed to small dams at survey time, 2.8% to large dams, and 1.4% to both types when using the 80th percentile of the distribution. Appendix Table I.3 report similar figures for exposure at birth. The ratio of children only exposed to small dams to those only exposed to large dams decreases with the threshold used to partition dams, by construction. Note that very large dams (>90th percentile) are rarely accompanied by small dams in their vicinity, as the share of individuals exposed to both types of dams based on this threshold is very low (Appendix Table I.3).

The heterogeneous effect of dam size is estimated through two separate estimations, one for each type of dams.²³ Hence, I split the Dam_{jtbc} variable into two distinct indicators, Dam_{jtbc}^{Small} and Dam_{jtbc}^{Large} , depending on the size of dams the child is exposed to in cluster j at time t . To maintain the validity of the exclusion restriction, I exclude from this heterogeneity analysis children exposed to both types of dams (0.8% to 1.3% of the sample depending on the threshold used): in this case, river gradient will directly affect child health through the presence of the other type of dams in the locality.

5.2.2. *The positive effect of dams on child nutritional status is exclusively driven by small dams*

Results are reported in Appendix Figures I.4 to I.6, and are very similar across the different thresholds used. Both small and large dams have similar effects on child malaria-related outcomes. The risk of being anemic or severely anemic increases near large and small dams, respectively. In both cases, living close to a dam has a negative and significant effect on hemoglobin level, suggesting that whatever the size of the dam, individuals living in surrounding areas exhibit a higher risk of being infected by malaria. However, the positive effect of dams on child nutritional status is exclusively driven by small dams, a conclusion in line with the negative impact of large dams on cropland productivity in their vicinity. Overall, both types of dams have similar effects on child mortality risk, the impact of large dams on infant and post-neonatal mortality being larger though not significantly different from the impact of small dams.

²³ Note that it is not possible to run the heterogeneity analysis for dam size using an interaction term in the basic 2SLS approach nor the control function approach since it would require to control for the presence of a large dam near cluster j in the first-stage regression. As mentioned before, most of the dams located near DHS localities were built before survey time. Hence, exposure and proximity to a dam are highly correlated.

5.3. The role of upstream dams

Spatial distributional effects of dams have first been highlighted by [Duflo and Pande \[2007\]](#) based on an administratively-defined upstream-downstream relationship between districts. [Strobl and Strobl \[2011\]](#) have refined this methodology by using river basins instead of administrative units to identify such a relationship. This approach has then been used by [Chakravarty \[2011\]](#), [Blanc and Strobl \[2013\]](#) and [Mettetal \[2019\]](#). Following my definition of exposure to a dam in the vicinity, I here adopt an even more precise approach to identify localities effectively located downstream of a dam.

5.3.1. Empirical approach

Watershed delineation – Using drainage lines position and elevation data, I delineate watershed boundaries for each DHS cluster, that is the upstream land area up to 100 kilometers from the DHS cluster that drains all rivers and rainfall into the 10 kilometers buffer surrounding each DHS cluster position. Examples are shown in Appendix Figure [J.1](#). I then use these boundaries to compute geographic covariates, including topographic information and drainage lines length, at the watershed level. Appendix Table [J.2](#) present the corresponding summary statistics. The average area of a watershed is 2,541 square kilometers for a cumulative drainage lines length of 658 kilometers.

Definition of exposure – Unlike previous studies, I do not use aggregate information at the administrative unit or river basin level to categorize a locality as exposed to an upstream dam. Instead, I use locality-specific information based on its delineated watershed. An individual is thus considered exposed to an upstream dam if there is at least one dam present within its specific watershed –but outside the 10 kilometers buffer around household location– by survey time (or by child birth for child mortality outcomes). 16.3% of the localities have a dam in their watershed by survey time (Appendix Table [J.2](#)).

Instrument at the watershed level – Following the same identification strategy as before, I use river gradient information at the watershed level to instrument for the presence of a dam upstream from each DHS cluster location.

5.3.2. Upstream dams have no discernible effect on both nutritional status and malaria-risk of under-five children

Point estimates of exposure to an upstream dam on child health outcomes are reported in Table [10](#). Living more than 10 kilometers downstream from a dam has virtually no effect on child health, whatever the indicator considered. The absence of effect on anthropometric

measures might suggest a more limited income effect of dams in downstream localities than in their direct vicinity, contrasting with the conclusions from [Duflo and Pande \[2007\]](#) for large dams in India. I find no effect of upstream dams on malaria-related outcomes among under-five children, a result consistent with the nature of the overwhelming majority of small dams present in the data, designed to keep water in their reservoir and not to supply water to downstream areas through a canal network. Localities present downstream from a small dam generally do not directly benefit from it but, in turn, appear to escape its adverse effects on malaria risk.

While being born near a dam significantly increases infant mortality risk, I find no discernible effect of upstream dams on it, with a non-significant 2.3 percentage point reduction (Table 10, column 4). This result echoes the one from [Mettetal \[2019\]](#) for large upstream dams in South Africa. Post-neonatal mortality slightly decreases with the presence of a dam upstream (-2 percentage points), but this effect is not significantly different from zero.

5.3.3. Health investment behaviors and adult health outcomes are not modified by upstream dams

Results suggest that health investment behaviors are not shaped by the presence of a dam upstream (Table 11, columns 5 to 9). This might either reflect a lower income effect of upstream dams, as suggested above, or that the marginal cost of not using a bed net is lower when the dam is located upstream and not in the direct vicinity of the household.

Except a weakly significant increase in body mass index for women (column 1), and similarly to what I found for child health, point estimates reported in columns 2 to 4 of Table 11 suggest that upstream dams have no discernible effect on adult health, and if they had one, it would be very low compared with the one of neighboring dams. Hemoglobin level is 0.191 g/dL lower among adults residing downstream from a dam, with a risk of anemia in adulthood 5.3 percentage points higher, but these differences are not statistically significant.

Last, individuals might decide to move to upstream areas with a dam to work. While it is not possible to directly investigate this assumption,²⁴ I find that upstream dams induce moderate labor market effects: the probability of working is not impacted, but workers tend to be more paid in cash rather than exclusively paid in kind and to have more permanent jobs (Table 11, columns 10 to 14) when having a dam upstream.

²⁴ DHS do not collect information on the place of work.

Table 10. Effect of upstream dams on child health

I. <i>Child mortality outcomes</i>								
	(1)	(2)	(3)	(4)	(5)			
	Died within one week of life	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality			
Presence of a dam in watershed at birth	-0.005 (0.012)	-0.008 (0.013)	-0.027 (0.016)	-0.023 (0.020)	-0.020 (0.013)			
Kleibergen-Paap F-statistic	54.975	55.385	55.340	55.928	54.566			
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000			
Mean of dependent variable	0.027	0.032	0.048	0.063	0.030			
N	909,804	901,277	816,985	721,733	872,603			
II. <i>Nutritional status</i>								
	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
	HAZ	WHZ	WAZ	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Presence of a dam in watershed at survey time	0.239 (0.220)	-0.128 (0.176)	-0.020 (0.177)	-0.059 (0.058)	-0.071 (0.046)	0.003 (0.032)	0.006 (0.018)	0.024 (0.036)
Kleibergen-Paap F-statistic	38.759	35.239	35.959	38.759	38.759	35.239	35.239	45.730
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Mean of dependent variable	-1.400	-0.253	-0.999	0.360	0.165	0.096	0.035	0.163
N	544,590	536,731	553,758	544,590	544,590	536,731	536,731	795,662
III. <i>Malaria-related outcomes</i>								
	(14)	(15)	(16)	(17)	(18)			
	Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test			
Presence of a dam in watershed at survey time	0.045 (0.050)	-0.141 (0.272)	0.042 (0.073)	0.020 (0.022)	0.202 (0.136)			
Kleibergen-Paap F-statistic	45.951	34.060	34.060	34.060	10.056			
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000			
Mean of dependent variable	0.244	10.260	0.641	0.036	0.184			
N	787,290	255,440	255,440	255,440	101,049			

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a birth (columns 1 to 5) or a child alive at survey time (columns 6 to 18). The table reports the point estimates of exposure to an upstream dam from Equation 1 on child health outcomes, the presence of an upstream dam being instrumented as in Equation 2. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature at birth (columns 1 to 5) or during survey month (columns 6 to 18) within the 10 kilometers buffer, and cluster *j*'s geographic coordinates. Other covariates include child's sex and year of birth in all columns, and a dummy for multiple birth in columns 1 to 5. Country, drainage basin, survey year, country \times survey year fixed effects are included in all columns, as well as survey month and basin \times survey month in columns 6 to 18. All columns also control for land gradient, elevation and rivers length at the watershed level.

p*<.10; *p*<.05; ****p*<.01

Household material wealth non-significantly increases by 1.1% with the presence of a dam upstream (Table 11, column 15). This effect is much lower than the 18% increase observed when residing near a dam, a difference significant at the 1% level.

Similar to my criticism of using an overly aggregated level to define exposure to a dam, one might worry that considering wide watersheds could lead to the risk of diluting the corresponding point estimates. Moreover, closer upstream dams might have stronger effects than dams located further away within the watershed. To investigate these points, I re-run these analyses but now restrict the maximal upstream distance from the DHS cluster to 30, 50 or 70 kilometers from its location instead of 100. Corresponding point estimates and 95% confidence intervals are shown in Appendix Figures J.3 and J.4. Conclusions remain unchanged for child health outcomes. Results also barely change for adult health, health investments, and labor market effects. Workers tend to have better remuneration conditions and more permanent jobs, and households to be wealthier when having a dam up to 50 kilometers upstream.

Overall, it suggests that the effects of dams in Sub-Saharan Africa are essentially local, a conclusion consistent with the overwhelming majority of small dams present on the subcontinent.

Table 11. Effect of upstream dams on adult health, health investments, labor market and material wealth

I. Adult health					
	(1)	(2)	(3)	(4)	
	Body Mass Index	Hemoglobin level	Anemic	Severe anemia	
Presence of a dam in watershed at survey time	0.893* (0.532)	-0.191 (0.198)	0.053 (0.043)	0.001 (0.008)	
Kleibergen-Paap F-statistic	39.751	37.064	37.064	37.064	
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	
Mean of dependent variable	22.824	12.710	0.351	0.014	
N	436,733	515,558	515,558	515,558	

II. Health investments					
	(5)	(6)	(7)	(8)	(9)
	Up-to-date vaccinations	Bed net ownership	Bed net use	Iron supplementation	Deworming
Presence of a dam in watershed at survey time	-0.026 (0.049)	0.054 (0.079)	-0.020 (0.075)	0.000 (0.068)	0.067 (0.061)
Kleibergen-Paap F-statistic	49.780	32.768	31.064	22.284	39.504
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Mean of dependent variable	0.342	0.627	0.413	0.130	0.419
N	723,094	450,295	665,595	485,892	555,151

III. Labor market and wealth						
	(10)	(11)	(12)	(13)	(14)	(15)
	Currently working or worked in the past year	Paid in cash	Paid exclusively in-kind	Not paid	Seasonal, occasional or temporary job	Log. wealth index
Presence of a dam in watershed at survey time	0.054 (0.043)	0.119* (0.071)	-0.084** (0.036)	-0.035 (0.070)	-0.256*** (0.070)	0.011 (0.018)
Kleibergen-Paap F-statistic	49.320	42.169	42.169	42.169	40.103	51.194
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000	0.000
Mean of dependent variable	0.698	0.675	0.053	0.273	0.415	3.002
N	1,518,289	986,187	986,187	986,187	1,019,930	1,599,002

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a mother of children born in the five years preceding the survey due to data limitation and not currently pregnant in column 1, an adult in columns 2 to 4 and 10 to 15, a child alive at survey time in columns 5 and 7 to 9, and a household in column 6. The table reports the point estimates of exposure to an upstream dam by survey time from Equation 1 on adult health, child health investment, labor market and wealth outcomes, the presence of an upstream dam being instrumented as in Equation 2. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer and cluster *j*'s geographic coordinates. Other covariates include mother's year of birth and the interval between the last birth and survey time in months in column 1, adult's sex and year of birth in columns 2 to 4 and 10 to 15, and child's sex and year of birth in columns 2 and 7 to 9. Country, drainage basin, survey year, country \times survey year, survey month and basin \times survey month fixed effects are included. All columns also control for land gradient, elevation and rivers length at the watershed level.

* $p < .10$; ** $p < .05$; *** $p < .01$

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6. Discussion and Concluding Remarks

This chapter presents new evidence on the effect of dams on population health at a very local level in Sub-Saharan Africa. Exploiting river gradient as an exogenous source of variation in dam placement, it points to conflicting effects, with a significant improvement of child nutritional status around dams accompanied by a higher risk of malaria transmission. There is suggestive evidence that adults are also more exposed to malaria, with an increase in their risk of being severely anemic, one of the main consequences of malaria infection. These findings are consistent with the higher post-neonatal mortality risk found around dams, a period during which young babies' natural immunity against malaria acquired during childbearing fades away.

This increased malaria risk is not driven by a lower use of mosquito bed nets, as both ownership and utilization are higher around dams than farther away. This result suggests that individuals living near a dam are aware of the increased risk of mosquito bites in its vicinity. Households living near a dam tend to be better off, and individuals are more willing to occupy a permanent job than a seasonal one and to be paid in cash rather than not paid at all. The results also point to an improvement in health care access, potentially driven by the local development impact of dams and their positive income effect for households. However, there is no discernible effect of dam proximity on fertility behaviors, and so despite the positive income effect.

Heterogeneity analysis reveals extremely similar effects across the different Sub-Saharan African regions, but also across child's sex and rank of birth, suggesting that dams do not differentially affect siblings, either directly or indirectly through households' decisions taken in reaction to the presence of a dam. However, children from agricultural households are significantly more at risk of contracting malaria as a result of proximity to a dam than others. Our results also highlight the importance of the size of the dam, the reduction in chronic malnutrition being exclusively driven by small ones. Finally, we observe no effect of upstream dams on child health. However, upstream dams induce significant labor market effects for adults living in downstream communities, with an increase in the probability of having a permanent job and an improvement in remuneration conditions.

From policy makers' perspective, these results are particularly informative since they are only driven by variation coming from rivers' suitability according to the engineering litera-

ture. This chapter contributes to the understanding of how such infrastructures may have both positive and adverse effects on the local population. In particular, malaria prevalence is higher in the vicinity of dams, despite a more extensive use of mosquito bed nets in these localities. While irrigation infrastructures may significantly improve the life of the local population through a positive income effect, massive open surface water bodies create life-threatening environments in such contexts.

By no means is the policy implication of these results that dams should be banned but that complementary policies are needed to mitigate their adverse effect on malaria risk. If the local population had a better capacity to protect against mosquito bites and malaria infection, the net effect of the dams could turn out to be positive. It calls for large prevention and vaccination campaigns against malaria around dams, as well as free distribution of prevention equipment not limited to bed nets. From a more global perspective, the development of innovative prevention tools to cope with increasing insecticide resistance and prevent outdoor biting must be encouraged.

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APPENDICES TO CHAPTER 3

Appendix A. Examples of newspaper articles on adverse effects of dams

Figure A.1. Examples of newspaper articles pointing to adverse health effects of dams in the United States of America at the beginning of the 20th century

The New York Times.

SATURDAY, MARCH 20, 1937.

Increase in Malaria Laid To Power Dams and Autos

Special to THE NEW YORK TIMES.
ATLANTIC CITY, March 19.—Malaria is on the increase in both the North and South because of a combination of cheap automobiles, good roads and the multiplication of power dams and recreational lakes in malarious areas, according to Dr. L. L. Williams of the United States Public Health Service in Washington.

Speaking today at the closing session of the convention of the New Jersey Mosquito Extermination Association here, Dr. Williams said it was significant that most of the cases in the past two years occurred in parts of the country that had no malaria for twenty-five years previously.

He said tourists were flocking to resorts created around artificial lakes, particularly in the South. Bitten by mosquitos, they return to their homes and small outbreaks of the disease appear, he contended.

THE WALL STREET JOURNAL.

THURSDAY, APRIL 22, 1915

Damages for Malaria

Soon after the Alabama Power Co. completed its dam and hydro-electric development at Lock 12 on the Coosa river in Georgia, there was an outbreak of malaria in the territory surrounding the dam. The cause was alleged to be the great increase in malarial from the formation of the large lake above the dam.

Some lawyer suggested the bringing of suits against the company for damages and over 700 such suits were filed. The first one tried resulted in a decision for the company as did the second suit. Then the power company secured an injunction against bringing any of the remaining suits to trial until a decision had been reached on the first suits in the higher court. The principal involved in these suits is one of interest to every hydro-electric company in the country and the final decision in the cases will be closely watched.

Figure A.2. A newspaper article pointing to adverse health effects of dams in Sub-Saharan Africa (*Le Monde*, 2000)

AUJOURD'HUI
LE MONDE / JEUDI 21 DÉCEMBRE 2000

SCIENCES Les retenues d'eau créées dans les pays pauvres pour résoudre des problèmes alimentaires ont parfois des conséquences sanitaires dramatiques. ● DES ÉTUDES de paludisme, d'onchocercose (cécité des rivières) ou de bilharziose jalonnent ainsi les constructions de barrages au cours du siècle. ● DES ÉTUDES analysent les causes du développement de telles maladies et les moyens de les prévenir. Elles prônent le recours à des études d'impact sanitaire préalables à la construction d'un nouvel ouvrage.

● LA CONSULTATION de toutes les parties concernées, en particulier des femmes, et la sensibilisation des familles aux problèmes de nutrition permettraient d'éviter la plupart des drames. ● L'ENTRETIEN des installations apparaît indispensable pour éviter une dégradation rapide par comblement, eutrophisation ou transformation en marécage.

Les barrages africains peuvent engendrer des catastrophes sanitaires

Le développement de l'irrigation dans les pays pauvres ne s'accompagne pas suffisamment d'études d'impact préalables sur les populations locales. D'où la prolifération de maladies graves favorisées par la mise en eau permanente des terres agricoles

OUAGADOUGOU (Burkina Faso)
de notre envoyé spécial

La création de 1 200 hectares de rizières dans la plaine de Loumana, en 1957-1958, a suscité une flambée d'onchocercose (cécité des rivières) qui a ruiné l'aménagement de cette région du Burkina Faso. Des moins de 15% des femmes et de 20% des hommes adultes sont devenus aveugles en cinq ans. Entre les casiers rizières, de petites chèvres d'eau fournissent des conditions de paludisme, la plus grave jamais observée dans le monde sans doute, y fit également 130 000 morts en 1942-1945. En 1968, au Ghana, 90% des enfants de 10 à 14 ans habitant près des rives du barrage d'Alomso-Bo ont été atteints par la bilharziose en quelques mois à la suite de la création de la retenue d'eau. Actuellement, les effets les plus néfastes sont dus au barrage artésien de Diama, entre Sénégal et Mauritanie, qui a provoqué une importante épidémie de bilharziose intestinale.

Les hydro-aménagements en question

Faut-il stopper la construction de barrages dans les pays pauvres et, en particulier, en Afrique ? Cette question, a priori paradoxale lorsqu'elle concerne des pays durement touchés par la sécheresse, a été posée par les spécialistes du développement et les professionnels de la santé réunis lors du colloque international « Eau et santé - Impacts sanitaires et nutritionnels des hydro-aménagements en Afrique ». Cette manifestation, patronnée par le Centre national pour la recherche scientifique et technologique (CNRS) du Burkina Faso, l'école internationale d'ingénieurs de l'équipement rural de Ouagadougou, l'Institut français de recherche pour le développement (IRD), et l'Association pour le développement de la riziculture en Afrique de l'Ouest (Adira), a permis de mesurer l'ampleur des conséquences négatives des retenues d'eau pourtant destinées à l'alimentation en eau potable et à l'irrigation des cultures.

LEUR D'ESPoir

Si les risques sanitaires liés à l'eau sont en général connus, les aménagés eux-mêmes les considèrent encore trop souvent comme secondaires. Bien souvent, « personne ne prend en compte les conséquences d'une retenue d'eau sur la santé car nous manquons d'une culture de la

envisageable en Afrique, car « l'objectif de l'autosuffisance alimentaire n'est pas encore atteint », a rappelé Moussa Sotou, responsable de la mise en valeur des eaux au bureau régional pour l'Afrique de la FAO. « Un hectare irrigué produit deux à trois fois plus qu'un hectare non irrigué », a-t-il précisé. Actuellement, le rendement moyen des zones irriguées en Afrique est de 4 à 5 tonnes à l'hectare, et celui de la riziculture pluviale sans irrigation de 1 tonne à l'hectare.

Au sud du Sahara, seulement 3% des terres cultivées sont irriguées. « D'ici 2025, la pression démographique rendra le problème de l'eau encore plus crucial. Une dizaine de pays se retrouveront en état de stress hydrique, avec moins de 1 000 mètres cubes d'eau par tête d'habitant », ajoute le spécialiste. Il faudra alors trouver d'autres stratégies que la simple construction de barrages, en améliorant la « productivité de chaque goutte d'eau » grâce à des techniques d'économie de l'eau ou de collecte plus efficace de l'eau de pluie.

Christiane Galus

Assurer l'autosuffisance alimentaire

La Burkina Faso a construit de nouveaux barrages sur son territoire, destinés à la production d'énergie hydro-électrique ou à la culture par irrigation. Le barrage de Bagna, le plus important du pays, fournit les deux fonctions. Avec une capacité maximale de 1,2 milliard de m³, il est destiné à fournir 44 000 m³ de barrage par an et à produire à terme 200 000 tonnes de riz par an pour atteindre l'autosuffisance alimentaire.

Appendix B. Data Appendix

B.1. Demographic and Health Surveys

B.1.1. Sampling frame

The DHS sample design is based on a two-stage sampling procedure. First, enumeration areas, also called clusters, are selected from a list of all enumeration areas coming from the most recent national population census. The sampling frame is stratified by province and urban/rural areas within each province, and enumeration areas are randomly selected using a probability proportional to size method. Second, an exhaustive listing of all the households present in each cluster is carried out. A given number of households (usually between 20 and 30) per cluster are randomly selected with equal probability. Within sampled households, all women aged 15-49 who are either permanent residents of the household or visitors present on the night before the survey are eligible for survey interview. Male questionnaire is only administered in a randomly-selected sub-sample of households. Among households selected for male questionnaire, all men aged 15-59 (in some cases 15-54) who are either permanent residents of the household or visitors present on the night before the survey are eligible for survey interview. The sample design and questionnaires are standardized across survey waves, allowing for pooled cross-section analysis.

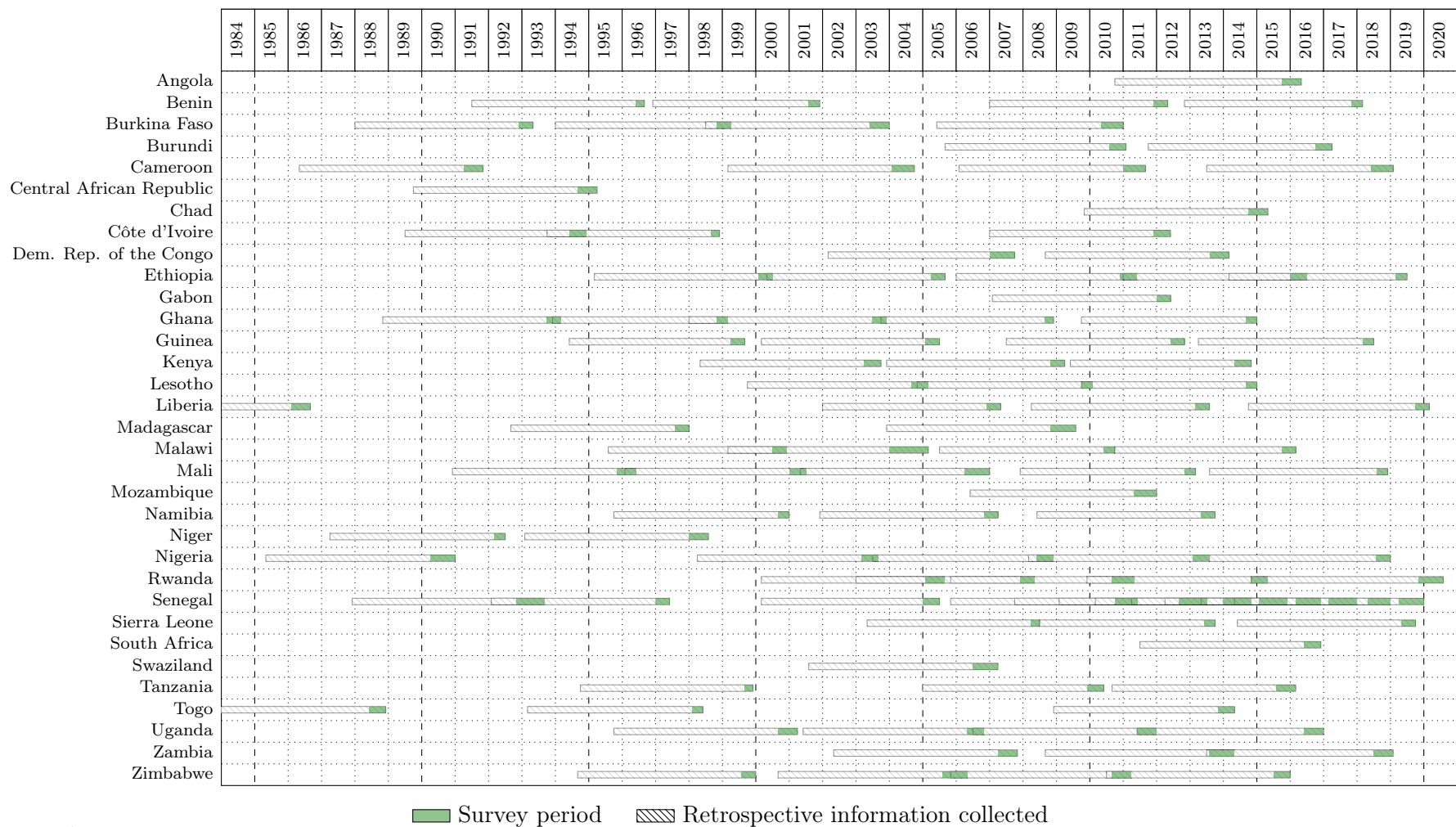
B.1.2. Scrambling procedure

To ensure that respondents cannot be identified, the DHS randomly displace the true GPS coordinates of the clusters. Urban clusters are randomly displaced within a radius of 2 kilometers around their true location, creating a positional error ranging from a minimum of 0 and a maximum of 2 kilometers. Rural clusters are randomly displaced within a radius of 5 kilometers around their true location and up to 10 kilometers for a further 1% of them, creating a positional error ranging from a minimum of 0 and a maximum of 10 kilometers. See the [DHS methodology for geographic data](#)²⁵ for additional information. This potential error being randomly assigned to clusters, the measurement error created by the scrambling procedure only leads to an attenuation bias. To limit this bias, I use buffers of 10 kilometers in line with the DHS recommendations.

B.1.3. Timeline

²⁵ <https://dhsprogram.com/What-We-Do/GPS-Data-Collection.cfm>

Figure B.1. Timeline of DHS Surveys and retrospective periods



Source: Author's computation

Notes: The figure shows the timeline of georeferenced DHS surveys (green) and retrospective periods (gray) for which information is collected.

B.2. Dams databases

I use the four main existing sources of dams location across the continent: the Global Reservoir and Dam Database (GRaND), the GLObal geOreferenced Database of Dams (GOODD), the FAO's African Geo-referenced Dams Database (AQUASTAT), and the South African Register of Large Dams.

If a dam appears in several databases, I keep the line with the most information on it. I exclude several records which point to areas where there is no dam. The number of dams I get from each database after cleaning is thus smaller than the original number of records.

GRaND. The Global Reservoir and Dam Database (GRaND v1.3) is part of the Global Water System Project, an international team of scientists created to address the lack of a reliable, global database of large dams and reservoirs across the World [Lehner et al., 2011]. The last version (at time of writing) of the database was released in 2019 and is available at <http://globaldamwatch.org/grand/>. It contains information on 7,320 geo-referenced dams with a height greater than 15 meters or with a reservoir of more than 0.1km³. It covers 392 dams in Sub-Saharan Africa after data cleaning. For 54 dams, the construction year was missing, and for 5 dams it was incorrect: in both cases, I retrieved it from the Landsat program.

AQUASTAT. The FAO's Geo-referenced Dams Database (AQUASTAT) gathers information from different data sources, including from the International Commission on Large Dams (ICOLD), national reports, and AQUASTAT national surveys. It focuses almost exclusively on dams with a height greater than 15 meters or with a reservoir capacity of at least 3 million cubic meters. The last version was updated in 2016 and is available at <http://www.fao.org/aquastat/en/databases/dams>. It contains information on over 14,000 dams around the world, of which 196 are located in Sub-Saharan Africa after cleaning. For 31 dams, the construction year was missing: I retrieved it from the Landsat program.

GOODD. The Global Geo-referenced Database of Dams (GOODD) contains a unique record of 38,667 geo-referenced dams obtained by digitizing visible dams on Google Earth satellite imagery [Mulligan et al., 2020], available at <http://globaldamwatch.org/goodd/>. However, it only contains the exact geographic coordinates of dams position but no information on dams construction dates, which I retrieved based on historical monthly-based satellite imagery from the NASA/USGS Landsat program, starting 1972 and available at <https://earthexplorer.usgs.gov/>. It covers 1,720 dams in Sub-Saharan Africa after

cleaning.

South African Register of Large Dams. South Africa is the first Sub-Saharan African country in terms of number of dams. The South African Register of Dams is produced by the Dam Safety Office of the Department of Water and Sanitation Affairs. The last version was released in 2018 and is available at <http://www.dwa.gov.za/DSO/>. It covers 5,271 small and large dams after cleaning. For 437 dams, the construction year was missing, and for 492 dams it was incorrect: in both cases, I retrieved it from the Landsat program.

I complement these databases with 4,189 unreferenced dams I found based on satellite imagery, for which I extract the exact location and construction year based on historical Landsat satellite imagery. It corresponds almost exclusively to unregistered, small dams.

B.3. Geographic and topographic data

Altitude. Elevation data are from the digital elevation model of the Shuttle Radar Topography Mission (SRTM) at a 3 arc-second resolution (8.3×10^{-4} degree, $\approx 90\text{m}$ at the equator), a joint work of the NASA and the U.S. National Geospatial-Intelligence Agency (GSA). Data are available at <https://www.hydrosheds.org/downloads>. For each DHS cluster, I calculate the average elevation in meters within the 10 kilometers buffer surrounding the DHS cluster position.

Land gradient. Based on elevation data, I construct land gradient under ArcGIS, and calculate the average land gradient in percent within the 10 kilometers buffer surrounding the DHS cluster position, excluding the river network from it.

River network. I exploit hydrologically-conditioned SRTM data at a 3 arc-second resolution (8.3×10^{-4} degree, $\approx 90\text{m}$ at the equator) from [Lehner et al. \[2008\]²⁶](#) to derive river courses (drainage lines) position throughout the continent under ArcGIS, as shown in [Figure B.2](#). Each drainage line is then split into 500 meters-long sections, and elevation data are used to calculate drainage lines slope.

Figure B.2. River network



Source: Author's computation based on HydroSHEDS data

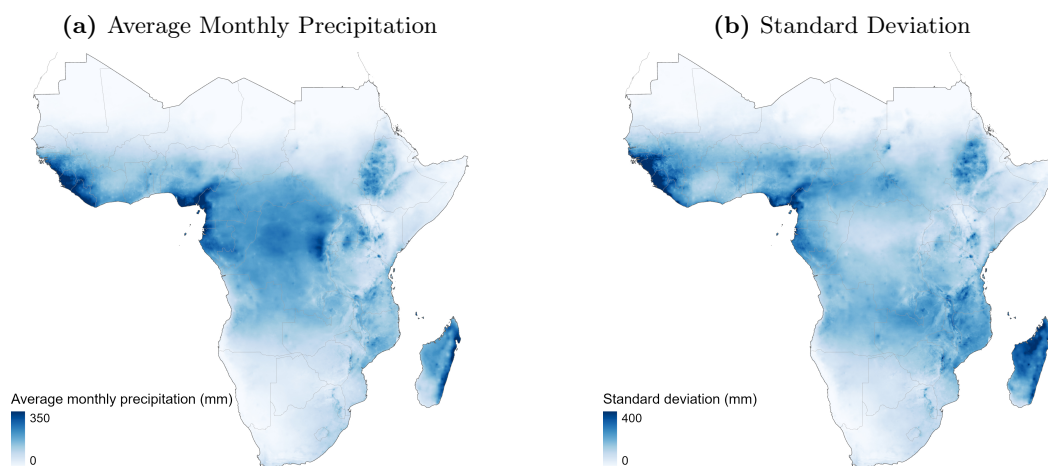
Notes: The map shows drainage lines created from elevation data. The intensity of the color reflects the Strahler stream order, an indicator of stream size based on its tributaries. Darker colors (resp. lighter) represent higher (resp. smaller) Strahler orders.

²⁶ Available at <https://www.hydrosheds.org/downloads>.

B.4. Other data

Rainfall. Historical monthly precipitation data are extracted from the Climate Hazards Group InfraRed Precipitation with Station (CHIRPS) database [Funk et al., 2015] available at <https://www.chc.ucsb.edu/data/chirps>. Data are available at a 0.05° resolution ($\approx 5\text{km}$ at the equator) from 1981 to present. For each DHS cluster, I compute the average monthly precipitation level within the 10 kilometers buffer from January 1981 to December 2020. Each individual is then assigned the average monthly rainfall level corresponding to its interview date for the analysis of contemporaneous outcomes. For child mortality analysis, each newborn is assigned the average monthly rainfall level from her birth date. The long-term average and standard deviation of monthly precipitation are shown in Figure B.3.

Figure B.3. Long-term average and standard deviation of monthly precipitation



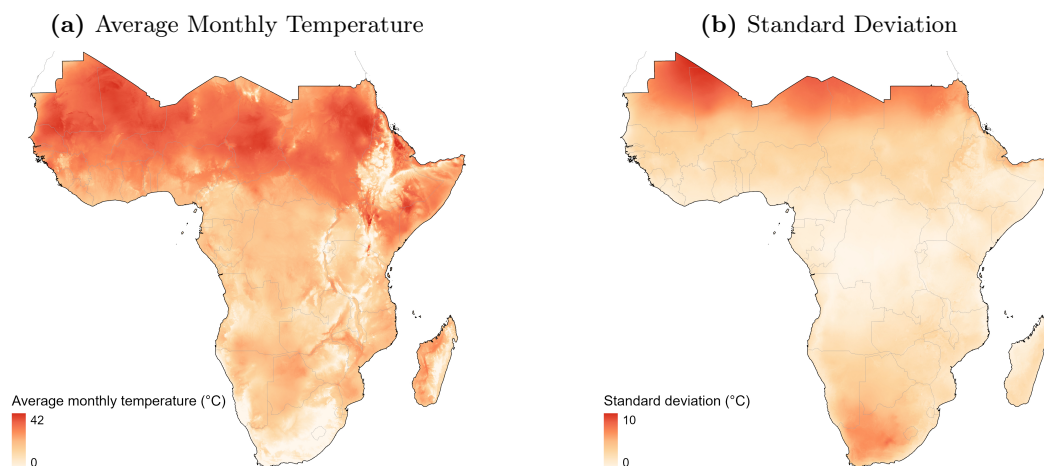
Source: Author's calculations based on CHIRPS data

Notes: The maps show the (a) long-term average monthly precipitation and (b) standard deviation of monthly precipitation over the 1981-2020 period.

Hydrologic basins. Hydrologic basins delineation is from the HydroSHEDS database [Lehner and Grill, 2013] available at <https://www.hydrosheds.org/page/hydrobasins>. It is used to capture regional effects related to the hydrological network.

Temperature. Historical monthly air temperature data are from the Terra Climate database [Abatzoglou et al., 2018] available at <http://www.climatologylab.org/terraclimate.html>. Data are available at a 0.04° resolution ($\approx 4.5\text{km}$ at the equator) from 1958 to present. I calculate the average monthly temperature level within the 10 kilometers buffer from January 1981 to December 2020. Assignment of the average monthly temperatures to individuals is the same as for the precipitation data. The long-term average and standard deviation of monthly temperature are shown in Figure B.4.

Figure B.4. Long-term average and standard deviation of monthly temperature

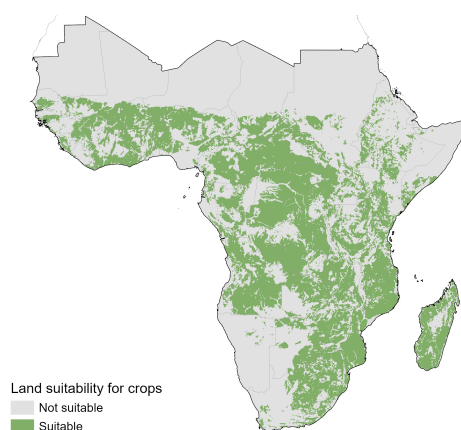


Source: Author's calculations based on TerraClimate data

Notes: The maps show the (a) long-term average monthly temperature and (b) standard deviation of monthly temperature over the 1981-2020 period.

Land suitability for food and cash crops. Land suitability for food and cash crops is derived from the GAEZ dataset [Fischer et al., 2021] available at <https://gaez.fao.org/>. Data are available at a 0.08° resolution ($\approx 9\text{km}$ at the equator). I extract land suitability index for 43 food and cash crops, supposing rainfed production with a low input level for the 1981-2010 period. I then determine whether a cell is suitable for at least one crop (good, high or very high suitability index, see Figure B.5) and compute the share of land (cells) suitable for at least one crop production within the 10 kilometers buffer around each DHS cluster.

Figure B.5. Land suitability for food and cash crops



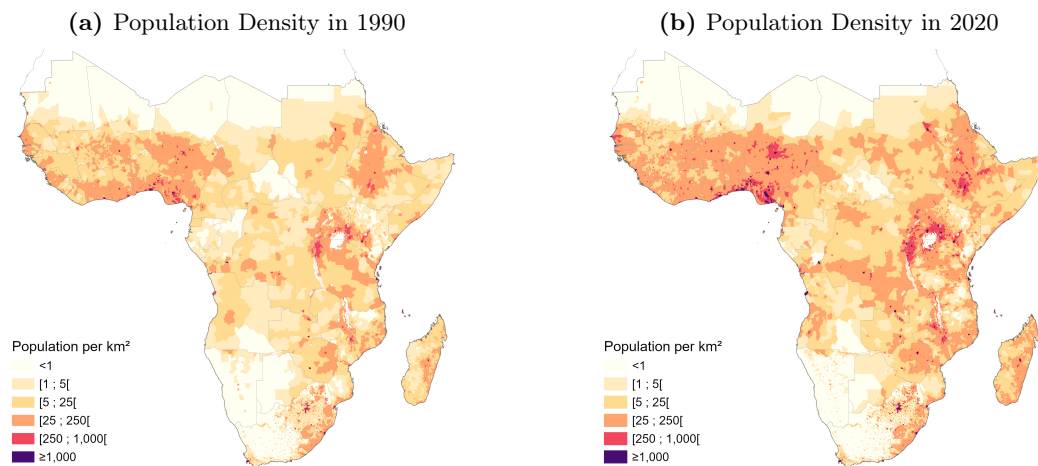
Source: Author's calculations based on GAEZ data

Notes: The map shows areas suitable for at least one food or cash crop in green, based on GAEZ dataset for rainfed production with low input level over the period 1981-2010.

Data on food crops cover barley, buckwheat, cabbage, carrot, cassava, chickpea, cowpea, dry pea, dryland rice, foxtail millet, gram, maize, oat, onion, pearl millet, phaseolus bean, pigeon pea, rye, sorghum, sweet potato, tomato, wetland rice, wheat, white potato, yam. Data on cash crops cover banana, citrus, cocoa, coconut, coffee, cotton, flax, groundnut, jatropha, olive, oil palm, rapeseed, sunflower, soybean, tea, tobacco, sugarbeet, sugarcane.

Population density. Population density data are extracted from the Gridded Population of the World (GPW) dataset [Doxsey-Whitfield et al., 2015] for the years 1990 and 1995 (available at <https://sedac.ciesin.columbia.edu/data/collection/gpw-v3>), as well as 2000, 2005, 2010, 2015 and 2020 (available at <https://sedac.ciesin.columbia.edu/data/collection/gpw-v4>). Data are adjusted to match United Nations population estimates and are available at a 0.04° resolution ($\approx 4.5\text{km}$ at the equator). I compute the average population density within the 10 kilometers buffer around the DHS clusters for each year. I then assign the population density measure to DHS clusters based on the survey date (for contemporaneous outcomes) or birth date (for retrospective outcomes), using the closest year for which this information is available.

Figure B.6. Population density in 1990 and 2020



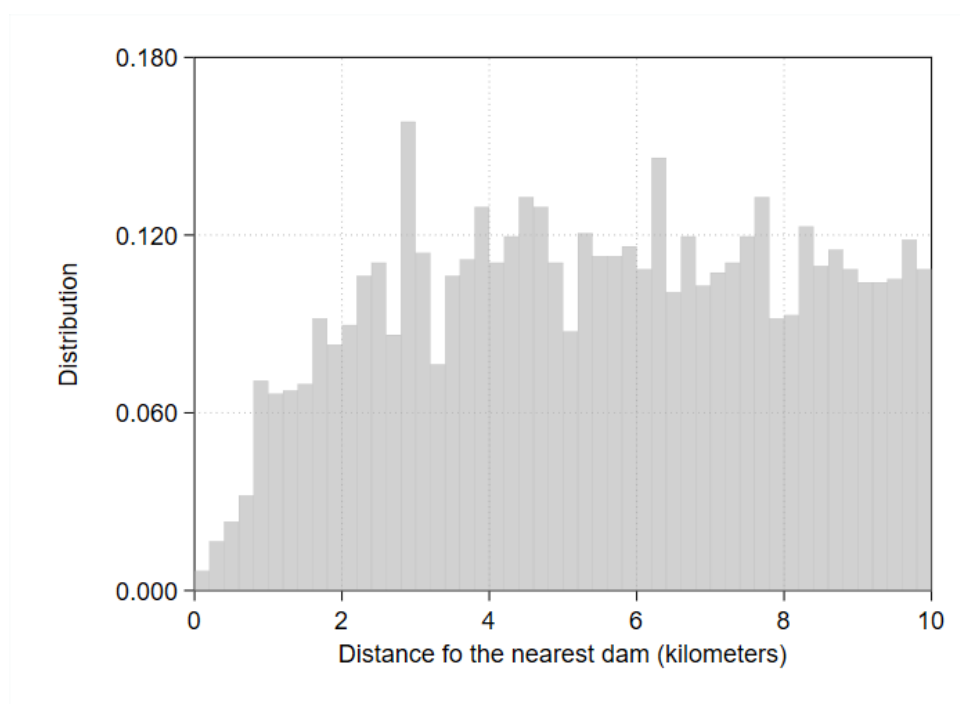
Source: Author's calculations based on GPW data

Notes: The maps show the population density in (a) 1990 and (b) 2020.

Appendix C. Additional summary statistics

C.1. Distance to the nearest dam within 10 kilometers

Figure C.1. Distribution of the distance to the nearest dam within 10 kilometers at the DHS cluster level



Source: Author's computation

Notes: The figure plots the distribution of the distance to the nearest dam at the DHS cluster level in kilometers when the sample is restricted to clusters with at least one dam within 10 kilometers. See Figure 4 of the chapter for the full distribution.

C.2. Summary statistics at the cluster level

Table C.2. Summary statistics at the DHS cluster level

	(1) Whole sample	(2) ≤ 10km from a dam	(3) > 10 km from dam	(4) (2) = (3) <i>p</i> -value
<i>Panel A. Geographic characteristics</i>				
Elevation	741.432	937.574	720.279	0.000
Terrain gradient (%)	7.173	6.533	7.242	0.000
Land suitability for crops	0.464	0.647	0.444	0.000
Temperature (°C)	30.354	28.989	30.502	0.000
Rainfall (mm/month)	104.977	77.736	107.914	0.000
Population density (/km ²)	726.226	855.825	712.249	0.000
Drainage lines length (km)	78.389	78.836	78.341	0.190
Drainage lines gradient (%)	0.931	0.840	0.941	0.000
<i>Panel B. Dam exposure</i>				
Distance to nearest dam (km)	99.425	5.489	109.556	0.000
Exposed to a dam by survey time	0.095	0.980	0.000	0.000
Number of dams by survey time	0.273	2.801	0.000	0.000
N	45,476	4,427	41,049	45,476

Source: Author's calculations from DHS and satellite data.

Notes: Unweighted statistics. The first column reports summary statistics for the whole sample of DHS clusters. The following two columns report the same statistics when the sample is restricted to DHS clusters with (column 2) or without (column 3) a dam at less than 10 kilometers. Column 4 reports the *p*-values from *t*-tests on the equality of means reported in columns 2 and 3.

C.3. Summary statistics for the sample of adults

Table C.3. Summary statistics at the adult level

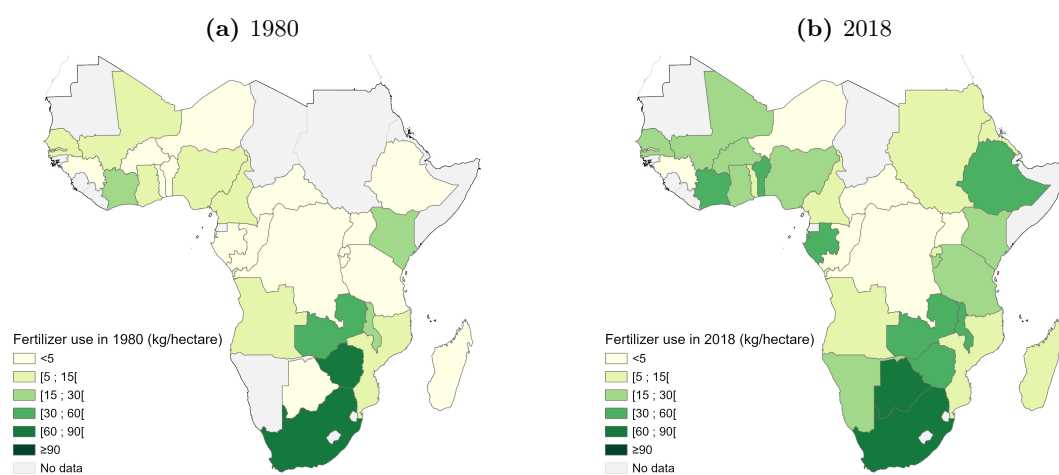
	(1) Whole sample	(2) ≤ 10km from a dam	(3) > 10 km from dam	(4) (2) = (3) <i>p</i> -value
<i>Panel A. Adult's characteristics</i>				
Woman	0.710	0.677	0.713	0.000
Age	28.867	28.712	28.883	0.000
Lowest two quintiles of material wealth	0.379	0.227	0.395	0.000
<i>Panel B. Topographic characteristics</i>				
Elevation	712.784	910.833	692.665	0.000
Terrain gradient (%)	6.928	6.208	7.001	0.000
Drainage lines length (km)	78.130	78.468	78.096	0.000
Drainage lines gradient (%)	0.896	0.805	0.905	0.000
<i>Panel C. Dam exposure</i>				
Distance to nearest dam (km)	100.514	5.576	110.158	0.000
Exposed to a dam by survey time	0.090	0.978	0.000	0.000
Number of dams by survey time	0.240	2.608	0.000	0.000
<i>Panel D. Malaria-related outcomes</i>				
Hemoglobin level	12.706	13.091	12.656	0.000
Anemic	0.352	0.292	0.359	0.000
Severe anemia	0.014	0.013	0.014	0.000
<i>Panel E. Employment</i>				
Currently working or worked in the past year	0.697	0.656	0.701	0.000
Paid in cash	0.676	0.782	0.666	0.000
Paid exclusively in kind	0.052	0.038	0.054	0.000
Not paid	0.272	0.181	0.280	0.000
Seasonal, occasional or temporary job	0.414	0.350	0.420	0.000
N	1,652,864	152,420	1,500,444	1,652,864

Source: Author's calculations from DHS and satellite data.

Notes: Unweighted statistics. The first column reports summary statistics for the whole sample of adults. The following two columns report the same statistics when the sample is restricted to households with (column 2) or without (column 3) a dam at less than 10 kilometers. Column 4 reports the *p*-values from *t*-tests on the equality of means reported in columns 2 and 3.

Appendix D. Fertilizer use

Figure D.1. Fertilizer use in kilograms per hectare of arable land



Source: Author's calculations based on World Development Indicators and FAO data

Notes: The maps show the average national fertilizer use in kilograms per hectare of arable land in (a) 1980 and (b) 2018. Fertilizer data cover nitrogenous, potash and phosphate fertilizers but not traditional nutrients (animal and plant manures). Data available at <https://data.worldbank.org/indicator/AG.CON.FERT.ZS>.

Appendix E. Inference

E.1. p -value adjustment for multiple hypothesis testing and spatial correlation

Table E.1. Unadjusted and Romano-Wolf adjusted p -values for the effect of dam exposure

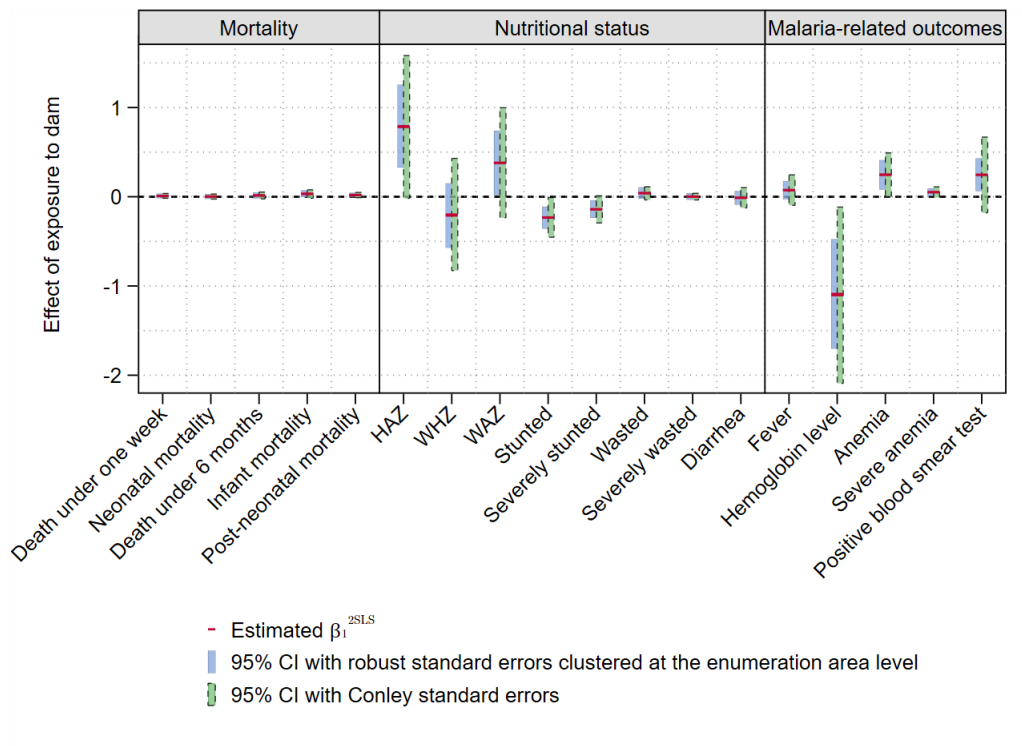
Outcomes		(1) Unadjusted p -values	(2) Romano-Wolf adjusted p -values
<i>Mortality</i>	Died within one week of life	0.453	0.598
	Neonatal mortality	0.836	0.954
	Died before 6 months of life	0.363	0.478
	Infant mortality	0.085	0.071
	Post-neonatal mortality	0.089	0.072
<i>Nutritional status</i>	Height-for-Age z -score	0.001	0.001
	Weight-for-Height z -score	0.251	0.317
	Weight-for-Age z -score	0.040	0.016
	Stunted	0.000	0.001
	Severely stunted	0.005	0.001
	Wasted	0.194	0.225
	Severely wasted	0.876	0.954
	Diarrhea	0.744	0.954
<i>Malaria sympt.</i>	Fever	0.147	0.138
	Hemoglobin level	0.000	0.001
	Anemia	0.004	0.001
	Severe anemia	0.028	0.010
	Positive blood smear test	0.008	0.001

Source: Author's calculations from DHS and satellite data.

Notes: Column 1 reports the unadjusted p -values with robust standard errors clustered at the enumeration area level obtained from Tables 3, 4 and 5. Column 2 reports the corresponding Romano-Wolf adjusted p -values accounting for multiple hypothesis testing with 1,000 replications [Romano and Wolf, 2005, 2016].

E.2. Inference with standard errors corrected for spatial correlation following [Conley \[1999\]](#)

Figure E.2. Confidence intervals obtained with standard errors corrected for spatial correlation



Source: Author's calculations from DHS and satellite data.
Notes: This figure plots the point estimates along with 95% confidence intervals of the effect of living near a dam on child health outcomes. Confidence intervals with robust standard errors clustered at the enumeration area level as in Tables 3 to 5 are in blue. Green confidence intervals with a dashed outline are obtained when allowing error terms to be spatially correlated within a 100 kilometers radius with a linear decay (Bartlett kernel) following [Conley \[1999\]](#). Control variables and fixed effects are the same as in Tables 3 to 5.

Appendix F. Robustness Checks

F.1. Recall bias and age heaping

Table F.1. Robustness of mortality results to age heaping

	(1)	(2)	(3)
	Died within first month of life	Died within first year of life	Died between 1 month and 12 months of life
<i>Panel A. OLS</i>			
Exposed at birth	-0.003*** (0.001)	-0.004*** (0.002)	-0.003*** (0.001)
R ²	0.020	0.027	0.015
<i>Panel B. 2SLS</i>			
Exposed at birth	0.006 (0.014)	0.035* (0.021)	0.030** (0.015)
Kleibergen-Paap F-Statistic	72.429	74.198	74.220
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.830	0.258	0.083
Mean of dependent variable	0.036	0.069	0.039
N	910,893	729,423	706,210

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child. The table reports the point estimates of exposure to a dam at birth from Equation 1 on the probability for a child to die within her first month of life (column 1), within her first year of life (column 2), or between her first month and first year of life, the presence of dam being instrumented following Equation 2. All regressions control for land gradient, elevation, land suitability for crops, rivers length, as well as population density, rainfall and temperature at birth within the 10 kilometers buffer. Other covariates include a dummy for multiple birth, child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year and country × survey year fixed effects are included. Children who did not reach the corresponding age at survey time are excluded to avoid censoring bias. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

p*<.10; *p*<.05; ****p*<.01

F.2. Continuous definition of exposure to dams

F.2.1. Child health

Table F.2. Effect of the number of dams nearby at birth on child mortality risk

	(1)	(2)	(3)	(4)	(5)
	Perinatal mortality	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality
<i>Panel A. OLS</i>					
Number of dams	0.000 (0.000)	0.000* (0.000)	0.000* (0.000)	0.000 (0.000)	0.000 (0.000)
R ²	0.017	0.019	0.022	0.025	0.011
<i>Panel B. 2SLS</i>					
Number of dams	0.005 (0.004)	0.002 (0.005)	0.006 (0.006)	0.014* (0.007)	0.008* (0.004)
Kleibergen-Paap F-Statistic	56.075	55.642	55.737	57.015	55.226
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Reduced-Form <i>p</i> -value	0.391	0.705	0.674	0.303	0.137
Mean of dependent variable	0.026	0.032	0.048	0.063	0.030
N	919,508	910,886	825,691	729,423	881,938

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child. The table reports the point estimates of the effect of the number of dams at birth within 10 kilometers from Equation 1 on child mortality, the number of dams being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, as well as population density, rainfall and temperature at birth within the 10 kilometers buffer. Other covariates include a dummy for multiple birth, child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year and country \times survey year fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table F.3. Effect of the number of dams nearby on child nutritional status

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Height-for-Age z-score	Weight-for-Height z-score	Weight-for-Age z-score	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Panel A. OLS								
Number of dams by survey time	0.004 (0.003)	-0.002 (0.002)	0.002 (0.002)	-0.001* (0.001)	-0.001* (0.001)	0.000 (0.000)	0.000 (0.000)	0.000 (0.001)
R ²	0.109	0.085	0.108	0.084	0.065	0.054	0.036	0.048
Panel B. 2SLS								
Number of dams by survey time	0.210*** (0.069)	-0.075 (0.053)	0.092* (0.054)	-0.063*** (0.018)	-0.035** (0.014)	0.013 (0.010)	0.003 (0.005)	0.009 (0.013)
Kleibergen-Paap F-Statistic	38.545	38.140	37.578	38.545	38.545	38.140	38.140	44.511
Kleibergen-Paap p-value	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
Reduced-Form p-value	0.000	0.115	0.012	0.000	0.000	0.372	0.642	0.011
Mean of dependent variable	-1.395	-0.253	-0.996	0.358	0.164	0.096	0.035	0.163
N	550,980	543,046	560,336	550,980	550,980	543,046	543,046	804,442

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child alive at survey time. The table reports the point estimates of the effect of the number of dams by survey time within 10 kilometers from Equation 1 on child nutritional status, the number of dams being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster j 's geographic coordinates. Country, drainage basin, survey year, country \times survey year, survey month and basin \times survey month fixed effects are included. Reduced-form p -values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

* $p < .10$; ** $p < .05$; *** $p < .01$

F.2.2. Adult Health

Table F.5. Effect of the number of dams nearby on adult health

	(1)	(2)	(3)	(4)
	Body Mass Index	Hemoglobin level	Anemia	Severe anemia
<i>Panel A. OLS</i>				
Number of dams by survey time	0.018* (0.009)	0.001 (0.003)	0.000 (0.001)	0.000 (0.000)
R ²	0.194	0.291	0.106	0.014
<i>Panel B. 2SLS</i>				
Number of dams by survey time	-0.082 (0.146)	-0.171*** (0.057)	0.053*** (0.013)	0.003 (0.002)
Kleibergen-Paap F-Statistic	40.400	28.287	28.287	28.287
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.000	0.000	0.000	0.000
Mean of dependent variable	22.893	12.707	0.351	0.014
N	442,186	521,264	521,264	521,264

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is an adult, except in column 1 where the analysis is done among mothers of children born in the five years preceding the survey due to data limitation and not currently pregnant. The table reports the point estimates of the effect of the number of dams by survey time within 10 kilometers from Equation 1 on adult health outcomes, the presence of dam being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include adult's sex (except in column 1, where the analysis is only done for women due to data limitation) and year of birth, as well as cluster *j*'s geographic coordinates. Column 1 also controls for the interval between the last birth and survey time in months. Country, drainage basin, survey year, country × survey year, survey month and basin × survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

p*<.10; *p*<.05; ****p*<.01

Table F.4. Effect of the number of dams nearby on risk of malaria for children

	(1)	(2)	(3)	(4)	(5)
	Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test
<i>Panel A. OLS</i>					
Number of dams by survey time	0.000 (0.001)	-0.002 (0.004)	0.001 (0.001)	0.000 (0.000)	-0.009*** (0.003)
R ²	0.070	0.172	0.129	0.040	0.303
<i>Panel B. 2SLS</i>					
Number of dams by survey time	0.052*** (0.018)	-0.273*** (0.092)	0.051** (0.024)	0.012 (0.007)	0.269*** (0.088)
Kleibergen-Paap Wald F-Statistic	44.084	26.799	26.799	26.799	12.861
Kleibergen-Paap <i>p</i> -value	0.000	0.000	0.000	0.000	0.000
Reduced Form <i>p</i> -value	0.000	0.000	0.000	0.001	0.000
Mean of dependent variable	0.244	10.261	0.641	0.036	0.182
N	795,714	258,036	258,036	258,036	102,499

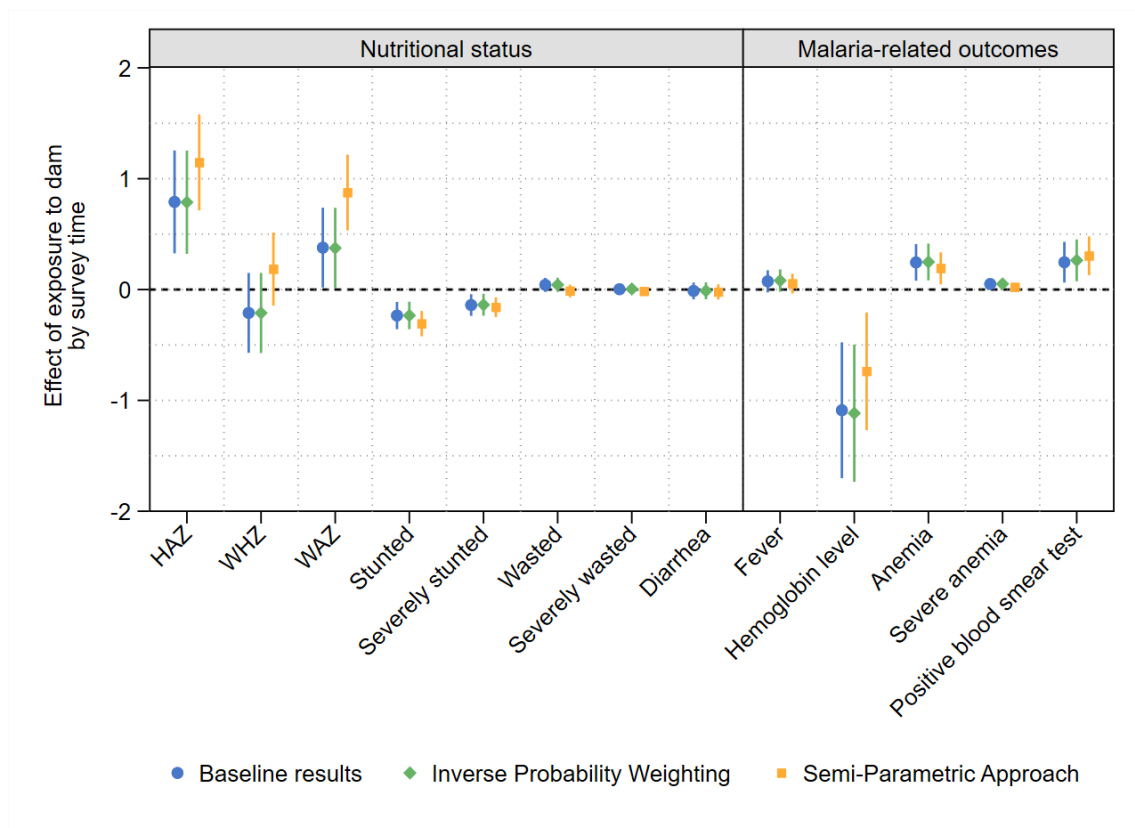
Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child alive at survey time. The table reports the point estimates of the effect of the number of dams by survey time within 10 kilometers from Equation 1 on child malaria-related outcomes, the number of dams being instrumented following Equation 2 in Panel B. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year, country × survey year, survey month and basin × survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero.

p*<.10; *p*<.05; ****p*<.01

F.3. Selective mortality

Figure F.6. Effect of residing near a dam on child contemporaneous health outcomes when considering selective mortality



Source: Author's calculations from DHS and satellite data.

Notes: This figure plots the point estimates along with 95% confidence intervals of the effect of living near a dam on child nutritional status and malaria-related outcomes. Blue dots corresponds to the baseline estimates from Tables 4 and 5. Green diamonds correspond to the point estimates obtained with inverse probability weighting of the observations according to their predicted survival probability at survey time. Yellow squares are the point estimates obtained following the semi-parametric approach from Cosslett [1991] by including one indicator variable for each centile of predicted survival probabilities at survey time. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Tables 4 and 5.

F.4. Alternative blood test for malaria detection

Table F.7. Effect of dam exposure on positive rapid diagnostic test

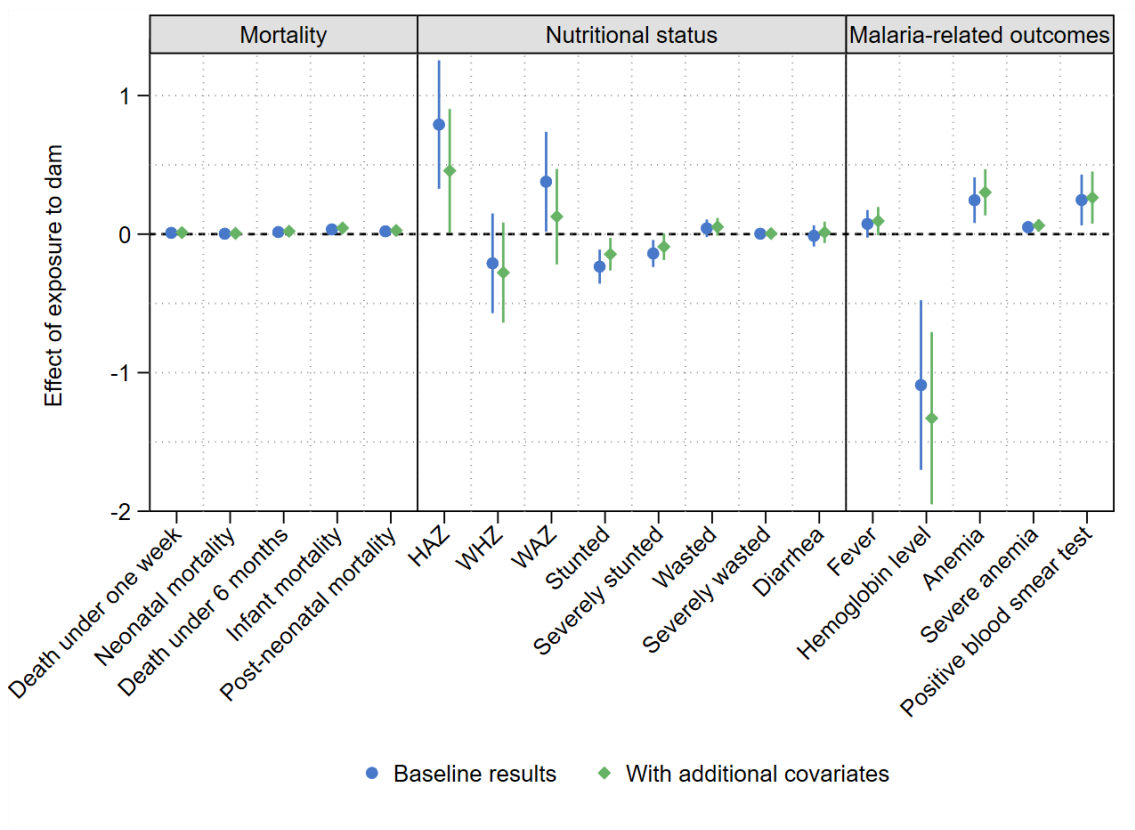
	(1)	(2)
	Positive rapid diagnostic test	
<i>Panel A. OLS</i>		
Exposed by survey time	-0.057*** (0.010)	
Number of dams by survey time		-0.013*** (0.003)
R ²	0.301	0.301
<i>Panel B. 2SLS</i>		
Exposed by survey time	0.332** (0.136)	
Number of dams by survey time		0.488*** (0.134)
Kleibergen-Paap F-Statistic	15.661	12.483
Kleibergen-Paap <i>p</i> -value	0.000	0.000
Reduced Form <i>p</i> -value	0.000	0.000
Mean of dependent variable	0.228	0.228
N	122,990	122,990

Source: Author's calculations from DHS and satellite data.

Notes: Robust standard errors clustered at the enumeration area level reported in parentheses. The unit of observation is a child alive at survey time. The table reports the point estimates of the effect of exposure to a dam (column 1) or the number of dams (column 2) by survey time within 10 kilometers from Equation 1 on child rapid diagnostic test for malaria, exposure to dam being instrumented following Equation 2. All regressions control for land gradient, elevation, land suitability for crops, rivers length, population density at survey time, as well as rainfall and temperature during survey month within the 10 kilometers buffer. Other covariates include child's sex and year of birth, as well as cluster *j*'s geographic coordinates. Country, drainage basin, survey year, country × survey year, survey month and basin × survey month fixed effects are included. Reduced-form *p*-values are obtained from testing the null hypothesis that all coefficients α_k from Equation 3 are jointly equal to zero. **p*<.10; ***p*<.05; ****p*<.01

F.5. Additional covariates

Figure F.8. Robustness to the inclusion of additional covariates

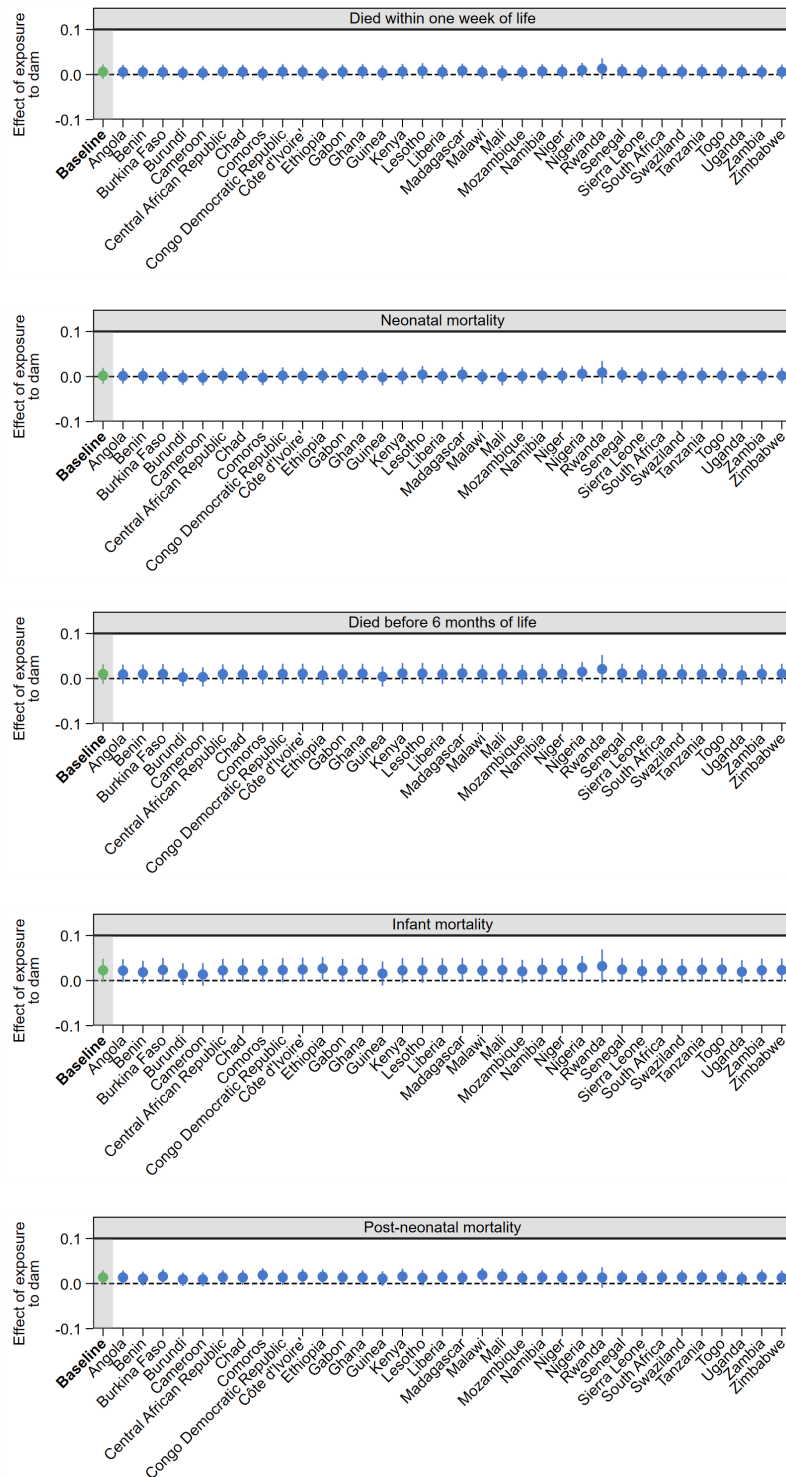


Source: Author's calculations from DHS and satellite data.

Notes: This figure plots the point estimates along with 95% confidence intervals of the effect of living near a dam on child health outcomes. Blue dots corresponds to the baseline estimates from Tables 3 to 5. Green diamonds correspond to the point estimates obtained when controlling for mother's years of education and age at birth, material wealth index, sex and age of household head as well as child's rank of birth. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Tables 3 to 5.

F.6. Leave-one-out analysis

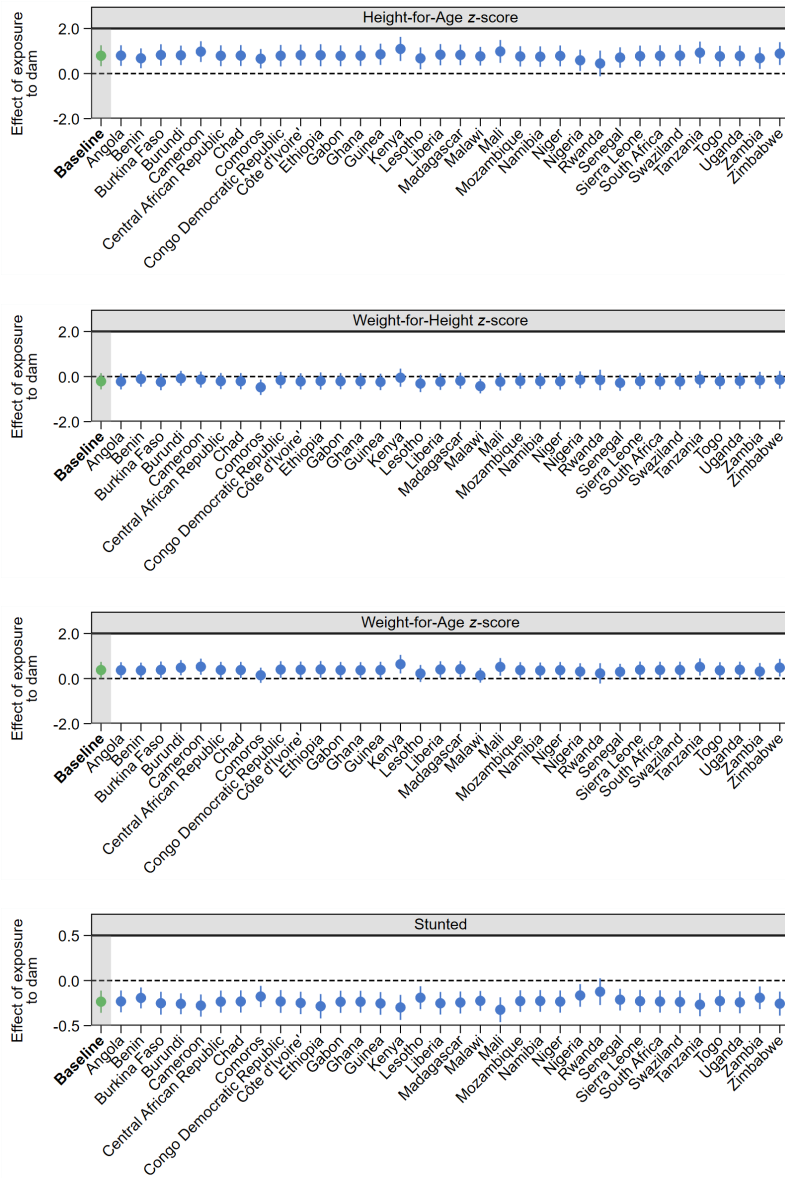
Figure F.9. Leave-one-out analysis for child mortality outcomes



Source: Author's calculations from DHS and satellite data.

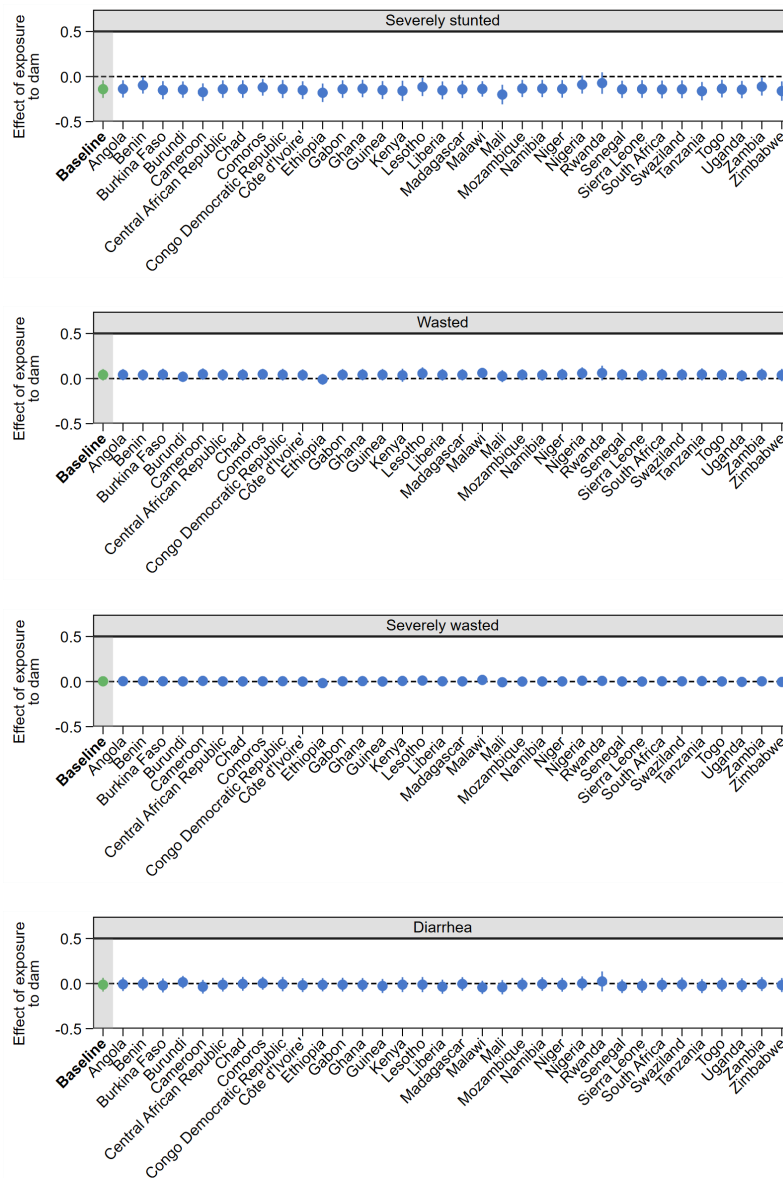
Notes: These figures report the point estimates along with 95% confidence intervals of the effect of being exposed to a dam at birth on child mortality outcomes for the baseline estimates and when dropping one country at a time. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3.

Figure F.10. Leave-one-out analysis for nutritional status outcomes



continued →

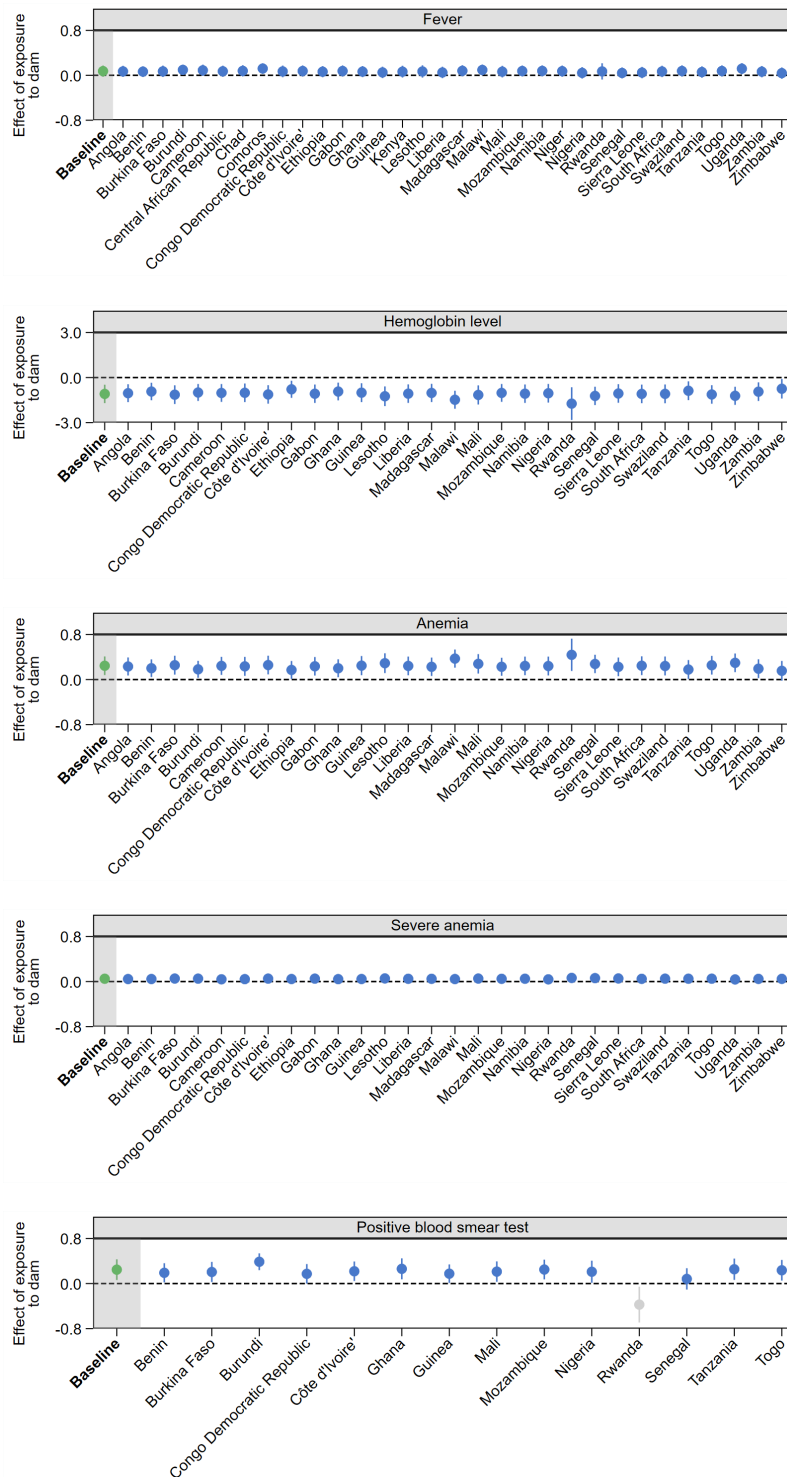
Figure F.10 (continued). Leave-one-out analysis for nutritional status outcomes



Source: Author's calculations from DHS and satellite data.

Notes: These figures report the point estimates along with 95% confidence intervals of the effect of living near a dam on child nutritional status for the baseline estimates and when dropping one country at a time. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 4.

Figure F.11. Leave-one-out analysis for malaria-related outcomes

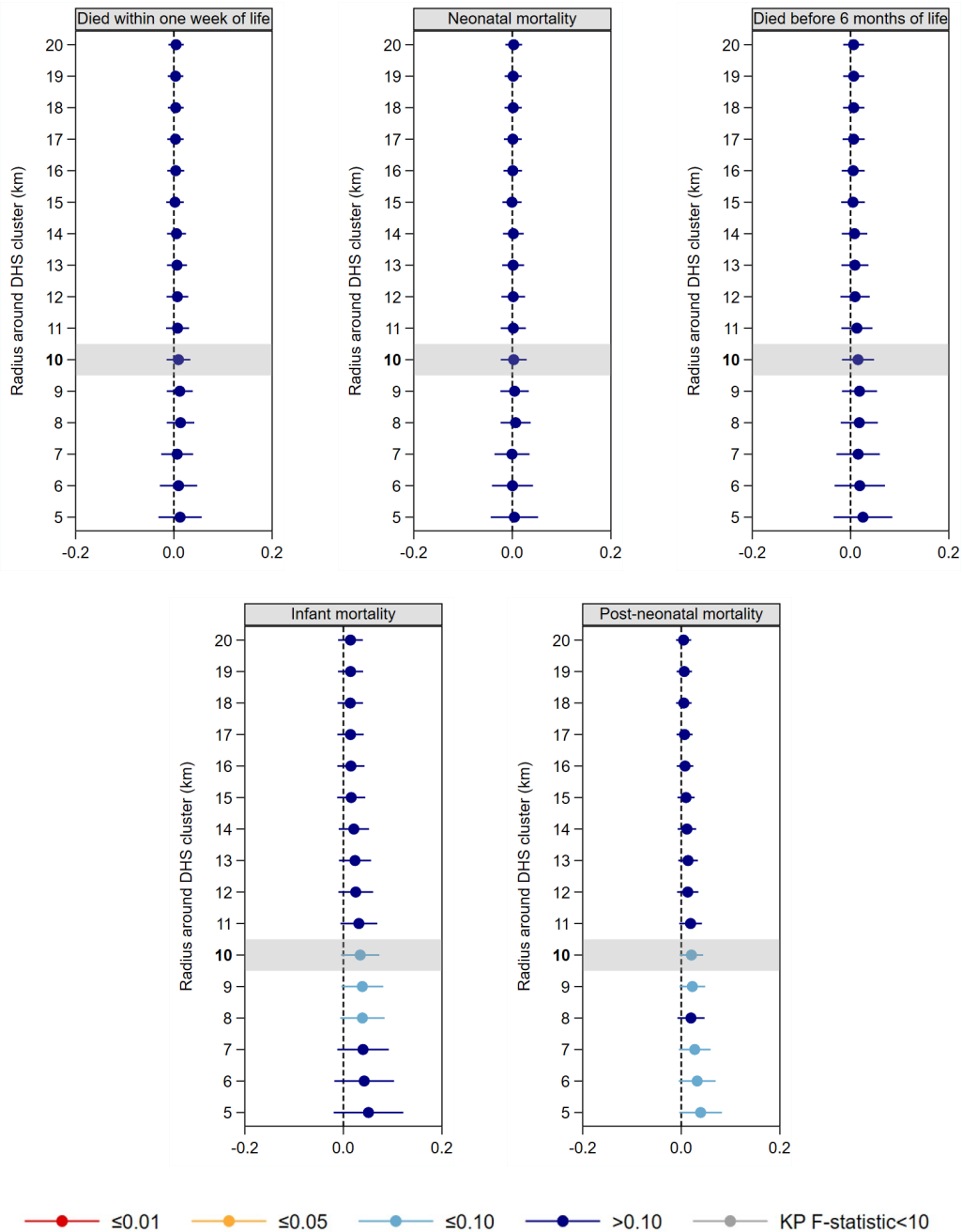


Source: Author's calculations from DHS and satellite data.

Notes: These figures report the point estimates along with 95% confidence intervals of the effect of living near a dam on child malaria-related outcomes for the baseline estimates and when dropping one country at a time. A gray dot indicates a Kleibergen-Paap F-statistic below the rule-of-thumb of 10. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 5.

F.7. Alternative radii for definition of exposure to dams

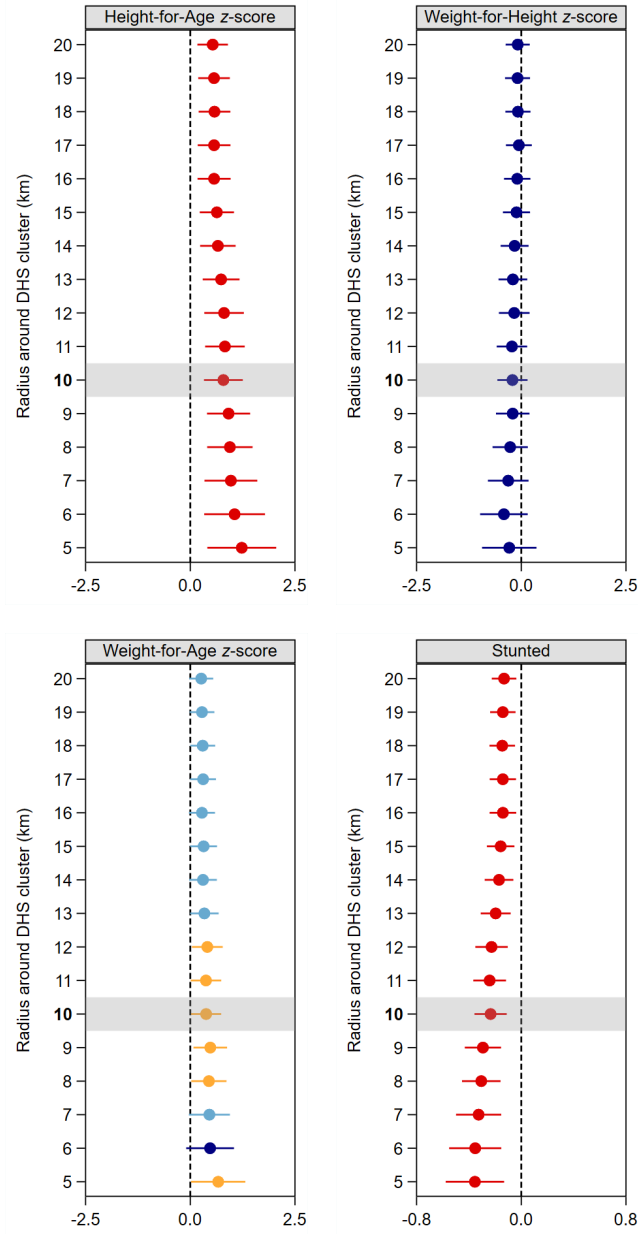
Figure F.12. Effect of dam exposure based on alternative radii on child mortality outcomes



Source: Author's calculations from DHS and satellite data.

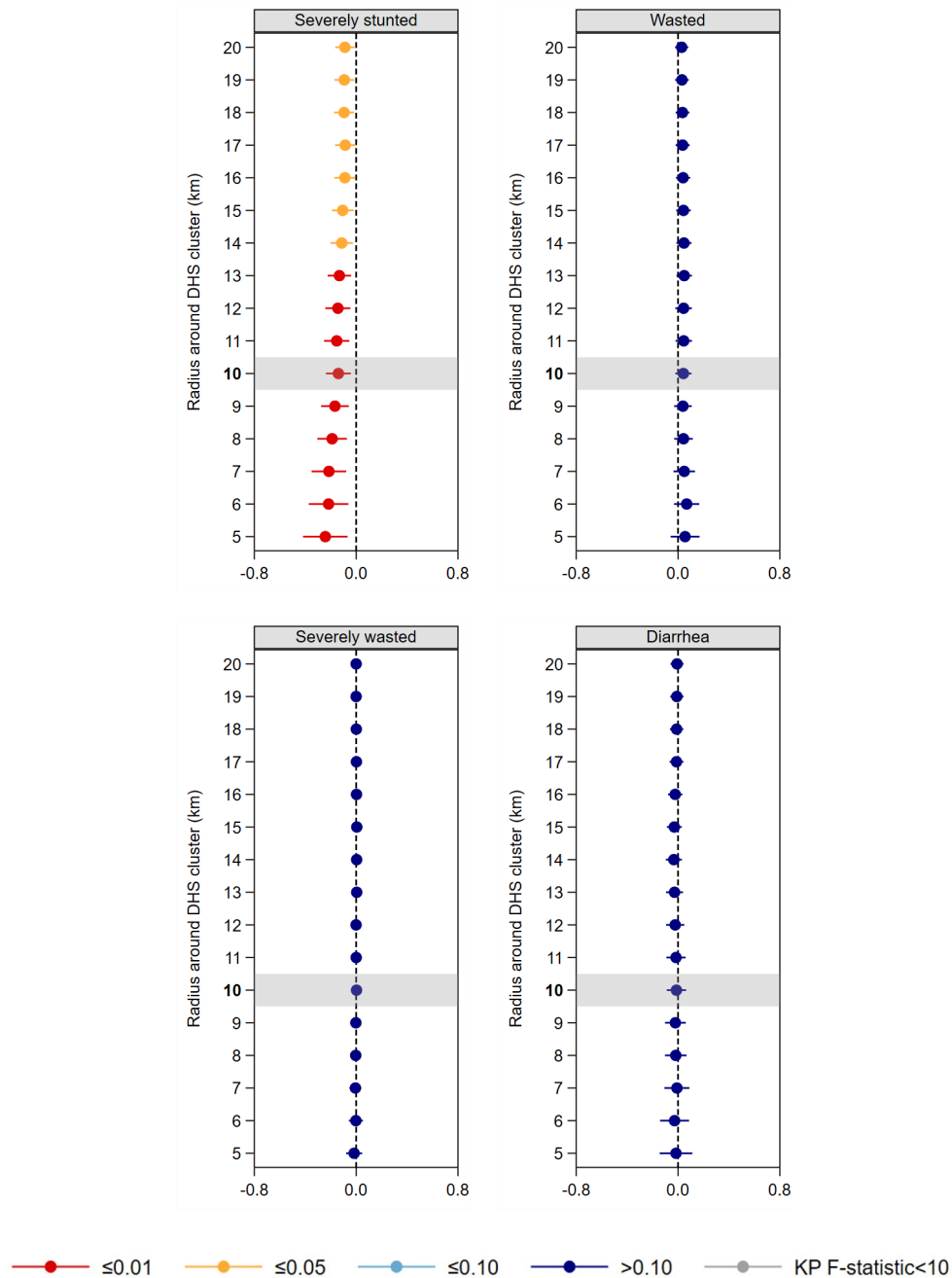
Notes: These figures report the point estimates along with 95% confidence intervals of the effect of living near a dam on child mortality outcomes for the baseline estimates and when using alternative distance thresholds to define exposure to a dam. Red, yellow, light blue and blue dots correspond to a significance level of 1%, 5%, 10%, and above 10%, respectively. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3. Geographic covariates are now defined at the radius level specified on the y -axis.

Figure F.13. Effect of dam exposure based on alternative radii on child nutritional status



continued →

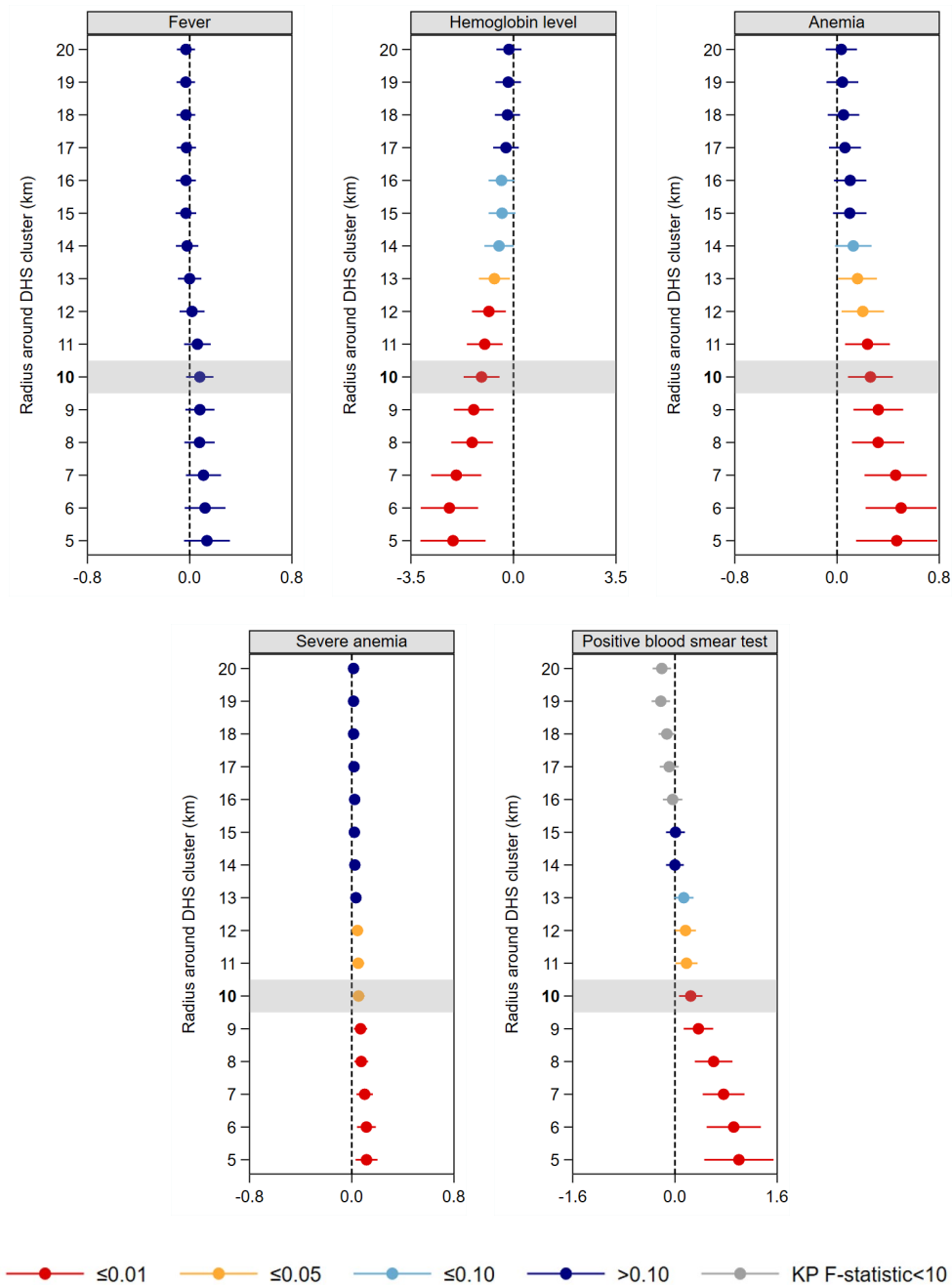
Figure F.13 (*continued*). Effect of dam exposure based on alternative radii on child nutritional status



Source: Author's calculations from DHS and satellite data.

Notes: These figures report the point estimates along with 95% confidence intervals of the effect of living near a dam on child nutritional status for the baseline estimates and when using alternative distance thresholds to define exposure to a dam. Red, yellow, light blue and blue dots correspond to a significance level of 1%, 5%, 10%, and above 10%, respectively. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 4. Geographic covariates are now defined at the radius level specified on the *y*-axis.

Figure F.14. Effect of dam exposure based on alternative radii on child malaria-related outcomes



Source: Author's calculations from DHS and satellite data.

Notes: These figures report the point estimates along with 95% confidence intervals of the effect of living near a dam on child malaria-related outcomes for the baseline estimates and when using alternative distance thresholds to define exposure to a dam. Red, yellow, light blue and blue dots correspond to a significance level of 1%, 5%, 10%, and above 10%, respectively. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 5. Geographic covariates are now defined at the radius level specified on the y-axis.

Appendix G. Control function approach

G.1. Baseline results

Table G.1. Effect of exposure to a dam on child health outcomes using the control function approach

		I. Child mortality outcomes							
		(1)	(2)	(3)	(4)	(5)			
		Died within one week of life	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality			
Exposed at birth		0.009 (0.011)	0.003 (0.012)	0.015 (0.016)	0.034** (0.017)	0.021* (0.011)			
	$\widehat{\varepsilon}_{jtsbc}$	-0.011 (0.011)	-0.005 (0.012)	-0.018 (0.016)	-0.038** (0.017)	-0.023** (0.011)			
Mean of dependent variable		0.026	0.032	0.048	0.063	0.030			
N		919,508	910,886	825,691	729,423	881,938			
		II. Nutritional status							
		(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
		HAZ	WHZ	WAZ	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Exposed at survey time		0.791*** (0.171)	-0.211 (0.133)	0.378*** (0.132)	-0.234*** (0.048)	-0.140*** (0.037)	0.042 (0.026)	0.003 (0.016)	-0.013 (0.030)
	$\widehat{\varepsilon}_{jtsbc}$	-0.740*** (0.171)	0.216 (0.133)	-0.332** (0.133)	0.218*** (0.048)	0.129*** (0.037)	-0.043* (0.026)	-0.004 (0.016)	0.009 (0.030)
Mean of dependent variable		-1.395	-0.253	-0.996	0.358	0.164	0.096	0.035	0.163
N		550,980	543,046	560,336	550,980	550,980	543,046	543,046	804,442
		III. Malaria-related outcomes							
		(14)	(15)	(16)	(17)	(18)			
		Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test			
Exposed at survey time		0.074** (0.033)	-1.089*** (0.218)	0.245*** (0.066)	0.050** (0.021)	0.247*** (0.067)			
	$\widehat{\varepsilon}_{jtsbc}$	-0.078** (0.033)	1.108*** (0.219)	-0.244*** (0.066)	-0.053** (0.021)	-0.296*** (0.068)			
Mean of dependent variable		0.244	10.261	0.641	0.036	0.182			
N		795,714	258,036	258,036	258,036	102,499			

Source: Author's calculations from DHS and satellite data.

Notes: Bootstrapped standard errors clustered at the enumeration area level reported in parentheses (500 replications). The unit of observation is a birth (columns 1 to 5) or a child alive at survey time (columns 6 to 18). The table reports the point estimates of exposure to a dam from Equation 4 on child health outcomes. In addition to the estimated residuals $\widehat{\varepsilon}_{jtsbc}$ from the first-stage equation (Equation 2), control variables and fixed effects are the same as in Table 3 for columns 1 to 5, as in Table 4 for columns 6 to 13, and as in Table 5 for columns 14 to 18.

* $p < .10$; ** $p < .05$; *** $p < .01$

G.2. Heterogeneous effects

Table G.2. Heterogeneous effects according to household agricultural status

I. Child mortality outcomes								
	(1)	(2)	(3)	(4)	(5)			
	Died within one week of life	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality			
Exposed at birth	0.008 (0.012)	0.002 (0.013)	0.017 (0.017)	0.042** (0.019)	0.027** (0.012)			
Exposed at birth × Agric. household	0.000 (0.001)	0.000 (0.001)	0.000 (0.002)	−0.002 (0.002)	−0.001 (0.002)			
Agricultural household	0.001** (0.000)	0.002*** (0.000)	0.004*** (0.001)	0.005*** (0.001)	0.003*** (0.000)			
$\widehat{\varepsilon}_{jtsbc}$	−0.010 (0.012)	−0.004 (0.013)	−0.020 (0.017)	−0.045** (0.019)	−0.029** (0.012)			
<i>p</i> -value $\beta_1 + \beta_2 = 0$	0.497	0.854	0.335	0.040	0.030			
Mean of dependent variable	0.026	0.032	0.048	0.063	0.030			
N	919,508	910,886	825,691	729,423	881,938			
II. Nutritional status								
	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
	HAZ	WHZ	WAZ	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Exposed at survey time	0.635*** (0.190)	−0.101 (0.153)	0.350** (0.136)	−0.215*** (0.052)	−0.107** (0.044)	0.022 (0.029)	−0.001 (0.018)	−0.004 (0.033)
Exposed at survey time × Agric. household	0.012 (0.019)	−0.023 (0.016)	−0.020 (0.015)	−0.005 (0.005)	−0.004 (0.004)	0.006* (0.003)	0.003 (0.002)	0.001 (0.003)
Agricultural household	−0.191*** (0.006)	−0.030*** (0.004)	−0.139*** (0.004)	0.050*** (0.002)	0.028*** (0.001)	0.005*** (0.001)	0.001** (0.001)	0.011*** (0.001)
$\widehat{\varepsilon}_{jtsbc}$	−0.595*** (0.190)	0.116 (0.153)	−0.298** (0.137)	0.202*** (0.052)	0.098** (0.044)	−0.025 (0.029)	−0.001 (0.018)	0.000 (0.033)
<i>p</i> -value $\beta_1 + \beta_2$	0.001	0.415	0.015	0.000	0.011	0.334	0.922	0.936
Mean of dependent variable	−1.400	−0.252	−1.000	0.360	0.166	0.097	0.035	0.163
N	532,482	524,589	541,579	532,482	532,482	524,589	524,589	777,086
III. Malaria-related outcomes								
	(14)	(15)	(16)	(17)	(18)			
	Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test			
Exposed at survey time	0.087** (0.038)	−1.296*** (0.238)	0.289*** (0.071)	0.060** (0.023)	0.165** (0.070)			
Exposed at survey time × Agric. household	0.000 (0.004)	0.061** (0.023)	−0.015** (0.007)	−0.003 (0.003)	0.077*** (0.011)			
Agricultural household	0.022*** (0.001)	−0.185*** (0.008)	0.039*** (0.002)	0.011*** (0.001)	0.048*** (0.003)			
$\widehat{\varepsilon}_{jtsbc}$	−0.091** (0.038)	1.286*** (0.239)	−0.282*** (0.071)	−0.061*** (0.023)	−0.252*** (0.070)			
<i>p</i> -value $\beta_1 + \beta_2$	0.020	0.000	0.000	0.012	0.001			
Mean of dependent variable	0.244	10.244	0.645	0.037	0.183			
N	770,895	251,914	251,914	251,914	101,344			

Source: Author's calculations from DHS and satellite data.

Notes: Bootstrapped standard errors clustered at the enumeration area level reported in parentheses (500 replications). The unit of observation is a birth (columns 1 to 5) or a child alive at survey time (columns 6 to 18). The table reports the point estimates of exposure to a dam from Equation 5 on child health outcomes depending on the agricultural status of the household. Control variables and fixed effects are the same as in Table 3 for columns 1 to 5, as in Table 4 for columns 6 to 13, and as in Table 5 for columns 14 to 18.

* $p < .10$; ** $p < .05$; *** $p < .01$

Table G.3. Heterogeneous effects according to child's sex

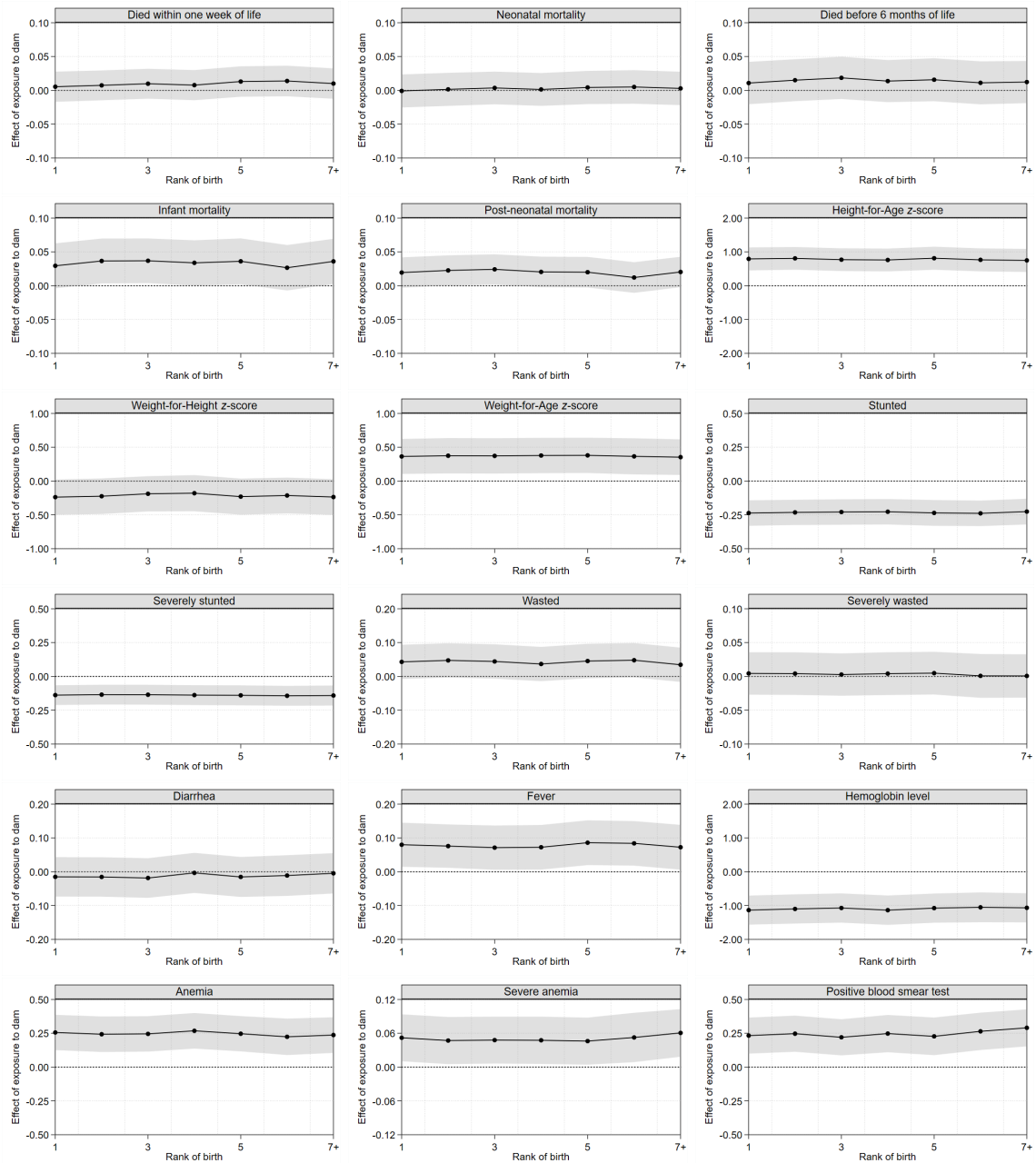
I. Child mortality outcomes								
	(1)	(2)	(3)	(4)	(5)			
	Died within one week of life	Neonatal mortality	Died before 6 months of life	Infant mortality	Post-neonatal mortality			
Exposed at birth	0.010 (0.011)	0.004 (0.012)	0.015 (0.014)	0.035** (0.017)	0.021* (0.012)			
Exposed at birth × Girl	-0.001 (0.001)	-0.002 (0.001)	-0.001 (0.002)	-0.002 (0.002)	0.000 (0.001)			
Girl	-0.008*** (0.000)	-0.009*** (0.000)	-0.010*** (0.000)	-0.011*** (0.001)	-0.002*** (0.000)			
$\widehat{\varepsilon}_{jtsbc}$	-0.011 (0.012)	-0.005 (0.012)	-0.018 (0.014)	-0.038** (0.017)	-0.023* (0.012)			
p -value $\beta_1 + \beta_2 = 0$	0.450	0.891	0.294	0.053	0.077			
Mean of dependent variable	0.026	0.032	0.048	0.063	0.030			
N	919,508	910,886	825,691	729,423	881,938			
II. Nutritional status								
	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)
	HAZ	WHZ	WAZ	Stunted	Severely stunted	Wasted	Severely wasted	Diarrhea
Exposed at survey time	0.788*** (0.180)	-0.208 (0.141)	0.371*** (0.125)	-0.234*** (0.050)	-0.141*** (0.039)	0.042 (0.026)	0.004 (0.016)	-0.012 (0.030)
Exposed at survey time × Girl	0.005 (0.017)	-0.005 (0.014)	0.014 (0.013)	-0.001 (0.005)	0.002 (0.003)	0.001 (0.003)	-0.002 (0.002)	-0.002 (0.003)
Girl	0.160*** (0.005)	0.024*** (0.004)	0.102*** (0.004)	-0.046*** (0.001)	-0.031*** (0.001)	-0.015*** (0.001)	-0.007*** (0.000)	-0.011*** (0.001)
$\widehat{\varepsilon}_{jtsbc}$	-0.740*** (0.179)	0.216 (0.141)	-0.332*** (0.126)	0.218*** (0.050)	0.129*** (0.039)	-0.043 (0.026)	-0.004 (0.016)	0.009 (0.030)
p -value $\beta_1 + \beta_2$	0.000	0.130	0.002	0.000	0.000	0.109	0.903	0.646
Mean of dependent variable	-1.395	-0.253	-0.996	0.358	0.164	0.096	0.035	0.163
N	550,980	543,046	560,336	550,980	550,980	543,046	543,046	804,442
III. Malaria-related outcomes								
	(14)	(15)	(16)	(17)	(18)			
	Fever	Hemoglobin level	Anemia	Severe anemia	Positive blood smear test			
Exposed at survey time	0.077** (0.034)	-1.087*** (0.215)	0.246*** (0.065)	0.050** (0.020)	0.250*** (0.065)			
Exposed at survey time × Girl	-0.007* (0.003)	-0.005 (0.023)	-0.002 (0.007)	-0.001 (0.003)	-0.008 (0.010)			
Girl	-0.007*** (0.001)	0.121*** (0.007)	-0.027*** (0.002)	-0.005*** (0.001)	-0.001 (0.002)			
$\widehat{\varepsilon}_{jtsbc}$	-0.078** (0.034)	1.108*** (0.214)	-0.244*** (0.065)	-0.053*** (0.020)	-0.296*** (0.066)			
p -value $\beta_1 + \beta_2$	0.036	0.000	0.000	0.013	0.000			
Mean of dependent variable	0.244	10.261	0.641	0.036	0.182			
N	795,714	258,036	258,036	258,036	102,499			

Source: Author's calculations from DHS and satellite data.

Notes: Bootstrapped standard errors clustered at the enumeration area level reported in parentheses (500 replications). The unit of observation is a birth (columns 1 to 5) or a child alive at survey time (columns 6 to 18). The table reports the point estimates of exposure to a dam from Equation 5 on child health outcomes depending on child's sex. Control variables and fixed effects are the same as in Table 3 for columns 1 to 5, as in Table 4 for columns 6 to 13, and as in Table 5 for columns 14 to 18.

* $p < .10$; ** $p < .05$; *** $p < .01$

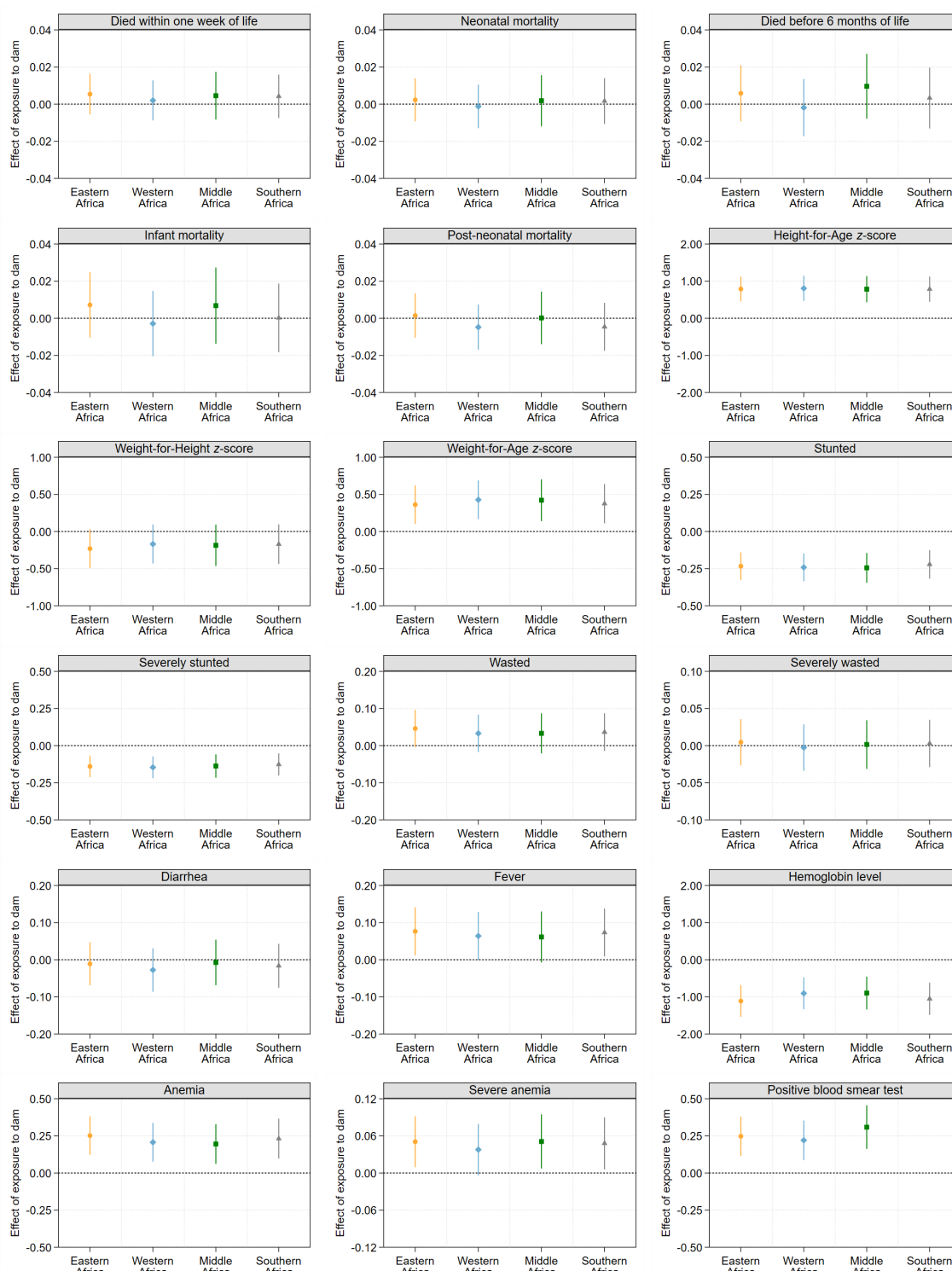
Figure G.4. Heterogeneous effects according to child's rank of birth



Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figures plot the point estimates along with 95% confidence intervals from interaction terms between exposure to a dam and a set of dummies for child's rank of birth. Each figure is from a separate estimation. Bootstrapped standard errors (500 replications) are clustered at the enumeration area level. In addition to child's rank of birth, control variables and fixed effects are the same as in Table 3, 4 or 5 depending on the outcome.

Figure G.5. Heterogeneous effects across Sub-Saharan African regions

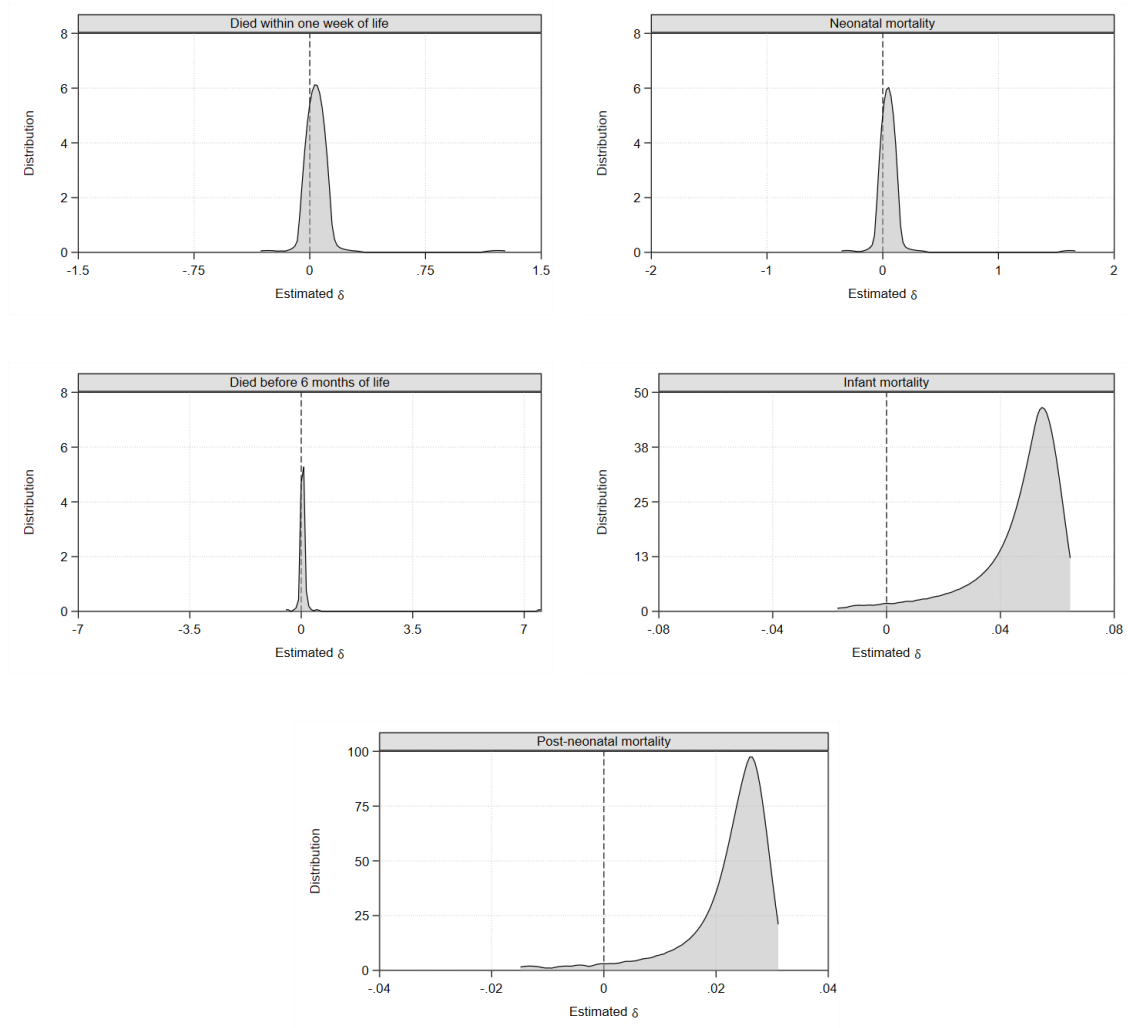


Source: Author's calculations from DHS 1996, 2001, 2007 and 2013.

Notes: The figures plot the point estimates along with 95% confidence intervals of the effect of exposure to a dam across the different Sub-Saharan African regions. Each figure is from a separate estimation. Bootstrapped standard errors (500 replications) are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3, 4 or 5 depending on the outcome. **Eastern Africa:** Burundi, Comoros, Ethiopia, Kenya, Malawi, Mozambique, Rwanda, Tanzania, Uganda, Zambia, Zimbabwe. **Middle Africa:** Angola, Cameroon, Central African Republic, Chad, Democratic Republic of the Congo, Gabon. **Southern Africa:** Lesotho, Madagascar, Namibia, South Africa, Swaziland. **Western Africa:** Benin, Burkina Faso, Côte d'Ivoire, Ghana, Guinea, Liberia, Mali, Niger, Nigeria, Senegal, Sierra Leone, Togo.

Appendix H. Sensitivity of OLS estimates to selection on unobservables

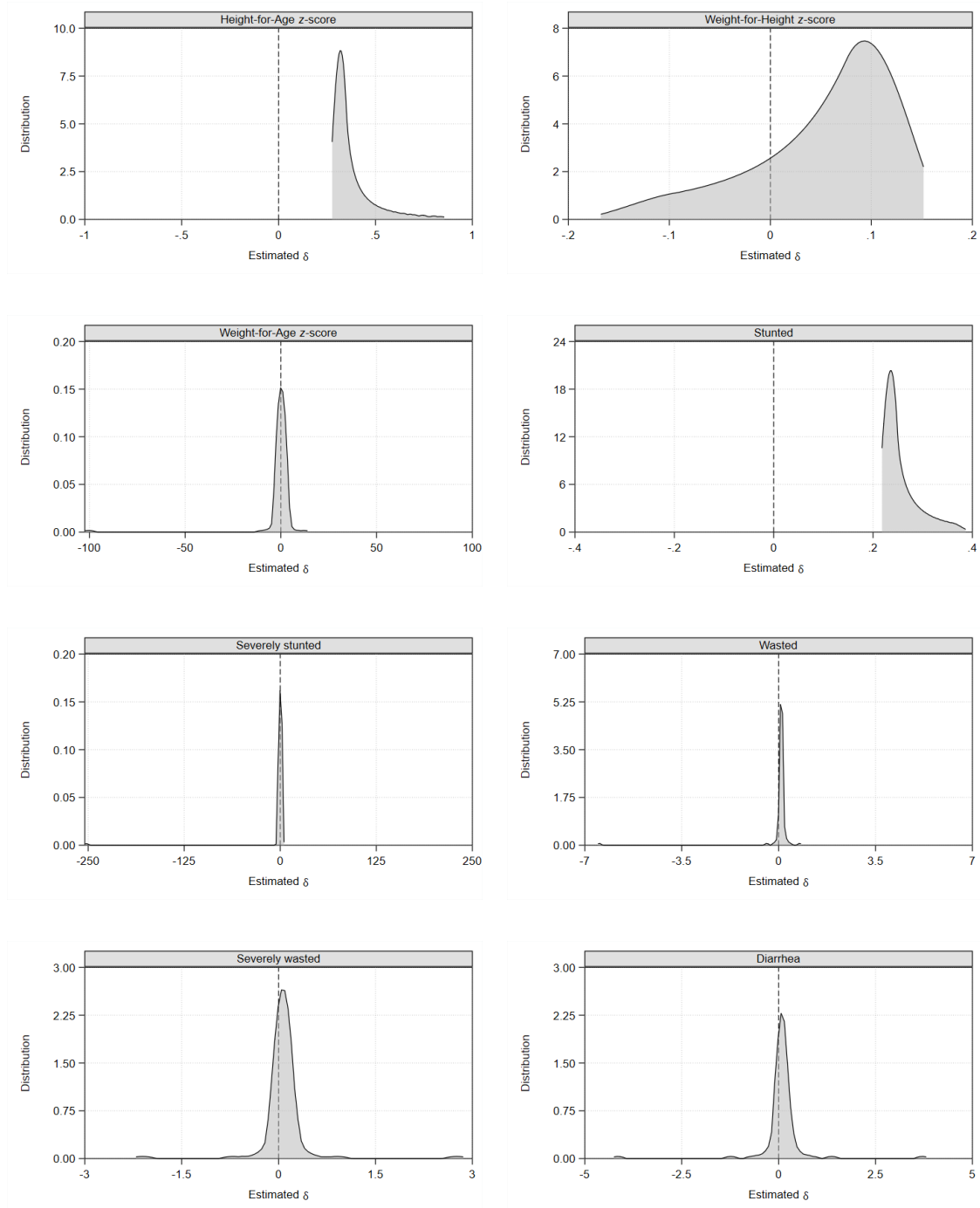
Figure H.1. Distribution of $\hat{\delta}$ for child mortality outcomes



Source: Author's calculations from DHS and satellite data.

Notes: These figures plot the distribution of the $\hat{\delta}$ from Oster [2019] estimated for 100 equally-spaced values x_i with $i \in \{1, \dots, 100\}$ from the corresponding 2SLS 95% confidence interval obtained from Panel B of Table 3. δ is the coefficient of proportionality of selection on unobservables relative to selection on control variables that would be necessary for the OLS point estimate to reach the value x_i . Control variables and fixed effects are the same as in Table 3.

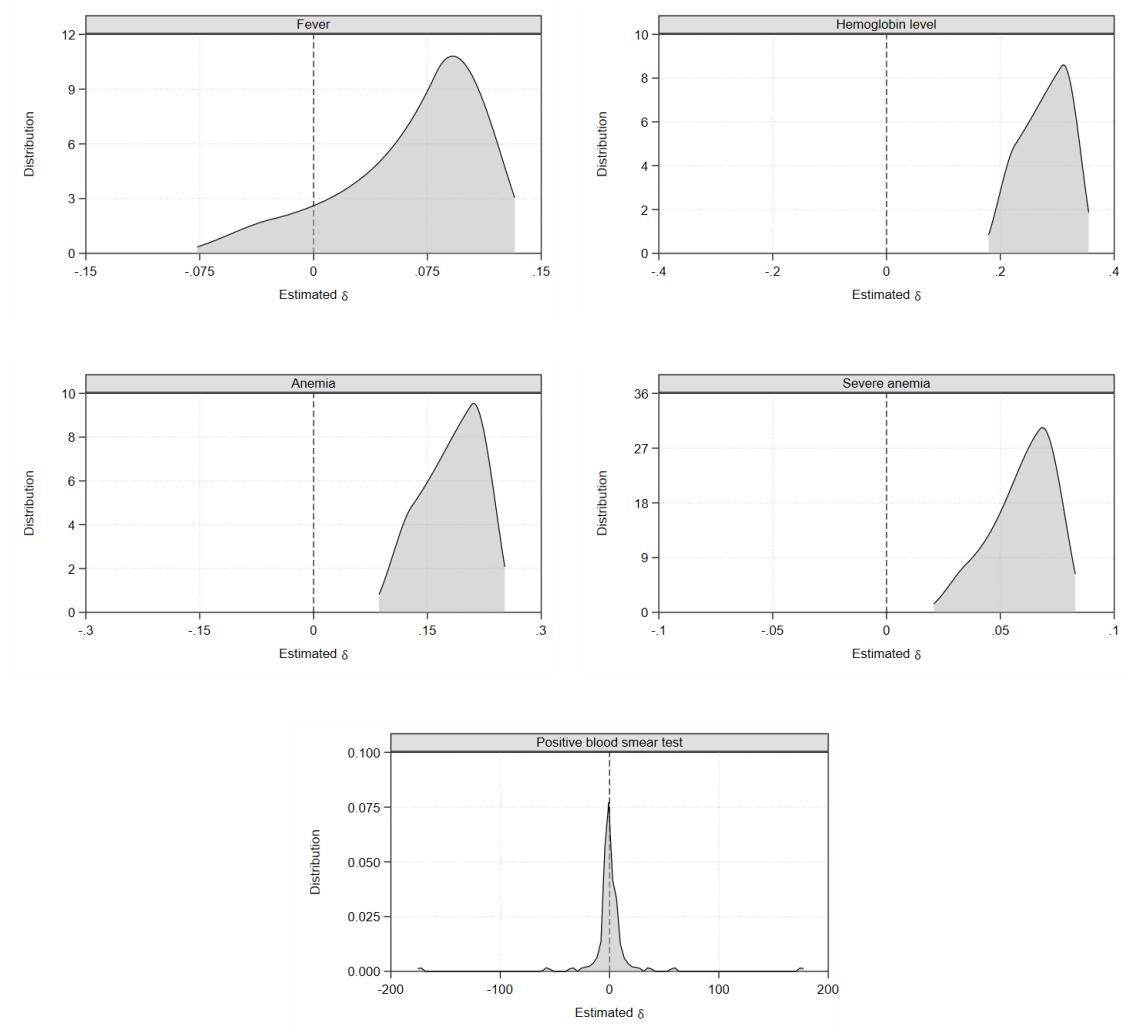
Figure H.2. Distribution of $\hat{\delta}$ for nutritional status outcomes



Source: Author's calculations from DHS and satellite data.

Notes: These figures plot the distribution of the $\hat{\delta}$ from Oster [2019] estimated for 100 equally-spaced values x_i with $i \in \{1, \dots, 100\}$ from the corresponding 2SLS 95% confidence interval obtained from Panel B of Table 4. δ is the coefficient of proportionality of selection on unobservables relative to selection on control variables that would be necessary for the OLS point estimate to reach the value x_i . Control variables and fixed effects are the same as in Table 4.

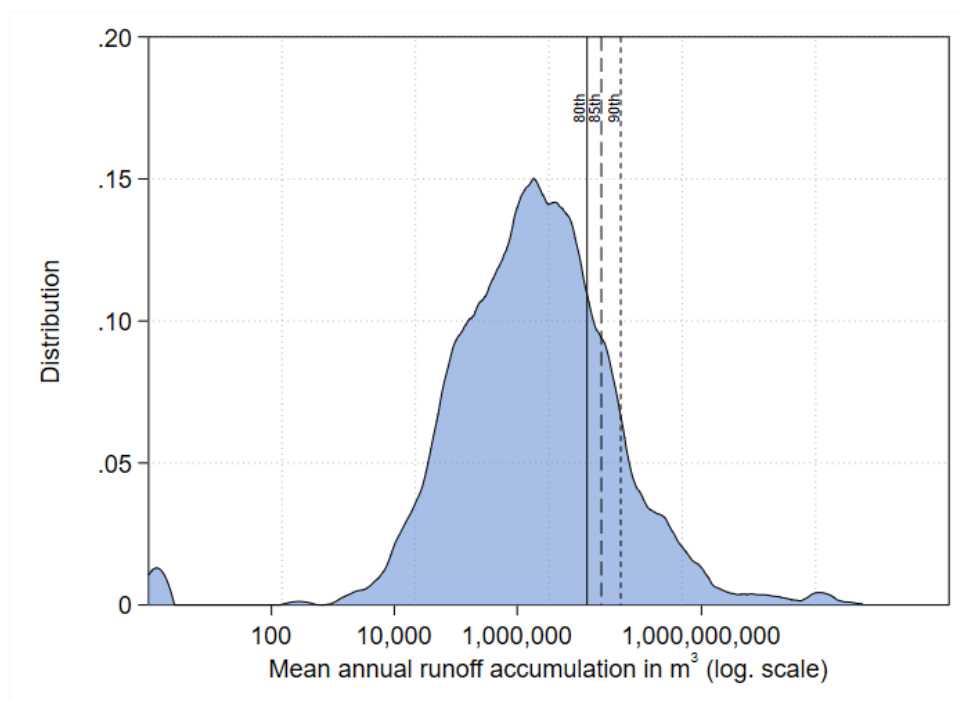
Figure H.3. Distribution of $\hat{\delta}$ for malaria-related outcomes



Source: Author's calculations from DHS and satellite data.

Notes: These figures plot the distribution of the $\hat{\delta}$ from Oster [2019] estimated for 100 equally-spaced values x_i with $i \in \{1, \dots, 100\}$ from the corresponding 2SLS 95% confidence interval obtained from Panel B of Table 5. δ is the coefficient of proportionality of selection on unobservables relative to selection on control variables that would be necessary for the OLS point estimate to reach the value x_i . Control variables and fixed effects are the same as in Table 5.

Figure I.2. Distribution of mean annual runoff accumulation in dam’s watershed



Source: Author’s calculations from NASA-GLDAS and satellite data.

Notes: The figure plots the distribution of the mean annual runoff accumulation in dam’s watershed in cubic meters. x -axis is in log-scale. Vertical lines correspond to the different percentiles of the distribution (in level) used to partition dams between small and large ones: solid line indicates the 80th percentile, long dash pattern the 85th percentile and short dash pattern the 90th percentile.

I.2. Exposure according to dam size

Table I.3. Share of under-5 children exposed to a dam by size

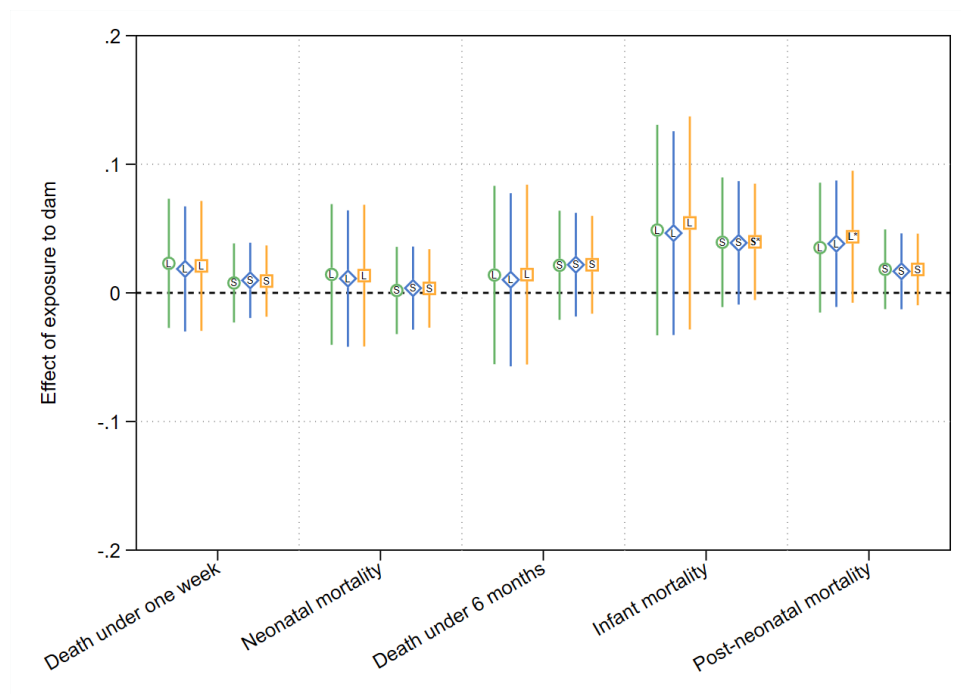
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	Threshold used to partition dams									
Any dam	80 th percentile			85 th percentile			90 th percentile			
	Small	Large	Both	Small	Large	Both	Small	Large	Both	
Exposed at birth	0.073	0.031	0.028	0.013	0.039	0.023	0.011	0.046	0.019	0.008
Exposed at survey time	0.073	0.031	0.028	0.014	0.040	0.023	0.011	0.047	0.018	0.008

Source: Author’s calculations from DHS, NASA-GLDAS and satellite data.

Notes: Unweighted statistics. The first column reports the share of children exposed to a dam, whatever its size. The following columns decompose exposure to a dam according to its size, based on alternative thresholds from the distribution of mean annual runoff draining into the dam. For each threshold, the table reports the share of children exposed only to dams considered as small (columns 2, 5, and 8), as large (columns 3, 6, and 9) or to both types of dams (columns 4, 7, and 10).

I.3. Heterogeneous effects according to dam size

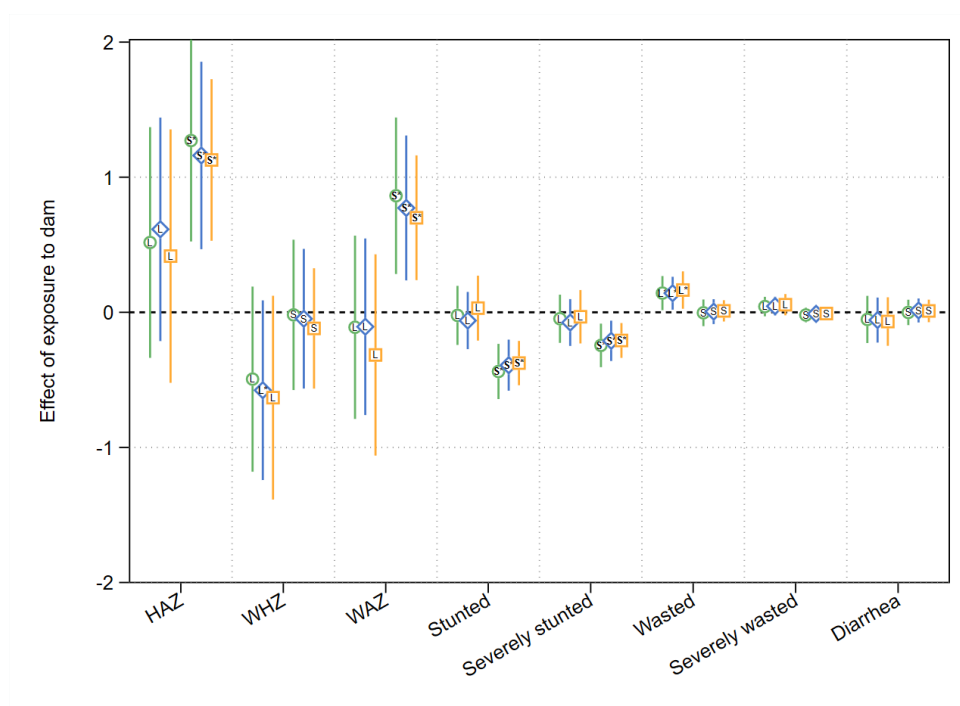
Figure I.4. Heterogeneous effect of exposure to a dam on child mortality depending on dam size



Source: Author's calculations from DHS, NASA-GLDAS and satellite data.

Notes: The figure plots the point estimates along with 95% confidence intervals of the effect of being exposed to a large (L) or a small (S) dam at birth on child mortality outcomes. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 3. Point estimates are represented by green circles when using the 80th percentile of the distribution of mean annual runoff in dam's watershed to partition small and large dams, by blue diamonds when using the 85th percentile, and by yellow squares when using the 90th percentile. * are shown for point estimates significantly different from zero for a significance level of at least 10% ($p < .10$).

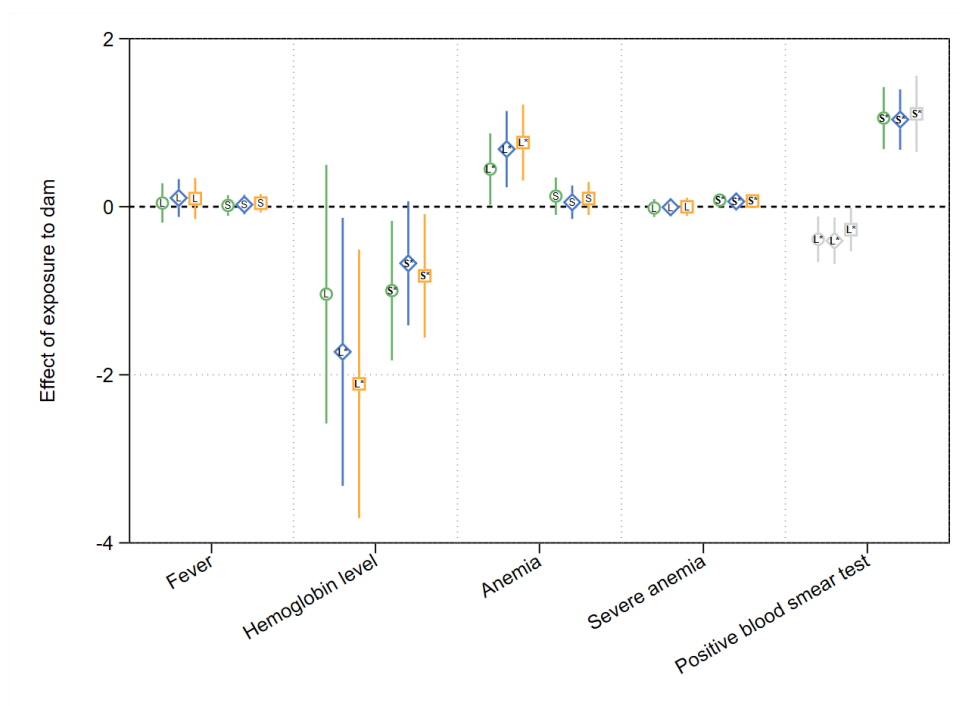
Figure I.5. Heterogeneous effect of exposure to a dam on child nutritional status depending on dam size



Source: Author's calculations from DHS, NASA-GLDAS and satellite data.

Notes: The figure plots the point estimates along with 95% confidence intervals of the effect of living near a large (L) or a small (S) dam on child nutritional status. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 4. Point estimates are represented by green circles when using the 80th percentile of the distribution of mean annual runoff in dam's watershed to partition small and large dams, by blue diamonds when using the 85th percentile, and by yellow squares when using the 90th percentile. * are shown for point estimates significantly different from zero for a significance level of at least 10% ($p < .10$).

Figure I.6. Heterogeneous effect of exposure to a dam on child malaria-related outcomes depending on dam size



Source: Author's calculations from DHS, NASA-GLDAS and satellite data.

Notes: The figure plots the point estimates along with 95% confidence intervals of the effect of living near a large (L) or a small (S) dam on child malaria-related outcomes. Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 5. Point estimates are represented by green circles when using the 80th percentile of the distribution of mean annual runoff in dam's watershed to partition small and large dams, by blue diamonds when using the 85th percentile, and by yellow squares when using the 90th percentile. Point estimates for which the Kleibergen-Paap F-statistic is below 10 are shown in gray. * indicates point estimates significantly different from zero for a significance level of at least 10% ($p < .10$).

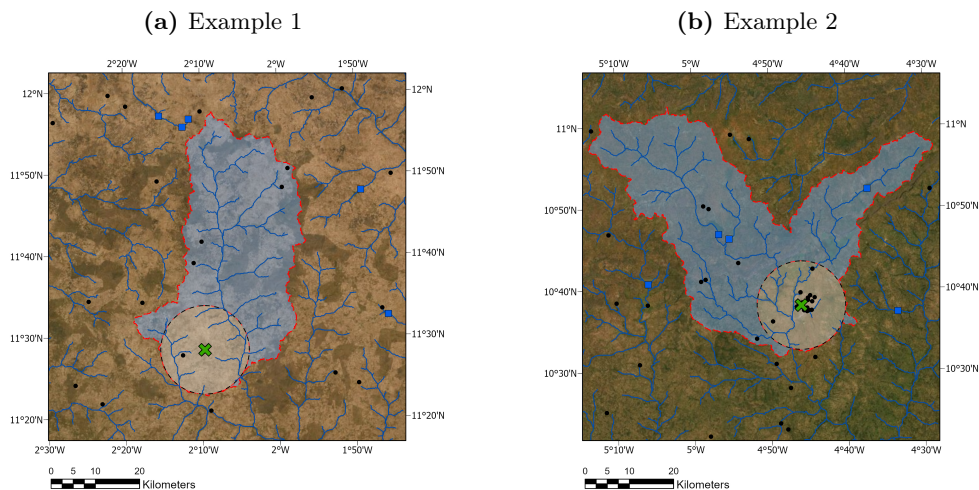
Appendix J. Watersheds

J.1. Watersheds delineation and exposure to an upstream dam

For each DHS cluster, I delineate the upstream land area up to 100 kilometers from its GPS coordinates that drains all rivers and rainfall into the 10 kilometers buffer around it. To do so, I exploit the drainage lines position as well as elevation data. I represent two examples by the blue area in Figure J.1 below.

Based on these boundaries, I define as exposed to an upstream dam at time t all individuals from a DHS cluster with a dam in this area by time t . Following this definition, in example 1 (Figure J.1 (a)), the DHS cluster represented by the green cross does not have a dam in its watershed: there is no dam (blue square) in the blue area. Hence, individuals from this DHS cluster are not exposed to an upstream dam. Conversely, in example 2 (Figure J.1 (b)), the DHS cluster represented by the green cross has three different dams located upstream. Individuals from this DHS cluster will be considered as exposed to an upstream dam at time t if at least one of these dams as been built before time t .

Figure J.1. Examples of watersheds in Burkina Faso



Source: Author's computation

Notes: The maps show two examples of watersheds delineated for two DHS clusters in Burkina Faso. In subfigure (a), there is no dam within the 10km buffer around the DHS cluster, nor on its watershed. In subfigure (b), there is no dam within the 10km buffer, but three dams within its watershed.

J.2. Summary statistics on watersheds characteristics

Table J.2. Summary statistics at the DHS cluster level on watersheds characteristics

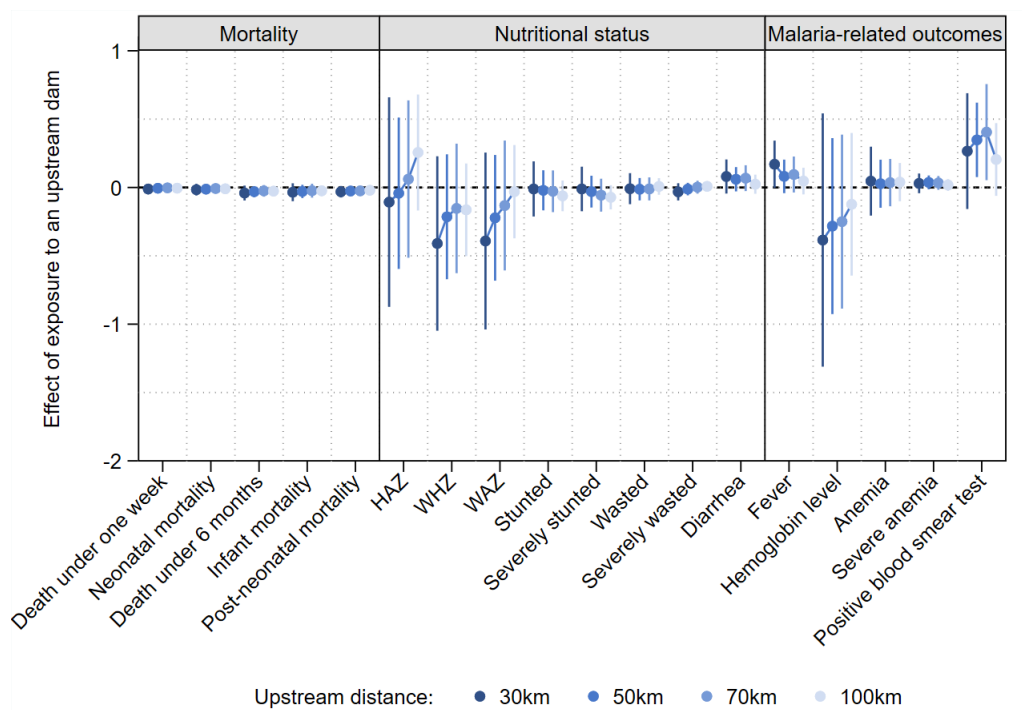
	(1) Whole sample	(2) Dam present in watershed	(3) Dam not present in watershed	(4) (2) = (3) <i>p</i> -value
<i>Panel A. Geographic characteristics of watersheds</i>				
Area	2578.604	5261.752	2044.387	0.000
Elevation	882.682	1054.235	848.525	0.000
Terrain gradient (%)	8.454	8.385	8.468	0.387
Drainage lines length (km)	658.173	1344.992	521.426	0.000
Drainage lines gradient (%)	1.044	1.025	1.048	0.145
<i>Panel B. Upstream dam exposure</i>				
Exposed to an upstream dam by survey time	0.163	0.982	0.000	0.000
Number of upstream dams by survey time	1.628	9.806	0.000	0.000
N	44,808	7,440	37,368	44,808

Source: Author's calculations from DHS and satellite data.

Notes: Unweighted statistics. The first column reports summary statistics for the whole sample of DHS clusters. The following two columns report the same statistics when the sample is restricted to DHS clusters with (column 2) or without (column 3) a dam in its watershed. Column 4 reports the *p*-values from *t*-tests on the equality of means reported in columns 2 and 3.

J.3. Results when using restricted watersheds

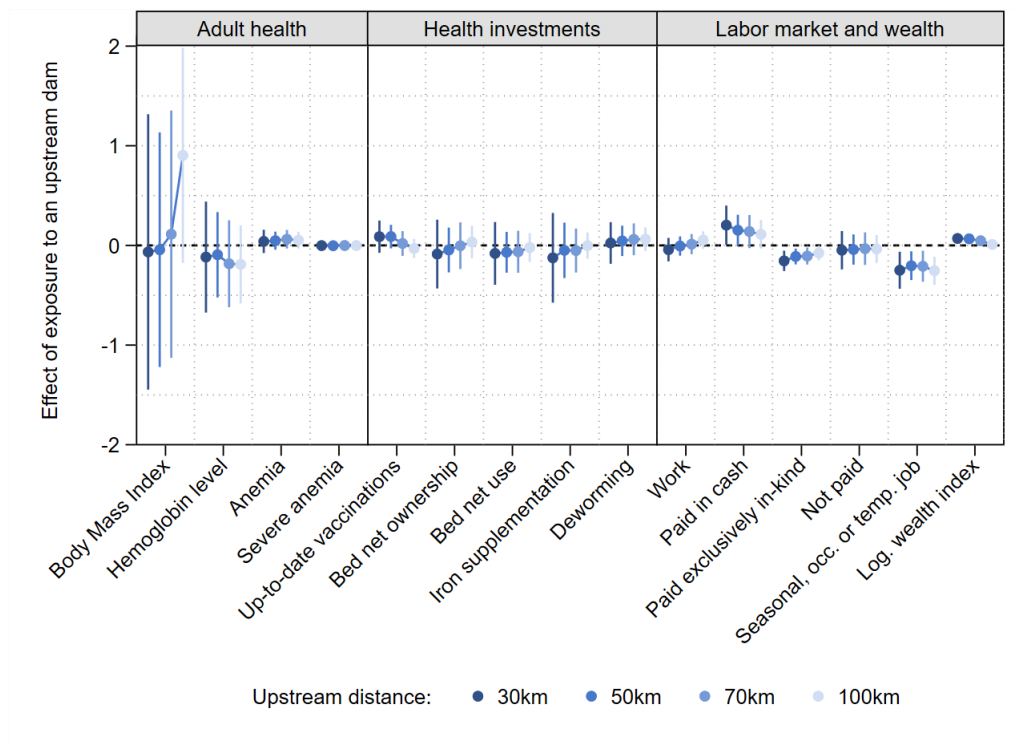
Figure J.3. Effect of exposure to an upstream dam on child health using alternative distance thresholds to delineate the watershed



Source: Author's calculations from DHS and satellite data.

Notes: The figure plots the point estimates along with 95% confidence intervals of the effect of living downstream to a dam on child health outcomes using alternative distance thresholds to delineate the watershed (30, 50, 70 and 100 kilometers). Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 10.

Figure J.4. Effect of exposure to an upstream dam on adult health, health investments, labor market and wealth using alternative distance thresholds to delineate the watershed



Source: Author's calculations from DHS, NASA-GLDAS and satellite data.
Notes: The figure plots the point estimates along with 95% confidence intervals of the effect of living downstream to a dam on adult health, health investments, labor market and wealth outcomes using alternative distance thresholds to delineate the watershed (30, 50, 70 and 100 kilometers). Robust standard errors are clustered at the enumeration area level. Control variables and fixed effects are the same as in Table 11.

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General Conclusion

The substantial progress made over the last forty years is undeniable, but most Sub-Saharan African countries are still lagging behind on the road to *health for all*. Development efforts and public policies do not always succeed in achieving their objectives, inviting researchers and policy makers to identify and understand factors that either bolster or slow down progress in improving population health.

This thesis has humbly sought to uncover and better understand some of these factors in the context of Sub-Saharan African countries. It first presents an empirical assessment of the effectiveness of a free health care policy in increasing the use of health services and improving child health. It then proposes to revisit the literature on the intergenerational effect of education by disentangling the improvement in health investments behaviors and child health brought about by higher levels of mother's and father's education, respectively. Finally, it explores the local health consequences of water reservoir construction in Sub-Saharan Africa. I here summarize the main findings from each chapter and discuss their policy implications.

*
* *

How do maternal health care utilization and child health outcomes respond to a drop in the price of primary health services?

The analysis of the effects of a free primary health care policy implemented in Zambia has revealed that demand for maternal health care services strongly reacts to a fall in price, with a large and sustained increase in institutional deliveries. As a result, the probability of being assisted by a skilled birth attendant during childbirth has sharply increased. A higher share of women has benefited from a postnatal check-up within the first 24 hours following childbirth thanks to the policy. Still, the results suggest that this procedure is far from universal as many women did not benefit from it despite delivering in a health facility. The analysis has then shown that removing user fees has had a limited impact on child health. Chronic malnutrition decreased, but there is no discernible average change in infant mortality risk following the policy. Further investigations suggest that this result is not driven by potential fertility or selection effects set off by the removal. I then uncover important heterogeneity with respect to other supply-side factors. The positive effects on maternal health care utilization are limited in space, as households living more than twenty kilometers away from a health center did not benefit from the removal. The absence of effect on average mortality also masks a heterogeneous effect depending on the quality of

care as neonatal mortality decreased in the vicinity of health facilities providing essential emergency obstetric care and child health services. These findings have important policy implications as they illustrate the existence of a twin challenge: making health services both financially and physically accessible, and of better quality for all. Important investments in infrastructures, equipment, and personnel are necessary to achieve this goal. These results also question the capacity of such health care systems to make higher levels of care accessible for all, given the small number and high spatial concentration of hospitals.

*
* *

What is the respective causal effect of mother's and father's education on health investment behaviors and child health?

The empirical assessment of the respective causal effect of mother's and father's education departs from the current literature that has completely overlooked the role of father's education, which is at best considered as a mechanism through which mother's education affects child health. Our results confirm the existence of a high correlation between parents' education and both health investments and child health outcomes. Nevertheless, when the potential endogeneity of education variables and assortative mating are accounted for, our results reveal that father's education matters more than mother's education in raising health investments. The analysis then explores several mechanisms that may drive our results. We find a strong negative effect of father's education on fertility preferences within the household, while mother's education increases her use of modern contraception and delays her age at first birth. Additional results suggest that father's education not only affects investment in health indirectly through better living conditions but also has a direct positive effect on it. We find that living with both parents is a significant determinant of child health but does not affect the estimation of the intergenerational effect of education on health in our setting. Overall, our results underline the predominance of father's education in determining health behaviors within the household. An important implication for future research is that not considering both parents' education levels simultaneously may produce misleading conclusions. Our findings also have important policy implications as they suggest that fathers should not be left out of the design of health policies aiming to improve health investment decisions and child health. In particular, part of the expected health returns to rising education levels are likely to come from fathers.

*
* *

Do dams affect the health status of children living near them in Sub-Saharan Africa?

The analysis of rich microdata matched to a unique record of dam construction in Sub-Saharan Africa has revealed conflicting effects of exposure to a dam on child health, with a decrease in chronic malnutrition accompanied by an increase in malaria prevalence. Overall, the results point to a higher infant mortality risk in the vicinity of dams, exclusively driven by a rise in mortality in the post-neonatal period, when babies' natural immunity against malaria acquired from their mother during childbearing fades away. Conclusions drawn from adult health outcomes confirm the existence of an adverse effect of dams on health. Heterogeneity analysis then reveals that such effects are equally shared between siblings but that children from agricultural households suffer disproportionately from the increased malaria risk. The analysis has then scrutinized several channels through which the presence of a dam might affect child health. Results reveal a positive income effect of dams as well as important labor-market effects. There is no discernible effect of dams on fertility behaviors, but results suggest an improvement in access to health care in their vicinity, potentially driven by positive effects on local development and the loosening of households' financial constraints. Health investment behaviors are not affected, except those related to malaria prevention, as both ownership and utilization of mosquito bed nets are higher near dams. Additional results finally stress out the importance of the size of the dam but do not suggest any effect of upstream dams on child health. These findings have important policy implications as they shed new light on the local health effects of dams in Sub-Saharan Africa. They should not be interpreted as evidence against dam construction as the results point to positive effects on labor-market outcomes and household wealth, as well as substantial improvement in child nutritional status. However, complementary policies are needed to mitigate their adverse effect on malaria risk: if the local population had a better capacity to protect against mosquito bites, the net effect of dams on child health could turn out to be positive. Such policies include large prevention campaigns and free distribution of prevention equipment in their vicinity. The development of innovative prevention tools to cope with increasing insecticide resistance and prevent outdoor biting must also be encouraged.

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List of Figures

General Introduction

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RÉSUMÉ

Au-delà d'être un objectif de justice sociale en soi, garantir un niveau de santé suffisamment élevé à chaque enfant apparaît comme essentiel au regard des multiples conséquences tout au long de la vie d'un mauvais état de santé durant la petite enfance. Malgré les nombreux efforts déployés, la situation demeure très préoccupante, en particulier en Afrique Subsaharienne. Identifier les facteurs qui enravent ou ralentissent l'amélioration de l'état de santé des jeunes enfants est ainsi primordial. Cette thèse s'inscrit dans la nécessaire poursuite des efforts de recherche destinés à fournir des preuves empiriques pour informer les politiques publiques. Elle apporte un éclairage nouveau sur plusieurs enjeux liés à l'amélioration de l'état de santé des jeunes enfants, tels que l'accès aux soins, les comportements de santé des ménages ou encore les effets sanitaires du développement d'infrastructures économiques telles que les retenues d'eau, et ce dans des contextes où les ressources demeurent limitées. Le premier chapitre montre que la mise en place de la gratuité des soins primaires en Zambie a permis d'accroître l'utilisation des services de santé maternelle, mais n'a produit que des effets limités sur la santé des jeunes enfants. Il souligne également l'importance de combiner une telle politique avec l'amélioration de l'accès physique aux infrastructures sanitaires et de la qualité des soins. Le deuxième chapitre explore les effets intergénérationnels de l'éducation des parents à la fois sur les investissements en santé et l'état de santé de leurs enfants au Zimbabwe. Les résultats mettent en évidence un rôle important de l'éducation du père dans l'amélioration des comportements de prévention adoptés au sein du ménage. Cette relation avait jusqu'ici été largement ignorée par la littérature. Le dernier chapitre s'interroge sur les conséquences sanitaires de la construction de retenues d'eau dans 34 pays d'Afrique Subsaharienne. Les résultats suggèrent l'existence d'un effet contrasté : la malnutrition chronique recule, là où l'exposition au paludisme s'accroît. Ces résultats appellent à la mise en place de politiques complémentaires visant à prévenir cet effet néfaste. Une attention particulière est systématiquement apportée à l'identification d'effets causaux ainsi qu'aux implications en matière de politique publique.

MOTS CLÉS

Santé des enfants, Comportements de santé, Accès aux soins, Infrastructures, Capital humain, Microéconométrie, Évaluation de politique publique

ABSTRACT

Ensuring a sufficiently high level of health for every child is both a social justice goal *per se* and undoubtedly an essential investment given the extensive empirical evidence on the long-run consequences of early life health on later life outcomes. Despite numerous efforts, the situation still gives cause for concern, especially in Sub-Saharan Africa. Identifying the factors that hinder or slow down child health improvement is thus of primary interest. This thesis is part of the continued efforts to provide empirical evidence to inform public policy. It sheds new light on several issues related to the improvement of the health status of young children, such as access to care, household health behaviors, and the health effects of the construction of economic infrastructures such as water reservoirs, in resource-limited settings. The first chapter shows that the introduction of free primary care in Zambia has led to a substantial increase in maternal health care utilization but has only had limited effects on the health of young children. It also highlights the importance of combining such a policy with improvements in physical access to health facilities and quality of care. The second chapter explores the intergenerational effects of parental education on both health investments and young child health in Zimbabwe. It uncovers an important role of paternal education in improving prevention behaviors in the household. This relationship had previously been largely ignored in the literature. The final chapter examines the health consequences of water reservoir construction in 34 Sub-Saharan African countries. The results suggest the existence of conflicting effects: chronic malnutrition decreases, while exposure to malaria risk increases with proximity to a water reservoir. These results call for complementary policies to mitigate this adverse effect. Particular attention is systematically paid to the identification of causal effects and to the public policy implications of these findings.

KEYWORDS

Child health, Health behaviors, Health care access, Infrastructures, Human capital, Microeconometrics, Public Policy Evaluation