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Paludisme et développement économique

Josselin Thuilliez

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THESE

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PALUDISME ET DÉVELOPPEMENT ECONOMIQUE

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« And therefore in the later stages of economics, when we are approaching nearly to the conditions of life, biological analogies are to be preferred to mechanical, other things being equal. [...] 'Progress' or 'evolution', industrial and social, is not mere increase and decrease. It is organic growth, chastened and confined and occasionally reversed by decay and innumerable factors, each of which influences and is influenced by those around it; and every such mutual influence varies with the stages which the respective factors have already reached in their growth »

« Je pense qu'en économie, dans les étapes avancées du raisonnement, les analogies biologiques sont plus appropriées que celles issues de la physique. [...] Le progrès ou l'évolution économique et sociale ne se réduit pas à un accroissement ou à une diminution de grandeurs. C'est la croissance organique, contenue, bornée et parfois contrariée par une multitude de facteurs décadents qui s'influencent mutuellement et dont l'effet varie en fonction de l'état de croissance déjà atteint par chacun d'eux. »

Alfred Marshall – *Distribution and exchange*, Economic Journal, Volume 8, N°29, 1898. Traduction parue dans la Revue française d'économie, Volume 6, N°1, 1991.

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ABBREVIATIONS

API: Annual Parasite Incidence
CDC : Center for disease control and prevention
CNS : Central Nervous System
DALY: Disability-Adjusted Life Year
DHS : Demographic and Health Survey
GIS: Geographic Information Systems
EIR: Entomological Inoculation Rate
GFATM: Global Fund for Aid, Tuberculosis and Malaria
IPT: Intermittent Preventive Treatment
ITN: Insecticide-Treated Net
IV: Instrumental Variables
DEAP: Département D'épidémiologie et des Affections Parasitaires
MAP: Malaria Atlas Project
MARA/ARMA: Mapping Malaria Risk in Africa / Atlas du Risque de la Malaria en Afrique
MLA: Monitoring Learning Achievement
MRTC : Malaria Research and Training Center
PCA: Principal Component Analysis
PNLP: Programme National de lutte contre le Paludisme
NIH: National Institutes of Health
IRB: Institutional Review Board
IRS: Indoor residual spraying
PED: Pays en développement
PASEC: Programme d'Analyse des Systèmes Educatifs de la CONFEMEN
QIHC: Qualitative Indicator of Human Capital
R₀: Basic Reproductive Rate
SACMEQ: Consortium for Monitoring Educational Quality
SPR: Slide Positive Rate
SR: Spleen Rate
SSA: Sub-Saharan Africa
UNPD: United Nation Population Database
WHO: World Health Organisation

PRÉAMBULE

La question initiale qui motive cette thèse est l'identification de facteurs et de mécanismes pouvant conduire des pays pauvres à rester dans un état de pauvreté. Une multitude de facteurs peut, bien entendu, être évoquée et a été énumérée dans la littérature macroéconomique (Azariadis & Stachurski, 2005). Les imperfections de marchés ainsi que les déficiences institutionnelles sont les deux arguments principaux généralement proposés pour expliquer des processus cumulatifs conduisant à des cercles vicieux ou vertueux de développement. Ces mécanismes ont en effet en commun de bloquer l'accumulation de capital physique, humain et l'adoption de nouvelles technologies. D'autres mécanismes moins organisationnels et plus directement liés à l'accumulation de facteurs ont également été étudiés, mais ont fait l'objet d'un consensus moins large, notamment la santé d'une population. De nombreuses combinaisons entre ces différents facteurs et mécanismes peuvent interagir à différents stades du processus de développement. Au plan microéconomique, les causes expliquant pourquoi un individu pauvre reste pauvre, sont nombreuses et détaillées dans une littérature déjà riche. Mais la somme des facteurs microéconomiques provoquant un cercle vicieux pour les individus n'explique pas, à elle seule, la situation d'un pays. Diverses externalités viennent se greffer sur ces relations microéconomiques pour former un système complexe.

À partir de cette question initiale se pose celle de l'approche à adopter pour identifier ces combinaisons et ces externalités. Une voie naturelle proposée par Rodrik & Rodriguez (2000) dans les travaux sur la croissance consiste à explorer en détail les canaux de transmission possibles. Une analyse approfondie d'une hypothèse préalablement identifiée comme plausible peut venir confirmer ou écarter certains liens voire en identifier de nouveaux. Ce retour sur des aspects déjà étudiés doit cependant s'accompagner d'un regard neuf, non seulement pour identifier la relation à approfondir mais aussi pour l'analyser sous un angle différent. L'approfondissement de différents liens et l'exploration de pistes négligées permettront sans doute à terme d'obtenir une vision simplifiée et modélisée relativement proche de la réalité, en quelque sorte, une cartographie du développement économique. La confrontation de différentes études sur le même lien permettra sans doute également d'identifier les facteurs contextuels dans le processus de développement des facteurs structurels en relation avec le lieu et la période historique. Par conséquent, les modèles utilisés dans cette thèse cherchent à éclairer une infime partie du processus économique d'ensemble.

Pour répondre à ces exigences, nous avons identifié deux facteurs qui nous semblent au centre du développement d'un pays: la santé et l'éducation. Plus particulièrement, car les problématiques de santé dans les pays en développement sont nombreuses, nous nous sommes intéressés à un phénomène de grande ampleur: l'endémie palustre. De même, nous nous sommes focalisés sur des aspects qualitatifs de l'éducation plus que sur des aspects quantitatifs. Certaines évaluations macroéconomiques en forme réduite prédisent une influence très grande du paludisme sur la croissance et le développement. Selon J.D. Sachs (2001), la croissance du PIB/tête des pays avec une forte prévalence de paludisme serait plus faible de 1,3% par an. Une diminution de 10% du paludisme serait associée à une croissance supérieure de 0,3%. La relation entre paludisme et développement n'est donc pas une question nouvelle, mais n'a pas non plus fait l'objet de beaucoup d'attention en économie. De plus, la littérature sur la croissance a montré que ce type de résultats n'était souvent pas très robuste, en raison notamment de problèmes d'endogénéité, et nous invite à la prudence.

Un certain nombre de chiffres indique pourtant que le paludisme est un phénomène qui présente des coûts élevés pour les pays en développement. Le paludisme se localise essentiellement dans la ceinture de pauvreté du globe. Près de 41% de la population mondiale (environ 3,3 milliards de personnes) vit dans des zones où le paludisme est transmis (Center for disease control and prevention - CDC). Le nombre de cas cliniques est estimé entre 250 et 500 millions par an (World Malaria Report 2005). Le nombre de morts est compris entre 700000 et 2.7 millions par an dont 75% d'enfants africains (CDC). C'est justement parce que le paludisme touche particulièrement les enfants qu'il nous a semblé essentiel de nous focaliser sur le lien entre paludisme et éducation. À l'école, le paludisme est un obstacle supplémentaire. De nombreux témoignages l'évoquent. Cependant, dès qu'il s'agit de quantifier l'ampleur du phénomène, il existe un manque dans la littérature. Le paludisme fait partie de la vie quotidienne et n'affecte pas uniquement les enfants scolarisés. C'est sans doute ce qui explique pourquoi cet effet est noyé dans la masse des autres effets économiques du paludisme et qu'il n'a pas été étudié précisément.

Nous pensons donc que le paludisme peut être un facteur non négligeable de blocage du développement des pays les plus touchés. En d'autres termes, le paludisme n'est certes pas LA cause de la pauvreté et de la faible croissance des PED, mais il peut venir accentuer certains phénomènes sous certaines conditions. Nous avons choisi de nous concentrer sur la relation entre paludisme et formation de

capital humain et d'analyser cet aspect en profondeur tant sur le plan macroéconomique que microéconomique. Peu d'études ont, jusqu'à présent, quantifié cet impact de façon rigoureuse bien que de nombreuses études médicales fassent un constat alarmant de l'impact du paludisme sur les capacités de développement créatif et intellectuel chez l'enfant. L'étude de ce lien sert également de prétexte et d'illustration à l'analyse du caractère endogène de la santé dans le processus de développement.

Notre étude, dans ses parties microéconomiques, se focalise sur le Mali pour deux raisons essentielles: le rôle moteur joué par le Mali dans la recherche sur le paludisme depuis le début des années 90 et l'excellence de ses structures de recherche à ce niveau. Harold Warmus, prix Nobel de médecine 1989, n'a-t-il pas dit du MRTC : « *Consider one example that I have visited myself, in Bamako, the capital of Mali, with an average income of \$300 per person per year, the NIH and the US Agency for International Development have established a Malaria Research and Training Centre (MRTC) that is staffed mainly by Malian scientists and technicians, has good internet connectivity, serves as a regional institution for training, receives its own grant money from the NIH, and hosts scientific visitors from NIH and Tulane University for collaborative projects. The centre is well known and well respected by the political leadership of Mali, is a source of public pride, and offers a positive view of health research to students at the adjacent school of medicine. Who would not find this to be an exciting environment in which to work, to learn, and to train—with the added benefit of spreading the culture of science?* » (Warmus, 2002).

La lutte contre le paludisme ne peut attendre un décollage économique. L'intégration d'une approche économique à l'approche épidémiologique et médicale peut permettre une meilleure compréhension des interactions entre progrès sanitaires et développement économique.

RÉSUMÉ

Le paludisme est une maladie parasitaire transmise par des moustiques de type *Anophèles* qui résulte du développement de parasites protozoaires. L'incidence et la sévérité du paludisme dépendent de nombreux facteurs entomologiques, environnementaux et humains. Les combinaisons possibles entre ces facteurs sont nombreuses. Le faciès clinique du paludisme varie, entre autres facteurs comme le comportement personnel et les caractéristiques génétiques de l'individu, selon le statut immunitaire de l'individu. Les populations exposées à des infections fréquentes acquièrent une immunité partielle et labile contre les effets du paludisme. Le niveau d'immunité affecte non seulement la mortalité, la sévérité du paludisme mais aussi les accès palustres dits « simples ». Il est donc paradoxalement plus difficile d'évaluer le véritable fardeau de la pathologie en zone fortement endémique où les individus sont plus exposés à des piqûres infectantes de moustiques puisque la parasitémie n'est pas directement et proportionnellement associée à des symptômes cliniques apparents. Même en zone non-endémique, il peut être difficile de déceler le paludisme puisque les symptômes associés sont transversaux et la maladie n'est pas immédiatement suspectée.

Chez l'enfant, l'immunité acquise ne joue un rôle protecteur efficace qu'à partir de l'âge de 5-6 ans en zone fortement endémique. C'est principalement la raison pour laquelle le paludisme est une menace majeure pour la survie des enfants. En 2000-2003, le paludisme était responsable de 8% des 10,6 millions de décès annuels chez les enfants de moins de 5 ans (OMS-CHERG). Il représentait la troisième cause de mortalité des enfants de moins de 5 ans. 94% du total des décès attribuables à la maladie ont lieu en Afrique (Bryce et al., 2005). En Afrique, la maladie représente 20 à 25% de toutes les causes de mortalité des moins de 5 ans, 25% des hospitalisations pédiatriques et le taux de létalité est d'environ 15%. Les enfants d'âge scolaire sont également très touchés par le paludisme (Clarke et al. 2004).

Le point de départ de cette thèse est de fournir un cadre théorique à l'analyse de la relation entre paludisme et développement économique. Les pays en développement sont soumis à des risques particuliers dans des contextes complexes. En supposant que des traitements adaptés ou des vaccins soient découverts, la résurgence de la Poliomyélite en Afrique de l'Ouest a montré que les progrès biomédicaux ne suffisent pas à éradiquer une maladie et que, dans les pays en développement, la compréhension des comportements à risque reste un enjeu majeur. Dans le domaine de la santé, les croyances, plus que les connaissances, semblent jouer un rôle important dans l'adaptation des comportements. Différents modèles ont été utilisés

pour analyser la persistance des comportements à risque : les modèles empiriques d'analyse des déterminants des comportements à risque, les modèles de croyance pour la santé, les modèles inspirés des théories de l'apprentissage social, les modèles de choix rationnels. Les modèles de choix rationnels se sont imposés en épidémiologie économique pendant les années 1990 pour expliquer la persistance des comportements à risque relatifs à des maladies à transmission interhumaine (principalement le VIH). Cependant, les comportements d'arbitrage face à des maladies à transmission vectorielle restent peu connus, certainement faute de données. Nous cherchons donc à combler ce manque dans la littérature en focalisant notre attention sur le paludisme.

Les moustiquaires imprégnées d'insecticide sont considérées comme l'un des moyens essentiels de contrôle du paludisme. Les études menées par l'Organisation Mondiale de la Santé en Zambie, au Ghana, au Rwanda, en Ethiopie et en Tanzanie, confirment que les moustiquaires imprégnées d'insecticide peuvent réduire la mortalité infantile d'environ un tiers en zone d'endémie. Cette mesure est comparable à la vaccination contre la rougeole en termes de coût-efficacité à prévenir les décès et la morbidité. Les moustiquaires imprégnées permettent non seulement de réduire le nombre de cas de paludisme chez les enfants de moins de 5 ans, mais aussi de diminuer théoriquement l'intensité de la transmission (via l'effet des insecticides sur les vecteurs). Pourtant les taux d'utilisation de ces moustiquaires restent relativement bas dans de nombreux pays sans explication convaincante. De même une plus grande gratuité n'est pas obligatoirement suivie d'un meilleur recours à la prévention et d'une meilleure utilisation des moustiquaires. En effet, Rhee et al. (2005) ont testé, dans le cadre d'un essai clinique, l'hypothèse selon laquelle le manque d'éducation et de connaissances sur le paludisme constitue un frein à l'utilisation des moustiquaires. Ils montrent qu'il est possible d'informer les individus sur le paludisme et de mettre en place des services d'imprégnation des moustiquaires avec des ressources limitées. Si l'accessibilité des moustiquaires et de leur imprégnation est nécessaire, elle n'est pas suffisante. L'utilisation effective des moustiquaires (définie comme l'état imprégné de la moustiquaire à la fin de l'étude) était significativement supérieure chez ceux qui avaient reçu une éducation (48% contre 33% chez ceux qui n'avaient pas reçu d'information). Néanmoins ces chiffres restent relativement bas dans tous les cas (<50%). Le manque de connaissances ne serait pas non plus la seule contrainte. L'un des grands enjeux en santé publique dans les pays d'Afrique depuis plusieurs décennies est de promouvoir à grande échelle l'utilisation de ces moustiquaires.

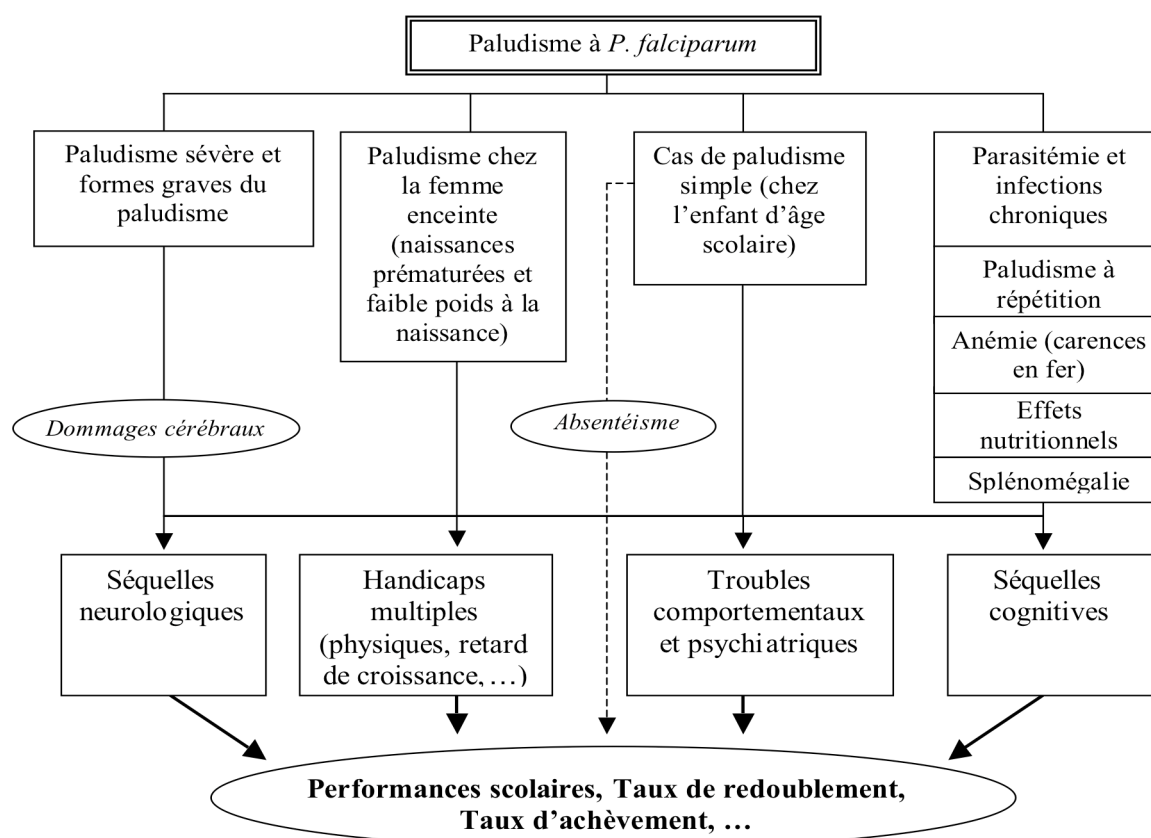
Dans le premier chapitre de cette thèse, nous construisons un modèle théorique montrant que, compte tenu des caractéristiques de la maladie (transmission

vectorielle et intensité de la transmission principalement), il peut être rationnel pour un individu de ne pas utiliser de moustiquaires imprégnées d'insecticide en dessous d'un certain seuil de prévalence de la maladie. Cette étude confirme les comportements dit « prévalence-élastiques » rencontrés en épidémiologie économique : l'utilisation de mesures préventives augmente lorsque le niveau de prévalence de la maladie augmente et décroît lorsque celui-ci diminue. Nous montrons également que ces comportements, associés à certaines spécifications des paramètres du modèle conduisent à de possibles équilibres multiples et donc potentiellement à des « pièges de paludisme ». Ces « pièges » sont définis comme des processus cumulatifs conduisant à une hausse de la transmission de la maladie quand la prévalence de celle-ci est au-dessus d'un certain seuil et à un déclin de celle-ci en dessous de ce seuil. Ce modèle débouche sur une implication forte par rapport au caractère exogène du paludisme. Pour les communautés « piégées » dans de telles trappes à paludisme, nous pouvons effectivement considérer la maladie comme exogène : c'est le facteur qui s'impose comme déterminant possible d'autres caractéristiques du village comme le niveau de revenus. Dans une telle situation, les individus peuvent également devenir fatalistes par rapport au paludisme ce qui affectera le coût d'opportunité des moyens de prévention à long terme. Si à son tour, le paludisme affecte le niveau d'éducation de la population et que ce niveau d'éducation affecte le coût d'opportunité des mesures préventives, le paludisme pourrait créer à long-terme des conditions propices à sa stabilité et des entraves à certains types de politiques publiques. Il est donc nécessaire d'étudier les effets du paludisme sur l'éducation. D'autres implications en termes de politiques publiques sont discutées dans le modèle, notamment la nécessité d'un choc exogène pour sortir de la trappe ainsi que les effets de chocs de différentes natures. Une autre explication hypothétique de ces pièges de santé pourrait être la relation bidirectionnelle entre santé et éducation. Les effets de l'éducation des mères sur les comportements préventifs et d'accès aux soins ont déjà été étudiés dans la littérature. Par contre, l'effet inverse du paludisme sur l'éducation est moins connu. C'est pourquoi les Chapitres 2, 3 et 4 de cette thèse se focalisent spécifiquement sur cette relation.

Quelques études ont mis l'accent sur les conséquences du paludisme sur le comportement de l'enfant, son développement cognitif, ses performances d'apprentissage et l'assiduité en classe. Cependant, la relation commence à peine à être explorée. Nous pouvons résumer les liens directs entre paludisme et performances scolaires à l'aide du schéma simplifié donné en Figure 1. Ces études tendent à décrire une relation complexe entre le paludisme, la formation de capital humain et le grand nombre de facteurs à risque, qui eux-mêmes peuvent avoir un effet sur les performances scolaires. Holding & Kitsao-Wekulo (2004) décrivent les

priorités pour la recherche future dans ce domaine. Le grand nombre de canaux potentiels qui conduisent à des troubles du développement chez l'enfant doit être étudié en utilisant différentes méthodes d'investigation. Comprendre les sources de la variabilité de ces troubles (différences de statut socioéconomique, présence d'autres infections, âge, environnement) pourrait également aider à isoler l'impact spécifique du paludisme, à identifier les enfants les plus à risque et à définir des modalités d'intervention. La littérature en microéconomie de la santé reconnaît également que les problèmes de santé physique et mentale peuvent empêcher l'accumulation de capital humain chez l'enfant (Currie & Stabile, 2006) mais les études en économie du développement dans ce domaine se sont principalement focalisées sur des indicateurs nutritionnels, la présence d'une infirmité visuelle ou de parasitoses intestinales. Au niveau des indicateurs de réussite scolaire utilisés, ces études se réfèrent soit à des tests de connaissance, soit à des indicateurs plus agrégés tels que le retard scolaire ou les taux de redoublement (Dumont, 2000).

FIGURE 1: Impacts du Paludisme sur les Performances scolaires (*source: auteur*)



Le point de départ de notre analyse empirique est une étude macroéconomique en coupe transversale sur 125 pays présentée dans le Chapitre 2. Ce travail montre que le lien entre les performances des enfants dans le primaire (mesurées par les taux de

redoublement et d'achèvement dans le primaire) et l'indice de paludisme à *P.falciparum* utilisé est fort et robuste. Nos régressions en doubles moindres carrés ordinaires montrent que les pays intensivement touchés par le paludisme ont des taux de redoublement dans le primaire au moins entre 11 et 18% supérieurs et des taux d'achèvement dans le primaire entre 48 et 54% inférieurs à ceux qui ne sont pas touchés par le paludisme, toutes choses égales par ailleurs. Cette analyse est ensuite confortée à partir de données de panel et d'autres tests de robustesse ont été effectués en utilisant des données de sources différentes. Nous trouvons également un effet négatif et significatif du paludisme sur les taux d'alphabétisation, toutes choses égales par ailleurs.

Ce type d'approche présente des limites, principalement liées à la précision des données de paludisme disponibles, mais également plusieurs intérêts. Premièrement, les études macroéconomiques sur le paludisme sont rares et se sont focalisées sur les effets de la maladie sur la croissance. Cette étude vient donc combler une partie du fossé qui sépare les estimations microéconomiques des estimations macroéconomiques en identifiant l'un des canaux d'impact potentiels. Deuxièmement, compte tenu du fait que la maladie touche massivement les enfants, il serait étonnant de ne pas trouver un impact sur l'éducation au niveau macroéconomique si une telle relation de causalité entre paludisme et éducation existait. Une fois ce risque identifié au niveau macroéconomique, il devient cohérent d'investir dans des données microéconomiques pour tester plus précisément cette hypothèse. Troisièmement, il est plus facile de communiquer ces résultats synthétiques à des organismes internationaux ou des bailleurs de fonds (UNESCO-EFA, 2009).

Afin de dépasser les limites intrinsèques aux données macroéconomiques et de donner des fondements microéconomiques à notre argumentation, cette approche est ensuite complétée et approfondie par des travaux microéconomiques réalisés à différentes échelles: au niveau communautaire dans le Chapitre 3 et au niveau individuel dans le Chapitre 4.

Nous utilisons premièrement les données des *Demographic and Health Surveys* 2001 et 2006 disponibles sur le Mali (MDHS) pour étudier la relation entre la prévalence de la fièvre, la prévalence du paludisme et les performances scolaires au niveau communautaire. Comme les enquêtes MDHS 2001 et 2006 présentent l'avantage d'avoir été réalisées pendant des périodes différentes de l'année (saison sèche et saison des pluies), il est également possible de fournir une discussion critique sur l'indicateur de prévalence de la fièvre, variable utilisée comme variable approximative du paludisme dans un grand nombre d'études sur le contrôle de l'endémie palustre. Pour cela nous utilisons des données GIS (Geographic

Information System) et le système de positionnement géographique des enquêtes MDHS pour extraire des données de paludisme ajustées sur l'âge provenant de cartes fiables et très détaillées de la prévalence du paludisme et de l'intensité de la transmission au Mali. Ces cartes sont issues d'une étude épidémiologique (Gemperli et al. 2006) et les modèles utilisés pour leur réalisation ont été validés sur des données de surveillance épidémiologique de plusieurs sites du *Malaria Research and Training Center* au Mali. Nous montrons ainsi que la fièvre n'est pas une variable approximative fiable pour le paludisme et nous recommandons l'utilisation de données géographiques de la prévalence du paludisme pour les recherches futures. Notre analyse en coupe transversale pour les deux années (incluant entre 310 et 380 villages différents) suggère qu'une prévalence plus forte du paludisme dans une communauté est liée à de forts taux de redoublement dans le primaire. Les villages les plus affectés ont des taux de redoublement 14% supérieurs à des villages non touchés. Ces résultats sont en accord avec nos précédents résultats. Cependant des études confirmatoires en données de panel sont nécessaires. Dans cette étude, nous contrôlons pour le biais potentiel de sélection en restreignant l'analyse aux enfants scolarisés dans le primaire. Nous incorporons également dans l'analyse l'endogénéité probable de la variable de paludisme.

Nous nous intéressons ensuite à cette relation au niveau individuel en utilisant des données plus précises sur le paludisme et sur les variables d'éducation. Cette étude a été effectuée en collaboration avec le *Malaria Research and Training Center* (Bamako, Mali) du Professeur O.K. Doumbo, centre de recherche de référence sur le paludisme. Elle a été financée par le National Institutes of Health et la Fondation Mérieux. J'ai été l'investigateur principal de cette étude pendant toute l'année scolaire 2007-2008. À ce titre, j'ai été en charge, en collaboration avec le Dr M.S Sissoko et O.B Touré, de la réalisation de l'ensemble des étapes de cette recherche: la recherche de financements, la définition de l'approche conceptuelle, l'élaboration des procédures méthodologiques, la conception des questionnaires et des tests, la validation éthique de l'étude, la supervision sur le terrain de la collecte des données et de l'achat du matériel, le contrôle des questionnaires et des tests scolaires, le suivi des opérations de saisie et le nettoyage des fichiers, l'analyse des données.

Cette étude a pour principal atout, par rapport à la littérature existante, d'utiliser des indicateurs de paludisme appropriés et correctement mesurés ainsi qu'un protocole de recherche et des modèles économétriques adaptés. Nous avons délibérément privilégié une approche non expérimentale à une approche expérimentale. Par ailleurs nous précisons les canaux d'impact du paludisme sur les performances scolaires. L'hypothèse principale testée ici est que la parasitémie palustre provoque en soi de mauvaises performances scolaires.

Tous les élèves de l'école de Donéguébougou dont les parents ont donné leur consentement éclairé pour la participation à l'étude ont été inclus (227 élèves) et suivis sur une période de 8 mois de façon active (huit passages transversaux mensuels) et passive (suivi continu des élèves au centre de soin et à l'école). Nous avons utilisé plusieurs mesures de l'incidence du paludisme chez l'enfant: le passé convulsif de l'enfant, le paludisme clinique via un examen médical, le nombre d'épisodes palustres par mois, la parasitémie via des tests sanguins (*P. falciparum* trophozoites/ μ L), l'anémie palustre, l'indice splénomégalique proposé par Hackett (1944). Nous avons également relevé d'autres indicateurs anthropométriques et d'autres variables de santé (épisodes morbides non palustres, anémie, infirmités, etc.)

Les tests cognitifs utilisés ont été construits à partir de modèles déjà utilisés dans d'autres études en les adaptant au contexte du village (langue, niveaux, avis des enseignants). Quatre catégories de tests ont été définies: connaissances et compréhension (adaptation du test de vocabulaire de Mill Hill), écriture (reproduction de symboles, mots ou paragraphes), mémoire visuelle (recherche d'images ciblées parmi d'autres, Jukes et al. 2006), raisonnement inductif (exercices mathématiques sur des combinaisons de nombres). Dix-huit tests adaptés au niveau des élèves ont été conçus au total. Tous les tests ont été administrés en une cession de 45 minutes à l'école à chaque passage transversal mensuel. Une analyse en composante principale a ensuite été réalisée pour créer un indicateur synthétique. La variable obtenue est fortement corrélée en moyenne aux résultats scolaires de l'enfant ($r > 0,8$) et la cohérence interne des différents tests est forte (α de Cronbach $> 0,7$). À ces tests s'ajoutent les notes des élèves au cours de l'année, l'absentéisme scolaire et une variable de retard scolaire pour tester la robustesse de nos résultats.

Le niveau socio-économique des familles a été évalué via un questionnaire adressé aux chefs de famille dans lequel d'autres variables d'intérêt sur l'enfant ont également été relevées.

Les résultats de l'étude montrent que le paludisme est la première cause d'absentéisme scolaire. Les modèles de régression à effets fixes intra-individuels montrent que le paludisme asymptomatique et le paludisme clinique affectent significativement et négativement l'indicateur synthétique des performances cognitives des enfants et les résultats scolaires académiques des enfants. Les modèles incluent d'autres variables de santé (anémie palustre, splénomégalie, indicateurs anthropométriques) et d'autres indicateurs socioéconomiques (entre autres: le travail des enfants, le niveau d'éducation des parents, la richesse du ménage).

Par exemple, les enfants qui ont eu une crise de paludisme clinique ont des résultats inférieurs de 0,40 points par rapport aux enfants qui n'ont pas eu de crise

(l'indice synthétique de performances cognitives varie entre -5 et +4). L'un des résultats les plus alarmants concerne les effets de la parasitémie asymptomatique (infection sans symptôme clinique apparent) sur les performances des élèves. Bien qu'inférieurs aux effets du paludisme clinique, nos résultats montrent que le paludisme asymptomatique affecte significativement les performances des enfants d'environ -0,10 points. Nous montrons que l'impact du paludisme asymptomatique sur les performances scolaires est expliqué par les effets de la parasitémie sur les performances scolaires, toutes choses égales par ailleurs et notamment en contrôlant pour l'anémie et la splénomégalie associées au paludisme chronique et au paludisme à répétition. Une parasitémie supérieure à 1200 parasites / μL (avec ou sans symptômes cliniques) est associée à une baisse significative de cet indicateur de 0.35 points en moyenne par rapport à des enfants dont la parasitémie est inférieure à 1200 parasites / μL le jour des tests. Les résultats laissent également penser à des effets cumulatifs du paludisme et à une persistance dans le temps (au moins un mois) de ces effets. Par contre, nos résultats sur les effets de la splénomégalie et de l'anémie palustre sur les performances des enfants restent ambigus et appellent des études confirmatoires.

La robustesse de ces résultats a été testée via des régressions sur d'autres mesures de performances cognitives, sur les résultats scolaires académiques des enfants et sur le retard scolaire. La moyenne géométrique de la parasitémie de l'enfant affecte significativement et négativement les notes académiques des enfants. Les enfants qui ont une parasitémie annuelle plus élevée sont en moyenne en retard dans leur scolarité par rapport aux autres enfants du même niveau. Comme les enfants ont été traités rapidement et gratuitement au cours de l'étude, on peut s'attendre à des effets plus importants encore du paludisme sur les capacités cognitives de l'enfant et les résultats scolaires en l'absence de traitement.

Comparativement à d'autres problèmes de santé (malnutrition et helminthiases principalement), les effets du paludisme à *P. falciparum* sur l'éducation semblent plus préoccupants compte tenu de la fréquence des infections. Ces aspects sont discutés dans le Chapitre 4. Enfin, les enfants qui dorment sous moustiquaires imprégnées ont, en moyenne, des résultats significativement plus élevés. D'autres questions économiques sont également abordées en discussion (effets du niveau de richesse des parents sur la variation des effets du paludisme sur les performances scolaires et cognitives des enfants).

Si la méthode des variables instrumentales n'a pas été utilisée dans cette étude - l'un des seuls instruments véritablement pertinents dans le cadre du protocole défini aurait été de relever une variable entomologique par enfant et par passage, variable impossible à mesurer - il est néanmoins possible d'envisager un lien de causalité. En

effet, les arguments suivants sont réunis: nos résultats concordent avec d'autres études indépendantes, il y a plausibilité biologique de l'hypothèse avancée, la séquence temporelle entre la cause et l'effet est appropriée, il y a une relation claire entre l'intensité de l'infection et l'effet, les résultats des modèles à effets fixes intra-individuels diffèrent peu des estimations en moindres carrés ordinaires.

Cette thèse se situe donc à la frontière de plusieurs disciplines de l'économie: économie du développement, économie de la santé, économie de l'éducation. Elle tente également d'intégrer plusieurs autres disciplines afin d'être le plus précis possible sur les phénomènes étudiés et les données utilisées. Nous avons donc travaillé avec des spécialistes d'horizons variés: épidémiologistes, biostatisticiens, médecins, cartographes et psychologues.

L'une des conclusions majeures de cette thèse est que le paludisme a des effets négatifs sur l'accumulation de capital humain chez l'enfant. Cette conclusion a été confirmée à différents niveaux d'étude et semble robuste à l'utilisation de données et de modèles différents. Nous précisons également les canaux d'impact. L'une de nos découvertes principales est que le paludisme asymptomatique affecte les résultats scolaires de l'enfant en zone d'endémie via les effets du parasite à *P. falciparum*, représentant un risque important pour son développement futur.

Néanmoins, cette thèse appelle des études confirmatoires sur la relation étudiée. Nous suggérons notamment de tester cette relation pour différents niveaux d'endémicité sur un nombre d'années étendu. L'effet de politiques de prévention sur l'amélioration des résultats scolaires devrait également faire l'objet d'une attention particulière. Nos résultats pourraient être approfondis à partir des quelques idées détaillées ci-dessous:

- En analysant les effets de la prévention du paludisme sur les performances scolaires des enfants via une étude expérimentale.
- En élargissant l'analyse à un plus grand nombre d'écoles et à des zones d'endémicité différentes en conservant une approche non expérimentale. Il est possible d'envisager pour cela d'envisager un partenariat de recherche avec la PASEC qui réalise des tests scolaires au Mali (Programme d'Analyse des Systèmes Educatifs de la Confemen).
- En utilisant des marqueurs génétiques comme variables instrumentales du paludisme dans des études incluant moins d'enfants mais sur une durée plus longue. De nombreuses études sont en cours sur les marqueurs de différence ethnique dans la susceptibilité du paludisme à *P. falciparum*. D'autres études étudient le rôle des expressions différentielles de certains gènes (chez l'hôte ou le parasite) et de la distribution temporelle de l'expression génétique différentielle dans la réponse immunitaire de l'hôte. Exploiter la variation

naturelle de ces marqueurs au sein d'une population peut permettre de traiter efficacement l'endogénéité des variables de paludisme à condition que ces marqueurs soient spécifiquement et exclusivement liés aux différentes formes de paludisme mais non corrélés aux variables d'éducation. Cette voie prometteuse (tant d'un point de vue méthodologique que des résultats qui en découlent) reste à approfondir en économie de la santé pour établir des liens de causalité (Ding et al. 2009).

GENERAL INTRODUCTION

After Nobel Laureate Arrow's contribution on malaria (2004) and the increased resources allocated to malaria control since the Abuja declaration (2000), it seems natural to wonder how economists can still contribute to the debate on the economic effects of malaria and help malaria control policies advance. This introduction starts with the presentation of malaria control strategies (Section 1). Section 2 provides the general framework used to explore the macroeconomic and microeconomic effects of malaria and its limitations and next focuses on the theoretical arguments explaining the effects of health (and particularly malaria) on economic development following recent development of the human capital theory. We present some stylized facts on the relationship between malaria and education in Section 3. Section 4 describes the empirical difficulties dealing with the estimation of education production functions. Section 5 provides the structure of our thesis.

I. Malaria control strategies: an overview

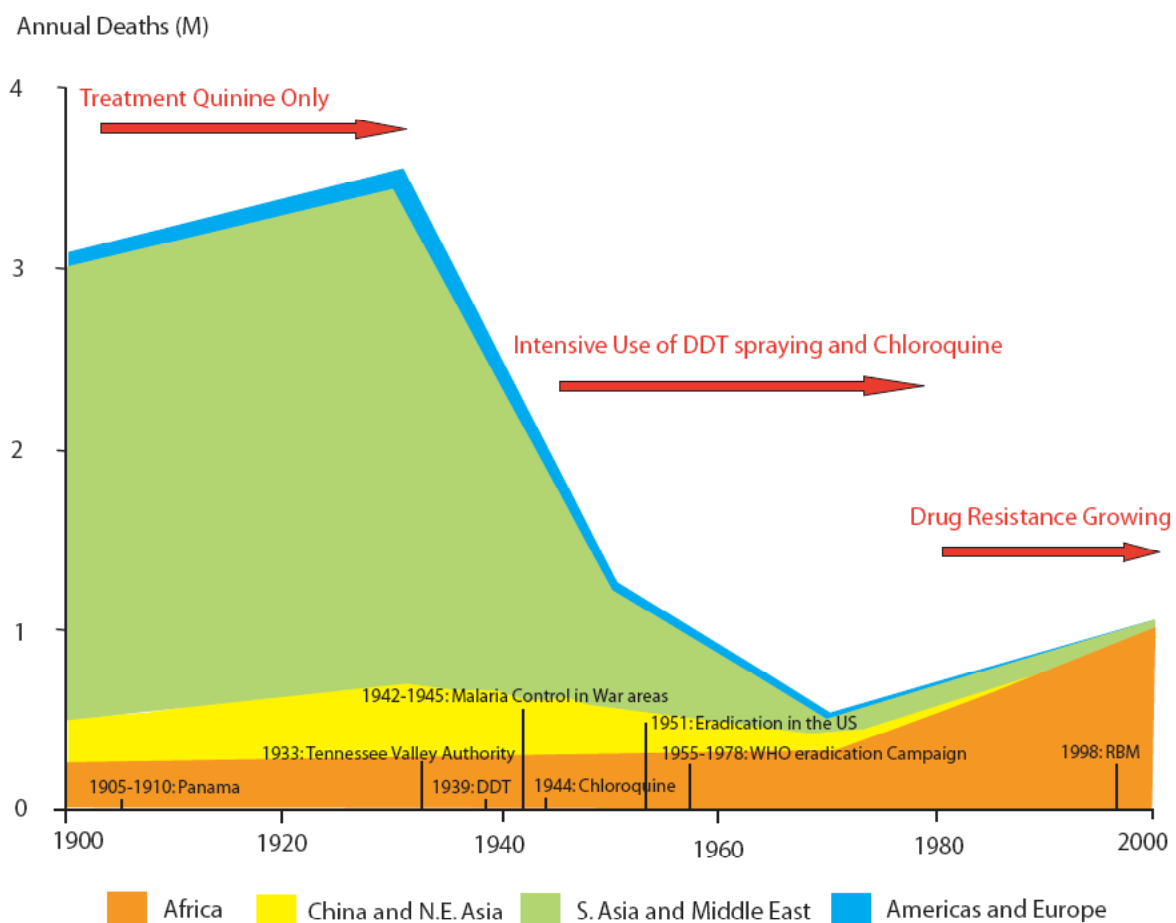
Malaria is prevalent throughout most of the tropical world, producing a situation in which 40% of the world's population is at risk for acquiring this disease. There were an estimated 247 million malaria cases among 3.3 billion people at risk in 2006, causing nearly a million deaths, mostly of children under 5 years. 109 countries were endemic for malaria in 2008, 45 within the WHO African region (World Malaria Report, 2008). Malaria is considered one of the most common infectious diseases and the most important of the parasitic diseases. Infections in humans are caused by four different species of the genus *Plasmodium*. *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium vivax*, and *Plasmodium ovale* are associated with different clinical presentations, progression, prevalence, and antimalarial resistance patterns. *P. falciparum* infection results in the highest morbidity and mortality, accounting for almost all of the over 1 million deaths caused by malaria annually, most of them in young children in Africa (see Appendix 1 for more details).

The WHO, the GFATM, the World Bank Booster program, the malaria vaccine initiative, the Bill and Melinda Gates Foundation and the President's malaria initiative have recently demonstrated a deep commitment to combating malaria. This commitment is providing a rare hope in the fight against malaria, similar to that which occurred during earlier eradication programs in the 1950s.

A. Policies and strategies for malaria control: past and present

Figure 2 provides a very schematic history of malaria control since 1900. There is evidence that the WHO global initiative to fight malaria launched in the 1950s has been much more effective in Asia than in Africa. The emergence and spread of chloroquine-resistant *Plasmodium falciparum* malaria parasites has been a disaster for world health.

FIGURE 2: Malaria Control since 1900 (schematic historical perspective – source: author from Carter & Mendis, 2002)



In 1998, WHO launched a new campaign to roll back malaria with the stated goal to halve malaria deaths worldwide by 2010 (Nabarro & Tayler, 1998). The Global Fund for Aid, Tuberculosis and Malaria (GFATM) was created in 2002 and has become the main source of finance for programs to fight AIDS, tuberculosis and malaria, with approved funding of US\$ 15.6 billion (malaria programs represents 25% of this amount) for more than 572 programs in 140 countries. It provides officially three quarters of all international financing for malaria.

The primary objective of malaria control is to reduce deaths and illness from the disease. Elimination of infection is far more difficult. The policies and strategies for malaria control are based on long-lasting insecticidal nets (LLINs) and artemisinin-based combination therapy (ACT), plus a revival of support for indoor residual spraying of insecticide (IRS). Firstly, insecticide-treated nets are comparable to measles vaccination in their cost-effectiveness in preventing malaria deaths and morbidity. Secondly, the demise of chloroquine and sulfadoxine-pyrimethamine leave artemisinin-class combination therapies (ACT) as the best treatment option. Patients given two or more effective drugs are less likely to encounter drug resistance and fail treatment. The malaria treatment guidelines (WHO, 2006) recommend that antimalarial treatment policy should be changed at treatment failure rates considerably lower than those recommended previously. *“This major change reflects the availability of highly effective drugs, and the recognition both of the consequences of drug resistance, in terms of morbidity and mortality, and the importance of high cure rates in malaria control”*. Thirdly, IRS is the application of insecticides to the inner surfaces of dwellings, where endophilic anopheline mosquitoes often rest after taking a blood meal. According to WHO, several years of consecutive rounds of IRS are effective in reducing malaria parasite prevalence and incidence in areas of high transmission and can be used in combination with ITNs.

According to the 2008 World Malaria Report (WHO, 2008), among 41 African countries that provided case and death reports over the period 1997–2006, the most persuasive evidence for impact of these “malaria control strategies” comes from four countries, or parts of countries, with relatively small populations, good surveillance, and high intervention coverage. They are Eritrea, Rwanda, Sao Tome and Principe, and Zanzibar (United Republic of Tanzania). All four areas reduced the malaria burden by 50% or more between 2000 and 2006–2007, in line with the World Health Assembly (WHA) targets. However, it is difficult to assert a direct causal relationship between malaria control policies and the decrease in the malaria burden in these countries because no rigorous evaluation has been undertaken. In other African countries where a high proportion of people have access to antimalarial drugs or insecticidal nets, such as Ethiopia, Gambia, Kenya, Mali, Niger and Togo, routine surveillance data do not yet show, unequivocally, the expected reductions in morbidity and mortality. Either the data are incomplete, or the effects of interventions are small. The reportedly high coverage of indoor residual spraying (IRS) in Namibia, South Africa and Swaziland is consistent with the observed declines in case numbers in these countries, and evidently builds on earlier successes achieved with IRS. Surveillance reports for many countries outside Africa indicate that malaria declined during the decade 1997–2006. Malaria cases were falling in at

least 25 endemic countries in five WHO regions. In 22 of these countries, the number of reported cases fell by 50% or more between 2000 and 2006–2007, in line with WHA targets.

In general, however, the links between interventions and trends remain ambiguous, and more careful investigations of the effects of control are needed in most countries. Reported malaria cases and deaths are often crude data. Surveillance systems and research studies supported by WHO to monitor antimalarial drug efficacy in countries are also providing new evidence that parasites resistant to artemisinin have emerged along the border between Cambodia and Thailand. When treatment failure becomes frequent, malaria deaths rise greatly, especially in children (Attaran et al., 2004). The main actions recommended by WHO to delay drug resistance are:

- Eliminate artemisinin-tolerant parasites by detecting all malaria cases in target areas and ensuring effective treatment.
- Reduce exposure of the parasites to artemisinin to limit emergence of resistance.
- Prevent transmission of artemisinin-tolerant malaria parasites through mosquito control and personal protection.
- Limit the spread of artemisinin-tolerant malaria parasites by mobile populations.
- Support the containment and elimination of artemisinin-tolerant parasites through comprehensive behaviour change, communication, community mobilization and advocacy.
- Undertake basic and operational research to fill knowledge gaps and ensure that strategies applied are evidence-based.
- Provide effective management, surveillance and coordination to enable a rapid and high-quality implementation of the strategy.

B. Preventing and treating malaria

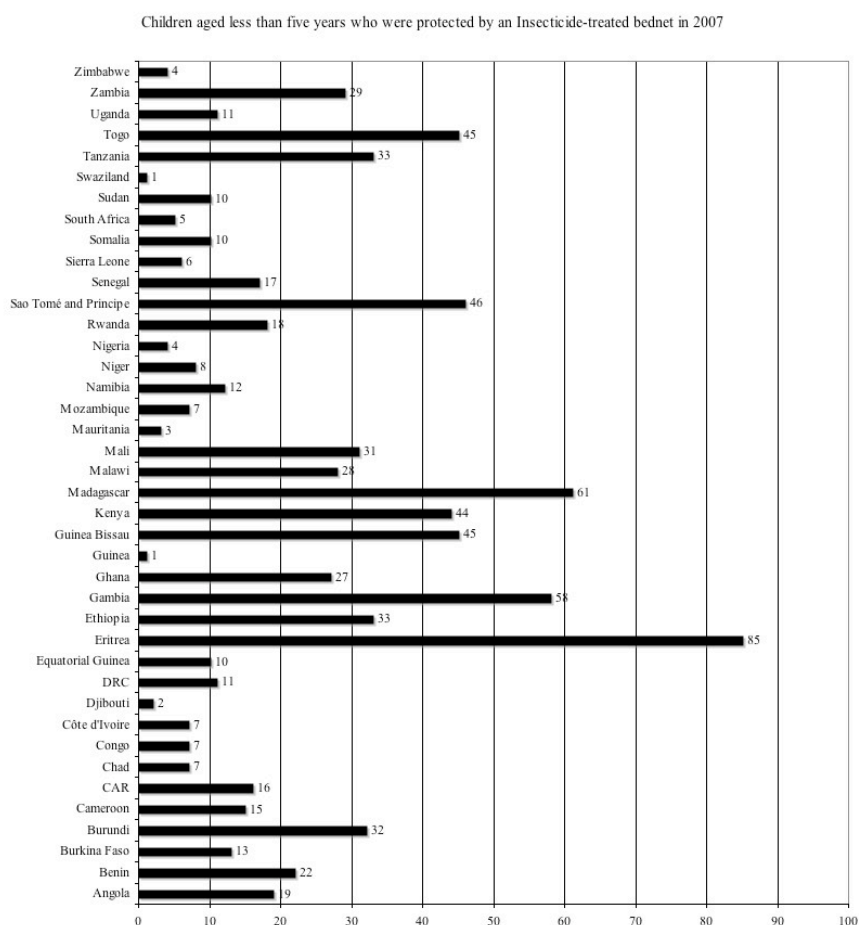
1. Preventing: determinants of household demand for ITN and household's use of ITN

As we said, ITN and IRS are the main preventive measures recommended by WHO (we do not consider recommendations for travellers). Besides, the systematic use of intermittent preventive treatment (IPT) in pregnancy is restricted to the African Region; 33 of the 45 African countries had adopted IPT as national policy by the end of 2006 (WHO World Malaria Report, 2008). Intermittent preventive treatment of malaria refers to the administration of doses of an antimalarial at prespecified intervals to prevent malaria - essentially a form of chemoprophylaxis. The most

widely used and evaluated drug has been sulfadoxine-pyrimethamine, an antimalarial that for a period succeeded chloroquine as first-line treatment for *falciparum* malaria but then rapidly succumbed to resistance in many areas. Interestingly, Barger et al. (2009) find that IPT using artemisinin-based combination therapy reduces malaria morbidity, asymptomatic parasitemia and anaemia among school-aged children in Mali. However, as one of the major public health challenges (for many decades) remains to scale up ITN coverage, we focus here on ITNs.

Figure 3 provides some figures about insecticide-treated net coverage in Africa (% of children aged less than five years who were protected by an ITN in 2007). Nearly all of the 45 countries in the WHO African Region had adopted, by the end of 2006, the policy of providing insecticidal nets free of charge to children and pregnant women, but only 16 aimed to cover all age groups at risk. However, the proportion of children sleeping under an ITN is far from reaching 50% in many African countries. Moreover, even when ITNs are available, use may be limited: greater accessibility to net-impregnation services is necessary but not sufficient to increase ITN use (Rhee et al., 2005).

FIGURE 3: ITN Use in July 2007 (source: author from Noor et al. dataset, 2008)



Studies on local perceptions, acceptance and use of insecticide-treated bednets are innumerable. Various cultural and behavioural factors may influence bednet acceptance and use. From them, we have evidences that demographic and political patterns (rural versus urban areas, factionalism), gender and age differences within the household, use of other anti-mosquito methods, financial considerations such as costs and income, means of payment, and income distribution are important patterns. Mothers' attitudes and behaviours regarding malaria and its prevention are also important determinants of success in malaria control programs promoting ITNs in rural Africa. In a randomised experimental study realised in western Kenya, Alaii et al. (2003) found that the proportion of mothers mentioning mosquitoes (anytime and alone) as a cause of malaria increased with educational level. They also show that mothers were willing to pay approximately U.S. \$ 4.5 for a regular bed net, but only U.S. \$ 10.5 cents (in the intervention group) and 0.036 (in the control group) for re-treating a bed net. The study suggests that, despite two years of experience with use, bed nets and insecticides would not be purchased as a household priority in this area. Dupas & Cohen (2008) also provide support to free distribution. They show that free distribution is more effective and more cost-effective than cost-sharing despite the fact that the private returns of preventing malaria are potentially large.

2. Treating: ACTs, Malpractice in Malaria treatment and diagnostic testing

By June 2008, all except four countries and territories worldwide had adopted ACT as the first-line treatment for *P. falciparum*. Free treatment with ACT was available in 8 of 10 countries in the South-East Asia Region, but a smaller proportion of countries in other regions. According to the World Malaria Report 2008, doses of ACT supplied through public health services increased from 6 million in 2005 to 49 million in 2006, of which 45 million were for African countries. These National Malaria Control Programs figures probably underestimate usage, and the exact consumption of ACT is not known. According to the same NMCP data, only 16 million rapid diagnostic tests (RDT) were delivered in 2006, of which 11 million were for countries in Africa, a small quantity in comparison with the estimated number of malaria episodes.

Following Arrow's recommendations (2004), the Affordable Medicine Facility-Malaria (AMFm) aims at expanding access to affordable artemisinin-based combinations therapies (ACTs) by reducing cost of these drugs in malaria-endemic countries and delay resistance to artemisinin. The AMFm is managed by the GFATM and is supposed to promote the use of effective antimalarial and drive out ineffective medicines from the market by: 1) reducing consumer prices to an affordable level

through price negotiations and a buyer co-payment and 2) ensuring safe and effective scale-up of ACT use by introducing in-country supporting interventions¹. Only 11 countries have been designated to participate to the first phase of AMFm starting in November 2009: Benin, Cambodia, Ghana, Kenya, Madagascar, Niger, Nigeria, Rwanda, Senegal, Tanzania and Uganda.

The question of malpractice in malaria treatment, malaria misdiagnosis, and malaria epidemiologic surveillance is a key issue to delay ACTs resistance. The use of rapid diagnostic tests (RDTs) for *Plasmodium falciparum* malaria is being suggested to improve diagnostic efficiency in peripheral health care settings in Africa. RDTs generally cost less than a full course of ACT. However, the wide-scale use of RDTs will not be funded through the AMFm co-payment mechanism. On the GFATM website one can read: « Surveys in 18 African countries found that 38 percent of children with fever were treated with antimalarial drugs, but only 3 percent with ACTs »². Fever is not malaria, however. On the one hand, the overuse of ACTs may lead to additional costs and increased resistance risks. As we will discuss in Chapter 3, the use of fever as an indicator of malaria has to be taken with caution. In a recent study made in Zanzibar, Msellem et al. (2009) find that RDTs results in improved adequate treatment and health outcomes without increased cost per patient. They conclude that RDTs may represent a tool for improved management of patients with fever in peripheral health care settings. On the other hand, several researches have shown that clinicians are reluctant to refrain from treating for malaria after a negative test (Bisoffi et al., 2009). If the result of a test is not going to influence management, then doing the test is a waste of money (Bisoffi & Van den Ende, 2008). These issues are crucial for the future of malaria control strategies, as poor implementation strategies of new combination therapies will obviously doom them to failure. Losing ACTs would be a disaster for malaria control, as was the lost of chloroquine.

C. Financing Health in developing countries

In December 2001, the commission on Macroeconomics & Health recommended an overall \$31 billion of donor aid *per year* to country programs by 2015. Total annual health costs to save around 8 million lives *per year* were estimated to \$94 billion dollars *per year* and countries would commit \$63 billion of this value by 2015. \$2 to \$3.5 billion in annual spending (US\$4.02 per African at risk) would be needed

¹ see AMFm FAQs for more details (Accessed: Sept 2009):

http://www.theglobalfund.org/documents/amfm/AMFmFAQs_en.pdf

² <http://www.theglobalfund.org/en/malaria/> (Accessed: Sept 2009)

to control malaria in Africa (Teklehaimanot et al., 2007). It is often asked where the money needed for expanding health care (and education) in developing countries will be found. Nobel laureate Sen (1998) argues that education, health services, and many other means of improving the quality of life are typically highly labor-intensive and are thus relatively inexpensive in poor countries (because of low wages). While poor countries have less money to spend, they also need less money to provide these services. However, the ethical and social responsibilities of States have to face some macroeconomic constraints (macroeconomic equilibria, the human resources crisis and brain drain, increased dependency toward vertical programs, etc). We should note that, the Abuja declaration, where African States commit to dedicate 15% of government expenditure to health, has been reduced to rhetoric for most countries. In 2006, in the African WHO region, total expenditure on health averaged 5.5% of GDP. The general government expenditure on health represented 47.1% of total expenditure on health (in comparison, in Europe, this figure was 75.6% in 2006). General expenditure on health as a % of total government expenditure averaged 8.7 % in 2006 against 8.2% in 2000 and external resources for health represented 10.7% of total expenditure on health (World Health Statistics, 2009).

Moatti & Ventelou (2009) have shown, in a critical review, how the main paradigms of the health economics literature applied to developing countries have evolved since 2000. Nobel laureates Fogel (1993) and Sen (1998) as well as the commission on Macroeconomic & Health have largely contributed to this change, while the AMFm results from this recent evolution. In particular, disease-targeted “vertical programs” are viewed as a way to improve efficiency of health care system as a whole. This situation is comparable to the mid-1950s when vertical programs replaced integrated disease-control and development programs. Instead, Moatti & Ventelou (2009) suggest a “diagonal approach” to bridge the gap between “vertical” and “horizontal” programs. The second paradigm is related to health care financing: prepayment and health insurance mechanisms are replacing cost recovery policies and direct payment. These new paradigms justify the increase of international transfer dedicated to healthcare programs.

However, the notion that health spending is often inefficient and that more spending does not necessarily result in better outcomes is well known and remains poorly understood. Unfortunately current research on these topics does not provide clear-cut answers to carry out an arbitrage on different investments and resources allocation options. The disparities of health production costs and efficiency between and within developing countries, failures in the monitoring of public health systems performance and the deficiencies in how health care systems help patients to make correct choices are some of the remaining barriers.

To conclude this section on malaria control strategies, it is very interesting to observe that conclusions and their interpretations about the relationship between malaria and economic development are related to the history of disease-control programs³. Gallup and Sachs' kinds of arguments had obviously more chance to stand out from the rest when the Roll Back Malaria program was being implemented between 1998 and 2001, as they provide a clear opportunity to legitimize the funding of malaria control program. The same arguments were used in the mid-1950s when the Director-General of WHO asserted: *"there can be no doubt about the general economic and social benefits that malaria eradication brings to countries cleared of the disease"* (Packard, 2009; quoting the proposal by Director-General for malaria eradication, WHO 1955). This can be a dangerous strategy as the multiplicity of malaria economic effects is not well understood yet. However, studying the economic effects of malaria remains necessary. First, the relationship between health and economic development is an important theoretical question to understand the development of nations. Second, it is necessary to understand better how to implement efficiently malaria control strategies.

II. Economic analysis of malaria

A. Economic effects of Malaria: the "impact approach"

1. Macroeconomic level

Why are malarious countries poorer? Why do they experience lower economic growth rates than non-malarious countries do? Recently, a series of papers has explored the link between economic development and malaria⁴. Contrary to other tropical diseases and since the failure of eradication efforts in the 1980s, malaria has been described as an unavoidable effect of tropical location and natural forces (i.e., heavy rains, floods). Therefore recent empirical studies have used malaria as an exogenous variable in regressions considering the effect of malaria endemicity on economic growth and income level. Nevertheless, malaria transmission channels are still not well-known.

³ For a literature survey of studies assessing the economic burden of malaria since 1909, see Packard (2009).

⁴ See for instance McDonald (1950), Barlow (1967), Newman (1968), Gomes (1993) and Audibert et al. (1999), Bowden et al. (2008).

Gallup & Sachs (2001), using a malaria exposure index defined as the fraction of population at risk of contracting *falciparum* malaria in a country, show that poverty is not a leading cause of malaria, *ceteris paribus*. The ecological conditions that allow the reproduction and development of vectors determine the intensity and distribution of the disease. In a cross-country regression framework, the authors find that countries with intensive malaria are poorer and have grown 1.3% less per person per year. These results point to malaria's causing poverty, not vice versa.

In another article, McCarthy et al. (2000), using estimates of malaria morbidity from the World Health Organization (WHO), suggest that economic development may influence malaria control. They confirm the dominant role of climate in determining malaria intensity and the negative correlation between malaria and growth but they find that access to rural healthcare and income equality also influence malaria morbidity once one controls for climate. This finding raises the problem of the endogeneity of malaria with respect to growth and the robustness of the results in this field of research.

In contrast, in Acemoglu et al. (2003), malaria "*is unlikely to be the reason why many countries in Africa and Asia are very poor today*". "*Moreover, it is hard to imagine how malaria could have such a large effect on economic performance*" Acemoglu, Johnson & Robinson (2001)⁵. Acemoglu & Johnson (2007) also cast doubt on the causal relationship between bad health conditions and poverty. Using a large cross-section time series data set, they find that health progresses have not improved economic performance in the 1960s to 1990s decades.

Consequently, Gallup & Sachs (2001) or McCarthy et al. (2000) studies do not prove that malaria is a *cause* of low incomes and poor economic growth. Many other changes occurred simultaneously in the economies where malaria has been eliminated. Malaria eradication can be correlated with many other factors that influence economic growth including governance and the quality of public policies that have been omitted from econometric models. Lastly, the relationship between malaria and income growth is certainly bidirectional. Packard (2009) notes that "*given the multiple difficulties associated with past efforts to demonstrate the impact of malaria on economic development and the economic benefits of malaria control, it is surprising that Gallup and Sachs stated their conclusions with such confidence and that their conclusions have been accepted with so little question.*" Moreover, "*the vast majority of references to Sachs' research are not by economists*".

⁵ It should be noted that the relationship between malaria and growth is not the primary focus of these studies.

However, the terrible toll on the health and well-being of people living in malaria-endemic or epidemic areas and the convergence between different studies⁶ prove at least that the disease could be considered a potential legitimate contributor of poor economic performance (Arrow, 2004). The macroeconomic literature is relatively poor compared to the microeconomic literature on malaria. Even though macroeconomic and microeconomic studies are very complementary in this field of research, macroeconomic effects are not the mere aggregate of microeconomic results.

2. Microeconomic level

Microeconomic studies mostly use the Human Capital approach described in the next subsection of this introduction. This approach leads one to estimate the costs of malaria on various levels: those incurred by individuals and households, public health services and governments, and other private agents (firms, NGOs for instance). Direct or immediate costs are distinguished from indirect costs. Shepard et al. (1991) reported that the total cost of malaria for Sub-Saharan Africa was 0.6% of total SSA GDP in 1987 and estimated to increase to 1% in 1995.

a) Direct costs

Direct costs on household and individual level are expenditures related to prevention and treatment. Seeking treatments at health centres, buying drugs and arranging funerals entail obvious financial costs. For the private business sector, the cost of private health care provided by employers may rise. For public health services, public expenditures for malaria prevention and treatment may be substantial even if they are not easily quantified. This lack of quantification can result in a reduction in the standard of care for all patients. In Mali, only one study (Keita, Diakité & Diarra, 2004) tried to estimate the direct cost of malaria. The authors find that this cost amounted to 3.36% of GDP in 2003. The total costs due to malaria are around 74.1 billions CFA Francs, i.e. US \$ 10.6 billions (7000 CFA Francs per person per year). The burden is supported at a level of 92% by households, 6% by the state and 2% by other economic agents.

b) Indirect costs and loss of productivity

Indirect costs include, among others, lost time due to illness (such as time spent seeking care), long-term losses in household productivity and income, absenteeism from work or school, a decline in activity in various sectors, and excessive use of

⁶ See for instance Shepard et al. (1991) and Chima et al. (2003).

government resources on related expenditures. Studies on the indirect cost vary widely in their methods and in what they try to assess. The effect of malaria on farmers productivity and efficiency has been the subject of careful research (Conly, 1975; Audibert, 1986, 1993, 1997; Audibert et al., 1990, 1999, 2003, 2009; Leighton & Foster, 1993). The latest results from Audibert et al. (2009) suggest that the effect depends on the type of agricultural activity. Indeed malaria has no effect on coffee and cocoa productions but reduces technical efficiency in the cultivation of the cotton crop. One difficulty tackled in later articles on efficiency relates to coping or anticipatory coping strategies adopted by the families (particularly wealthy families) that bias the results indicating the estimated effects of the disease on production and productivity. The size and direction of this bias depends on the conditions of the labour market and on epidemiologic conditions. Other difficulties come from measurement errors related to health variables, omitted variables or reverse causality leading to endogeneity problems (Strauss, 1986).

However, it is often hard to design a standard methodological framework of the link between malaria and economic development from the wide variety of microeconomic studies. Moreover, malaria could affect individuals without destabilizing the economy of a country. A synthesis of the teachings from the macroeconomic and microeconomic literature on the link between malaria and poverty is necessary before starting our work.

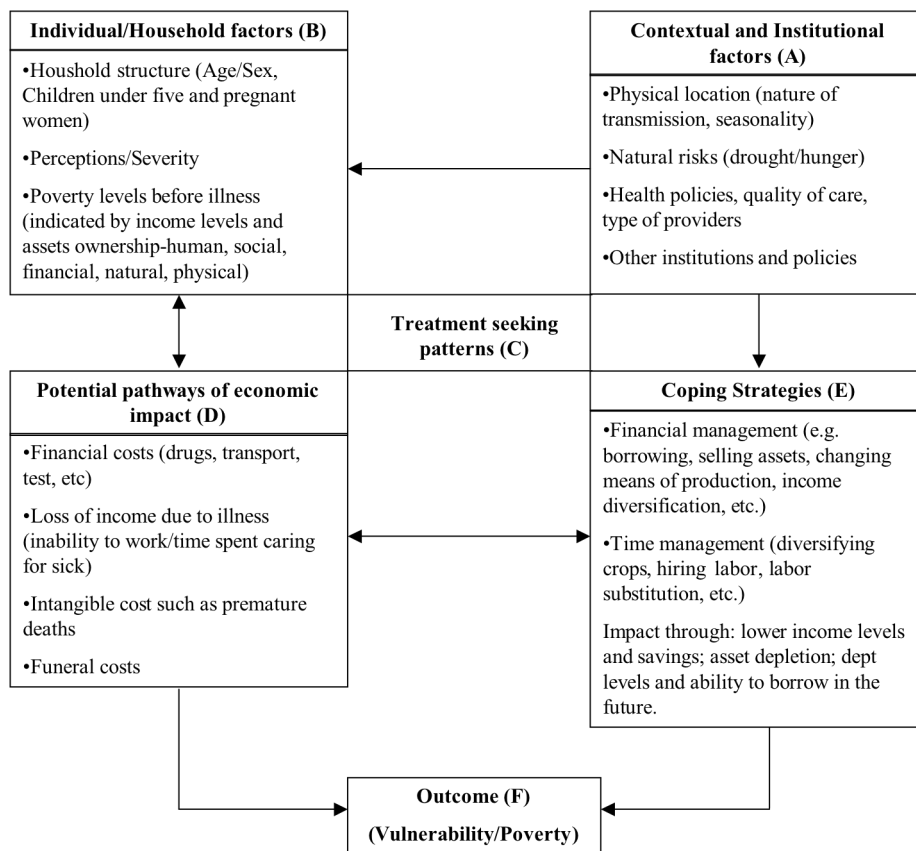
B. Malaria, poverty and growth: an endogenous relationship?

The debate between Acemoglu and Sachs on effects of malaria (and health in general) on growth is useful in raising new questions. These authors try to determine whether or not health progresses are sources or consequences of the development of nations. However, we argue that the fundamental question of this debate is not being asked correctly. We should instead try to understand how health interacts with economic development. We have some evidence that malaria and poverty are intimately correlated and that malaria is a costly disease. Whatever the conclusion of the debate between Acemoglu and Sachs may be, it will not improve our knowledge on the possible mechanisms of such a correlation. It should also be noted that studies published by Sachs or Acemoglu use approximately the same approach and have their own critics (Packard, 2009). Furthermore, all diseases have not the same characteristics and possibly not the same relationship with growth and poverty. The debate is becoming increasingly ideological in nature with both sides standing their

ground. The literature should rather focus on transmission channels and theoretical mechanisms as suggested by Rodriguez & Rodrik (2000).

What does the literature on poverty and malaria teach us? First, causation may run in both directions at the macroeconomic and the microeconomic level: from malaria to poverty and from poverty to malaria. Second, probable macroeconomic pathways are demographic effects, human capital, trade and foreign investment, reduced tourist activity, and limitations on the internal movement of a population. The complexity of these interactions and their failure to follow the predicted pattern point out the difficulty to assess the macroeconomic effects of malaria (Brown, 1986). On the other hand, the control of malaria requires financial resources. Third, potential microeconomic mechanisms involved in the relationship between malaria, poverty and vulnerability have been described in Chuma et al. (2006) and reproduced in Figure 2 (see also Appendix 1). This simplified scheme is a useful tool for assessing the economic costs of malaria at the household level.

FIGURE 4: Framework for analysing the relationship between malaria, poverty and vulnerability (source: Chuma et al. 2006 with their permission)



Exogenous factors (A) determine malaria transmission patterns. Factors in (A), (B), (D), (E) interact with individual treatment-seeking behaviours. Disease costs are detailed in Box D and determine coping strategies and intra-family labor replacement (already defined in the previous subsection). Wealthy households cope better than poorer household (relation between B and E). Worrall et al. (2005) provide an exhaustive literature survey and find mixed evidence on the effects of socioeconomic status on malaria incidence. These findings speak in favour of malaria exogeneity. However, they find strong evidence that malaria prevention and treatment is related to socioeconomic status. Fourth, there is a gap between macroeconomic and microeconomic estimates of the burden of malaria. It is possible that malaria affects individuals without disturbing macroeconomic conditions.

Nevertheless, some effects of malaria are not taken into account in this framework or have not been explored in depth, particularly the effects on future generations through education and human capital accumulation. This point was emphasized by Packard (2009): *“clearly, the one area where malaria may have a detrimental effect on economic growth is its impact on the health and mental development of children. To the extent that malaria retards the educational advancement of a country’s population, it may well have a long-term negative effect on development. Evidence also suggests that poor health in childhood is likely to impair the subsequent health of children over the life course, reducing productivity as adults. However, no one has calculated how these reductions in productivity might affect macroeconomic development.”* Second and most importantly, this literature generally presents a static picture of the relationship between the disease and poverty. Strauss & Thomas (1998) have shown the importance of including dynamic issues in the discussion. Human rational treatment and preventive behaviours (C) as a response to epidemiologic conditions are generally not integrated into a dynamical framework that allows for an explanation of the probable endogenous relationship between malaria and poverty, and an integration of possible externalities. Last but not least, there is no real tentative to reconcile Sachs and Acemoglu arguments, taking into account societal costs as a whole.

C. Malaria and economic development: analytical framework

1. Health, education and the Human Capital Theory

Becker (1964) defines investments in human capital as *« activities that influence future monetary and psychic incomes in increasing the resources of people [...] The*

many forms of such investments include schooling, on the job training, medical care, migration, and searching information about price and incomes. » Therefore, human capital refers to the stock of skills and knowledge involved in the capacity to produce economic value. Our purpose here is not to conduct an exhaustive review of the human capital literature from Schultz (1961), Becker (1964, 1975) and Mincer (1974) to Strauss & Thomas (1998) or Grossman (1999). Investments in education and health present numerous similarities and complementarities (Mushkin, 1962):

- Both are incorporated in the individual.
- Both may affect the productivity of factor and generate externalities.
- Both can be financed by public and private sources.
- Both can be perceived as consumption goods generating welfare (because they influence consumer's satisfaction gained from other goods) or investments (because they may enter in the production function or affect global and marginal productivity of factors).
- They are complementary: bad health reduces returns to education investments; good health reduces the depreciation of education investments; the level of education in the population affects health care system efficiency (through human resources and population basic knowledge on health and hygiene).

However, the differences between investments in health and education have to be emphasized. Education has different levels of distinction between formal, informal and non-formal education. Education is supposed to increase the efficiency of the production system in a society (Furtado, 1977). Formal education can be defined as the process of training and developing, deliberately and systematically, people in knowledge, skills, mind, and attitudes in a structured and certified way with set qualifications for teacher and learner. Informal education is the lifelong process whereby every individual acquires attitudes, values, skills and knowledge from daily experience and the educative influences and resources in his or her environment. Non-formal education is any organised educational activity outside the established formal system. In particular, formal primary education has two main purposes: to produce a literate and numerate population that can deal with problems encountered at home and at work, and to serve as a foundation on which further education is built.

This thesis relies on the impact of malaria on youths within the formal primary education system. However, there are other youths who are not within this formal educational sector and who may be affected by the disease. The relation between formal education (generally measured in terms of completed years of schooling), labour market outcomes, nonmarket outcomes, and input allocation at the microeconomic level has been well documented. The quantity of schooling is also highly and positively related to economic growth rates in macroeconomic studies.

However, quantity of schooling is a very approximate measure of the knowledge and cognitive skills of people and the labor force. Hanushek (2005) focuses specifically on school quality and suggests that school quality has direct implications for the productivity and earnings of individuals. The theoretical basis of this assumption is the “*Mincer equation*”, where quality of schooling is introduced and the return to quality is estimated. Studies that have examined the microeconomic effects of education quality (defined as the measure of knowledge of people) find that higher quality is related to higher individual productivity and earnings, all other things being equal (Jamison et al., 2007). Barro & Lee (2001) also find that higher levels of education quality (as measured by international student achievement tests or repetition rates) increase growth rates of national income. Hence, we have more evidence of the effects of education investments on income growth and level than for health investments.

The specific multidimensional nature of health also has to be underlined in the human capital theory: health evolves over an individual lifespan and health inputs can be health-increasing or health-decreasing, influencing investments in health. Consequently, health is a specific form of human capital that is difficult to measure. The effects of health on a particular outcome may also vary over time. Therefore the complex nature of health makes it more difficult to understand the returns and benefits from investments in health than in the case of education. This is particularly important when one tries to assess the time-course of the effects of health on learning, labour, productivity or income. This is also important because some changes in health status will be anticipated or/and compensated for, leading to feedback issues between health and the outcome measured (Strauss & Thomas, 1998). This will occur, for instance, if better health leads to a higher level of school achievement and if, in turn, higher income and a higher level of education lead to an input allocation in favour of health-increasing inputs. However, the health-productivity hypothesis and the nonconcave health-based efficiency wage models suggests that workers’ real wages vary with health outputs and that health affects productivity. Hence, household production studies should focus on health as a legitimate determinant of productivity, just as education. The general equilibrium effects of these models predict that the workers with the poorest health will be excluded from the market because they are too costly (Strauss & Thomas, 1998). Besides, investments in health differ from investments in education in two other points:

- First investments in health affect simultaneously labour force quality and quantity whereas investments in education influence only the quality of labour force.

- Second, the objective of health investments is to preserve health capital (through prevention and/or treatment). Education is a more creative process that aims at increasing the stock of knowledge. However, if health affect education performance, it could be relevant to consider health as a creative process as well.

The interactions between human capital accumulation (through health and education) and economic development are detailed in the following paragraph.

2. Malaria, education and economic development : an integrated approach

The effects of malaria cannot be understood without a comprehensive grasp of the interplay of demographic, cultural, economic, social, political, psychological and other relevant dimensions. Figure 5 proposes an integrated framework (from an economic perspective) for analysing the relationship between malaria and economic development, taking into account human capital accumulation and both micro and macro mechanisms. We do not pretend to answer which factor among the choice of institutions (Acemoglu and colleagues) or malaria and geography (Sachs and colleagues) explain predominantly cross-country time series patterns of income per capita. Relationships are certainly more complex than these authors pretend. We will rather focus on the main mechanisms that could be involved in the economic impact of malaria without judging their relative importance and starting from existing development models.

a) The traditional neoclassical approach

Drouhin et al. (2003) provide a discussion on the macroeconomic impacts of the AIDS epidemic in different development models. In the traditional neoclassical growth model developed by Solow (1956), the output of an economy grows in response to the relative availability of production factors (labor and capital). Savings are assumed to be a fixed fraction (determined by the population propensity to save) of output and technology improves at an exogenous rate. Variables such as human capital or human health variables do not enter in this model. Furthermore, the constant-returns-to-scale production function conforms to diminishing marginal returns in its two arguments (capital and labor). In this framework and holding saving and capital depreciation rates fixed, the dynamics of the population will determine the dynamics of national income, possibly adjusted by exogenous technology growth rate. The saving rate has no effect on the long run growth rate per capita but positively affects the level of per capita income in the steady state. The model implies that countries with similar production technologies, comparable

savings and population growth rates and the same physical capital depreciation rate, should converge to similar levels of per capita income because of diminishing marginal returns. In such a Solowian context the impact of malaria on the capital per capita in the permanent regime will be theoretically undetermined as in Drouhin et al. (2003): lower population growth rate leading to higher capital per capita by reducing demographic pressure on savings (positive Malthusian effect), lower savings rate (because of a high healthcare expenditure and because the increase in the risk of dying reinforces the preference for the present) and therefore lower per capita product (negative effect), lower labour productivity (absenteeism, fatigue) entailing in a reduction in per capita production (negative effect).

Barlow (1967) was one of the first economist to examine the economic effects of malaria taking into account the multiplicity of these effects and integrating general equilibrium effects in a macroeconomic demographic model. He identified four effects of malaria eradication on per capita income: effects on population size (increase in population resulting from both declining mortality and rising fertility), effects on labor inputs (increase in labor supply and efficiency), effects on capital inputs (limited and unproductive private saving due to population growth and public investments), effects on output (exploitation of new territories). In his calibrated model, Barlow found a positive impact of malaria eradication in the short run and a negative impact in the long run in Ceylon between 1947 and 1965. Eradication led to immediate increase in labor quality and quantity (through reductions in morbidity and debility decreased mortality). This increases income per equivalent consumer. After a while (1953-1955), the negative effects of malaria eradication became prominent (increase in the population, decreased savings, higher demand on the public sector). In his model, the main economic disadvantage of a malaria-eradication program is the rapid increase in the population of children. He suggested that these effects could be contained by birth control. However, Barlow's model is not off limits. Following Newman's (1970) findings, Barlow supposes that malaria eradication will raise fertility despite the fact that the demographic effects of malaria are theoretically undetermined. Malaria can have three instantaneous effects on fertility (Lucas, 2007): 1) a negative biological effect on fecundity; 2) economic theories predict that increased income and the decreased price of a surviving child will increase fertility (positive effect); 3) the demographic transition theories suggest that increased survival certainty and preferences for quality over quantity will decrease fertility (negative effect). There are two long-term demographic effects of malaria eradication on fertility: 1) women born after eradication will be healthier, leading to higher fertility; 2) if malaria has a negative effect on education, their additional education increases their opportunity cost of time, potentially lowering total fertility. For

instance, Lucas (2007) finds that the initial effect of malaria eradication in Sri-Lanka (from 1937 to 1953) was actually an increase in fertility (because of the biological effect) but the next generation had lower fertility and higher education. Therefore, while the initial population growth might be detrimental to GDP per capita, increased education and lower subsequent fertility can mitigate this initial negative growth effect. This seems contradictory to Barlow's findings. It is possible that Barlow's long run was insufficiently long... Two strong additional hypotheses made by Barlow are the negative effect of exogenous population growth on the availability of savings for capital formation and the adult equivalent consumption (assumption made on the consumption demand of children). As noted by Kelley (1974) "*macroeconomic demographic models simplicity entails sacrificing important hypotheses relating to economic-demographic interactions; and the economic-demographic hypotheses included are, in several instances, untested empirically and questionable theoretically. Second, the usefulness of these models for public policymaking is limited. The main policy implication is that slower population growth rates are desirable*" but they do not tell us how to accomplish this goal. Moreover, Barlow considers that the malaria eradication will "*impose burdens on the educational sector*" due to the massive enrolment of new cohorts of children as time progresses. Barlow acknowledged that investments in education could lead to improvements in the quality of the workforce after a substantial lag time but this would be due to public investments accompanying population growth and not to disease eradication by itself. In other words, investments in health are not supposed to improve the quality of the workforce through children human capital accumulation in his model. Instead, reducing mortality, morbidity and debility will increase the quality and quantity of labor inputs. The quantity of labor inputs will be increased through diminishing absence from work and the fall in deaths rate among younger age groups. However, Barlow recognises that "*a worker freed of debilitating effects of the disease can improve his performance both physically and mentally; he may complete a given manual task in a shorter time and also undertake activities more imaginative and ambitious than before.*" Malaria during adulthood has long been seen as a major impediment to agricultural production and particularly food production. The trade-off between population growth and increased farmers productive capacity following malaria control has long created anxiety among policy makers. Whether or not the productivity effects of malaria have been underestimated in past analyses is an important question. Drouhin et al. (2003) suggest that the impact of the disease will be differentiated according to the development level reached by a nation. We shall also consider the relative incidence of the disease in the labor force with respect to the rest of the population and the relative impact of the

disease on different segments of the labor force (low-income or high income segment, low productivity or high productivity segment) certainly involved in the long-term effects of malaria.

b) Endogenous growth and production externalities of human capital

Human capital is one particular source of externality that has been emphasized in the endogenous growth literature. Introducing externalities leads to a relaxation of the assumption of diminishing returns to capital and makes necessary investment in human capital to increase the equilibrium growth rate up to the level of the optimal growth rate. In Lucas (1988), externalities take the form of public learning, which increases the stock of human capital and affects the productivity of all factors of production. Interactions amongst agents cause the social returns to human capital to exceed the private ones: individual workers are more productive, regardless of their skill level, if other workers have more human capital (increase in total factor productivity in the economy). In other words, human capital is not just an input in the production process. An important implication in Lucas (1988) is that government investments in human capital can potentially result in a substantial increase in the rate of economic growth.

Azariadis & Drazen (1990) have shown how Lucas's model could lead to the existence of poverty traps and nonconvergent long term growth path, when human capital is subject to threshold externalities : *« sustainable differences in per capita growth rates could appear even among economies with identical structures. »* The attainment of a critical mass of human capital makes it easier to acquire future knowledge. They add : *« what is really at work here is the interchange of increasing with decreasing returns to social inputs, that is, the existence of ranges for the state variables over which social returns to scale alternate from low to high as the size of production externalities varies. Increasing social returns alone need not yield multiplicity; alternating increasing and decreasing returns in the production process may do so. »*

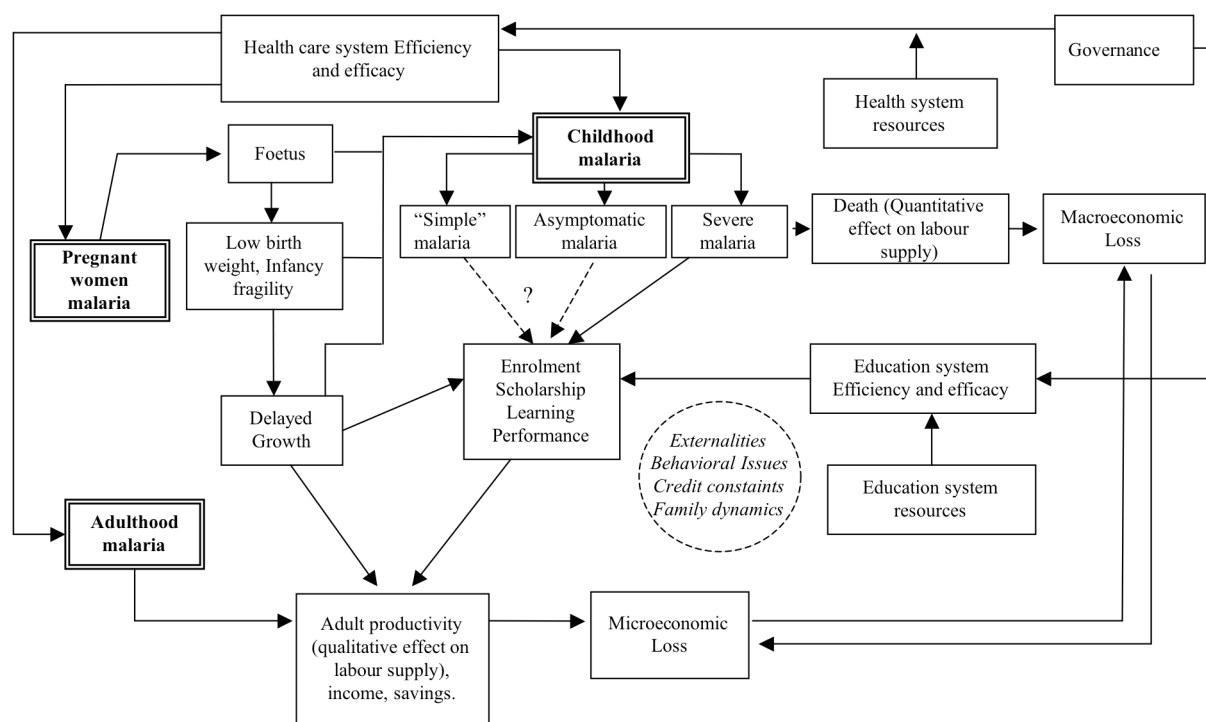
Drouhin et al. (2003) conclude their analysis by formulating a comprehensive model. As Barlow (1967), they propose to list the channels through which the disease affects economic growth and to distinguish long-term from short-term effects. AIDS is supposed to have a short-term impact on the flow of potential workers available and able to work. However and contrary to Barlow, AIDS has also a long-term impact on the stock of human capital i.e. the stock of workers' health or education status. They use an endogenous growth model as the starting point of their analysis and find that integrating these two impacts reverse the prognostic of linear evolution of the

economies: threshold effects lead to multiple equilibrium and “epidemiological traps”. Second, because not all countries are affected by the epidemic in the same way, they suggest that “*if the AIDS crisis, radically modifies the dynamics of the economy, it would be a serious mistake to take an approach which limits itself to measuring the distances between growth rate curves for the “base-case scenario” (before the AIDS crisis) and the growth rate curve after the crisis, as it would overlook the bifurcation. In other words, the comparison would be made with a base-case scenario (no AIDS) which already (incorrectly) includes the impact of the crisis!*”

c) A more qualitative and disaggregated approach

The framework presented in Figure 5 emphasizes the interactions between malaria, education and economic development and seeks to complete the different approaches in a more flexible and qualitative way.

FIGURE 5: Framework for analysing the relationship between malaria, education and economic development (source: adapted from Audibert personal communication, sept 2009)



Indeed, a separate analysis of the different impacts per isolated variable seems incomplete to understand the possible macroeconomic effects of malaria, as many variables and their interactions are involved in this process. In particular, the poor understanding of the interactions between health and education and their effects on

human capital accumulation and growth as well as the limitations of macroeconomic and econometric models to study these questions, justify a more comprehensive approach. Figure 5 is a convenient construct. In this framework, malaria and education may have proper effects on economic development. They can also play an interactive role in the long-term development process through the accumulated effect of malaria during prenatal development, infancy, childhood and adulthood.

Malaria will have quantitative effects on labor supply. These are long-term effects, as malaria mostly kills children. The effect of malaria on children's health and education will block human capital accumulation. This will impact adult productivity and possibly adult health, income and savings at the microeconomic level. There is growing evidence that child health affects adult health. Child health may also be directly associated with adult market success or through its effects on schooling as discussed earlier. The relationship between malaria and under-nutrition is unclear. Under-nutrition is highly prevalent in many areas where malaria is prevalent. Improved nutritional status could lessen the severity of malaria episodes. Reductions in clinical malaria episodes could also improve markers of nutritional status.

Governance matters in this framework. Government expenditure on health and education may be interpreted as investments in human capital and are supposed to be directly productive without failures. Both the level and composition of government expenditure matter. The return on these investments will be affected by the health and education status of individuals. The return on these investments will also be affected by the effectiveness of the public expenditure management system. Various other sources of inefficient use of resources are illustrated through externalities (local externalities in addition to aggregated externalities may exhibit multiple equilibria), individual behaviours, credit constraints and family dynamics (such as coping strategies and the productive structure of the household). The impact of public spending on health and education will be discussed in Chapter 1 and 2.

Nobel Laureate Sen has shown through the notion of *capabilities* (a person being able to do certain basic things she has reason to value) that the availability of health care, basic education and the access to modern medical knowledge in rural communities are among the "*important influences on life and death*" (Sen, 1998). Poverty is the lack of freedom to choose, in other words a capabilities deprivation. The capability to escape illiteracy, morbidity and mortality depends on various physical and social conditions, not only on the lack of material resources or incomes. The epidemiological conditions in which a person lives are part of them. The equality in the distribution of the national wealth seems to matter as well. The benefits of health exceed its exclusive role in the production function. This leads us to one important economic question formulated by Sen: why economic growth is sought in

the first place? Of course, economic prosperity helps people to lead freer and more fulfilling lives. But health and education also causally influence the way people enjoy their lives, in addition to the role they have in promoting productivity. Sen (1997) underlines the need to integrate the relationship between growth and other factors into a more foundational understanding of the process of development. However useful the concept of human capital may be, it only focuses on one side of the coin. Human beings are not merely means of production. *Human capabilities* in contrast to *human capital* serves directly the well-being and freedom of people; and indirectly through economic production and social development. Basic education may improve the quality of public debate and consequently, improve governance. That is principally why the model presented in Chapter 1 starts from the disease and human behaviors to understand the development process involved in the understanding of what we study in this thesis (i.e. the relationship between malaria and economic development), whereas the traditional approach suggests to start from aggregated production functions.

III. The impact of malaria on education: justifications

A number of studies have emphasised the consequences of malaria for child behavioural and cognitive development, learning performance and school attendance. Nevertheless, the relationship is only beginning to be explored. Economists developed the hypothesis of an impact of malaria on human capital accumulation through its effects on education at the beginning of the 21st century:

« Effects on Human Capital: Infants and children carry the greatest burden of malaria morbidity and mortality. Those who survive may have lasting effects on their physical and mental—hence, economic—potential. The physical and cognitive effects are recognized, though poorly quantified. Even without direct effects of the disease, however, children with malaria lose out by missing school. In the few studies that have examined the relationship, upward of 10 percent (up to 50 percent) of school days lost to illness in sub-Saharan Africa are due to malaria (Sachs and Malaney, 2002). Across a population, this means higher failure rates, higher dropout rates, and poorer achievement. The total decrement in human capital development due to malaria is, however, unknown. » Arrow (2004).

« *Malaria has lifelong effects on cognitive development and education levels through the impact of chronic malaria-induced anaemia and time lost or wasted in the classroom due to illness. The importance of these effects is speculative, however, because their impact is virtually unstudied.* » Gallup & Sachs (2001).

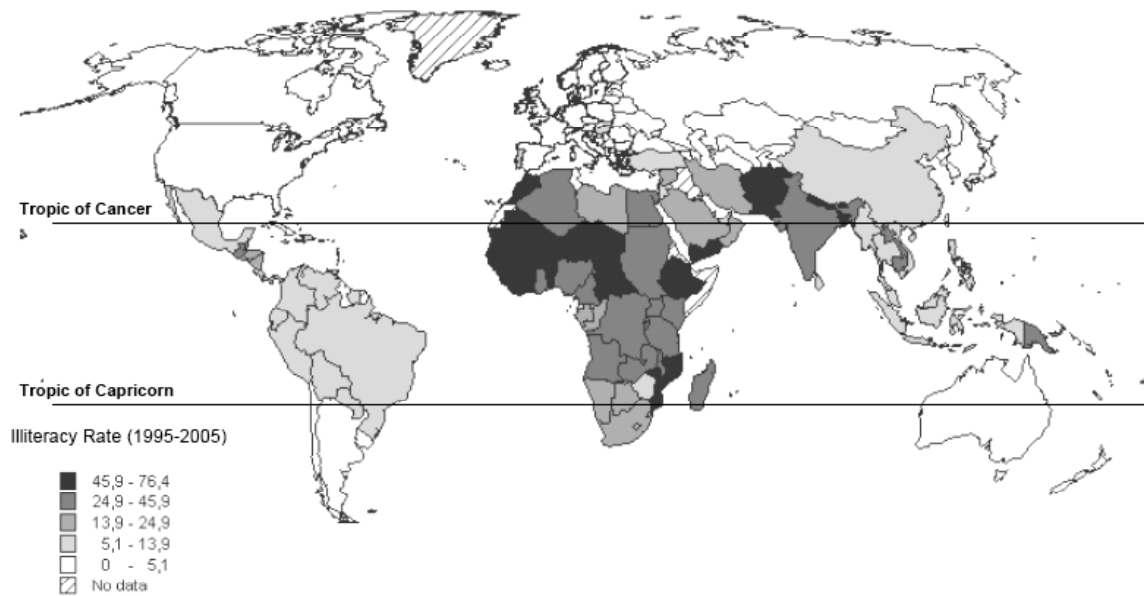
A. Some stylised facts on malaria and education in less developed countries

1. Geographic arguments

As shown in Figures 3 and 4, most of the countries with a high rate of illiteracy are located in the same regions as malaria (Figure 2 give the distribution of *falciparum* malaria only). However, poor countries with high under-5 mortality rates predominate in the same regions as malaria. It is plain to see that, unsurprisingly, a number of confounding factors are correlated with both education levels and malaria. In a panel regression framework, Table 1 tries to tackle this problem and gives some evidence that illiteracy and malaria are intimately connected and that the link between malaria and education is more than a fortuitous correlation. Method and indicators used in this table are detailed in Chapter 2. Nevertheless, illiteracy (the proportion of the adult population aged 15 years and over that is illiterate) is a very imperfect indicator (because of deviations from the standard definition of illiteracy and variations in the quality of data collected between countries) and can only give a general vision of the impact of malaria on human capital accumulation. Country-level malaria indices are also subject to measurement errors.

FIGURE 6: Illiteracy Rate, 15 and above (1995 – 2005)

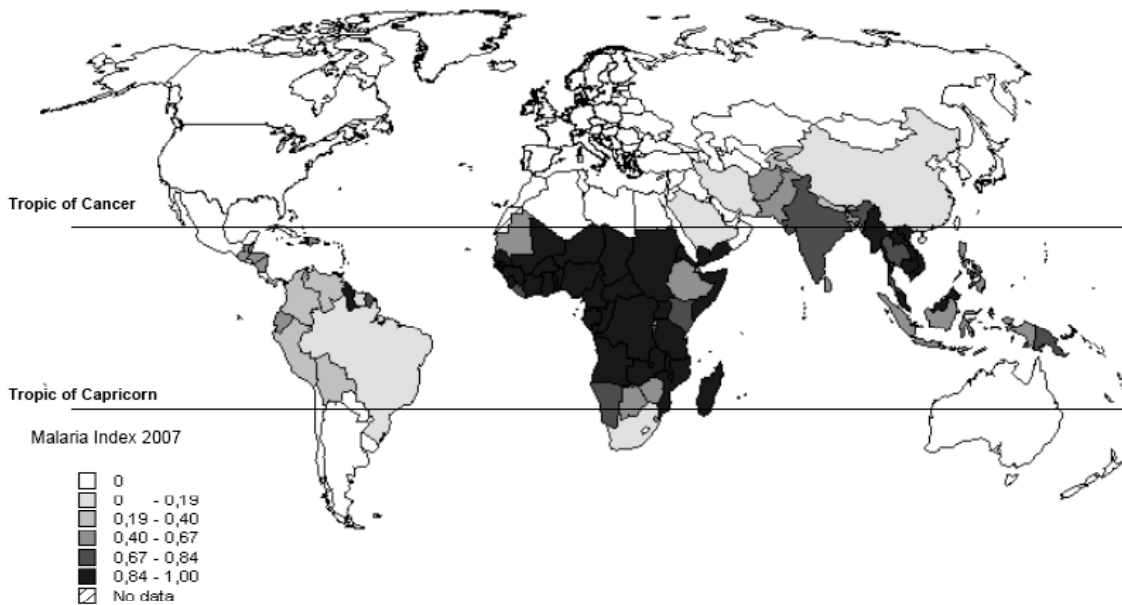
ILLITERACY RATE (1995-2005)



Source: Author from UNDP Data – HDR 2006
<http://hdr.undp.org/en/humandev/>. Accessed: March 2008.

FIGURE 7: *P. falciparum* Malaria Index (2007)

MALARIA INDEX 2007



Source: Author from MAP (Malaria Atlas Project) - 2008
http://www.map.ox.ac.uk/MAP_publications.html. Accessed: April 2008.

TABLE 1: Panel data estimates of the effects of Malaria on Illiteracy Rates

	Dependant Variable is Illiteracy Rate (Panel Dataset 1950-1995) - Random effects Model					
	(1)	(2)	(3)	(4)	(5)	(6)
Sachs & Gallup Malaria index	0.102*** (0.019)	0.049*** (0.018)	0.044* (0.023)	.	.	.
Sachs & Gallup <i>falciparum</i> Malaria index	.	.	.	0.100*** (0.035)	0.075** (0.030)	0.065* (0.034)
Log GNP per capita PPP (\$ constant 95)	-0.017 (0.014)	0.025* (0.014)	-0.000 (0.021)	-0.044*** (0.013)	0.026* (0.014)	-0.001 (0.020)
South Asia	0.141** (0.070)	0.158** (0.066)	0.190*** (0.073)	0.127* (0.075)	0.166** (0.069)	0.193*** (0.074)
Latin America and the Caribbean	0.100** (0.041)	0.043 (0.040)	0.060 (0.046)	0.103** (0.045)	0.046 (0.042)	0.063 (0.047)
East Asia	-0.044 (0.049)	-0.046 (0.050)	0.004 (0.059)	-0.061 (0.054)	-0.048 (0.052)	0.005 (0.059)
Middle East and North Africa	0.307*** (0.040)	0.231*** (0.039)	0.217*** (0.045)	0.314*** (0.043)	0.231*** (0.040)	0.218*** (0.046)
SSA	0.135*** (0.038)	0.134*** (0.039)	0.103** (0.047)	0.081* (0.048)	0.105** (0.045)	0.077 (0.051)
Log Urban Pop (% total pop)	-0.211*** (0.014)	-0.163*** (0.017)	-0.130*** (0.025)	-0.215*** (0.015)	-0.164*** (0.017)	-0.130*** (0.025)
Log Under 5 Child mortality	.	0.086*** (0.010)	0.085*** (0.016)	.	0.090*** (0.010)	0.086*** (0.016)
Log Primary expenditure per pupil PPP (% of GDP)	.	.	0.049*** (0.016)	.	.	0.050*** (0.015)
Log Prim School pupil-teacher ratio	.	.	0.015 (0.034)	.	.	0.018 (0.034)
Constant	1.082*** (0.106)	0.245* (0.146)	0.155 (0.236)	1.332*** (0.092)	0.230 (0.146)	0.150 (0.236)
<i>Number of observations</i>	347	266	178	347	266	178
<i>Overall R squared</i>	0.669	0.723	0.723	0.657	0.723	0.724

Notes:

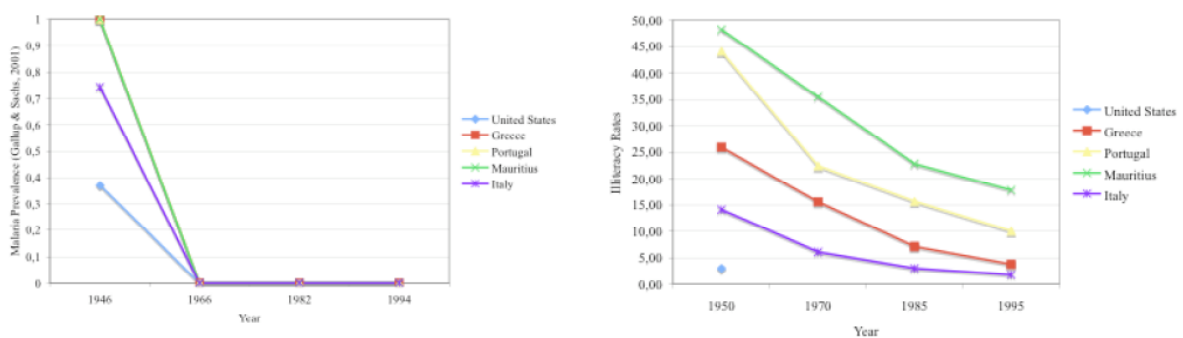
1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

2. Historical arguments

In 1917, the Texas State Health Department, the University of Texas Extension Department and the International Health Board made a first attempt to determine the effects of malaria upon physical and mental development (Kelley, 1917). One interesting conclusion is that the cure from malaria leads to an immediate increase in physical and mental development but the article does not give any explanation of the causal relationship. Moreover, the method and data used in this study remain very crude. McDonald (1950) also underlines the consequences of malaria on child education in terms of absenteeism and chronic infections. Until the 1980s, research in this field had not progressed and the indirect and direct consequences of the disease on child development had been neglected. Snowden (2006) and Amorosa et al. (2005) analyse the Italian anti-malaria campaign. The authors all believe that the campaign has had an under-estimated durable effect on the achievement of alphabetisation in the country. Moreover, education and schools have been used as tools to attack the disease. In a recent paper with an historical perspective, Bleakley (2007) considers the malaria-eradication campaigns in the United-States (1920), Brazil, Colombia and Mexico (1950) to assess the impact of childhood exposure to

malaria on labour productivity. Using a cohort-level dataset based on microeconomic data, the author finds that the cohorts born after eradication enjoyed higher adult literacy than did the preceding generation. The effect of childhood malaria exposure on adults is similar for the four countries considered. Lucas (2005) finds similar conclusions with a quasi-experimental procedure in Paraguay and Sri Lanka. Figure 8 shows (for some countries that have eradicated malaria) that countries with higher prevalence rates before eradication have also taken more time to reach low levels of illiteracy rates.

FIGURE 8: Malaria Index and Illiteracy rates for some countries that have eradicated malaria (*source:author*)



Even if data for United-States after 1950 are not available, the order between countries seems to be identical in both graphs all the more that Mauritius is a particular case and area. Indeed, there are still some areas at very low risk of malaria in Mauritius. *P. falciparum* was the predominant form of malaria in Mauritius from the early 19th century to its eradication in 1965 whereas it was not in Europe nor in the United-States. Mauritius is still an area of unstable malaria and the resurgence of the disease in the 1980's suggests that the country is not totally protected from re-introduction of the disease from imported malaria cases that can deteriorate to reach epidemic proportion. Of course this is not sufficient to conclude that malaria is one *cause* of illiteracy but it is an interesting insight however.

3. Key figures for SSA

In 2000-2003, malaria accounted for 8% of the 10.6 million yearly deaths in children younger than age 5 years (WHO-CHERG). This was the third cause of under-5-years mortality. 94% of global child deaths attributable to the disease occur in Africa (Bryce et al., 2005). In holoendemic areas of Africa, the disease represents the cause of between 20 to 25% of instances of 0-5-child mortality, 25% of paediatrics hospitalisations, and 15% of instances where hospitalisations ends in death. Because

of the decrease in acquired immunity that occurs during pregnancy, malaria also causes acute disease during pregnancy, with severe health consequences for both the mother and the child⁷.

As the burden of disease has been the subject of numerous publications, Tables 2 and 3 focus here on Sub-Saharan Africa (SSA), which is the continent most affected by *falciparum* malaria. They provide a summary of data on the burden of malaria and learning in SSA. Much of the burden of disease in SSA is due to premature mortality. Malaria is the disease with the largest contribution to the global burden of disease among infectious and parasitic diseases in children younger than age 10 years. Mali was ranked 28th in terms of age-standardised DALY rates per 100,000 due to Malaria in 2004. The primary gross enrolment rate in Mali is rather high (83%). With an official primary completion rate of 52.17% and 16.95% of primary education repeaters, Mali was ranked respectively 13rd and 8th among 31 and 32 SSA countries with available data.

In addition to these figures, Gwatkin et al. (2007) and Davoodi et al. (2003) analysed within-countries health and education inequities. The authors find that the poor are less likely to benefit from effective primary education and public health intervention in Sub-Saharan Africa. In particular, the ratio of access to medical treatment of fever between the poorest and the richest quintiles is 0.57 in Africa. The ratio between the poorest and the richest quintiles for fever is 2.63 in Africa. With respect to primary education, the poor's share is less than 13% in SSA. The overall spending on education is also pro-rich in this part of the globe. Berthélemy (2005) shows that sub-Saharan African countries have paid little attention to primary education to the benefit of secondary education. This pattern is explained, among others things, by the post-colonial legacy. Berthélemy & Seban (2009) find that access to health services depends considerably on the socioeconomic characteristics of households and particularly on the mother's education level. Governance efficiency is identified as an important determinant in this analysis.

⁷ For an historical perspective on childhood malaria mortality in Africa, see Snow et al. (2001). For a review of the economic effects of malaria in pregnancy, see Worrall et al. (2007).

TABLE 2: Estimated burden of disease for children in Sub-Saharan Africa (DALYs, GDB 2004).

	SSA			
	Male	Female	Total	%
Children age 0-4 Years				
All causes	98 972 417	90 371 751	189344169	
Communicable diseases	89 603 172	82 312 544	171915716	90,80%
Infectious and parasitic diseases	46 204 658	42 825 200	89029858	47,02%
Tuberculosis	584 573	495 570	1080143	0,57%
HIV/AIDS	4 112 534	4 021 507	8134042	4,30%
Diarrhoeal diseases	14 358 928	13 236 332	27595260	14,57%
Poliomyelitis	8 944	9 027	17971	0,01%
Measles	3 455 345	3 286 693	6742038	3,56%
Tetanus	1 545 051	848 968	2394018	1,26%
Malaria	14 654 803	13 967 096	28621899	15,12%
Respiratory infections	18 280 069	16 594 626	34874696	18,42%
Nutritional deficiencies	4 466 505	4 259 829	8726335	4,61%
Protein-energy malnutrition	3 276 990	3 147 034	6424024	3,39%
Iodine deficiency	482 015	450 144	932159	0,49%
Vitamin A deficiency	257 573	235 705	493278	0,26%
Iron-deficiency anaemia	426 348	403 985	830333	0,44%
Other	23 579	22 961	46540	0,02%
Non communicable diseases	6 303 400	5 793 781	12097180	6,39%
Children age 5-10 Years				
All causes	17 744 302	16 696 670	34440972	
Communicable diseases	10 123 984	10 578 333	20702318	60,11%
Infectious and parasitic diseases	7 277 432	6 912 428	14189860	41,20%
Tuberculosis	470 508	440 594	911102	2,65%
HIV/AIDS	801 541	788 605	1590147	4,62%
Diarrhoeal diseases	1 212 244	1 138 968	2351212	6,83%
Poliomyelitis	0	0	0	0,00%
Measles	58 823	70 140	128963	0,37%
Tetanus	5 594	2 435	8029	0,02%
Malaria	1 300 769	1 306 385	2607153	7,57%
Respiratory infections	2 096 811	2 562 015	4658826	13,53%
Nutritional deficiencies	749 479	863 599	1613078	4,68%
Protein-energy malnutrition	200 384	294 114	494498	1,44%
Iodine deficiency	141 222	131 028	272250	0,79%
Vitamin A deficiency	4 560	4 849	9409	0,03%
Iron-deficiency anaemia	396 377	417 422	813799	2,36%
Other	6 936	16 186	23122	0,07%
Non communicable diseases	3 546 634	3 428 358	6974993	20,25%

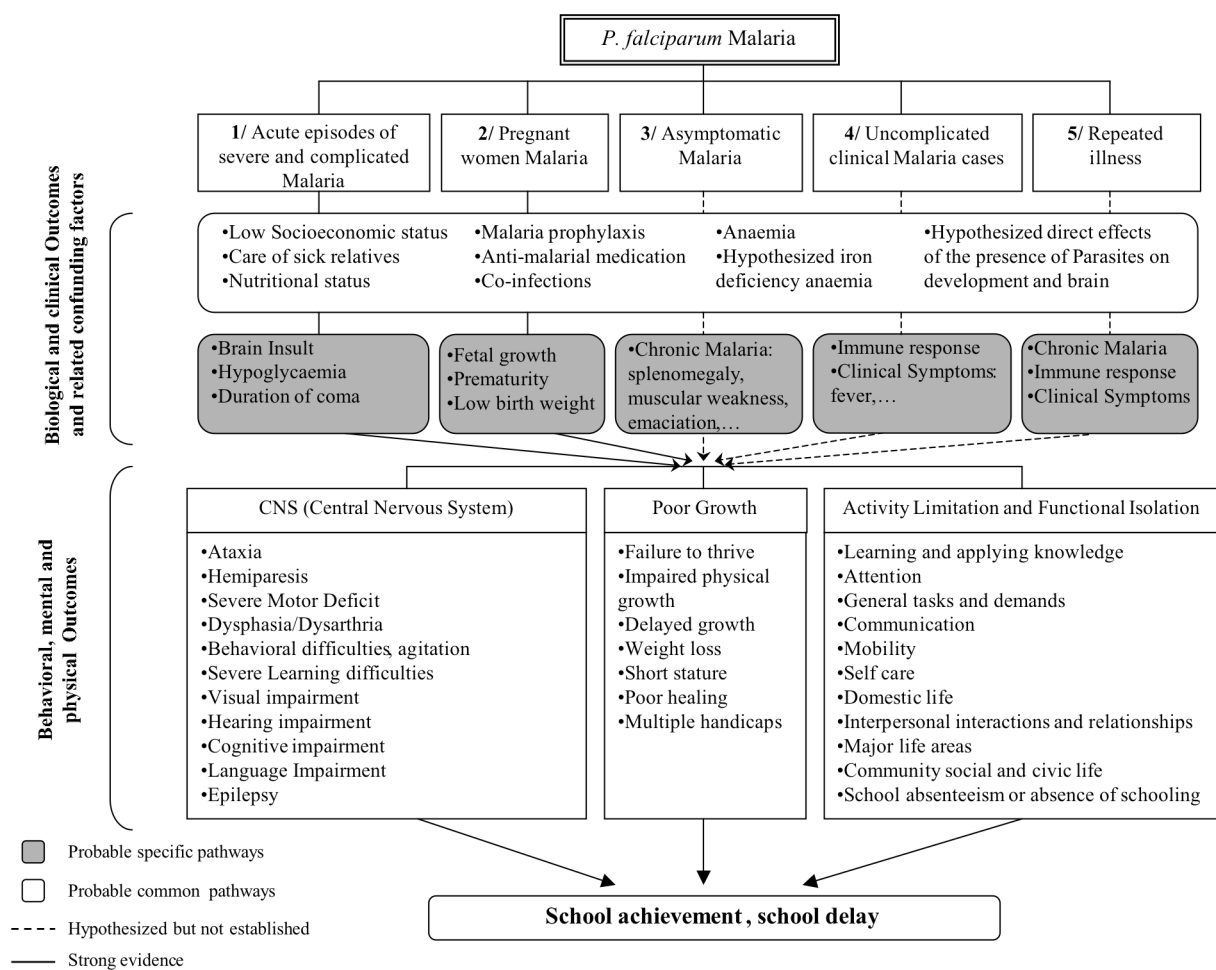
TABLE 3: Data on learning and DALY rates for SSA countries (2004 and 2007)

Country	Age-Std DALY rates per 100 000 due to Malaria - 2004	Gross enrollment rate (%), primary - 2007	Gross enrollment rate (%), secondary - 2007	Primary completion rate - 2007	Percentage of repeaters (%), primary - 2007
Angola	2596.921
Benin	3108.717
Botswana	40.541
Burkina Faso	3368.694	65.307	15.509	33.295	11.750
Burundi	2168.742	114.459	15.242	39.230	31.957
Cameroon	2833.835	109.637	25.153	55.474	20.067
Cape Verde	80.595	101.452	79.349	86.239	12.885
Central African Republic	2784.263	70.670	.	30.244	26.817
Chad	3209.871	73.983	18.832	30.433	21.804
Comoros	1078.534
Congo, Dem. Rep.	3074.221	85.134	33.350	50.683	15.925
Congo, Rep.	2884.79	105.861	.	72.267	21.218
Cote d'Ivoire	2493.343	72.128	.	44.693	21.591
Equatorial Guinea	3826.899	124.236	.	66.723	24.267
Eritrea	87.7141	54.945	29.241	46.381	14.597
Ethiopia	1381.675	90.753	30.468	46.277	6.037
Gabon	1908.337
Gambia, The	2823.284	86.417	48.766	71.583	5.751
Ghana	2707.557	97.671	49.251	.	.
Guinea	3127.484	90.831	37.623	64.200	9.072
Guinea-Bissau	3260.058
Kenya	1796.887	112.642	52.787	.	.
Lesotho	4.726
Liberia	3263.543
Madagascar	288.811	141.378	26.354	61.515	19.140
Malawi	2502.99	116.496	28.270	55.363	20.678
Mali	3123.871	83.127	31.630	52.176	16.956
Mauritania	1859.196	103.238	25.162	59.438	3.400
Mauritius	1.245	101.385	.	93.529	3.722
Mozambique	2500.01	111.019	18.339	46.322	5.923
Namibia	585.233	109.213	58.987	77.082	16.423
Niger	3749.37	53.338	10.611	39.604	4.895
Nigeria	3323.237
Rwanda	1822.062	147.360	18.075	.	14.609
Sao Tome and Principe	347.815	127.355	49.767	72.136	25.948
Senegal	1818.495	83.542	26.276	50.126	10.634
Sierra Leone	3376.271	147.083	31.631	80.754	9.942
Somalia	891.204
South Africa	13.315	102.504	97.075	84.454	7.960
Sudan	1979.75	66.366	33.354	49.973	2.811
Swaziland	34.747	113.425	54.421	74.623	18.035
Tanzania	2379.689	111.854	.	85.410	4.220
Togo	2978.417	97.099	39.345	57.380	23.730
Uganda	2591.993	116.166	22.525	.	.
Zambia	2700.077	118.958	43.143	88.086	6.499
Zimbabwe	381.857

B. Hypothesised Medical pathways and associated confounding factors

The question of the link between malaria, school achievements and cognitive abilities is not a new one but is far from resolved. As we said, one of the first studies about the impact of malaria on mental development was conducted in Texas by Kelley (1917). However, the mechanisms for the effects of malaria on education and learning remain unknown. Figure 9 proposes a schematic diagram that models the pathways through which malaria may affect children’s schooling attainment. The possible dependent mechanisms have been divided into biological, clinical and behavioural outcomes, common and specific probable pathways for each form of malaria. We define cognition as the mental process involved in knowing, learning, and understanding things. We are specifically interested in educational achievement.

FIGURE 9: Probable effects and pathways of *falciparum* Malaria on School Performance (source: author).



1. Effects of malaria on pre-school aged children

Malaria is a parasitic disease transmitted by anopheles mosquitoes that results from the biological development of protozoal parasites (see Appendix 1 for more details). In this thesis, we mainly focus on *P. falciparum* malaria, which is far more severe than the other types of malaria. It is estimated that for 400 subjects that receive an *Anopheles*-infesting bite, 200 will be infected by *falciparum* malaria, 100 will have “simple” clinical malaria, 2 will have cerebral malaria and 1 will die (Marsh, 1992). The effects of malaria on pre-school aged children involve mainly channels 1/ and 2/ of Figure 9.

Acquired immunity in children does not play an efficient protective role until the age of 5-6 years, even in highly endemic areas. That is partly why malaria is a major threat to child survival. Particularly among children under the age of five, complicated forms of malaria may develop rapidly. The effects of severe malaria, better known as cerebral malaria (CM), have been quantified by numerous studies. A literature review (from 1960 to 2002) can be found in Mung'Ala-Odera et al. (2004). Ngoungou et al. (2007) provide a quantification of the burden in Mali. 101 subjects (mean age was 5.6 ± 3.6 years) who had had cerebral malaria in Mali were followed from 1999 to 2001. The authors find that twenty-eight children presented persistent neurological sequelae (26.7%). Among them, eight (7.9%) children had developed these sequelae just after CM and 20 (19.8%) a few months later. These included headaches, mental retardation, speech delay, bucco-facial dyspraxia, diplegia and frontal syndrome (one case each), dystonia (two cases), epilepsy (five cases) and behaviours and attention disorders (15 cases). This study shows that neurological signs due to cerebral malaria can persist in the long run (or at least a few months after). Many other studies have analysed the relationship between severe *falciparum* malaria and developmental and long-term cognitive impairment (Carter et al., 2005; Boivin et al., 2007; John et al., 2008).

Because of depressed acquired immunity during pregnancy, malaria is a real treat for both pregnant women and the foetus with increased risk of premature delivery. Indeed, malaria can lead to anaemia, low birth weight and foetal growth retardation. All of these consequences of malaria during pregnancy can lead to reduced cognitive, physical or development impairments. Barreca (2007, unpublished paper) analyses the long-term economic impact of *in utero* and postnatal exposure to malaria and finds substantial effects on educational attainment in adulthood.

2. Malaria's effects on school-aged children

The effects of malaria on school-aged children mainly involve channels 3/, 4/ and 5/ of Figure 9 and the long-term consequences of malaria during early childhood. Even for older children the protection conferred by acquired immunity is partial. It is well understood in the medical literature that cerebral malaria, malaria among pregnant women and iron deficiency anaemia can impact the architecture of a developing brain. It is much less well understood how other forms of malaria (3/ asymptomatic malaria, 4/ uncomplicated clinical malaria cases, and 5/ repeated illness) impacts the cognitive abilities and school performance of school children.

The main hypothesised mechanisms identified in the literature for Channels 3/, 4/ and 5/, are iron deficiency anaemia after repeated illness or after asymptomatic malaria and school absenteeism after clinical malaria. Indeed one common probable pathway of the effects of all forms of malaria on cognitive achievement is through malaria-induced anaemia. However, the cognitive effects of anaemia due to causes other than iron deficiency are not well understood and it is still not clear whether anaemia in African children is caused predominantly by malaria or iron deficiency (Müller, Traoré, Jahn & Becher, 2003). Hence, there are likely to be more than these two mechanisms.

Tables 4 and 5 give a synthesis of methods, outputs and results of recent medical studies in this field of research. This literature review was conducted using a Pubmed search of relevant published literature from 2003 to 2008. We excluded studies about cognitive impairment after cerebral malaria or long-term handicaps and cognitive impairment as a consequence of malaria during pregnancy. These studies have already been the subject of numerous literature reviews. As we said, a previous literature review (from 1960 to 2002) can be found in Mung'Ala-Odera et al.(2004) but also in Snow & Newton (2004) for cerebral malaria and in Holding & Snow (2001) or Kihara, Carter & Newton (2006) for the probable pathways of cognitive impairment after *P. falciparum* malaria.

First, simple and repeated clinical malaria cases can have non-cognitive consequences through school absenteeism, general health conditions, or other socio-economic conditions and behaviours associated with the disease. For instance, in a case study done in Kenya, Brooker et al. (2000) attribute 13-50% of medically related school absences to malaria. Leighton & Foster (1993) also provide evidence of the number of school days lost due to malaria in Kenya and Nigeria. In Kenya, primary school students were determined to miss 11% of the school year (20 school days missed per child per year). In Nigeria, school days missed varied between 2% to 6% of the school year (3 to 12 days per year per student). Social and economic costs of the disease can amplify absences: nutrition is highly related to socioeconomic conditions,

and parents can retain children at home to take care of younger siblings who are sick or to complete other household or productive tasks. This may occur especially in agricultural economies where farming activities are a family affair and because of coping mechanisms (Audibert et al., 2009). Nevertheless, these conclusions remain speculative because we still know very little about the impact of malaria on school attendance. Absenteeism in developing countries can be due to many reasons unrelated to malaria.

Second, malaria is associated with a number of neglected tropical diseases like hookworm, schistosomiasis, onchocerciasis, filariasis, dengue fever, and trypanosomiasis. Brooker et al. (2006) suggest that the control of malaria and parasitic helminths in school children could be viewed as an essential co-contributor in the promotion of health in schoolchildren. Malaria incidence can be increased by the coinfection with the other tropical diseases.

Third, specific mechanisms of asymptomatic malaria and repeated illness could function through the effects of chronic malaria, characterised among others by enlargement of the spleen, emaciation and muscular weakness.

Fourth, another common probable pathway to the effects of all forms of malaria on cognitive achievement is through parasitaemia *per se*. In Chapter 4, we will test this hypothesis: the direct effects of the presence of *falciparum* malaria parasites *per se* on school achievement, all other things being equal. Until now, the focus has been limited to the indirect effects of exposure *in utero* on birth outcomes (Holding & Kitsao-Wekulo, 2004), and the limited number of investigations among school age children has focused on the effect of parasite clearance (Clarke et al., 2008). However, the randomised control trials may have affected the educational outcomes or may have omitted other confounding factors correlated with both parasitisation and school achievement in the post-intervention analysis. The mechanism could include a “*toxicity effect, leading to biochemical changes in the central nervous system (CNS); excitation of the immune system, leading to changes in behaviours related to appetite and reaction time; and physiological effects such as discomfort and disturbed sleep, leading to reductions in activity levels or causing behavioural change*” (Holding & Snow, 2001). Neurotoxin released from infected red blood cells may also damage the cortical areas of the brain (Kihara et al., 2006). As there is a wide range of potential pathways to impaired educational development and because combinations of different sources of variability in outcomes are plausible, the relative contribution of different pathways will be tested in the last chapter of this thesis. Studies that examined the effects of asymptomatic malaria before 2003 (Boivin et al., 1993; Al Serouri et al., 2000) did not detect any appreciable differences between children when they were parasitaemic and when they were non-parasitaemic.

TABLE 4: Studies examining cognitive function following *falciparum* and *vivax* malaria or evaluating the effect of malaria prevention on school outputs since 2003: study objectives and design.

Study	Objectives	Study Design	Study Place	Duration	Population and Sample Size
1 Fernando et al. (2003a)	To assess the short term impact of an acute attack of malaria on cognitive performance of schoolchildren.	Matched Case / Control study. Passive case detection using microscopy.	Sri Lanka (Moneragala)	Prospective study from January 1998 to November 1999. 14 days follow-up in November 1999	Age: 6-11 years (Grade 1 to 5). 648 schoolchildren: 199 children with malaria (MC), 144 children with non-malarial fever (NMFC), 305 healthy children (HC).
2 Fernando et al. (2003b)	To measure the impact of repeated attacks of malaria infection on the cognitive performances of children at school entry.	Cross sectional Study. Past history of malarial attacks were collected from the parent or legal guardian by interview.	Sri Lanka (Moneragala and Anuradhapura)	One month follow-up in January 1997 (school entry).	Age: 5-6 years (Grade 1). 325 schoolchildren
3 Fernando et al. (2003c)	To measure the impact of repeated attacks of malaria infection on the school performances of schoolchildren.	Historical cohort study. Passive case detection using microscopy.	Sri Lanka (Kataragama)	Prospective study from January 1992 to November 1997.	Age: 6-14 years (Grade 1 to 7). 571 schoolchildren
4 Fernando et al. (2006)	To investigate the impact of malaria and its prevention on educational attainment.	Randomized, double-blind, Placebo / Control, Clinical Trial.	Sri Lanka (Kataragama)	Clinical trial from March to November 1999.	Age: 6-11 years (Grade 1 to 5). 587 schoolchildren
5 Jukes et al. (2006)	To assess long-term impact of early childhood malaria prophylaxis on educational outcomes.	Follow-up of a randomized, double-blind, Placebo / Control, Clinical Trial.	Gambia (Farafenni)	Intervention follow-up from April 1984 to March 1988. Data collection and cognitive assessment from May to November 2001.	Age: 3-59 months at the beginning of the survey. 579 traced children.
6 Clarke et al. (2008)	To assess the effect of Intermittent preventive treatment (IPT) of malaria on health and education in schoolchildren.	Cluster-randomised, double-blind, placebo-controlled clinical trial.	Bondo District (Western Kenya)	Clinical trial from March 2005 to January 2006.	Age: 11-16 years (Grade 5 to 6). 818 schoolchildren included in the analysis of educational outcomes (intention to treat population).

TABLE 5: Studies examining cognitive function following *falciparum* and *vivax* malaria or evaluating the effect of malaria prevention on school outputs since 2003: school outputs used and results.

Study	School Outputs	Main Results
1 Fernando et al. (2003a)	Two cross-sectional educational assessments in November 1999. Grade specific examination papers (mathematics and language), absenteeism.	At the time of presentation and 2 weeks later, children diagnosed with malaria had significantly lower scores in mathematics and language ($P < 0.001$) than NMFC and HC. The impairment seems to be cumulative with repeated attacks. A child having an acute attack of uncomplicated malaria was absent from school 5.4 days in average against 3.4 days for a child with non-malarial fever. Children absent for long duration due to a malaria episode have significantly lower scores.
2 Fernando et al. (2003b)	One cross-sectional educational assessment in January 1997. Cognitive performance (writing skill, reading skill, ability to count, ability to identify numbers,...)	All indices of cognitive performance were poorer in children who had experienced more than 5 attacks of malaria during their lifetime compared to children who had had less than 4 attacks.
3 Fernando et al. (2003c)	One cross-sectional assessment in November 1997. Grade specific special examination designed for the study (25 questions, mathematics and language), 3 routine end of school examinations for 1997.	Significant negative correlation between the total number of malarial attacks experienced by the child during the six years follow-up and tests and school scores. Experiencing more than six malarial infections decreased language and mathematic scores by 15% relative to those who had experienced fewer than 3 malarial infections. There is no significant association between anthropometric indices and school performances.
4 Fernando et al. (2006)	Two assessments pre and post-intervention. End-of-term examinations (mathematics and language), Absenteeism.	School absenteeism due to malaria was reduced by 62.5%. Post-intervention, children who received chloroquine scored 26% higher in language and mathematics than children who received placebo. Educational attainment was significantly associated with taking chloroquine prophylaxis and absenteeism due to malaria.
5 Jukes et al. (2006)	One cross-sectional assessment in November 2001. Cognitive tests (visual, Raven's matrices, Digit span, Fluency, Proverbs, Vocabulary) Years at school, School enrolment.	There were no significant difference between treated and control group in mental development scores but scores appeared significantly higher for children who received malaria prevention for the longest period. Children who received malaria prevention during the trial achieved half a grade higher in school.
6 Clarke et al. (2008)	Two cross-sectional assessments pre and post-intervention. Tests of everyday attention for children (TEA-Ch), behaviour in class (teacher ratings of inattentive and hyperactive-impulsive behaviours), educational attainments (end of school term exam).	Significant improvements in TEA-Ch (counting sounds and code transmission) were seen in the IPT group compared with control group. No effect was shown for end of school term exam and inattentive behaviour.

IV. Estimation strategies and empirical issues in the estimation of education production functions: application to the case of malaria

A. The education Production function

Within the Human Capital Theory framework, economists have conceptualised learning as a production process (Ben-Porath, 1967)⁸. The economic conceptualisation of cognitive development and educational achievement through a production function is rather simplistic. In particular, one main limitation is that it does not account for psychological concepts related to the dynamic and interacting nature of the developmental trajectories of specific skills or knowledge. Another limitation is that it assumes a unitary household model. For our purposes and given the complexity of what we study (the impact of child health on education outcomes), some simplicity is gained by defining:

$$E_{ijt} = E(S_{ijt}, Q, C_{ijt}, H_{ijt}, I_{ijt}, M_{ijt}) \quad (1)$$

where E_{ijt} is the educational achievement for child i residing in household j at time t ; S is years of schooling until time t ; Q is a vector of school and teacher characteristics supposed to be exogenous and fixed; C is a vector of child characteristics at time t (including innate ability); H is a vector of household characteristics at time t ; I is a vector of educational inputs under the control of parents applied at any time up until time t such as time spent by brothers or sisters with the child that has a pedagogical value; M is the health history of the child, including his/her malaria history until time t . The production function emphasises the role of malaria, health and health dynamics in determining educational skills. We focus on primary education, which is considered in this thesis as the basis of formal education. All the variables included directly affects educational outcomes. Indirect effects are also possible, but we are mainly interested in the direct effects of malaria *per se* on school achievement. The effect of invariant innate ability on human capital accumulation is ambiguous and controversial: more innate ability could lead to less schooling or make learning easier and promote schooling (Cunha et al., 2006). Moreover, human capital has often been seen as the opposite of human abilities, which are supposed to be innate and age-invariant. However, this traditional debate has been renewed by Cunha & Heckman

⁸ For some of the more recent research on the effects of health on human capital accumulation, see Behrman and Rosenzweig (2004), Miguel & Kremer (2004) and Schultz (2002).

(2006). Indeed, abilities are not only the expression of genes. Environment interacts with genes to produce cognitive and noncognitive abilities or skills. The formation cycle involves different inputs at different stages of the life cycle to produce different outcomes. In this framework, skills or abilities are self-reinforcing and persist in time so that Equation (1) can be written in a recursive form (the so-called “added-value models”):

$$E_{ijt} = E(S_{ijt}, Q, C_{ijt}, H_{ijt}, I_{ijt}, M_{ijt}, E_{ijt-1}) \quad (2)$$

Nevertheless, the model defined in Equation (2) is more useful when one wants to assess determinants of the acquisition of skills and abilities at different stages of life and the dynamics of child investments (stage-specific or general investments). As we focus only on primary school-aged children, Equation (1) seems more appropriate to our subject. In our analysis, innate ability has to be seen merely as another skill.

Finding an empirical specification of (1) and (2) given data limitations (particularly missing data for inputs) has numerous challenges and requires restrictions on the original empirical structure, which will be discussed according to the given level of study in the following chapters. Empirical issues and study designs to estimate education production functions are numerous, however. One has to navigate the advantages and disadvantages of every approach presented in the next section.

B. Problems and solutions in assessing the effects of malaria on primary education

The discussion presented in this subsection is based on chapters of the Handbooks of Development Economics and the Handbooks of the Economics of Education applied to our subject.

1. Retrospective estimates from cross-sectional and Panel data

Retrospective data are observational settings where no attempt is made by the researchers to alter the behaviour of the communities, households or individuals from whom the data are collected (Glewwe & Miguel, 2008).

a) Cross-sectional data

Tables 4 and 5 presented results and methods for four studies (Fernando et al., 2003 a, 2003b, 2003c; Jukes et al., 2006) that have examined the effects of malaria with cross-sectional data since 2003. The identification strategy is based on variation

across schools, households or pupils. There is no estimation of the effect of malaria on education across countries. However, it is impossible to achieve data on all unobservables in (1) possibly correlated with both observable inputs and outcomes. Therefore, researchers have to identify sources of bias and either collect this information or find estimation strategies to control for these possible sources of bias such as fixed-effect models or instrumental variables. However, fixed-effect models are still imperfect. Unobservable and random shocks to health among children may lead to the unobserved reallocation of education resources across different children within a family (Glewwe & Jacoby, 1995). Instrumental variables (IVs) that affect child health but should have no causal impact on education outcomes after controlling for child health status raise the question of the validity of instruments and their correlation with the error term. Another main limitation of these cross-sectional studies is that they can only inform us as to the nature and extent of the burden at point in time, post-infection. Child development is a dynamic process and would be best investigated in longitudinal surveys.

b) Panel data

To our knowledge, there is no study that examines the effects of malaria on the performances of school-aged children using panel data strictly speaking (panel data collected on the same children for more than two periods). That is principally the reason why Chapter 4 uses this strategy to fill in this gap in the literature. The main advantages are that unobserved variables that do not change over time can be differenced out and need not be measured, and that variables measures of past events are much better than in cross-sectional surveys. The main problems are that panel data econometrics can still suffer from time-variant unobserved shocks, selection (on pupils included in the study) and attrition bias. The impact could be under- or over-estimated depending on the correlation of the omitted children or household characteristics with both the malaria indices and the educational outcomes. One possible solution is again to use IV estimations. A valid instrument is a variable that is: 1) correlated with the observed variables that are likely to have bias problems; 2) uncorrelated with the error term, i.e. uncorrelated with all unobserved variables and any measurement errors; 3) not already included as an explanatory variable (Wooldridge, 2002; 2003). The difficulty of finding valid instruments has led some economists to favour other identification strategies.

2. Natural experiments

Natural experiments do not estimate an education production function but can provide estimates of an impact of changes in M_{ijt} in equation (1). Moreover, the short-

versus long-term effects are expected to be different, as other variables in the model may be adjusted over different time horizons (Glewwe & Kremer, 2006). To our knowledge, there are only two studies that have applied natural experimental methods to estimate the effects of malaria on educational levels: Bleakley (2007) and Lucas (2005). These studies provide good additional evidence of the effects of malaria on human capital accumulation. The estimation and identification strategies of these studies are based on the follow-up of year-of-birth cohorts and national malaria eradication campaigns. The supposed exogenous implementation of the eradication program, the relatively rapid impact of the treatment campaigns and the well-defined treatment period are combined with cross-area heterogeneity to identify the effect. Lucas (2005) finds that the decline in malaria explains 57-90% of the increase in the years of female education in the malarious zone and 37-62% of the increase in female literacy. Bleakley (2007) finds that both in absolute terms and relative to the comparison group in non-malarious areas, cohorts born after eradication had higher incomes and literacy rates as adults than the preceding generation (with an increase of around five percentage points for literacy and around 0.1–0.6 years for schooling in Colombia, for instance). Lucas (2005) also argues that her findings contrast with Acemoglu & Johnson (2007)'s cross-country findings because the cohort level entails a more precise measurement than is available in a cross-country approach.

One limitation of these studies is that they used data from what Adrienne Lucas calls the « malaria periphery » defined as areas in which malaria transmission was primarily seasonal or epidemic before eradication. Countries included in both studies are: Colombia, United-States, Mexico, Brazil (Bleakley, 2007) and Paraguay, Sri Lanka, Trinidad and Tobago (Lucas, 2005). These countries are particular epidemiological settings and were classified as *P. vivax*-dominant areas before eradication campaigns. Therefore, the external validity of these results is limited to these areas. Another limitation is that the measures of the severity of malaria before eradication are often very crude so that it is difficult to quantify how areas with high infection rates benefited more from the eradication campaign than other areas. In the case of Bleakley, however, 2SLS estimates are used to correct for measurement errors. A third limitation is that they may fail to control for individual or household-specific-time-variant unobservable variables. Lastly, selection bias in the surveys used to construct the year-of-birth cohorts is still possible: birth year and region of residence at birth year are often based on declarations.

3. Randomised evaluations and ethical questions

Randomised evaluations of the effects of malaria treatments on educational attainment are also rather scarce. Like natural experiments, they do not estimate an education production function strictly speaking. This method, increasingly common in development economics since Esther Duflo's work, has the main advantage that all observed and unobserved characteristics of the treatment and control groups are uncorrelated with the treatment status if the random assignment is not violated in practice. They also limit measurement errors but have failed to address selection and attrition bias (Glewwe & Kremer, 2006).

Two studies provided in Tables 4 and 5 are excellent randomised clinical trials. Fernando et al. (2006) analyse the effect of chloroquine prophylaxis on the educational attainment of children, and Clarke et al. (2008) estimate the effect of intermittent preventive treatment (IPT) for malaria on education. Nevertheless, they do not identify precisely possible mechanisms. Clarke et al. (2008) provide strong evidence of a link between asymptomatic malaria and sustained attention and Fernando et al. (2006) show that during intervention malaria decreased by 55% in the treatment group as compared with the placebo group and that educational attainment was significantly associated with taking chloroquine prophylaxis and was significantly higher among children with better compliance with preventive treatment.

However, the randomized control trials may affect educational outcomes or may omit other confounding factors correlated with both parasitisation and school achievement in the post-intervention analysis. Indeed, human behaviour may change during intervention and adapt itself to it. Weaker students from the treatment may be less likely to drop out than those from the control group, leading to underestimated effects. Like natural experiments, these trials can only estimate *partial equilibrium effects*. Studies should also collect panel data before and after the intervention because this allows one to look at changes in the outcome variables over time. Another important question deals with randomised economic evaluations concerns ethics. Randomised control trials have to respect ethical rules and not violate regulations on human subjects research. For instance in the Clarke et al. (2008) study, 8 children died among the 6758 pupils (aged 5-18 years) with parental consent and enrolled in both placebo and control groups. Even if this rate is lower than the population mortality rate (around 1571 per 100,000 in 2004 according to the Burden of Disease tables - WHO), even if the protocol was approved by the ethics review board of Kenyatta National Hospital and even if the study was published in a scientific review that is vigilant with respect to ethical matters, the question is raised

of the medical surveillance of children included in large-scale randomised studies in developing countries.

C. Measurement issues

Measurement issues concerning health variables have been described in Strauss & Thomas (2008). This paper provides an instructive discussion of mortality, life expectancy and DALYs at the macroeconomic level; and anthropometry, self-assessed health, physical health and biomarkers at the microeconomic level. However the specific case of malaria and the difficulty of understanding and collecting malaria indicators are not analysed. Strauss & Thomas (1998) recommend the use of multiple health indicators simultaneously, wherever possible, to take into account the multidimensionality of health.

One of the main barriers to the control of malaria is that we are still not able to clearly determine the number of malaria cases and deaths worldwide:

- There are no direct counts of the number of malaria cases and deaths that occur each year.
- A very wide range of estimates has been made using a variety of definitions and consequently, there is a dramatic lack of precision.
- Drug resistance introduces a supplemental difficulty of correctly estimating the number of malaria cases and deaths.

Hence, there is a clear information gap, with vast uncertainties in this field of research. Without reliable data, estimating the economic effects of malaria is not an easy task. Different methodological and technical barriers impede us from measuring precisely the number of malaria cases (Audibert et al., 2003). The incidence and severity of *P. falciparum* malaria depends on various entomological, environmental and human factors. Possible combinations of factors are numerous. The clinical presentation of malaria varies with immune status, among other factors such as personal behaviour or genetic characteristics. Populations exposed to frequent infections acquire partial labile protective malarial immunity. The level of immunity affects not only mortality and the severity of malaria but also the number of uncomplicated malarial attacks. Therefore and paradoxically, it is more difficult to assess the real burden of the pathology in highly endemic areas where people are more exposed to mosquitoes infected bites since parasitisation is not directly and proportionally associated with apparent clinical symptoms. On the contrary, diagnosis is supposed to be easier in low endemic areas where symptoms are directly caused by infection in the absence of immunity. Nevertheless, malaria symptoms are

common to a wide variety of diseases so that it is not easy to make a correct diagnosis even in epidemic areas where malaria is not suspected as the first cause of illness.

However, we see different solutions to tackling this lack of data and this thesis is at the same time a reflection on how to tackle health data deficiencies in developing countries:

- First, it is always possible to use some proxies. Fever is a good candidate as a proxy of malaria. The number of fever cases (managed as malaria) is often used as a representative measure of the burden of the disease in a country. Death is considered relatively infrequent compared to illness. Moreover, a self-reported 14-day history of fever is a commonly used measure in standardised surveys of child health. DALYs or national reported cases of malaria from national monitoring and evaluation systems are also potential candidates. Acemoglu & Jonhson (2007) also use estimates of mortality by disease before the 1940s from the League of Nations and national public health sources. However national reporting systems are systematically different between countries. We will show that conclusions based on proxies of malaria have to be made prudently and can be misleading in some cases.
- Second, spatial statistical models and the use of GIS (Geographic Information Systems) have become fundamental tools for the epidemiology and control of malaria. One of the leaders in the field of macroeconomic research on malaria, Jeffrey Sachs, provides the percentage of the population living in areas of high malaria risk in a country via the Earth Institute website, calculating the figures using GIS software from a digitised WHO map (1997). The MARA (Mapping Malaria Risk in Africa) helps provide a geo-positioned series of malariometric data for Africa. The MAP (Malaria Atlas Project) assemble different data source on malaria risk, disease incidence and intervention coverage to create reliable maps of the prevalence and transmission intensity of malaria. Individual initiatives help to map malaria in specific countries using transmission models (Gemperli et al., 2005).
- Third, economists, epidemiologists and physicians can work together. It is feasible to look for the data where they actually are that is, in the field. The quality of these data on the severity and intensity of the disease would be, of course, much better (Audibert, 1986).

These points will be discussed in the following chapters, which will progress from macroeconomic to microeconomic levels.

Measurement issues concerning education variables have been the subject of more research. It is generally assumed that measurement errors in schooling are random

(Griliches, 1977). In many studies, human capital is captured by figures on completed years of schooling, which are a measure of education quantity more than education quality as defined earlier. In this thesis we will use qualitative indicators of human capital (QIHC) at different levels of study. The measures used will be discussed in detail in Chapters 2, 3 and 4.

V. Thesis Structure and hypothesis

The **first chapter** of this thesis proposes to rethink malaria economic analysis by combining economic epidemiology tools with the poverty trap literature. The endogeneity of malaria transmission with respect to households and individuals socioeconomic characteristics remains a particularly relevant and unresolved issue. We design a theoretical model of rational preventive behaviours in response to the disease, which includes endogenous externalities. Two important conclusions emerge from the model. First, agents increase their preventive behaviours when the degree of prevalence of malaria in a society is more severe. This result is consistent with the literature on “prevalence-elastic behaviour”. Second, we show that the “Malaria trap”, defined as any self-reinforcing mechanism that causes malaria to persist, is a plausible explanation for the “vicious circle, which makes the poor malarious and the malarious poor” (Watts, 1999). This chapter contributes to the existing economic epidemiology literature by adding new foundations for understanding the dynamic of a disease. Even though it is very difficult to empirically test the validity of this theory for malaria mainly because of the lack of long-term longitudinal surveys on the disease, the theoretical results of this chapter question the nature of the possible self-reinforcing mechanisms that could lead to a disease trap. One main implication of this model concerns the endogeneity of malaria. If a community is trapped in a high and stable level of malaria prevalence, malaria may be considered exogenous in this particular community. Moreover, the disease could generate some socioeconomic barriers to its control. Disease traps could also interact with other mechanisms such as poverty traps. Hence, understanding the negative effects of malaria on economic variables is crucial to improve malaria control programs. On this basis, the following three chapters focus on the causal effects of malaria on education at different levels of study. Macroeconomic and microeconomic studies are very complementary in the

study of the economic burden of malaria. The scope of these chapters has been limited to only observing the impact of malaria on school performance and very little on the reverse; that is how education affects malaria. The effect of mother and household education on child health has already been the subject of careful research.

The **second chapter** explores the link between malaria and human capital accumulation in malarious countries in comparison with those of non-malarious countries. If malaria has such a huge impact on childhood morbidity, it would be surprising not to encounter the impact of malaria on education at the macroeconomic stage. Cross-country and panel regression analysis show that the relation between school results (measured by repetition and completion rates) and the malaria index is strong. The potential endogenous nature of malaria is taken into account using the Kiszewski et al. (2004) instrument variable. These estimates capture both the direct and the indirect effect of malaria in addition to general equilibrium effects.

The **third chapter** estimates the relative importance for child school performance (indicated by primary repetition) of fever, malaria and some social determinants. It uses individual, household and cluster surveys from the Demographic and Health Surveys conducted in Mali in 2001 and 2006 (MDHS). It also provides a discussion of the use of fever as an indicator of malaria in large cross-sectional surveys by comparing the 2001 and 2006 MDHS, which were realized during two different transmission seasons (dry and rainy seasons). The Geographic Information System and DHS Global Positioning System dataset were used to extract age-specific malariological measures from reliable maps of the prevalence and transmission intensity of malaria. We show that fever is not a reliable proxy for malaria and we recommend the use of spatial measures of malaria prevalence or the use of reliable measures of malaria parasitaemia in addition to fever in future research. Cross-sectional regression analysis suggests that greater prevalence of malaria in a community is linked to higher primary repetition rates, but confirmatory studies are needed. We control potential selectivity bias by restricting the sample to primary school pupils. This study also incorporates the endogenous nature of child health into the analysis.

The **fourth chapter** assesses the role of malaria on primary education and especially on school achievements in Donéguébougou, a small village located in a malaria-endemic area near Bamako, Mali. We estimate an education production function of educational achievement were the index of malaria is refined by

distinguishing different forms of malaria. Potential pathways of the effects of malaria on educational achievement are described with the help of the medical literature. As we said, finding an empirical specification of the education function given data limitations (particularly missing data for inputs) has numerous challenges. In order to tackle these difficulties and compute estimations with an appropriate dataset, a study protocol was designed specifically in partnership with the Malaria Research and Training Centre, Mali. Field data were collected between November 2007 and June 2008 on 227 schoolchildren living in Donéguébougou. The probable endogeneity of malaria indices due to omitted variables is dealt with using panel data. We test the direct effects of the presence of *falciparum* malaria parasites *per se* on school achievement and cognitive performance. We find suggestive evidence that the effect is causal. We conclude that it is time to consider malaria as one of the most common chronic mental health problems among school children in endemic areas with large effects on children's human capital accumulation and education.

**CHAPTER I: ECONOMIC EPIDEMIOLOGY OF MALARIA
AND PREVENTIVE BEHAVIOURS: IS THERE A MALARIA
TRAP?**

Insecticide-treated nets (ITNs) are seen as an essential malaria control technique that provides personal and community protection. Studies conducted by the World Health Organization (WHO) in Zambia, Ghana, Ethiopia, Rwanda, and Tanzania confirm that mosquito nets treated with insecticide can reduce childhood mortality by as much as one-third in malarious-endemic parts of Africa. Insecticide-treated nets are comparable to measles vaccination in their cost-effectiveness in preventing deaths and morbidity. Recent studies at the national level have tried to assess knowledge and beliefs about malaria and nets; access, ownership, treatment, and use of mosquito nets; consumer preferences regarding mosquito nets; and usage and attitudes regarding other mosquito control products. Nevertheless, one of the major public health challenges remains to scale up ITN coverage and only a small number of studies have attempted to describe patterns of use and possession of ITN. Moreover the mechanisms underlying the personal protection are still unclear and the conclusions are often contradictory. For instance, in studies on a national scale, the nets provide an exceptionally high level of personal rather than community protection, but these conclusions are based on a sample of communities with less than 50% of villagers sleeping under treated nets (Thomson et al., 1995; Gosoni et al. 2008).

This chapter provides a theoretical model of rational preventive behaviours in response to the disease. This chapter proceeds as follows. Section 1 reviews briefly some arguments of the economic epidemiology literature and determinants of households' demand for and use of ITNs. In section 2, we present the hypothesis and analytical framework and the population dynamic of disease in an original model of economic epidemiology of malaria. Section 3 discusses the policy implications of the model. Section 4 concludes.

I. Previous Literature: economic epidemiology of malaria

On the one hand, even though infectious diseases (tuberculosis, malaria, acute respiratory infection and HIV) represent the primary cause of mortality worldwide, their dynamics are not yet well understood by economists (Philipson, 2000). On the

other hand, while the idea of multiple steady-state equilibria leading to poverty cycle is not a recent one, especially in the development economics literature, the focus of the recent research has been on exploring mechanisms that lead to such multiple equilibria. There are many mechanisms that can lead to poverty traps, in particular human capital accumulation in a broad sense and associated threshold externalities. Comparing emerging countries with non-emerging countries, Berthélemy (2006, 2008) shows that initial education and health policies have played a critical role in take-off; he suggests that there could be “several multiple equilibria”. The relationship between health and development is interpreted as resulting from a bidirectional interaction between these two variables, which may lead a country into a poverty trap or, alternatively, into a virtuous circle of economic take-off. In this field of research, economic epidemiologic tools have not been exploited yet. They could provide a useful explanation of some probable pathways that explain this bidirectional interaction.

Gergovitz & Hammer (2003) provide a critical discussion of how rational choices help us to understand how people respond to infectious diseases: people maximise their well-being by choosing levels of prevention and therapy subject to the constraints they face. This consideration is obviously irrelevant if individuals lack choices, as suggested by Sen, but for infectious diseases such as malaria, one may consider that people can almost always choose among a range of preventive or curative actions. This is all the more true if we consider traditional healers, traditional plant-based prophylactics for malaria or informal access to healthcare and prevention. Even if their safety and efficacy are not well known, there is still a widespread use of traditional herbal medicines in rural areas. Of course, this way of looking at how people make decisions that affect their health is rather simplistic and makes strong assumptions on the nature of choices. Indeed, a person's decision affecting health may be determined by objectives and constraints but also by beliefs. However, this approach helps understanding what people do and what difficulties government interventions could face. Actually, Gergovitz & Hammer (2003) also discuss the implications of rational choices for public policy. These models are relevant for public policy because they take externalities into account: private choices do not produce socially optimal outcomes. If individuals' decisions are not compensated, coordinated or prompted to internalize the externalities by subsidies, taxes or nonmarket incentives, the decision making will probably not be socially optimal.

Until recently, the agent's prevention behaviour was not incorporated into epidemiological models. Private rational choices have been formalised by Geoffard & Philipson (1996) and Kremer (1996). Geoffard & Philipson (1996) focus on HIV/AIDS

and stress the possibility of high prevalence-elastic behaviours and proposes a method for estimating risk preferences that can be tested empirically with epidemiological cohort surveys and survival analysis. They emphasize the difference between standard epidemiological and economic models. As demand for protection is an increasing function of rising prevalence, the hazard rate from susceptibility into infection will be a decreasing function of the prevalence of the disease in economic models because remaining susceptible individuals face a larger risk and increase protection. Conversely, it will be an increasing function of the disease in standard epidemiological models. Models without behavioural response generate a unique stable steady state. Models that explicitly assume that behaviour is a function of lagged prevalence (as Geoffard & Philipson, 1996) will generate cycles in prevalence but not multiple steady states.

Taking into account the composition of the pool of available partners, Kremer (1996) analyses the feedbacks generated by the proportion of highly sexually active people changing their activity on the spread of the epidemic. One particularly interesting feature of this model is that individuals can become fatalistic in high-prevalence regions because the marginal probability of being infected for high-activity people changes only slightly or even decreases when prevalence increases. Indeed, in this model, marginal utility from an additional partner is a decreasing function of the number of partners. Marginal probability of infection is also a decreasing function of the number of partners but above a certain threshold of partners (high-activity people) this probability will be lower with high prevalence than with low prevalence. Under this threshold (low-activity people), the marginal probability of infection will be higher with high prevalence than with low prevalence. Therefore, in homogenous populations, low-activity people will tend to reduce their activity when prevalence increases, whereas for high-activity people, the optimal number of partners will tend to increase, a sort of “*rational fatalism*” (Gersovitz & Hammer, 2003). In heterogenous populations even if high-activity people reduce their activity in response to increased prevalence, if this decrease in activity is smaller in proportion than the decrease in low-activity people, the composition of the pool of available partnerships will worsen and will generate a positive feedback that may lead to multiple equilibria. For our purposes, this study is certainly one of the most interesting as Kremer illustrates how positive externalities in the disease dynamic can lead to multiple equilibria.

Auld (2003) integrates agents’ rational anticipations and shows that pessimistic expectations over the future of the epidemic induce more current risky behaviour. This results means that expectations over future interventions can complicate intervention analysis. All of these papers show how economic and biological

epidemiology models differ in terms of their predictions regarding the dynamic of the disease as for public policies for controlling the disease. However, this literature has mainly focused on the spread of HIV-Aids. Moreover, analytical treatments are often too complicated to understand how a disease can be a supplemental barrier in the development process of some countries or communities.

To our knowledge, only a few studies combine epidemiologic models for malaria with the consideration of economic behaviours (Gergovitz & Hammer, 2004; Momota et al., 2005; Raut, 2004; Gollin & Zimmermann, 2007). Using an overlapping-generation model, Momota et al. (2005) show that the agent's prevalence-elastic behavior, considered explicitly, may cause the cyclicity of the spread of the disease and that the effect of foreign aid on the welfare of agents could be ambiguous. Raut (2004) is one of the first attempts that seeks to model the dynamics of the disease incorporating private human behaviours and its effects on aggregate income growth through human and physical capital accumulation. However, his model includes too many concepts and tries to tackle too many problems at the same time. This lacks of clarity makes it difficult to interpret the results. Gollin & Zimmerman (2007) use a dynamic general equilibrium framework that incorporates epidemiological features and show that it is possible for an economy to display multiple steady states because of disease dynamics. They introduce the possibility of a "malaria trap" that is defined as a vicious circle between the disease and poverty that makes disease prevention unaffordable. They suggest that income per capita could be reduced by about half for countries trapped in such low-income level equilibrium. However, the concept of "malaria trap" in this paper is not very clear and could be rather be assimilated to a poverty trap. Moreover, the model cannot be considered as an epidemiological model strictly speaking. Gergovitz & Hammer (2004) provide a framework for the economic analysis of vector-borne diseases and associated externalities from a social planner perspective.

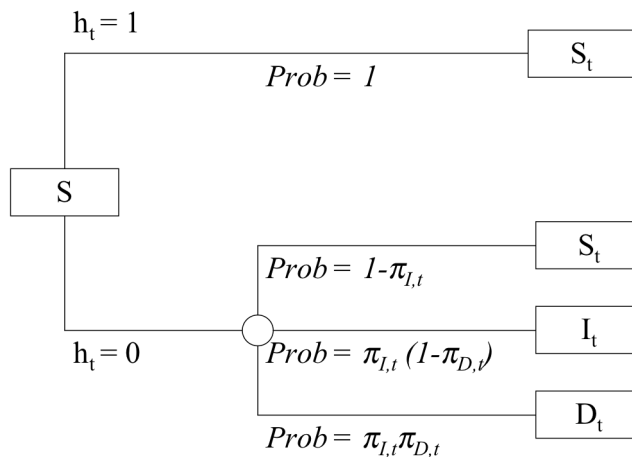
In this chapter, we propose an economic model of the epidemiology of malaria to analyse the external effects of individual decisions and study other possible mechanisms. The main epidemiological aspects of the model are similar to those presented by Geoffard & Philipson (1996) but adapted to vectorial transmission. Our results differ clearly from their results, as their model generates cycles in prevalence but not multiple equilibria.

II. Model

A. Hypothesis and analytical framework

Consider a simple model in which agents live only one period. The economy begins its process at time 0. Agents adopt a certain behaviour at the beginning of each period t : use of an insecticide treated net ($h_t = 1$) or exposure to malaria risk ($h_t = 0$). Malaria is a parasitic disease transmitted from person to person through *Anopheles* vectors (see Appendix 1). We suppose that the only means by which an agent can prevent himself from parasitic infection is to sleep under an insecticide treated net. The use of an ITN is supposed to be completely efficient in protecting from malaria infection. Formally, at any time t , the health status of the representative agent, $\sigma_t(h_t)$ can take three values: susceptible ($\sigma_t(h_t)=S$), infected ($\sigma_t(h_t)=I$), or dead ($\sigma_t(h_t)=D$). This is in keeping with the main simplified characteristics of *falciparum* malaria where most of deaths occur sudainly after cerebral malaria during early childhood. Those who survive to this stage acquire partial labile immunity against the disease (Figure I.1).

FIGURE I.1: Decision tree (source: author)



Following Momota et al. (2005), we designate M_t as the malaria level of the population at time t . As, we consider that dead agents have the time to infect other agents before dying:

$$M_t = I_t + D_t \tag{1}$$

A higher level of M_t means that the degree of the spread of malaria is more serious. We also express the aggregate level of preventive behaviours per unit of total

population at time t as H_t , and we define the opportunity cost θ of using an insecticide-treated net as a function of W :

$$H_t = \int_0^1 h_t f(W) dW \quad (2)$$

$$\theta = \theta(W)$$

where $W \in [0,1]$ is a scalar of individual characteristics; f is the probability density function of W . We first suppose that the distribution of W is uniform between $[0,1]$. We also suppose that θ is monotone and $\theta'(W) > 0$.

We denote the size of the total population at any time t by N_t . To keep the demographics of the population at a manageable level, we also assume that whenever a person dies, either naturally or from malaria, the same number of newborn agents are added to the population. Newborn agents are assumed to be identical and susceptible.

The lifetime expected utility maximised by a representative agent at any period t is a function of one's health and preventive behaviour at the end of t :

$$\max_{h_t} E[U(\sigma_t(h_t) - \theta(W)h_t)] \quad (3)$$

Subject to:

$$\begin{aligned} P(\sigma_t(h_t) = S_t | h_t = 1) &= 1 \\ P(\sigma_t(h_t) = S_t | h_t = 0) &= 1 - \pi_{I,t} \\ P(\sigma_t(h_t) = I_t | h_t = 0) &= \pi_{I,t}(1 - \pi_{D,t}) \\ P(\sigma_t(h_t) = D_t | h_t = 0) &= \pi_{I,t}\pi_{D,t} \end{aligned}$$

We suppose that $\pi_{D,t}$ is exogenous and constant and that the probability of infection $\pi_{I,t}$ conditional on exposure at any time t is a continuous function of the aggregate level of preventive behaviours and p_t , the probability of being infected conditional on contact with other infected people through mosquitoes:

$$\begin{aligned} \pi_{I,t} &= \pi(H_t, p_t) \\ \text{Where} & \\ p_t &= p(H_t, M_{t-1}) \end{aligned} \quad (4)$$

The use of an ITN affects 1) the probability that an infected person infects an uninfected vector, 2) the probability that an infected vector infects an uninfected and unprotected person and 3) the ratio of mosquitoes to people.

Hence, there exist two routes through which the aggregate use of ITNs affects the probability of being infected conditional on exposure. One is the route in which the use of ITN directly influences the probability of being infected conditional on exposure through two main channels. First, the mosquito population is reduced through the effect of insecticide so that contact with other infected people through mosquitoes will be reduced. Second, among the surviving mosquitoes, the proportion of infectious mosquitoes is reduced because man-vector contact is reduced so that the entomological inoculation rate (number of infective bites per person per period) decreases and leads to a change in p_t . Because mosquito dynamics operate on a much faster time-scale than human dynamics, it is plausible to assume that the effect of the use of ITN on mosquito population is instantaneous. The other route is an indirect route. The malaria level in the population at time $t-1$ also influences p_t . Indeed, we consider that malaria is transmitted from one generation to another through mosquitoes as newborn agents are assumed to be identical and susceptible. Hence, p_t is supposed to be a continuous function, p , of H_t and M_{t-1} . Finally this change in p_t influences the value of $\pi_{I,t}$.

We assume that π is twice continuously differentiable and as regards its derivatives, $\frac{\partial \pi}{\partial H_t} < 0$, $\frac{\partial \pi}{\partial p_t} > 0$, and taking p_t as given, π is convex i.e. $\frac{\partial^2 \pi}{\partial H_t^2} \geq 0$.

According to the discussion above, we express p_t as a function with respect to H_t and $\pi_{I,t-1}$ and we assume that $\frac{\partial p_t}{\partial H_t} < 0$ and $\frac{\partial p_t}{\partial M_{t-1}} > 0$. Furthermore, we impose the

following boundary condition: $\lim_{H_t \rightarrow N_t} \frac{\partial \pi(H_t, 0)}{\partial H_t} = 0$.

As in Geoffard & Philipson (1996), protection will occur when the instantaneous opportunity cost of using an insecticide-treated net (the loss in current utility from protection) is below the expected cost (the expected utility loss of risking infection through exposure):

$$E[U(\sigma_t(1)) - U(\sigma_t(0))] \geq \theta(W) \tag{5}$$

i.e.

$$v(s) - (1 - \pi_{I,t})v(s) - \pi_{I,t}(1 - \pi_{D,t})v(I) - \pi_{I,t}\pi_{D,t}v(D) \geq \theta(W)$$

where $v(\sigma_t(h_t))$ denotes the value of a health state and: $v(S) > v(I) > v(D), \forall h_t = (0, 1)$.

Hence, protection occurs if and only if:

$$\pi_{I,t} \geq \frac{\theta(W)}{\left[(v(s) - v(I)) + \pi_{D,t} (v(I) - v(D)) \right]} = C(W) \quad (6)$$

In other words, there is a threshold probability of infection above that an agent engages in protection. For notational convenience let us denote this threshold as $C(W)$. As θ is monotone and $\theta'(W) > 0$, note that C is strictly monotone and $C'(W) > 0$. Therefore, the inverse function of C , C^{-1} , exists and is an increasing function of $\pi_{I,t}$.

Hence, we have:

$$\begin{cases} \text{if } w \leq C^{-1}(\pi_{I,t}), h_t = 1 \\ \text{if } w > C^{-1}(\pi_{I,t}), h_t = 0 \end{cases} \quad (7)$$

B. The Population Dynamics of Disease

1. Determining the steady state equilibrium when M_{t-1} is exogenous

In this section, we examine how the decisions of individuals affect the probability of being infected conditional on exposure. Indeed an agent health decision has external effects in the present model. A dynamical path is obtained from (4) and (7). At the aggregate level, as w is uniformly distributed between $[0,1]$, we can also write:

$$H_t = \int_0^1 h_t f(W) dW = \int_0^1 h_t dW = \int_0^w h_t dW + \int_w^1 h_t dW \quad (8)$$

The equilibrium condition of this system is expressed as $W = C^{-1}(\pi_{I,t})$ and we denote the level of H_t at equilibrium by H^C . As a result, it is easy to show that the steady state is reached when:

$$H_t = \int_0^{C^{-1}(\pi_{I,t})} h_t dW + \int_{C^{-1}(\pi_{I,t})}^1 h_t dW = C^{-1}(\pi_{I,t}) \quad (9)$$

i.e.

$$\pi_{I,t} = C(H_t)$$

To begin, fixing the level of p_t in $\pi(H_t, p_t)$ at 0 and 1 and considering that the level of M_{t-1} is given, let us define $\pi(H_t, 0)$ and $\pi(H_t, 1)$ as $\underline{\xi}(H_t)$ and $\bar{\xi}(H_t)$. We can see that these two curves construct upper and lower boundaries, respectively of

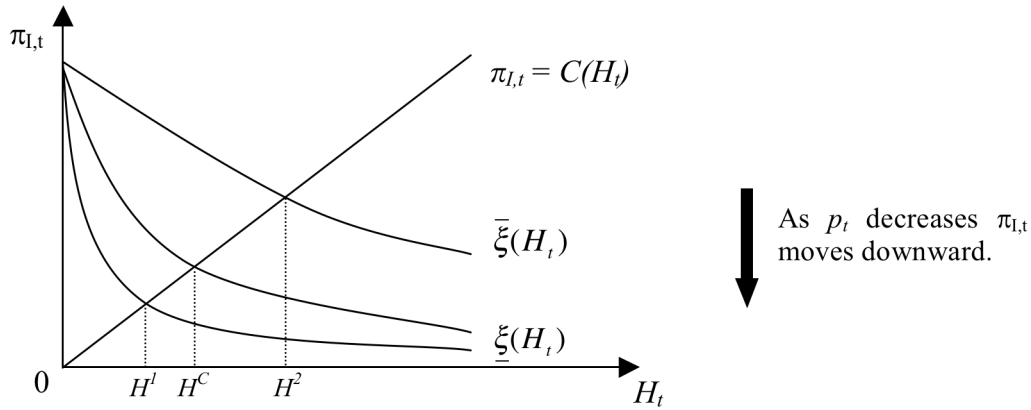
$\pi(H_t, p_t) = \phi(H_t)$. Since $\underline{\xi}(H) \leq \phi(H)$ holds, the phase diagram of $\phi(H_t)$ lies between $\underline{\xi}(H_t)$ and $\bar{\xi}(H_t)$.

With regard to the existence and stability of the steady state equilibrium, we state the following proposition:

Proposition 1.

- (i) If $\lim_{H_t \rightarrow N_t} p(H_t) = 0$ then there exists at least one steady state equilibrium.
- (ii) Under the above hypothesis, the steady state equilibrium exists uniquely.
- (iii) The equilibrium is stable, and in the neighbourhood of the steady state, equilibrium path is monotonic.

FIGURE I.2: Equilibrium path (source: author)



Proof. (i) To prove the existence of a steady state equilibrium, let us show that there exists an $H > 0$ that satisfies:

$$C(H) = \phi(H)$$

Showing that (1)-(3) hold true, we can confirm the existence of the non-trivial steady state equilibrium:

- (1) $\phi(H_t)$ is continuous.
- (2) There exists an H , which satisfies $\frac{\phi(H_t)}{C(H_t)} \leq 1$.
- (3) $\lim_{H \rightarrow 0} \frac{\phi(H_t)}{C(H_t)} > 1$ holds.

- To begin, let us examine that (1) holds. Since $\phi(H) = \pi(H, p(H))$ holds by definition and because $\pi(H_t, p_t)$ and $p(H_t)$ are assumed to be continuous, it can be confirmed that (1) holds trivially.

- Next, we prove (2). Recall that C is strictly monotone; $C(H) \geq 0$ and $C'(H) > 0$.

Hence, since $\phi(H) \leq \bar{\xi}(H)$ holds, $\frac{\phi(H)}{C(H)} \leq \frac{\bar{\xi}(H)}{C(H)}$ holds as well. When

$\lim_{H_t \rightarrow N_t} p(H_t) = 0$ holds, $\lim_{H \rightarrow N_t} \frac{\partial \pi(H, p)}{\partial H} = 0$ also holds and thus, $\lim_{H \rightarrow N_t} \bar{\xi}'(H) = 0$

holds. As the use of an ITN is supposed to be completely efficient to protect from malaria infection, $\lim_{H \rightarrow N_t} \partial \pi(H, p) = 0$. Therefore, noting that $\frac{\partial \pi}{\partial H_t} < 0$ and $\frac{\partial \pi}{\partial p_t} > 0$, it

can be confirmed that for a sufficiently large value of H , $\frac{\bar{\xi}(H)}{C(H)} \leq 1$ holds, and so is

$$\frac{\phi(H)}{C(H)}.$$

- Finally, let us prove (3). $\pi_{1,t}$ is the probability of infection so that $\lim_{H_t \rightarrow 0} \pi(H_t, p_t) = 1$.

As $\lim_{H_t \rightarrow 0} C(H_t) < 1$, then it can be confirmed that (3) holds.

(ii) Next, let us prove the uniqueness of the non-trivial steady state equilibrium by showing that

$$(4) \frac{\phi(H)}{C(H)} \text{ is a decreasing function with respect to } H.$$

As $\frac{\partial p}{\partial H} < 0$, C is strictly monotone, $C(W) \geq 0$ and $C'(W) > 0$, we can immediately

obtain that $C(H)\phi'(H) - \phi(H)C'(H) = \frac{\partial \pi}{\partial p}(H, p) \left[C(H)\frac{\partial p}{\partial H} - C'(H) \right] \leq 0$ and thus, (4) is

proven.

(iii) The stability of the equilibrium and monotony of the equilibrium path come

directly from $\frac{\partial^2 \pi}{\partial H_t^2} \geq 0$, $\frac{\partial \pi}{\partial H_t} < 0$ and $\frac{\partial p_t}{\partial H_t} < 0$.

Corollary. The convergence speed toward the steady state equilibrium depends on the level of p_t (the probability to be infected conditional on the contact with other infected people through mosquitoes), other things being equal.

2. Determining the population dynamics of disease when M_{t-1} is endogenous

a) The “prevalence-elastic behaviour”

In the last subsection, we have shown that if M_{t-1} is exogenous, there exists one stable steady state equilibrium, H^C . Proposition 2 presents an important result of economic epidemiology. It shows that agents increase their preventive behaviour when the degree of prevalence of malaria in a society is severer. This result is consistent with the literature on “prevalence-elastic behaviour”. Moreover, as ψ is monotone and $\psi'(M_{t-1}) > 0$, therefore, the inverse function of ψ , ψ^{-1} , exists and is an increasing function of H_t .

Proposition 2. $\psi'(M_{t-1}) > 0$, i.e. H_t is an increasing function of M_{t-1} .

Proof. The malaria level evolves according to:

$$\begin{cases} \text{The initial level of malaria, } M_0 \\ M_t = (N_t - H_t)\pi(H_t, p(H_t, M_{t-1})) \\ \pi(H_t, p(H_t, M_{t-1})) = C(H_t) \end{cases} \quad (10)$$

The equilibrium of this system is expressed as $M_t = M_{t-1}$. As H_t depends on M_{t-1} , let us suppose that there exists (H_t^C, M_{t-1}^C) such that:

$$\pi(H_t^C, p(H_t^C, M_{t-1}^C)) = C(H_t^C) \quad \text{and} \quad \frac{\partial \pi}{\partial H_t}(H_t^C, p(H_t^C, M_{t-1}^C)) \neq 0 \quad (11)$$

Then, applying the implicit function theorem, there exists an implicit function, ψ , $H_t = \psi(M_{t-1})$, such that:

$$\begin{aligned} (a) \quad & \pi(\psi(M_{t-1}), p(\psi(M_{t-1}), M_{t-1})) = C(H_t) \\ (b) \quad & H_t^C = \psi(M_{t-1}^C) \\ (c) \quad & \psi'(M_{t-1}^C) = -\frac{\frac{\partial \pi}{\partial M_{t-1}}(H_t^C, M_{t-1}^C)}{\frac{\partial \pi}{\partial H_t}(H_t^C, M_{t-1}^C)} \end{aligned} \quad (12)$$

Generalising this result from (10), it can also be shown that:

$$\psi' = \frac{\frac{\partial \pi}{\partial p} \frac{\partial p}{\partial M_{t-1}}}{C' - \frac{\partial \pi}{\partial H} - \frac{\partial \pi}{\partial p} \frac{\partial p}{\partial H}} \quad (13)$$

By definition $C'(W) > 0$, $\frac{\partial \pi}{\partial H} < 0$, $\frac{\partial \pi}{\partial p} > 0$, $\frac{\partial p}{\partial H} < 0$, $\frac{\partial p}{\partial M_{t-1}} > 0$.

Therefore, $\psi'(M_{t-1}) > 0$ holds.

b) Disease dynamic when θ is supposed to be linear

From (10), we have:

$$\begin{cases} M_t = (N_t - \psi)C(\psi) \\ \frac{\partial M_t}{\partial M_{t-1}} = -\psi' C(\psi) + (N - \psi)C' \psi' \\ \quad = \psi' [C'(N - \psi) - C(\psi)] \\ \frac{\partial^2 M_t}{\partial M_{t-1}^2} = \psi'' [C'(N - \psi) - C(\psi)] + \psi' [C'' \psi' (N - \psi) - 2C' \psi'] \end{cases} \quad (14)$$

As an example, suppose that θ is linear so that the functional form of C is also linear and:

$$C = \beta H \quad \text{and} \quad C' = \beta, \text{ with } \beta > 0 \quad (H1)$$

Then we have:

$$\begin{aligned} \frac{\partial M_t}{\partial M_{t-1}} &= \psi' \beta (N - 2\psi) \\ \text{Hence,} & \\ \frac{\partial M_t}{\partial M_{t-1}} &> 0 \quad \text{if } \psi(M_{t-1}) < N/2 \quad \text{i.e. } M_{t-1} < \psi^{-1}(N/2) \end{aligned} \quad (15)$$

Depending on the specification of the relationship between M_t and M_{t-1} different dynamic paths are possible. Some of them will lead to malaria traps.

Definition. A malaria trap is defined as any self-reinforcing mechanism that causes malaria to persist.

Under H1, many situations are possible (Figures I.4). Whatever the specification of the relationship between M_t and M_{t-1} is, we state the following proposition:

Proposition 3.

(i) Under H1, a non-trivial steady state equilibrium exists if and only if

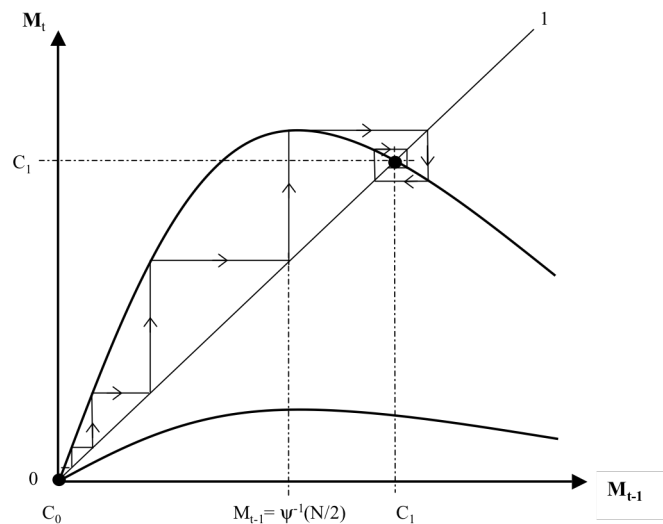
$$\lim_{M_t \rightarrow 0} \frac{\partial M_t}{\partial M_{t-1}} > 1.$$

(ii) The equilibrium is stable, and in the neighbourhood of the steady state, the

convergence path is cyclical if and only if $\frac{\partial M_t}{\partial M_{t-1}} > -1$.

Proof. We provide here an intuition with support for Figure I.3. Figure I.3 shows that when $\lim_{M_t \rightarrow 0} \frac{\partial M_t}{\partial M_{t-1}} > 1$, we have at least one non-trivial high stable steady state equilibrium that corresponds to a high level of malaria transmission. However H1 is not a sufficient condition to allow for the presence of multiple equilibria. Moreover, Figure I.3 depicts a rather pessimistic scenario.

FIGURE I.3: Disease Dynamics – Case 1: Phase Diagram (*source: author*)



Therefore, we impose the following boundary condition:

$$\lim_{M_t \rightarrow 0} \frac{\partial p_t}{\partial M_t} = 0 \tag{H2}$$

Proposition 4. H1 and H2 are sufficient conditions for the possibility of multiple equilibria.

Proof. When $\lim_{M_{t-1} \rightarrow 0} \frac{\partial p_t}{\partial M_{t-1}} = 0$ holds, $\lim_{M_{t-1} \rightarrow 0} \psi' = 0$ and thus $\lim_{M_{t-1} \rightarrow 0} \frac{\partial M_t}{\partial M_{t-1}} = 0$.

As $\frac{\partial M_t}{\partial M_{t-1}} > 0$ when $M_t > \psi^{-1}(N/2)$, $\exists M_{t-1} > 0$ such as $\frac{\partial M_t}{\partial M_{t-1}} = 0$.

H2 is justified by the epidemiologic literature on the relationship between seropositivity, prevalence, incidence, the entomological inoculation rate (EIR), the mosquitoes reproduction rate and vectorial capacity. Epidemiologic patterns of malaria infection are mainly governed by environmental parameters that regulate vector populations of *Anopheles* mosquitoes. The causal effects of EIR on malaria prevalence have been widely analysed and the majority of sites fit the concept of a linear relationship between malaria prevalence and the logarithm of the annual EIR. Figure I.4 provides the relationship between Annual Entomological Inoculation Rate (EIR) and the prevalence of *Plasmodium falciparum* malaria based on data obtained from 31 sites throughout Africa plotted on A, linear and B, logarithmic areas (Beier et al., 2004). Outliers excluded from the fit are in black. However, the bidirectional relationship between EIR and prevalence has not been the subject of careful research perhaps because of the limited number of successful malaria control programs. One study made in Papua New Guinea (Graves et al., 1987) suggests that the entomological inoculation rate decreased in intervention villages after net impregnation despite the increases in the mosquito population and while EIR increased in control villages. Mboera and Magesa (2001) also show that the fall and rise in the mosquito infectivity in Muheza, north-eastern Tanzania between the mid-1930s and the mid-1970s is attributed to the widespread use of antimalarial drugs, which initially tended to reduce the infectivity of patients relative to mosquitoes, and the subsequent development of resistance to these drugs in the malarial parasites. Hence, the epidemiologic literature seems to confirm the validity of H2 i.e.

$\lim_{M_t \rightarrow 0} \frac{\partial p_t}{\partial M_t} = 0$ and therefore, Figure I.5 seems to be a more realistic scenario.

Indeed, we can see from Figure I.5 that the existence of malaria traps is plausible. The relative position of the curve with respect to the 45° line will determine the number of equilibria, possibly characterising different epidemiological settings or other initial conditions.

FIGURE I.4: Relationship between Annual Entomological Inoculation Rate (EIR) and the prevalence of *P. falciparum* malaria (source: Beier et al., 2004).

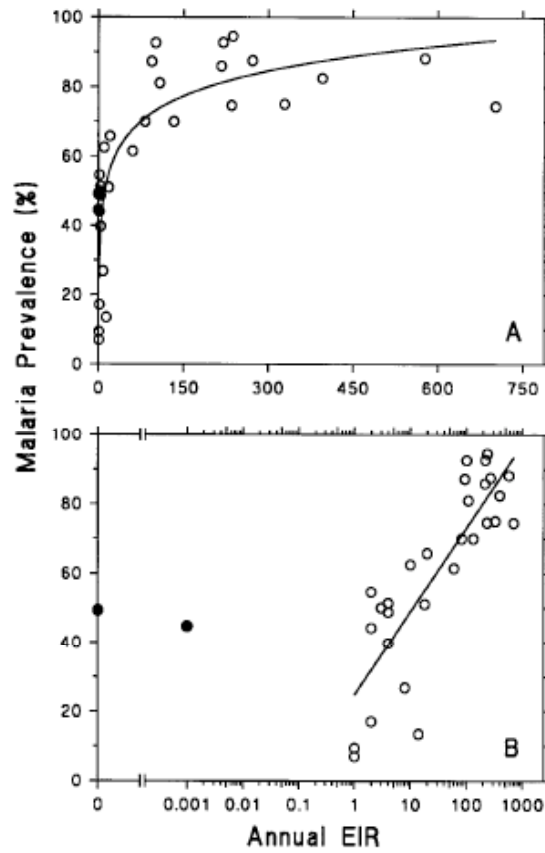
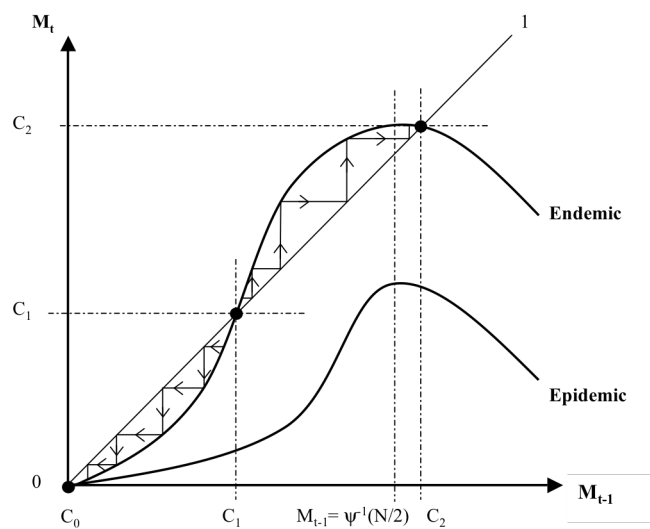


FIGURE I.5: Disease Dynamics – Case 2: Phase Diagram (source: author)



III. Policy implication

A. How to break the malaria trap?

How to break the “malaria trap”? This question has already and obviously been asked. However, in most of the research on malaria, the term “cycle” refers to the “biologic life cycle” of malaria (see Appendix 1). It may be useful to tackle the problem from a human behaviour perspective.

One main consequence of the existence of “Malaria traps” for public health is that it is not possible to get out off the disease trap without exogenous public policy interventions or exogenous change in climate or epidemiologic conditions (“natural” disappearance of malaria in certain regions of Europe for instance). Public policies will allow one community to jump out off the disease trap, (characterised by a high and stable level of disease prevalence) to reach a lower and still stable equilibrium (characterised by a lower level of disease prevalence) by modifying the initial conditions.

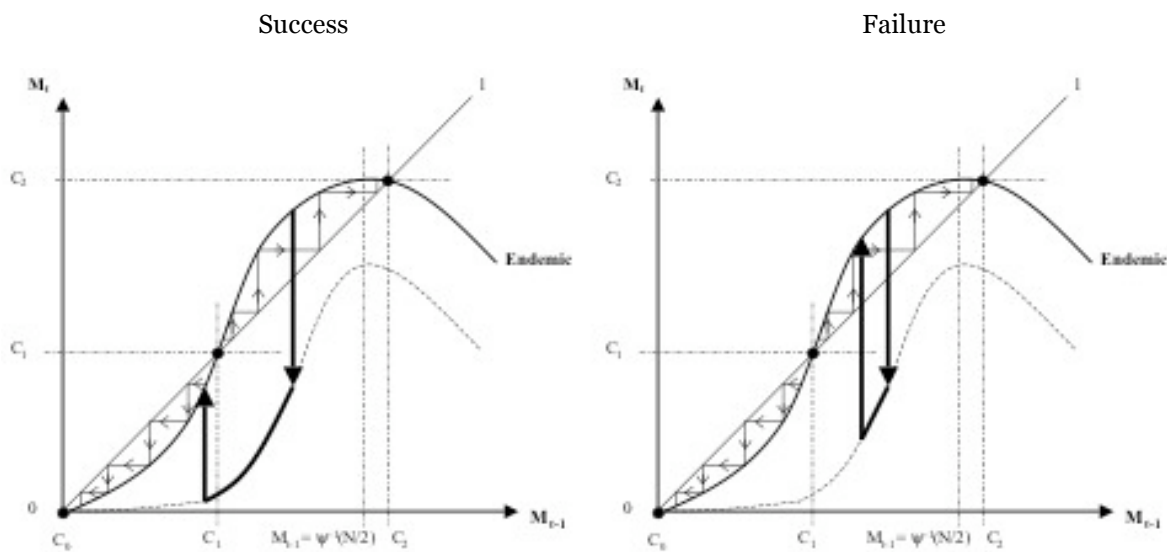
What are the effects of a transitory shock? In order to understand the complex moving of the curve that links M_t to M_{t-1} implied by a transitory shock we will try to decompose these effects into two shifts: a vertical or/and a horizontal shift. A control or an eradication program is certainly a combination of both vertical and horizontal shifts.

From (10) and (H1), we have: $\pi(H_t, p(H_t, M_{t-1})) = \beta H_t$. Holding H_t constant (suppose that H_t is equal to $N/2$ for instance), as $\frac{\partial \pi}{\partial H} < 0, \frac{\partial \pi}{\partial p} > 0, \frac{\partial p}{\partial H} < 0, \frac{\partial p}{\partial M_{t-1}} > 0$, when β increases, M_{t-1} also increases and this leads to a horizontal shift in ψ . Conversely, we also expect a horizontal shift in ψ in the opposite direction when β decreases. For instance, a decrease in β can be interpreted as a decrease in θ , the opportunity cost of using an insecticide-treated bednet, or a change in W , individual characteristics (a decrease in illiteracy rate for instance). Hence, a decrease in β induces a decrease in $\psi^{-1}(N/2)$ and a decrease in the high equilibrium level, other things being equal.

Instead, a vertical increase could be achieved through a change in p or π , holding $\psi^{-1}(N/2)$ constant. Figure I.6 provides one probable mechanism, which could lead a community to break the trap and eradicate the disease through a vertical shift. This situation can be compared to the Italian eradication program in Sardinia (1901-1949), a malaria endemic island in Italy with temporal increase in malaria incidence. It is still not clear whether or not the disease disappeared thanks to the intensive use

of DDT but the failure to eradicate mosquitoes led to a situation referred to as “anophelism without malaria”. A vertical shift could lead to change the growth path of the disease from an increase (toward the high level equilibrium) to a decrease (toward eradication). Holding $\psi^{-1}(N/2)$ constant, a permanent vertical shift could also lead to a lower steady state equilibrium but this reduction in the equilibrium level will always be limited to $\psi^{-1}(N/2)$, the point where the 45° line becomes the tangent line of the curve.

FIGURE I.6: Policy implications: Eradication following a vertical shift(*source: author*)



However, the important fact is that we do not know how to eradicate the disease. There are striking differences between and within countries and eradication success or failure will not be due invariably to the same causes and conditions. Thousands of communities are suffering from malaria for ages but we only begin to collect sufficient epidemiologic information. We face a high degree of uncertainty to understand the disease and its environment as a whole. In endemic areas in Africa, History suggests that we are not capable of eradicating malaria. RBM aims principally at reducing mortality caused by the disease. Hence, a more plausible scenario will be malaria control (Figure I.7) i.e. a combination of a horizontal and a vertical shift, where the level of endemicity will be reduced and maintained to a lower level. Control may be seen as a supplement constraint on the disease.

Comparing Figure I.6 and Figure I.7 is rather instructive to understand and interpret the history of malaria control programs. Figure I.6 could better correspond to past anti-malaria campaigns in Europe, in the United-States, or in some islands where the transmission cycle has probably been interrupted by increasing efforts

during a sufficient period of time. These efforts have concentrated on vector control programs, increased efficiency of personal protection measures through better coordination, or increased attention on most vulnerable groups in particular endemic or epidemic settings. A horizontal shift could rather correspond to more subtil interventions through a modification of psychological perception of prevention and a change in the opportunity cost.

FIGURE I.7: Policy implications: Control (*source: author*)

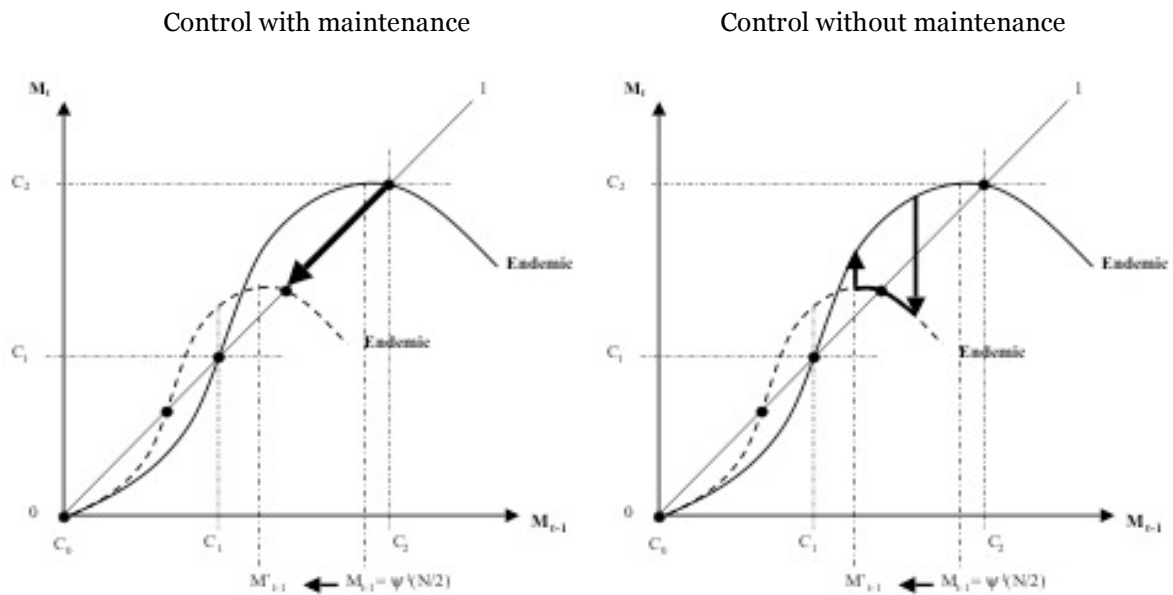
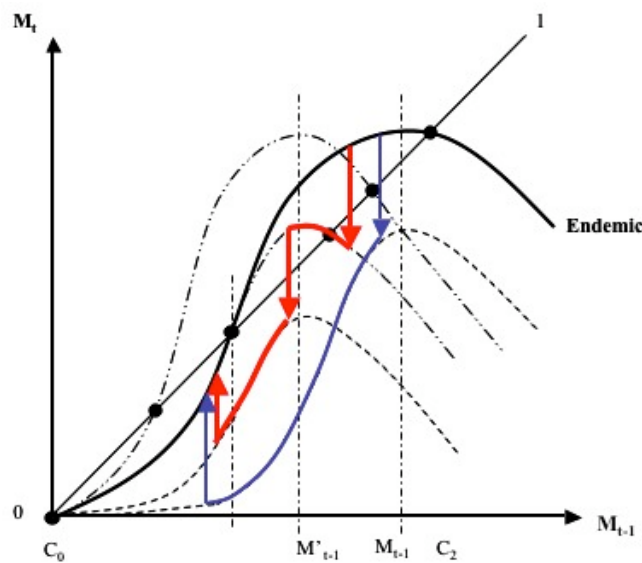


FIGURE I.8: Policy implications: Combining vertical and horizontal shifts (*source: author*)



The current RBM campaign in endemic areas of Africa and Asia could be a combination of vertical and horizontal shifts (Figure I.7). If not, policy makers should think about the implications and advantages of both types of actions, their potential interactions and the timing of each kind of interventions as illustrated very schematically in Figure I.8. Certain combinations will certainly be more efficient or effective and less time-costly than others. However, Figure I.8 is just a simplified illustration and we would need to calibrate the model to provide more precise recommendations.

B. Effect of health expenditure on health outcomes

“Disease traps” could also explain why empirical studies fail to find a significant positive effect of health expenditure on health. Key question for policy and health makers is whether or not health expenditure is instrumental in bringing about progress in health status. Early studies find no evidence that total spending on health is the principal determinant of child mortality outcomes (Musgrove, 1996; Filmer & Pritchett, 1997). In fact, these studies find no significant evidence of any impact of health spending on child mortality. The contribution of health expenditure could be relatively marginal in comparison to improvement in living conditions, institutional factors, female education and income inequality. Gupta et al. (1999, 2002) show that health expenditure reduces childhood mortality rates, though the association is not so strong. However, returns on health expenditure could be higher in low-income countries than in high-income countries (Gupta et al., 2003). Bokhari et al. (2006) suggest that government spending on health in developing countries is an important factor in reducing under-five mortality and maternal mortality, just as income per capita or economic growth. Mixed and heterogenous results in this field of research question the quality, the heterogeneity and the comparability of data. Good national data on the sources and uses of funds in the health system, comparable across countries, are necessary to infer valid causal relationship. Non-robustness of these studies may also be due to the absence of a strong statistical relationship or to model misspecifications. Indeed explanation of non-robustness could be linked to the existence of multiple equilibria: linear models are not satisfying answers to estimate such nonlinear relationships. Also, non-robustness may reflect treshold levels in the returns to increases in health spending. However, the composition of health expenditure and the quality of health services but also the dependency toward foreign health assistance and vertical programs could also explain why microeconomic as well as macroeconomic studies fail to identify mechanic effects of health expenditure on health outcomes.

C. Exogeneity of malaria?

Another implication of this model that can have immediate consequences for public health policies and research is about the exogenous nature of malaria. It is often said that malaria is a local disease strongly linked to environmental conditions. Malaria is also described as a disease of poverty. In the General Introduction, we asked whether or not malaria was endogenous to the development process. Something is endogenous to a system if it is determined within the system and if it is derived internally. In an econometric multiple regression model a variable is “*endogenous if it is correlated with the error term, either because of an omitted variable, measurement error, or simultaneity*” (Wooldridge, 2003). The model derived in this chapter treats malaria as an endogenous variable. We started from disease characteristics and adaptive human behaviours in response to the disease to understand malaria dynamics. Therefore, malaria is endogenously integrated in the model i.e. malaria dynamics is derived internally. However and interestingly, the main conclusion of this model leads us to consider malaria as an exogenous variable because of social externalities of agent decisions that lead to multiple equilibria. Indeed, we have shown that multiple equilibria are theoretically possible and so is the existence of malaria traps, defined as any self-reinforcing mechanism that causes malaria to persist. Hence, if a community is trapped in a high and stable level of malaria prevalence, malaria may be considered as exogenous in this particular community. Therefore, it is plausible that poverty has no influence on the probability of infection. This will lead to some kind of conditional exogeneity. Exogeneity does not mean that people have to suffer fatalistically from unjust background conditions. Exogeneity just implies that tackling malaria could have huge effects on economic development. Another implication of exogeneity is about rational fatalism. If a community is trapped in a high level of malaria stable equilibrium, people may think that there is no hope to escape to the trap (fatalism). This could induce a change in the opportunity cost of protection that may lead people to prefer risk (i.e. exposure) than prevention (rational fatalism, and consequently, increase the spread of the disease. If, in turn malaria also impacts education levels and education affects the opportunity cost of sleeping under an ITN, this could lead to a situation where malaria generates by itself a barrier to long-term interventions against the disease. This last observation justifies understanding better the effects of malaria on basic education. To conclude, we open the discussion by providing other possible mechanisms that could lead to disease traps.

IV. Conclusion

We have shown that social externalities of agents' decisions lead may lead to multiple equilibria. In conclusion and following Azariadis and Drazen (1990) and Azariadis and Stachuski (2005), we will try to provide additional hypothetical explanations of what we called "malaria traps" and, more generally, any "disease trap". One of the main plausible explanations could be the cumulative effect of low education on health and educational differences on health risks and illness. Empirical tests of the cumulative disadvantage theory applied to education and health provide good support this theory (Dupre, 2008). The initial conditions of the endogenous variables can generate long-run negative externalities. Another potential pathway could be the cumulative effect of poverty and income inequality. Of course, education and poverty are also linked. Accumulated evidence indicates that education is correlated with earnings and future labour participation. Individuals who lack knowledge of health risks, economic assets, and psychological buffers are at increasing risk of illness over the course of their lives (Dupre, 2008). Future studies should try to investigate by what precise mechanisms poverty traps and disease traps could interact. A third important pathway is more related to the epidemiological context: geographic and ecological conditions leading to "spatial disease traps" (Jalan and Ravallion, 2002). A fourth mechanism could be institutions, organisations and inequity. Other mechanisms could be the effects of demography, as we suggested in section 1. The effects of malaria control programs on mortality and fertility rates have been examined in the past (Newman, 1970). The lack of health insurance markets or the existence of health insurance markets with asymmetric information or high transaction costs could also be one potential pathway. Another hypothesis made by Sen is about the adaptative preferences of people. In other words, individuals can become accustomed to unfavorable circumstances. If people think that there is no hope to eradicate malaria or if they are in such material conditions that they do not anticipate their needs with respect to health, they will probably never escape the disease trap without an exogenous intervention. Empirical studies will be needed to test whether or not the "disease trap" is a plausible explanation at the microeconomic and macroeconomic levels. This could be particularly interesting in helping us to understand the epidemiologic transition in developed countries in the 19th century.

The model of this paper could be extended in many ways. First, it could be interesting to distinguish asymptomatic infections from symptomatic infections in this model. Second, one limitation of this study is that we did not take into account all potential externalities. In particular, the model is based on one strong hypothesis that the use of an ITN is completely efficient in protecting from malaria infection.

However, even with high coverage with ITNs, transmission still occurs, and individuals can still witness illness occurring. Third, resistance and immunity could also be integrated in the model. Last, the following step of this work will be to provide a calibration of this model and to simulate different thresholds effects.

In the following chapters we will focus on the relationship between malaria and education. As the effect of mother and household education has already been studied, we will analyse the causal effects of malaria on human capital accumulation, a relationship that has been poorly quantified.

**CHAPTER II: MALARIA AND HUMAN CAPITAL
ACCUMULATION: SOME MACROECONOMIC EVIDENCE⁹**

⁹ This chapter is based on Thuilliez (2009).

In this chapter, we focus on the macroeconomic effects of malaria for four main reasons:

- First, studies on the economic effects of malaria are mostly microeconomic studies. Macroeconomic studies, which assess the global cost for societies are rather scarce. Even though macroeconomic and microeconomic studies are very complementary in this field of research, macroeconomic effects are not the mere aggregate of microeconomic results. Indeed microeconomic studies do not incorporate general equilibrium effects or externalities and provide several quantifications of the relevant effect.
- Second, before investing in microeconomic data, **our hypothesis is that malaria has such a huge human impact on children that it will be surprising not to encounter the effects of malaria on education at the macroeconomic level.**
- Third, macroeconomic studies are very useful to communicate some key figures on the cost of malaria to International Organisations and to advocate for resources (UNESCO-EFA, 2009).
- Studying the effects of malaria on education is interesting by itself but Chapter 1 provided additional arguments to justify the study of the effects of malaria and education. The cumulative disadvantage theory applied to education and health also provides good support to explain the “malaria traps”.

This chapter proceeds as follows. Section 1 presents conceptual issues around human capital quality measures at the country level. Section 2 specifies the methodology and other variables included in the models. Results are presented in section 3 and some robustness analysis in section 4. Section 5 concludes.

I. Schooling quality at the Country level

Conceptually, education has two main qualitative goals. The first one is to develop cognitive capacities of students. The degree to which systems actually achieve this is one indicator of their quality. The second element is « education’s role in encouraging learners’ creative and emotional development, in supporting objectives of peace,

citizenship and security, in promoting equality and in passing global and local cultural values down to future generations » (EFA, Global Monitoring Report, 2005). It is difficult to measure education quality for a broad number of countries, especially if we consider the second objective. A small number of authors have tried to attack this problem. Barro & Lee (2001) provide a panel dataset that includes output and input measures of schooling quality from 1965 to 1990. Hanushek and Kimko (2000) also develop cross-country measures of labor force quality based on information about international differences in mathematics and science knowledge for 1991. Following a different methodology, Altinok & Murseli (2007) obtain qualitative indicators of human capital (QIHC). First, they compile recent surveys between 1995 and 2003 in a cross-country database level for 105 countries. Second they update Barro & Lee (2001) dataset. And third, they gather all international surveys on children's achievement in a panel dataset concerning the period between 1964 and 2005. However, as Altinok & Murseli (2007) dataset has not been used intensively yet, it is difficult to use it as a reference to compare different cross-country studies. Moreover the panel dataset on international tests scores is highly unbalanced and the cross-sectional indicator compiled different years so that it is difficult to interpret. Al-Samarrai (2006) proposed to use simple measures of education quality available on the UNESCO Institute for Statistics website (primary repetition rates, primary completion rates, primary survival rates) and frequently used to compare school outcomes across countries in international monitoring reports. As one of our objectives is to compare our results to other studies, these measures will be preferred.

Cross-country and Panel literature on the determinants of school quality tends to focus on education resources. Increasing investments in basic education has been seen as a key strategy to achieving the Education For All (EFA) goals (World Education Forum, 2000). Nevertheless, in cross-country comparisons, the link between resources and educational outputs is weak (Al-Samarrai, 2006). Increases in expenditure in primary education alone are unlikely to be sufficient to achieve the education millennium development goals. Other variables such as the effectiveness of the public expenditure management system, household spending and the composition of public education spending may explain this weak link. In this chapter, we explore another potential important factor that can influence schooling quality in developing countries and may contribute to make expenses in primary education ineffective. We use different datasets to estimate in a cross-country and panel regression framework the impact of an index of malaria endemicity on repetition rates and completion rates. One difference in our work is that we introduce a proxy for general child health conditions, climate variables and governance indicators in the model.

As we said in General Introduction, the relationship between school outputs and inputs is generally analysed with an education production function, particularly at the macroeconomic level. Nevertheless, health conditions and diseases that alter cognitive and learning abilities like malaria are not taken into account in macroeconomic studies of the determinants of education quality. Following Barro & Lee (2001), we define a very simplified education production function where health is taken into account:

$$Q = Q(F, R, H) + \varepsilon \quad (1)$$

Where Q denotes schooling quality, F , family factors (principally family incomes), R , public resources used for primary education, H , child health conditions and ε , unmeasured factors influencing schooling quality.

II. Data and Methodology

The main framework of this study has been designed for a cross-sectional dataset using Sachs & Gallup (2001) and Al-Samarrai (2006) indicators for two main reasons:

- 1- The widely different methodologies and sources of data are a real barrier to draw reliable conclusions on the economic costs of malaria. Our cross-sectional analysis based on published dataset enables comparing our results with other studies and going deeper than former analysis's. In other words, we need to compare what is comparable.
- 2- Panel data used to study the macroeconomic effects of malaria have not only advantages. First, macroeconomic panel data can be highly unbalanced which raises selection and attrition problems. The quality of data for oldest years is also obviously different from the quality of data collected during recent years. Second, the main criticisms addressed to Gallup & Sachs (2001) deals with the instrumentation strategy. Sachs & Gallup (2001) actually used a time-invariant variable to instrument for a change in the *falciparum* malaria index from 1965 to 1994. They argue that this instrument is exogenous to health interventions and economic conditions because the instrument is built upon climatological and vector characteristics on a country-by-country basis.

“There is no reason to think that the distribution of malaria mosquito vectors is a cause of economic growth apart from the direct influence of malaria, making vector prevalence an ideal instrument for malaria change.” Gallup & Sachs (2001)

However, one can argue that the discovery and widespread use of DDT (since 1939) against mosquito vectors is linked to initial economic conditions and public health interventions with major consequences on the change in the distribution of anopheles mosquitoes. In addition, as they only use one instrumental variable in this study, they cannot prove the validity of this instrument. However the partial R^2 of the first stage regression of the change in the malaria index on the instrument is 0.51, which confirms its relevance as an instrument candidate and the strength of this instrument. Moreover, the mathematical construction and the literary justification of this instrument is rather convincing for different reasons. Indeed, the control of malaria depends predominantly on entomological and ecological factors. It is much more difficult for instance in Africa where *An. gambiae s.l* and *An. funestus* are predominant and have biologic properties favourable to the spread of malaria.

“Anopheles mosquitoes species vary widely in their efficacy in transmitting human malaria so that their distribution is strongly correlated with malaria intensity and its change” Gallup & Sachs (2001).

The formula for the instrumental variable includes temperature, species abundance and entomological properties. Interestingly, Hamoudi & Sachs (1999) note that in many countries that have eradicated malaria, malaria vectors remain, suggesting that the danger of resurgence persists and explaining why most European countries take surveillance of malaria seriously. This last argument is certainly the most convincing one to explain why the distribution of malaria vectors is not strongly linked to economic conditions and health interventions but rather to ecological conditions. Similar instruments based on ecological conditions have been used and validated after this study. In this chapter, we will use the same instrumental variable with the notable exception that we will not use it to instrument a change of the endogenous variable. As the use of entomological variables seems to be a convincing instrumentation strategy, in Chapter 3, we will use a similar instrumental variable at a more microeconomic level and we will show that this instrument is strong and validated by usual tests.

Therefore, the lack of time-variant instrument has led us to favour cross-sectional analysis. However our robustness checks presented in section 5 provide different specifications applied to panel dataset.

A. Data

1. Dependant Variable: Primary Repetition and Completion rates.

As we are particularly interested in measuring the specific impact of malaria on learning abilities of children, we first chose to consider primary school repetition rates which reflect this particular aspect of education quality. Indeed, despite the fact that repetition rates are sometimes affected by school and educational policies (such as national regulations), they are more directly determined by school results. The choice of this variable and the large database we dispose allow us to have a reasonable size sample with good developing countries coverage (in particular African countries) to make our regressions (from 80 to 84 observations). As we said, other alternative indicators are test scores. Although, some cross-country studies have been undertaken using student's scores on internationally comparable tests of achievement in knowledge, the size of samples is often reduced because of limited availability of internationally comparable data. The recent efforts to define a standardized indicator from MLA (Monitoring Learning Achievement – UNESCO/UNICEF), SACMEQ (Consortium for Monitoring Educational Quality) and PASEC (“Programme d’Analyse des Systèmes Educatifs de la CONFEMEN”) still include a small sample of countries. Primary repetition rates are defined as the proportion of pupils enrolled in a given grade at a given school-year at primary level who study in the same grade in the following school-year (UNESCO).

In order to take into account other effects of malaria on school results we also use the primary completion rates as an alternative dependent variable. The choice of this variable gives us a smaller size sample due to the lack of data for both the explained and the explanatory variables in the models but still with good developing and African countries coverage (from 54 to 55 observations). Primary completion rates are defined as the total number of students (of any age) in the last grade of primary school, minus the number of repeaters in that grade, divided by the total number of children of official graduation age (World Bank).

Repetition and completion rates have been frequently used to compare school outcomes across countries in international monitoring reports.

Table 1 given in the introduction presented panel data estimates of the effects of malaria on illiteracy rates from 1950 to 1995. The illiteracy rates before 1980 have been gathered from personal researchs at the UNESCO archives (UNESCO basic facts and figures 1951-1958 p^o9, 1952; and UNESCO statistical yearbooks until 1990).

2. The *P. falciparum* malaria index

A major problem when assessing the economic cost of malaria - at the macroeconomic level (Sachs & Gallup, 2001) as well as at the microeconomic one (Audibert et al., 2003) - is to measure the exposure to malaria risk and the severity of the disease. This is clearly due to the lack of data on malaria incidence. As we said in General Introduction, different methodological and technical barriers impede to measure with precision the number of malaria cases.

As a proxy of child malaria morbidity, we first used the index described in Sachs & Gallup (2001) defined as the fraction of population at risk of contracting *falciparum* malaria in a country. This index use historical maps of the geographic extent of malaria prevalence combined with detailed data on the world population distribution and the fraction of cases of malaria that are *falciparum* malaria (for 1990). We computed this indicator for four different years (1946, 1966, 1982, 1994). This assumes that the relative share of *P. falciparum* cases did not change substantially from 1946 to 1994. As there are so many problems relative to the measurement of the number of malaria cases (even more for historical data), we consider these indexes as the best measures of *falciparum* malaria risk for these particular years.

Second, we extend these indicators with more recent data available from the Malaria Atlas Project. The main objective of this project is to develop a detailed model of the spatial limits of *P. falciparum* and *P. vivax* malaria at a global scale and its endemicity within this range (Guerra et al., 2008). The MAP data identified 87 countries at risk of *P. falciparum* transmission between 2002 and 2006. The raw estimated population at risk are projections for 2007 made from the Global Rural Mapping Project (GRUMP) alpha population surface (adjusted to United Nation national country estimates for 2000) and national medium variant population growth rate by country from the United Nation Population Database (UNPD). This work is protected under a Creative Commons (Attribution-Noncommercial-Share Alike 3.0) licence, which allows using it and adapting it, under the conditions detailed at <http://creativecommons.org/licenses/by-nc-sa/3.0/>. To create the percentage of population at risk for 2007 by country, we used the 2000 UNPD estimates as a baseline and the national population growth rate by country. This index has two main advantages. First it enables us to update and improve the Sachs & Gallup (2001) database on *falciparum* malaria risk and to make cross-sectional regressions with

recent years. Indeed, the MAP estimates are not calculated as the Sachs & Gallup (2001) Malaria index, and therefore this new index is not strictly comparable to the first one. Nevertheless, it provides an index constructed in a different way that can be used in a robustness analysis. Our assumption is that the quality of the Malaria Index is improved and actualised. Second, the MAP database also provides the projected populations at risk within areas of unstable and stable *P. falciparum* transmission. Therefore, among people at risk, it is possible to have the proportion of those who live in stable or unstable areas and to dissociate the effects of malaria in both cases.

Costs in terms of education are expected to differ with the level of endemicity and the percentage of areas in stable or unstable areas. Malaria is said to be stable if it is transmitted throughout the year by long-lived, anthropophilic vector anopheline mosquitoes (Kiszewski et al., 2004). MAP defines stability as a *P. falciparum* annual parasite prevalence ≥ 0.1 per thousand people per year (see appendix 1 for more details on the classification of malaria risk). We can distinguish three main situations that will have different costs in terms of human capital and basic education at the macroeconomic level in our study. In highly endemic regions (where the fraction of population at risk of contracting falciparum malaria is high), mortality and morbidity mainly occurs among children aged from 4 months to five years and pregnant women. We should then expect in these areas a bigger impact of malaria on primary school quality and school performances through the channels we already detailed in General Introduction (Figure 9) and because of the other external effects associated with the disease. By contrast, in regions where malaria transmission is less stable, where herd immunity is less, malaria and severe malaria can affect people of all ages but less frequently (mainly during epidemics or seasonal variations). Educational impacts are expected to be smaller. In areas free of malaria, there will be obviously no impact.

In this chapter, we will consider that a country is highly endemic when the *P. falciparum* malaria index is greater than 0,5 for both indexes. We will also try to assess the impact in stable and unstable areas.

Last, a third index of malaria derived from completely different data is used below in cross-country regressions. As the calculation of DALYs and weights used in the Burden Of Disease methodology account for neurologic sequelae of severe malaria, we use DALYs as a last indicator of malaria to compare the effects of the disease with other infections.

3. Other Independent Variables

Three variables of resources that have been identified previously as potential determinants of education outcomes in the literature are used to take into account the impact of public spending on repetition rates: primary current expenditure per pupil (PPP), public current expenditure in primary education as a percentage of GNP, primary school pupil-teacher ratio.

Income per capita is also included in the regressions for two main reasons. On the one hand, countries with higher income have theoretically better primary education access and internal efficiency indicators. Indeed, income per capita may be interpreted as a proxy for parents' income (Barro & Lee, 2000). Nevertheless, when it is associated with education quality indicators, the effect of GDP per capita is not so clear. This point will be discussed in Section V. On the other hand, malaria and income levels are intimately connected and our *falciparum* malaria index could just be a proxy for poverty.

Level of urbanisation, another explanatory variable that is a potential influential factor of primary repetition and completion rates, is included in the model. Indeed, in urban areas, school results are expected to be higher because travel costs linked with school attendance may be lower than in urban areas, children can have a better access to educational services or better general conditions to work especially in developing countries (electricity for instance).

As the literature already stressed it, effectiveness of the public expenditure management system could explain the weak link between resources and education quality (see for instance Reinikka & Svensson, 2004). In order to control for this factor we include in the regressions one measure of governance quality developed by Kaufmann et al. (2006): "government effectiveness 1996". Regression (4) of Table A2 also includes a measure of control of corruption for 1996. The Worldwide Governance Indicators are a compilation of information and perceptions from diverse groups of respondents but have to be seen as proxies of the complex phenomenon of governance. We do not pretend here to answer to the particular question of the impact of the quality of management system on education outcomes but to catch only the influential effects of these factors on our dependant variables, *ceteris paribus*.

Malaria could also be a proxy for a range of other variables, principally general health conditions, particularly child health conditions, climate and geographical location. Therefore, the regressions given in Table II.1 and II.2 include the under-5 mortality rate (as a proxy for child health conditions) and the percent of land area in subtropics and tropics (Sachs & Gallup, 2001). These last two variables allow us to control for climate, ecological conditions and diseases associated with tropical and subtropical location that could drive our coefficient estimates on the *falciparum*

malaria index. As an alternative measure of geographical location, regressions (1) and (5) in Table II.3 include regional dummies.

4. Descriptive statistics of Cross-Section data

Descriptive statistics, sources and definitions of the variables used in the main cross-sectional analysis (1996) presented in this chapter can be found in Table II.A1. Simple correlations between variables are given in Table II.A3. Panel data were drawn from the Barro & Lee (2001) dataset completed with the World Development Indicators (World Bank 2006). Data for robustness analysis are also from the World Development Indicators (World Bank, several years) and UNESCO database. As an example, take the 125 observations available for both the primary repetition rate in 1996 and the *falciparum* malaria index in 1994. Thirty-six countries of the 125 countries, or 29%, have intensive malaria (29 are African countries). Ranking the 125 countries by primary repetition rate, 25 of the 36 countries with severe malaria (69%) are in the bottom half of the ranking. A first look at the data lets us think however that malaria could only be a proxy for geographic location or other variables. In the next subsection we detail the methodology used in the study to explore the link between malaria and school results and treat the possible sources of errors. We recall that we only present our identification strategy for the cross-sectional regression analysis (1996), which is comparable to other studies.

B. Methodology

Our modelling approach is based on previous works (Barro & Lee, 2000; Al Samarai, 2006) and the relevance of including malaria in the model has been tested using stepwise selection procedure and usual tests (Appendix II.2).

We begin by estimating the regressions using Ordinary Least Squares (OLS). Standard errors are adjusted for heteroscedasticity in all cases. In this first step, we consider all the available observations. Outlying observations are not automatically mistakes in data entry and can provide important economic information by increasing variation in explanatory variables. In addition to these regressions, we also analyse how robust the OLS results given in Table II.1 and II.2 are to different estimation techniques.

Firstly, this chapter assess whether the coefficient estimate on the malaria index suffer from endogeneity bias (due to measurement errors or two-way causation principally but also omitted variables). The *falciparum* malaria index can clearly suffer from measurement errors. It is also possible that low education levels have a negative impact on malaria eradication directly or through other variables like

income level, general health conditions... It is possible that countries with bad educational outcomes will have more difficulties to control malaria. However, this last argument remains speculative, as it is not clear whether or not the general level of formal education in a country will improve malaria control. For instance, it can be argued that some MENA (Middle East and North Africa) countries succeeded in controlling malaria recently but still have relatively high rates of illiteracy. Indeed, according to the Malaria Report 2008, Algeria, Egypt, Iraq and Saudi Arabia are among 10 countries worldwide that have successfully reached the elimination phase for malaria. United Arab Emirates was malaria-free in January 2007. Much of this success in combating malaria may be attributed to improvements in malaria interventions. The educational achievement of MENA countries remains below other countries at similar levels of economic development. The 2007 TIMSS test on 8th grade Math and Science capabilities resulted in none of the 12 participating MENA countries reaching the average scale¹⁰. However, malaria epidemiology differs widely from SSA in this part of the world with a higher proportion of *P.vivax* malaria cases and different predominant *Anopheles* vectors. Moreover, despite low levels of education performance, MENA countries have almost reached full primary education enrolment and have halved illiteracy rates within the past 20 years. As emphasised in General Introduction and in Chapter 1, mother's education is a key determinant of access to health services and use of ITNs in Africa. Hence, a bidirectional relationship cannot be completely excluded.

To correct for the possible endogeneity bias, we use the spatial index of the stability of malaria transmission based on the interaction of climate with the dominant biologic properties of vector *Anopheles* of malaria (biting activity, proportion of blood meal taken from human hosts, daily survival of the vector, duration of the transmission season and of extrinsic incubation). This interaction determines vectorial capacity and explains a great part of the force, the stability and the regional variation of malaria transmission (Kiszewski et al., 2004). This index is measured on a highly disaggregated level and then averaged for the entire country weighted by population. The total number of countries in which malaria is endemic or potentially endemic has been divided into 260 different regions with their particular characteristics (to represent the diversity of habitats) and 34 vectors *Anopheles* were considered as dominant. Because it is built upon climatological and vector characteristics, "Malaria Ecology" is said to be exogenous to public health interventions and economic conditions. There is no reason to think that the ecology-based distribution of malaria mosquito vectors and the variation of their biologic

¹⁰ http://nces.ed.gov/timss/resultso7_math07.asp (accessed: November 2009)

properties are a cause of bad educational outcomes apart from the direct influence of malaria or through observable variables such as tropical location already included in our models. The variable is therefore a good instrument candidate for the *falciparum* malaria index. As we said, a similar instrument has already been used by Sachs & Gallup (2001). The first stage regression of the *falciparum* malaria index on the instrument (“Malaria Ecology”) has a R^2 of 0.56.

Secondly, because of the sample size, we can only introduce a limited number of control variables to isolate the causal effect of the *falciparum* malaria index. The specification models presented in Table II.1 and II.2 already include a relatively high number of independent variables. Introducing more variables may lead to a “small sample size problem” and multicollinearity can increase the variance of our unbiased estimators. Therefore, we present different specifications of the regression model with a restricted number of independent variables and a higher size sample (from 85 to 117 observations for regressions with repetition rates and from 56 to 73 observations for regressions with completion rates). We control separately for different factors by including in each regression successively a set of variables for geographical and climate characteristics, variables for health conditions, governance indicators and education resources (Appendix II.3, Table II.A.6).

Thirdly, OLS regressions assume that the residuals are independent. It is very possible that the primary repetition rates within each subcontinent may not be independent and this could lead to residuals that are not independent within subcontinents. We hence allow regressions observations to be clustered into subcontinents to take into account the probable spatial correlation of observations within subcontinents (Appendix II.3, Table II.A.7).

Fourthly, we explore whether our OLS and 2SLS results are driven by influential observations or not. OLS is susceptible to outlying observations because it minimizes the sum of squared residuals. Individual country with large residual and high leverage are identified and dropped for each regression¹¹. From 8 to 11 countries have been identified as outliers in the regressions and dropped (Appendix II.3, Table II.A.8).

¹¹ Studentized residuals and leverages were examined as a first approach for identifying outliers and observations that have potential great influence on regression coefficient estimates. Next, DFIT statistic was used as an overall measure of influence.

III. Results: Cross-Country Estimates

The results from the OLS and Two Stage Least Squares regression analysis with Samer Al Samarrai dataset for primary repetition and completion rates are presented in Table II.1 and Table II.2. Different models are reported to take into account different options chosen in the literature. There appears to be a very strong relationship between the *falciparum* malaria index and the primary repetition and completion rates. The positive coefficient on the malaria index (0.096; $\sigma=0.039$) in regression (1) of Table II.1 indicates that countries with intensive malaria have repetition rates in primary education 9% higher than countries without malaria. A one standard deviation increase in the *falciparum* malaria index is associated to a 0.43 standard deviation increase in primary repetition rates. The negative coefficient on the malaria index (-0.295; $\sigma=0.087$) in regression (1) of Table II.2 indicates that countries with intensive malaria have primary completion rates 29% lower than countries without malaria. A one standard deviation increase in the *falciparum* malaria index is associated to a 0.47 standard deviation decrease in primary completion rates.

The coefficient estimates on the malaria index are significantly different from 0 (at the 5% level) in regression (1) and (2) of Table II.1. In regression (3) of Table II.1, the *p value* of the coefficient estimate is 0.13 and the value of the coefficient estimate is lower than in the previous regressions. This may be due to the high correlation between the *falciparum* malaria index and the pupil-teacher ratio within the sample ($r = 0.73$). However, the corresponding Instrumental Variable estimation, given in regression (6) of Table II.1, shows that the impact of malaria is still significant, positive and high. In all OLS regressions of Table II.2, the coefficient estimates on the malaria index are significantly different from zero, negative and relatively stable.

Section 3 suggested that the *falciparum* malaria index may be endogenous and explained that instrumental variable estimation can be used to account for this problem. Two stages least squares regressions are given in column (4) to (6) of Table II.1 and II.2. We use the “Malaria Ecology” previously described as an instrument for the *falciparum* malaria index. If there were a reverse causation between the malaria index and primary repetition and completion rates, the coefficient estimates on the malaria index reported in columns (1) to (3) would be positively biased in Table II.1 and negatively biased in Table II.2.

TABLE II.1 : Cross-Country OLS and 2SLS estimates of the effects of Malaria on Primary Repetition Rates

Dependant variable is Primary Repetition Rate (1996)						
	OLS			2SLS		
	(1)	(2)	(3)	(4)	(5)	(6)
Sachs & Gallup falciparum Malaria index 1994	0.096** (0.039)	0.102** (0.045)	0.066 (0.043)	0.180*** (0.060)	0.185*** (0.059)	0.111* (0.067)
Log GNP per capita PPP 1996 (\$ constant 95)	0.023 (0.015)	0.034* (0.019)	0.037** (0.017)	0.019 (0.016)	0.030 (0.020)	0.034** (0.017)
Subtropics (% land area)	0.038 (0.029)	0.026 (0.029)	0.013 (0.030)	0.021 (0.034)	0.009 (0.035)	0.006 (0.034)
Tropics (% land area)	0.017 (0.043)	0.012 (0.045)	0.006 (0.042)	-0.019 (0.047)	-0.025 (0.048)	-0.011 (0.047)
Log Urban Pop 1995 (% total pop)	0.031 (0.032)	0.024 (0.036)	0.018 (0.032)	0.042 (0.034)	0.039 (0.038)	0.026 (0.035)
Log Under 5 Child mortality 1995	0.053*** (0.017)	0.049*** (0.017)	0.034** (0.015)	0.029 (0.023)	0.026 (0.023)	0.025 (0.019)
Log Publ Expend in Prim Educ (% GNP) 1996	-0.019 (0.014)	-0.013 (0.016)	-0.015 (0.014)	-0.017 (0.014)	-0.012 (0.017)	-0.014 (0.015)
Log Primary expenditure per pupil PPP (PPP) 1996	-	-0.012 (0.011)	-0.007 (0.010)	-	-0.010 (0.014)	-0.007 (0.011)
Log Prim School pupil-teacher ratio 1996	-	-	0.093*** (0.031)	-	-	0.083*** (0.033)
Government Effectiveness 1996	-0.001 (0.013)	-0.005 (0.014)	-0.012 (0.014)	-0.014 (0.016)	-0.017 (0.015)	-0.017 (0.015)
Intercept	-0.530*** 0.187	-0.470** (0.203)	-0.740*** (0.204)	-0.466** (0.197)	-0.432** (0.221)	-0.692*** (0.207)
<i>Number of observations</i>	84	82	80	84	82	80
<i>R squared</i>	0.51	0.52	0.59	0.46	0.48	0.58

Notes:

1. Robust standard errors adjusted for heteroscedasticity in parenthesis.

2. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

3. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

4. The p.value of the coefficient estimate on the falciparum malaria index 1994 in regression (3) is 0.13.

TABLE II.2 : Cross-Country OLS and 2SLS estimates of the effects of Malaria on Primary Completion Rates

Dependant variable is Primary Completion Rate (1996)						
	OLS			2SLS		
	(1)	(2)	(3)	(4)	(5)	(6)
Sachs & Gallup falciparum Malaria index 1994	-0.295*** (0.087)	-0.325*** (0.102)	-0.222* (0.112)	-0.543*** (0.184)	-0.554*** (0.188)	-0.480* (0.264)
Log GNP per capita PPP 1996 (\$ constant 95)	-0.003 (0.051)	-0.040 (0.045)	-0.053 (0.044)	0.029 (0.064)	0.002 (0.061)	-0.009 (0.068)
Subtropics (% land area)	0.008 (0.075)	0.045 (0.072)	0.096 (0.076)	0.013 (0.086)	0.047 (0.082)	0.065 (0.090)
Tropics (% land area)	-0.031 (0.078)	-0.009 (0.076)	-0.008 (0.075)	0.069 (0.108)	0.089 (0.108)	0.074 (0.103)
Log Urban Pop 1995 (% total pop)	0.072 (0.053)	0.104* (0.057)	0.124** (0.057)	0.042 (0.058)	0.053 (0.062)	0.068 (0.068)
Log Under 5 Child mortality 1995	-0.095 (0.063)	-0.079 (0.065)	-0.078 (0.064)	0.000 (0.098)	0.000 (0.098)	-0.012 (0.108)
Log Publ Expend in Prim Educ (% GNP) 1996	0.018 (0.030)	0.003 (0.032)	0.014 (0.033)	0.001 (0.030)	-0.002 (0.032)	0.002 (0.036)
Log Primary expenditure per pupil PPP (PPP) 1996	- (0.024)	0.036 (0.024)	0.028 (0.023)	- (0.027)	0.023 (0.027)	0.022 (0.026)
Log Prim School pupil-teacher ratio 1996	- (0.084)	- (0.084)	-0.150* (0.084)	- (0.114)	- (0.114)	-0.055 (0.114)
Government Effectiveness 1996	-0.016 (0.038)	-0.002 (0.035)	-0.006 (0.035)	0.003 (0.045)	0.005 (0.041)	0.002 (0.040)
Intercept	0.991* (0.561)	0.797 (0.605)	1.355* (0.694)	0.497 (0.763)	0.483 (0.783)	0.737 (1.071)
<i>Number of observations</i>	55	54	54	55	54	54
<i>R squared</i>	0.79	0.80	0.82	0.75	0.77	0.79

Notes:

1. Robust standard errors adjusted for heteroscedasticity in parenthesis.

2. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

3. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

After correcting for the possible endogeneity of the *falciparum* malaria index, the absolute value of the estimated effect of malaria on repetition and completion rates is higher than our previous results and still statistically significant in all regressions. This generally occurs when the endogenous variable suffers from measurement errors and the *falciparum* malaria index is actually relatively crude. Therefore, the 2SLS regression analysis confirms that the link between *falciparum* malaria and primary repetition and completion rate is strong. The positive coefficient on the malaria index (0.180; $\sigma=0.060$) in regression (4) of Table II.1 indicates that countries with intensive malaria have repetition rates in primary education 18% higher than countries without malaria. The negative coefficient on the malaria index (-0.543; $\sigma=0.184$) in regression (4) of Table II.2 indicates that countries with intensive malaria have primary completion rate 54% lower than countries without malaria. As 2SLS and OLS estimates on the *falciparum* malaria index differ significantly (because of measurement errors on the malaria index), the 2SLS estimator is more efficient than the OLS estimator. Therefore, we will now focus on regressions (4) to (6) of Table II.1 and II.2 for the analysis.

GNP per capita is only significant in regression (6) of Table II.1 and its impact on primary repetition rate is very small and positive. Coefficient estimates of GNP per capita in regressions for completion rates are not significantly different from zero (Table II.2). These results suggest that income per capita do not have a strong impact on primary repetition and completion rates.

Tropical and subtropical location does not appear to have a significant impact on primary repetition and completion rates. It could be argued that the impact of malaria on educational outcomes reflects the influence of a particular region and not the isolated effect of malaria. For example, Sub-Saharan Africa has in average high levels of malaria, high repetition rates and relatively low completion rates. Hence, the malaria index could just be a proxy for Sub-Saharan Africa. Regression (1) and (5) of Table II.A.6 include regional dummies (South Asia, East Asia, Sub-Saharan Africa, Middle East and North Africa, and Latin America and the Caribbean). The results are globally unchanged.

Whereas it was positively and significantly associated with repetition rates in OLS regressions, the Under-5 mortality rate does not appear to have a significant impact on repetition or completion rates in 2SLS regressions. In all 2SLS regressions of Table II.1 and II.2, the coefficient estimates on the Under-5 mortality rate are not significantly different to zero.

Level of urbanisation and the governance indicator have no significant impact on the educational outcomes under study here.

The coefficient estimates on public current primary expenditure (% of GNP) is negative in all regressions of Table II.1. It is positive in regressions (4) and (6) and negative in regression (5) of Table II.2 but never significantly different from 0. This insignificant effect of total educational spending on repetition or completion rates suggests that an increase in total school resources itself may not lead to an increase in student achievement. This conclusion is in accordance with Hanushek (1995) and Samer Al Samarrai (2006). There is no systematic relationship between school performance and general measure of school resources.

When regressions include two school variables (primary expenditure per pupil and public current primary expenditure as a proportion of GNP) the results are not dramatically changed. Results suggest that primary expenditure per pupil is also an insignificant determinant of repetition and completion rates, *ceteris paribus*.

Nevertheless, when the regressions include the three school variables, the pupil-teacher ratio is positively and significantly (at the 1% level) associated with repetition rates in regression (6) of Table II.1. This result is in keeping with Barro and Lee (2001) conclusions about the link between repetition rates and pupil-teacher ratio. A lower pupil-teacher ratio is estimated to improve repetition rates and test scores. Components of school expenditures allocated to lower the pupil-teacher ratio will reduce primary repetition rates whereas the other resources variables still have no significant impact. Nevertheless, the literature remains ambiguous on this point (Samer Al Samarrai, 2006). Moreover, the pupil-teacher ratio is not significant in the primary completion rate regression (regression (6) of Table II.2). Therefore, as in the literature, there seems to be an ambiguous relationship between educational outcomes and commonly measured school resources. Nevertheless, as this question is not the primary focus of our study we will now concentrate our efforts on testing the robustness of the coefficient estimates on the malaria index.

IV. Robustness analysis

A. Sachs & Gallup (2001) indicators for malaria

1. Cross-Country robustness analysis

Regressions in Table II.A6 present alternative specification models with a reduced number of variables. We control for different sets of independent variables. Only the 2SLS regressions have been presented. All regressions include the *falciparum* malaria index and GNP per capita. We control successively and separately for

geographical (regional dummies instead of climate variables), variables for health conditions, governance indicators (control of corruption and government effectiveness) and educational resources. Using these specification models does not alter the main conclusions of this chapter. The regional dummy variable that appears to play significant role on the 2SLS regressions with repetition rates is Latin America and the Caribbean. These countries, on average, have higher primary repetition rates than other countries, after controlling for the variables included in the model. No other dummy variable appears to play significant role on the 2SLS regressions. Malaria could also be a proxy for a range of tropical diseases that are not adequately controlled for by Under-5 child mortality (1995), tropical and subtropical location. To assess whether other diseases were responsible for the correlation of malaria with primary repetition and completion rates, we include jointly two relevant indicators for the time period under study here: an average of the proportion of the population living in areas with dengue fever from 1975 to 1995 and an estimating fraction of the population at risk of contracting yellow fever for 1996 (Sachs & Gallup, 2001). Descriptions and sources of these variables are given in Table II.A1. Results are given in regressions (2) and (6) of Table II.A.6. Unfortunately we do not have indicators neither for other tropical diseases nor for the acquired immunodeficiency syndrome (AIDS) at the macroeconomic level for 1996. As the spread of AIDS has evolved very quickly, we have not included recent data of this burden in the regressions. However, we do not think that our conclusions will be changed with the inclusion of such indicator because AIDS affects education by different channels than malaria (for instance through its impact on teachers). The estimated impact of malaria remains relatively stable and statistically significant at the 5% level for regression (2) and at the 1% level for regression (6). The two tropical diseases have no significant correlation with repetition and completion rates.

Table II.A.7 shows that our results do not change dramatically when observations are clustered within subcontinents. The 2SLS results are nearly the same of those presented in Table II.1 and II.2. Therefore, we cannot reject the assumption that the residuals are independent.

Table II.A.8 shows that our conclusions are not modified when influential observations are dropped. The size and significance of the coefficient estimates on the *falciparum* malaria index change only slightly.

2. Malaria and Primary repetition rates: Panel Dataset

The panel data models used to estimate the effect of malaria on primary repetition rates are random effects models (Table II.3). This is justified by the fact that 1) our

time dimension is reduced, 2) our between variation is higher than our within variation.

TABLE II.3 : Panel data estimates of the effects of Malaria on Primary Repetition Rates (1960-1995)

	Dependant Variable is Repetition Rate (Panel Dataset 1960-1995) - Random effects Model					
	(1)	(2)	(3)	(4)	(5)	(6)
Sachs & Gallup Malaria index	0.039*** (0.013)	0.024* (0.013)	0.019 (0.015)	.	.	.
Sachs & Gallup <i>falciparum</i> Malaria index	.	.	.	0.039* (0.021)	0.034* (0.020)	0.023 (0.021)
Log GNP per capita PPP (\$ constant 95)	-0.011 (0.008)	0.019** (0.009)	-0.001 (0.012)	-0.014* (0.008)	0.020** (0.009)	-0.001 (0.012)
South Asia	0.021 (0.039)	0.032 (0.038)	0.026 (0.043)	0.028 (0.039)	0.037 (0.038)	0.029 (0.043)
Latin America and the Caribbean	0.040** (0.019)	0.017 (0.019)	0.009 (0.022)	0.044** (0.019)	0.019 (0.019)	0.012 (0.022)
East Asia	-0.024 (0.024)	-0.016 (0.025)	-0.017 (0.029)	-0.021 (0.024)	-0.016 (0.025)	-0.015 (0.028)
Middle East and North Africa	0.029 (0.019)	0.002 (0.019)	-0.010 (0.023)	0.033* (0.019)	0.002 (0.019)	-0.009 (0.023)
SSA	0.060*** (0.021)	0.049** (0.021)	0.041 (0.025)	0.052** (0.025)	0.037 (0.024)	0.035 (0.028)
Log Urban Pop (% total pop)	-0.017* (0.010)	0.004 (0.010)	0.016 (0.013)	-0.019* (0.010)	0.005 (0.010)	0.016 (0.013)
Log Under 5 Child mortality	.	0.051*** (0.007)	0.047*** (0.009)	.	0.053*** (0.007)	0.047*** (0.009)
Log Primary expenditure per pupil PPP (% of GDP)	.	.	-0.005 (0.010)	.	.	-0.005 (0.010)
Log Prim School pupil-teacher ratio	.	.	-0.011 (0.018)	.	.	-0.008 (0.018)
Constant	0.213*** (0.068)	-0.299*** (0.101)	-0.107 (0.142)	0.247*** (0.067)	-0.313*** (0.102)	-0.113 (0.143)
<i>Number of observations</i>	291	274	206	291	274	206
<i>Overall R squared</i>	133	129	113	133	129	113

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

Because the Sachs & Gallup (2001) malaria measures are available for 1946, 1966, 1982, 1994 and the output measures for five-year intervals from 1960 to 1995, we matched the input measures with outputs in the nearest year for which the malaria measure is available. Completion rates are not available for this period. The results show similar patterns. However the coefficients seems to be lower than in our cross-country analysis. This may be due to the fact that we have no time-variant instrument for malaria for this period of analysis and thus the coefficient are certainly biased downward.

B. DALY rates (2002) as an index of malaria burden

TABLE II.4 : 2SLS Regressions of Primary Repetition and Completion Rates on malaria.

	2SLS Regressions							
	<i>Primary Repetition Rates (2002)</i>				<i>Primary Completion Rates (2002)</i>			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Age-std DALY rates (per 100,000):								
Malaria	0.020*** (0.007)	0.021*** (0.005)	0.024** (0.011)	0.022*** (0.006)	-0.034* (0.021)	-0.040** (0.019)	-0.017 (0.028)	-0.040** (0.019)
HIV/AIDS	0.002 (0.003)				-0.007 (0.008)			
Schistosomiasis		0.045*** (0.013)				0.017 (0.036)		
Hookworm disease			-0.001 (0.002)				-0.007 (0.005)	
Nutritional deficiencies (Protein-energy malnutrition, Iodine deficiency, Iron-deficiency anaemia)				-0.005 (0.012)				0.008 (0.036)
Other Controls								
Log Urban Pop (% of Total Pop)	-0.717 (1.721)	-0.748 (1.241)	-1.517 (1.542)	-1.328 (1.275)	10.801** (4.849)	12.793*** (4.598)	10.743*** (3.995)	12.507*** (4.409)
Log GDP Per Capita PPP (\$ constant 2005)	0.030 (1.236)	-0.034 (1.223)	-0.921 (1.604)	-0.645 (1.329)	-1.711 (4.107)	0.094 (3.992)	-4.196 (3.822)	0.013 (3.802)
Log Publ Expend In Educ (% of GDP)	2.342 (2.724)	2.861 (2.645)	2.200 (2.896)	2.110 (2.839)	6.566 (6.381)	7.189 (6.803)	6.116 (5.306)	7.166 (6.935)
Log Primary Expend per Pupil	-2.954 (2.622)	-2.558 (2.637)	-2.816 (3.047)	-2.654 (2.794)	-5.444 (5.024)	-6.245 (5.635)	-7.630 (5.013)	-6.329 (5.610)
Log Prim School Pupil-Teacher Ratio	0.533 (2.820)	1.328 (2.866)	0.449 (3.897)	0.542 (3.496)	-3.173 (5.937)	-4.230 (7.002)	-10.361 (8.326)	-3.696 (8.361)
Government effectiveness 2002	-0.734 (0.901)	-0.928 (0.812)	-0.452 (0.979)	-0.456 (0.995)	2.518 (2.793)	1.997 (2.898)	3.299 (2.633)	1.960 (2.797)
Intercept	7.662 (21.449)	4.481 (20.330)	20.339 (31.348)	16.814 (24.185)	82.853 (51.924)	61.161 (47.012)	133.854** (63.993)	61.054 (48.374)
<i>Number of observations</i>	65	65	65	65	64	64	64	64
<i>R-squared</i>	0.661	0.684	0.644	0.649	0.633	0.610	0.691	0.610

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
4. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

Because the sample size is maximum 65 observations, we only introduced a limited number of control variables in the regression. Moreover, some health variables are highly correlated (particularly malaria and hookworm disease, $r=0.80$; and malaria and HIV/AIDS, $r=0.79$). However, the effect of malaria on both primary repetition and completion rates seems to be robust to the change of malaria measurement. An increase of the DALY rate of 1 per 100,000 leads to an increase of 2% of the primary repetition rate and to a decrease of 3% of primary completion rate. We also included a variable for public health expenditure in these regressions, as this variable is available for 2002. The effect of public health expenditure (not presented here) does not change previous conclusions and is almost significant at the 10% level and positive in regressions (5) to (8) but not in regressions of primary repetition rates.

C. Using MAP (2007) to compute a malaria index

The following Table II.5 presents the results with the updated malaria index from MAP (2007). Because the dataset is more recent some variables are still uncomplete and have very few observations. They have been dropped from the analysis - it was the case for primary current expenditure per pupil (PPP) and public current expenditure in primary education as a percentage of GNP. As these variables had no significant effect in the previous analysis we suppose that they are controlled through the primary school pupil-teacher ratio, which is well documented. All other variables have been kept in the models. The malaria index used here and the Malaria Atlas Project have already been described earlier. The malaria index is the percentage of population at risk of *falciparum* malaria in 2007 (Figure 9 of introduction). This index is then decomposed into two indexes:

$$\% \text{ Tot Pop at risk of } falciparum \text{ malaria in 2007} = \% \text{ Tot Pop at risk of } falciparum \text{ malaria in 2007 living in stable transmission areas} + \% \text{ Tot Pop at risk of } falciparum \text{ malaria in 2007 living in unstable transmission areas}$$

According to Guerra et al. (2008), 1 billion people lived under unstable or extremely low malaria risk and 1.37 billions lived under stable and high malaria risk in 2007. Hence, we first use the global index of malaria separately and next we test the robustness of this index by introducing in the regression the % Tot Population at risk of *falciparum* malaria in 2007 living in stable transmission areas. This allows us to test the relative contribution of stability of the malaria burden.

We find similar patterns in the results as compared with our previous analysis for the *falciparum* malaria global index for both OLS and 2SLS analysis. However, it seems that the % of Population at risk of *falciparum* malaria living in stable transmission areas in 2007 drives our estimates of the coefficient associated with the *falciparum* malaria global index. Indeed, the partial effect of the *falciparum* malaria index is not statistically significant anymore once associated with the “*falciparum* malaria index – stable area” (regressions (2), (4), (6) and (8)), whereas the partial effect of the latter remains statistically significant. Holding other factors included in the models fixed and particularly the percent of population at risk of *falciparum* malaria in a country, countries with a high proportion of population living in stable transmission areas have repetition rates in primary education 13% higher than countries without stable transmission areas. This means that countries with relative higher proportions of population living in stable transmission areas (versus unstable areas) are more affected by the effects of malaria on human capital accumulation.

TABLE II.5 : OLS and 2SLS Regressions of Primary Repetition and completion rates on malaria MAP index.

	Dependant variable is Primary Repetition Rate (2007)				Dependant variable is Primary Completion Rate (2007)			
	OLS		2SLS		OLS		2SLS	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Updated <i>falciparum</i> Malaria Index 2007 (% of Tot pop at risk, stable and unstable areas)	0.054* (0.031)	-0.065 (0.054)	0.139* (0.070)	-0.068 (0.074)	-0.334*** (0.081)	-0.051 (0.151)	-0.960*** (0.236)	0.483 (0.302)
Updated <i>falciparum</i> Malaria Index 2007 - Stable Area (% of Tot pop at risk)		0.135*** (0.045)		0.139* (0.080)		-0.314** (0.136)		-0.908*** (0.305)
Log GNP per capita PPP (\$ constant 2007)	0.003 (0.016)	0.003 (0.014)	0.013 (0.018)	0.003 (0.014)	0.024 (0.032)	0.023 (0.029)	-0.051 (0.057)	0.020 (0.030)
Subtropics (% land area)	0.041* (0.023)	0.044** (0.022)	0.022 (0.031)	0.044** (0.021)	0.132** (0.060)	0.124** (0.059)	0.270** (0.104)	0.109 (0.069)
Tropics (% land area)	-0.004 (0.031)	-0.005 (0.028)	-0.045 (0.038)	-0.005 (0.028)	0.110 (0.097)	0.103 (0.090)	0.426*** (0.139)	0.089 (0.099)
Log Urban Pop 2007 (% total pop)	-0.004 (0.005)	-0.006 (0.005)	-0.003 (0.005)	-0.006 (0.005)	-0.001 (0.014)	0.007 (0.013)	-0.021 (0.021)	0.021 (0.016)
Log Under 5 Child mortality 2007	0.003 (0.010)	-0.003 (0.010)	-0.003 (0.011)	-0.003 (0.011)	-0.025 (0.032)	-0.010 (0.031)	0.032 (0.051)	0.018 (0.038)
Log Prim School pupil-teacher ratio 2007	0.041* (0.022)	0.038* (0.020)	0.033 (0.022)	0.038* (0.019)	-0.086 (0.069)	-0.084 (0.064)	-0.013 (0.081)	-0.079 (0.063)
Government Effectiveness 2007	-0.007 (0.012)	-0.013 (0.012)	-0.010 (0.012)	-0.014 (0.013)	-0.031 (0.027)	-0.018 (0.027)	0.027 (0.047)	0.007 (0.033)
Intercept	-0.119 (0.185)	-0.081 (0.171)	-0.178 (0.196)	-0.080 (0.165)	1.068*** (0.400)	0.991*** (0.362)	1.495** (0.611)	0.845** (0.369)
Number of observations	86	86	86	86	87	87	87	87
R squared	0.573	0.611	0.532	0.611	0.690	0.714	0.467	0.629

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
4. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

V. Conclusion

A. Malaria and primary education quality at the macroeconomic level: main results

Our cross-country and panel regression analysis has shown that the link between the level of *falciparum* malaria endemicity and primary repetition rates is strong and positive. The link between the level of *falciparum* malaria endemicity and primary completion rates is strong and negative. These results suggest that malaria contributes to impede children's human capital accumulation at the macroeconomic level.

Nevertheless, one may be careful when interpreting the results presented in the previous section. Why is there such a strong link? As we already explained it in the introductory chapter, malaria affects specifically child's cognitive and learning abilities and consequently school results. Our conjecture was that high repetition rates and low completion rates reflect particularly bad school results. Therefore, the strong link between malaria and primary repetition and completion rates reflects the impact of malaria on school results and does not imply that similar relation exists systematically between malaria and other educational outcomes. We did not explore here the relationship between *falciparum* malaria and other educational indicators.

Moreover, malaria is a complex economic and social phenomenon. It is very difficult to dissociate the direct medical impact of the disease from its numerous effects on society and human organization. In addition to its medical effects, the behavioural and social aspects linked with malaria may explain part of the estimated impact of malaria on repetition and completion rates given in this paper.

A major problem when assessing the impact of malaria on education is also the lack of good data. Our conclusions are valid in the limitation of data precision and availability. Some variables that may be expected to influence education outcomes or bias the coefficient values of the malaria index were not included because of the lack of cross-country data. We tried to account for this problem using different specifications of the regression model and different estimation techniques but it is still possible that variables correlated with the malaria index have been omitted. Besides, the conclusions we have made are only valid at the macroeconomic stage and are not a substitute for detailed country analysis or case studies. Microeconomic evaluations of the impact of malaria on school performances may differ from macroeconomic previsions.

Some variables included in the regression models have ambiguous effects. Notably, the GNP per capita has a positive effect on primary repetition rates in some regressions. However, this effect is not robust to different specifications of the model and to the use of actualised data. In regressions where GNP per capita is not associated with under-five mortality rate or the pupil-teacher ratio its effect is insignificant or negative (regression (4) of Table II.3 for instance). This may be due to the high correlation between GNP per capita, the malaria index, the under-five mortality rate and the pupil-teacher ratio. A discussion on potential multicollinearity is provided in Appendix II.2. The results are not changed when some variables are removed from the model (Table II.A2 and Table II.A5). Recent empirical literature on this topic suggests unstable effects of GNP or GDP per capita on education quality at the macroeconomic level. In Al-Samarrai (2006) this variable has a negative but non-significant effect on primary completion rate for instance. In Barro & Lee (2001)

the GDP variable is insignificantly associated with mathematics and science test scores but significantly associated with reading scores (positively) and repetition rates (negatively). Therefore our analysis casts serious doubts on the relevance of using macroeconomic data to study such important development issues because of data heterogeneity and quality. The same scepticism and criticism addressed to Sachs & Gallup (2001) or Acemoglu and colleagues with respect to data quality hold for this Chapter.

However, our results on the link between *falciparum* malaria and primary repetition rates suggest that the achievement of the education millennium development goals (in particular aspects concerning education quality) will require more than just focusing on expenditure in primary education. The results have been shown to be robust to a series of tests (different specification models, controls for influential observations, different estimation techniques, different dataset and different years). These results seem to provide a good idea of the burden at the macroeconomic stage. Regressions with completion rates also suggest that there are other externalities linked with the disease, as the impact is even higher. Hence our macroeconomic results remain useful to cast a first glance at the relationship. Nevertheless, because of questionable macroeconomic data quality discussed previously, these results are obviously insufficient to conclude that a *causal* relationship exists between these variables. It is possible that we are asking questions that are too subtle for the available data to answer with good precision.

B. The impact of public spending on education and health: does money matter?

There have been relatively few studies exploring the relationship between resources and health or educational outcomes at the macroeconomic level. Works that have been undertaken on this issue have mainly involved micro-level studies. At the microeconomic level, non-experimental studies show that there is not a strong or consistent relationship between student performance and school resources (Hanushek, 1997, 1995). Numerous randomized studies evaluating the effects of school resources (additional teachers, provision of additional official textbooks, flip charts) on educational achievement confirm that higher resources do not increase test scores for the average student (Glewwe et al., 2009; Kremer, 2003). Empirical microeconomic studies on the cost-effectiveness of primary preventive and curative measures suggest that under-five deaths could be reduced considerably at reasonable costs. However, studies using international dataset failed to establish a strong effect of public spending on both health (as suggested in Chapter 1) and educational

outcomes (Al-Samarrai, 2006).

Hence, our results on public spending are not a surprise. One explanation could be that data on public education expenditure and education are not comparable across countries. Our cross-country regressions could also suffer from unobserved heterogeneity that might be correlated with the variable of interest and not corrected adequately by our instrumentation strategy. However our results seems to be in keeping with the literature. It does not imply that resources in education are unnecessary but that increasing resources in education or improving education resources management alone are unlikely to be sufficient. Simply providing more resources in developing countries without focusing on weakest students, improved teacher incentives, and better budget allocation, will have a limited impact on school quality (Kremer, 2003). To the question: “*What are the most effective ways to increase primary school student learning?*”, we answer that diseases that alter cognitive capacities of children such as malaria should be taken into account much more seriously. This conclusion seems to be obvious but has not been the subject of careful researches in developing countries. Improving educational outcomes and especially school results in developing countries will require more attention on child health.

This chapter sees the need to place emphasis on the interactions between health and education policies in developing countries. In some cases, it might be more effective to invest in some specific health programs to improve educational quality rather than increasing public expenditure in education. Reciprocally, improving education could be one of the components that will improve the quality of interventions to prevent diseases like malaria. The impacts of school-based malaria control programs are still little known whereas education is one of the determinants of knowledge about malaria. Sharma et al. (2003) have shown that improvement in literacy and educational status will help in increasing the level of knowledge about various aspect of malaria. Knowledge about malaria and prevention measure is also one of the determinants of the use of insecticide-treated nets.

The following chapters will try to profundize the link between malaria and primary education at the community and individual levels.

**CHAPTER III: FEVER, MALARIA AND PRIMARY
REPETITION RATES IN MALI: COMBINING DHS DATASET
AND SPATIAL MALARIOLOGICAL MEASURES¹²**

¹² This chapter is based on a study under revision in *Social Science & Medicine*.

The Malian education system has been in crisis since the 1990s (Dumestre, 2000). Reasons behind this endemic crisis are numerous, involving political, social, financial and economic factors (Diakit , 2000). Within this context, the relationship between health and school has not been studied carefully despite the fact that schools are often squalid and schoolchildren do not have the access to appropriate healthcare to continue their studies under adequate conditions. The bidirectional relationship between health and education may be another neglected reason for the difficulties of numerous educational systems and the lack of educational achievement in Africa. As Chapter 2, this chapter focuses on one particular aspect of this relationship: the impact of malaria, one of the most common diseases in Mali, on repetition rates, an indicator of school performance. Indeed, the fight against malaria is far from over, and it is still difficult to understand why malaria has such an impact on economic development. This chapter also provides a new methodological approach to the use of malaria measures in economic assessments.

The chapter is structured as follow: Section 1 describes the data and methodology used to construct a database at the cluster level and provides a critical discussion about the use of fever as an index of malaria in endemic areas. Section 2 presents the results of the research. Finally, we conclude and discuss the main findings of this study in section 3. **Our primary hypothesis is that malaria prevalence is an important determinant of group-level educational outcomes.**

I. Data and Methodology

A. Defining the Cluster Level

The data used in this chapter are from Demographic and Health Surveys conducted in Mali (MDHS) in 2001 and 2006. The MDHS consist of a national representative household survey. The 2001 and 2006 MDHS have a sample size of 12,331 and 12,998 households. The primary sampling unit of the MDHS, or cluster, is defined as one enumeration area (EA) developed for the 1998 population census by the “Direction Nationale de la Statistique et de l’Informatique du Mali”. The largest EAs are divided before sample selection begins. The sample was selected using a stratified, two-stage cluster design. Once the number of households was allocated to

each administrative region by urban and rural areas, the number of clusters was calculated based on the 1995/1996 MDHS response rate for the 2001 MDHS and on the 2001 MDHS response rate for the 2006 MDHS. Then, clusters were selected proportionately to their size (number of households). For the 2001 MDHS, a total of 403 clusters were selected, 128 in urban and 275 in rural areas. For the 2006 MDHS, a total of 407 clusters were selected, 147 in urban and 260 in rural areas. Clusters interviewed are not the same in both surveys. In each selected cluster, a complete household listing operation was carried out and households were randomly selected to achieve a fixed sample take per cluster (for instance, 25 households in urban areas and 40 households in rural areas in the 2001 MDHS).

Because the MDHS sample are unbalanced by urban-rural residence and administrative regions, they require sampling weights to provide estimates at these domains of study but not necessarily at the cluster level, as households are randomly selected in each cluster. Moreover, the use of sample weights is inappropriate for estimating relationships such as regressions and correlation coefficients (Rutstein & Rojas, 2006). In this chapter, we focus on information at the cluster level because DHS do not provide health variables for school-age children. We compute indicators from individual or household surveys in order to conduct a cross-sectional multivariate analysis at the cluster level. We assume that the cluster level can be assimilated to a community level, and we do not use DHS population weights in this analysis.

B. Defining cluster health conditions

MDHS surveys do not provide health indicators for school-age children. The only health variables that are well documented are for children under the age of five. Therefore, if one wants to assess the effects of health on school-age children with national surveys, one has to find alternative strategies and use some proxies. The use of health variables of children under five can be justified by two arguments. Firstly, health variables of children under five are supposed to be good proxies for general health conditions in the cluster. Secondly, it is possible that pre-school health conditions affect school achievement. This point has been discussed in General Introduction. However, in both cases, one strong assumption is that pre-school health conditions are stable over time.

C. Methodological considerations on Malaria measurement

1. Fever Incidence

DHS children's recode files contain health data collected for the children born in the month of interview and the 59 months prior to the survey. The data for the mother of each of these children is also present. Questions examine immunization coverage, vitamin A supplementation, and recent occurrences of diarrhea, fever, anthropometric indicators and treatment of childhood diseases for young children.

Fevers in children can fall into three main categories (Larson et al., 2006): “(a) *uncomplicated fevers treated at home and/or through outpatient services at a health care facility and clinically resolve; (b) complicated febrile events that progress to life-threatening conditions such as cerebral malaria and severe malaria anaemia, requiring hospital care that if effective will result in cure; (c) febrile event that lead rapidly to death because of rapid pathological progression before reaching tertiary care or failed treatment in tertiary care.*” In this study, we used the DHS children's recode dataset to build an index of fever prevalence in children from 0 to 59 months at the cluster level. The questionnaire asks to the mother whether the child had an episode of fever in the two weeks prior the interview. The Fever index used here is then defined as the percent of children less than 59 months in the cluster who were reported to have fever in the 2 weeks prior the interview.

2. Malaria incidence and fever: some methodological considerations

In Mali, malaria is endemic among more than 90% of the total population. We first proposed to consider fever prevalence as a proxy of malaria prevalence in a cluster because DHS does not provide individual malaria parasitaemia or other malaria parameters in Mali. As indicated before, the number of fever cases (managed as malaria) is often used as a representative measure of the burden of the disease in a country. Death is considered relatively infrequent compared to illness. Moreover, self-reported 14 days history of fever is a commonly used measure in standardised surveys of child health.

However, most fevers are not a direct consequence of malaria infection and the relationship between fever and *Plasmodium falciparum* depends strongly on the season and area affecting the malaria-attributable fraction of fever cases. In the district of Bandiagara, an endemic area of Malaria in Mali, it was found that the overall proportion of fever attributable to malaria parasitaemia was only 33.6% during the rainy season and 23.3% during the dry season, with the highest proportion

occurring among the youngest children. Among children aged from 6-59 months, the malaria-attributable fraction of fevers was in average 53.9% during the rainy season and 30.7% during the dry season (Dicko et al. 2005).

The economic effects of malaria are likely to differ both qualitatively and quantitatively according seasons, endemicity and stability. For instance, in an area of low malaria endemicity in Senegal, during the period of maximum transmission, clinical malaria prevalence was 1.36% and malaria was responsible for 36% of school absences due to medical reasons. At the end of the period of minimum transmission, clinical malaria prevalence was 0.15% and malaria was responsible for 3% of school absences due to medical reasons (Trape et al., 1993). Most of the interviews of the MDHS 2001 have been made during the dry season in Mali (supposed low malaria transmission season). The MDHS 2006 has been made during the rainy season (supposed high malaria transmission season).

Figures III.1, III.2, III.3 and III.4 show that it can be misleading to use fever as an indicator of malaria, particularly during the dry season. For instance, during the dry season, the prevalence of fever in Kidal was 50% according to our personal calculations from MDHS 2001 (that corresponds to the MDHS reports) whereas Kidal is an epidemic area with a very low percentage of population at risk according to the Mapping Malaria Risk in Africa/Atlas du Risque de la Malaria en Afrique (MARA/ARMA) database (<http://www.mara.org.za/>, accessed in August 2008). The MARA/ARMA project does not provide data for Bamako or other cities in Mali but it has been documented elsewhere. Bamako and Mopti are considered as hypoendemic areas (Dumbo, 1992). In an urban area in Côte d'Ivoire (Abidjan) the fraction of malaria-attributable fever was only 22% among the 1 to 5 years-olds (Wang et al., 2006). In Benin, at the end of the dry season, the malaria-attributable fraction among presenting fever cases was only 4% among 1 to 5 years-olds (Wang et al., 2006).

Moreover, in Kenya during the rainy season, according to Clarke et al. (2004) malaria-attributable fraction of fevers among school-aged children was higher (60%) in an epidemic area (Nandi Highland) than in a holoendemic area (only 31%). In a community exposed to low level of malaria transmission in Madagascar highlands, a 4-year follow-up found a malaria-attributable proportion of fevers of 36.29% and the malaria transmission season was found to be significantly related to the risk of fever (Boisier et al., 2002). Therefore, the relationship between age, parasite density and occurrence of fever is clearly influenced by seasons and epidemiologic facieses.

Results from the literature suggest that it may be more accurate to use the 2006 MDHS than the 2001 MDHS in our analysis if we want to use fever as a proxy of malaria. Nevertheless, as we saw, even during the rainy season (RS), fever proportion can be a biased indicator of malaria intensity. It follows that fever is not a reliable

proxy for malaria in large cross-sectional analysis. Using reliable malariological measures is fundamental to estimate the scale of the problem and the resources needed to control malaria. The number of studies using fever or fever treatment to make recommendations to malaria control policy makers is innumerable (see for instance Filmer, 2005 or Njau et al., 2006) and can lead to misleading recommendations. Future investigations on malaria health policy should consider these points more seriously. In the next subsection, we propose to use a different proxy of malaria in economic assessments.

FIGURE III.1: Fever Prevalence by regions: author's calculation from DHS Dataset
(source: author from MDHS dataset)

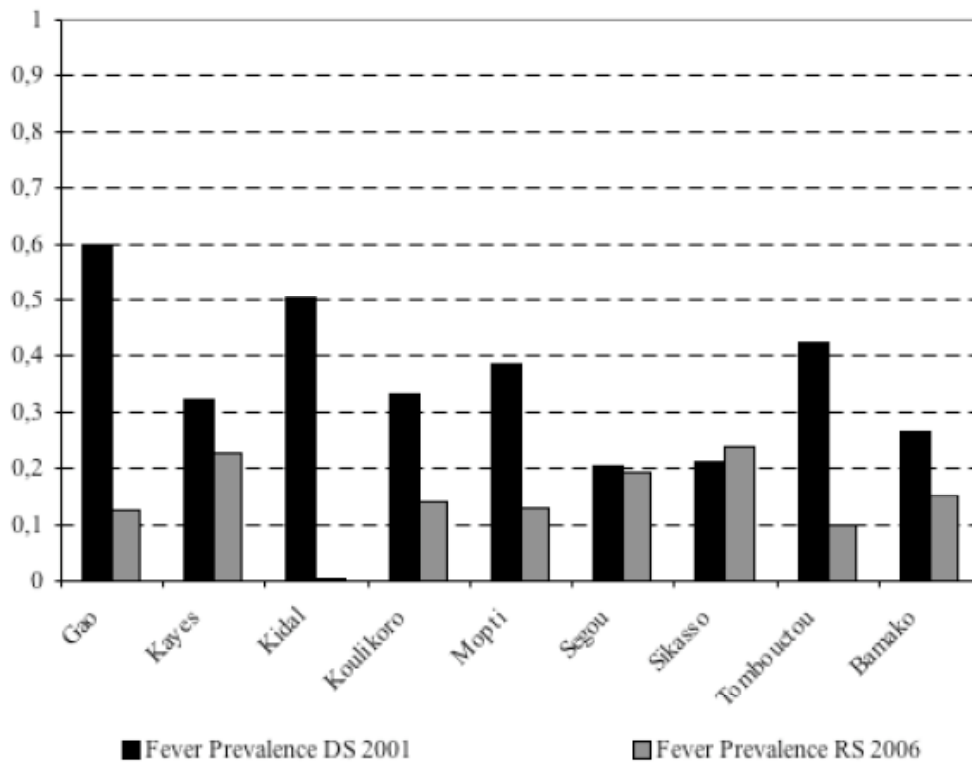


FIGURE III.2: Fraction of Population living in malaria stable areas by regions
(source: author from from MARA/ARMA)

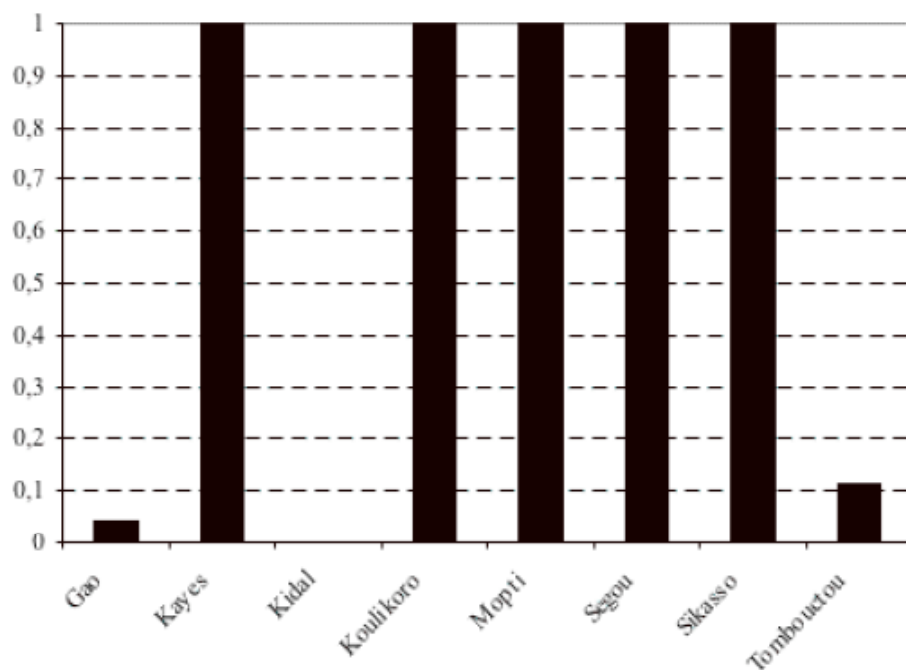


FIGURE III.3: Fraction of Population at risk given Malaria Prevalence by regions
(source: author from from MARA/ARMA)

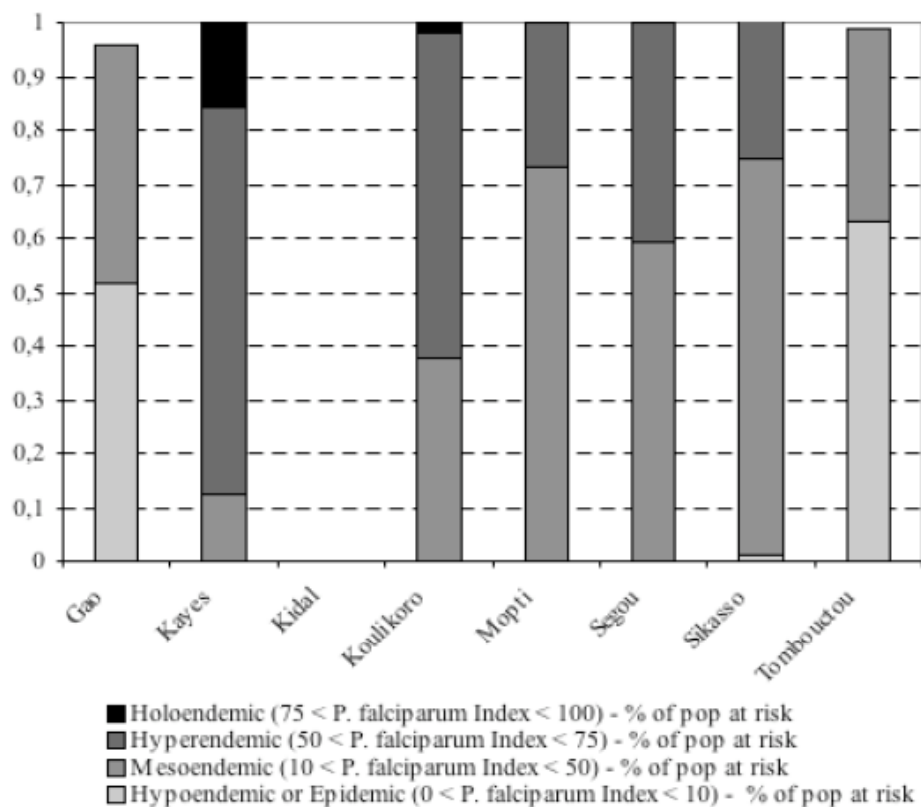
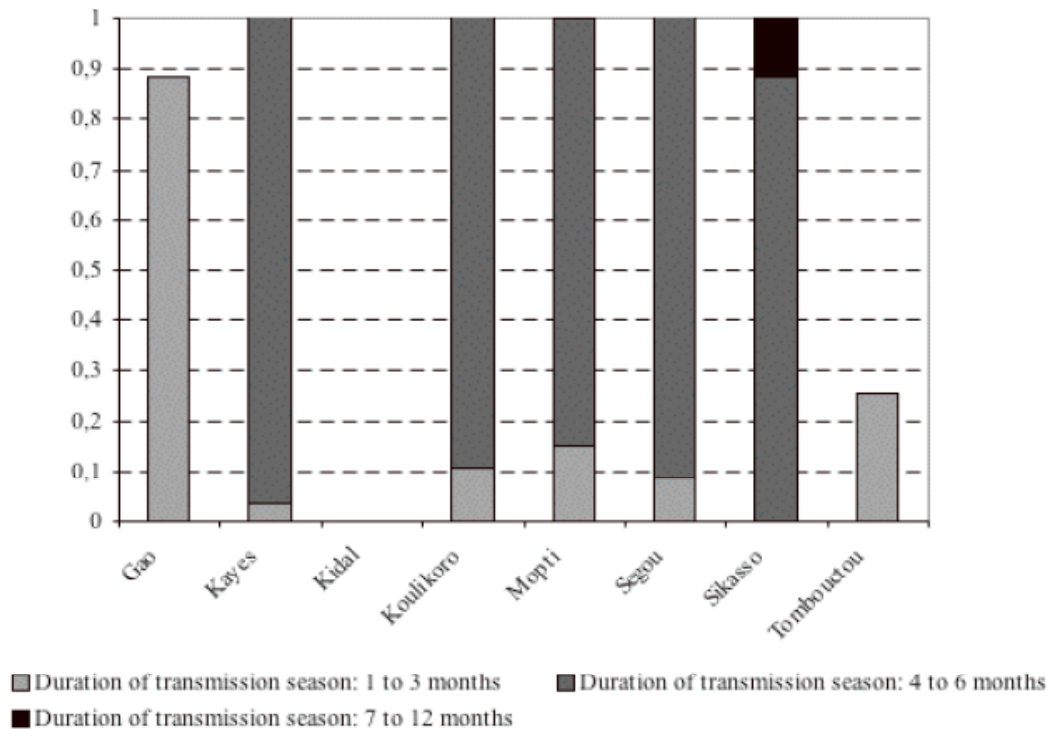


FIGURE III.4: Fraction of Population at risk given transmission season by regions
(source: author from from MARA/ARMA)



3. Using reliable Malaria Maps of malaria prevalence and geographic information system to compute malaria indices for economic assessments

Figures III.1, III.2, III.3 and III.4 provide a useful insight about malaria distribution by regions in Mali. However, malaria endemicity can vary widely over only short distances. Moreover the MARA/ARMA data presented here are not age specific. Symptomatic malaria is supposed to decline with age in hyperendemic as well as in low endemic areas. In a recent study, Gemperli et al. (2006) proposed to estimate malaria prevalence at unsampled locations by incorporating information from environmental covariates in spatial and geostatistical Bayesian models. They used mathematical models based on the Garki Project combined with smoothed data from the MARA/ARMA database, the Topographic and Climate database for Africa, the normalized difference vegetation index from the NOAA/NASA Pathfinder AVHRR Land Project database and historic surveys conducted by the Malaria Research and Training Center in Mali, to predict malariological measures. The data cover various years and various age-group ranges. The models used were validated against observed field-based data, have small prediction error and good spatial resolution. The spatial predictions given by the authors are particularly adapted to our analysis because they provide the annual Entomological Inoculation Rate (EIR)

and age-specific prevalence of malaria among children in Mali (malaria prevalence for children under five years, malaria prevalence for children from two to ten years of age). As geographic information is collected in the DHS surveys, it is possible to extract the spatially referenced malariological measures for the MDHS clusters, using geographic information system (MapInfo Professional 9.0, Pitney Bowes MapInfo Corporation, NY, USA) and the malaria maps computed by Gemperli et al. (2006). Figures III.5, III.6 and III.7 provide the map used to compute the spatial malaria indices from Gemperli et al. (2006). Figure III.8 gives the distribution of fever for children under-5 by categorized areas of malaria prevalence for children under-5 during the dry season (2001 MDHS) and the rainy season (2006 MDHS). Prevalence thresholds are given *a priori* by Gemperli et al. (2006). This figure confirms that fever is a better proxy of malaria during the rainy season than during the dry season. Studies that are using fever as a proxy for malaria should take into account this seasonal variation and rely more on surveys made during the rainy season. However, it does not mean that fever remains a good proxy for malaria as the percent of fever attributed to malaria is never 100% and can vary by areas during the rainy season as well as during the dry season.

FIGURE III.5: Predicted Annual Entomological Innoculation Rate (*source: Gemperli et al., 2006*)

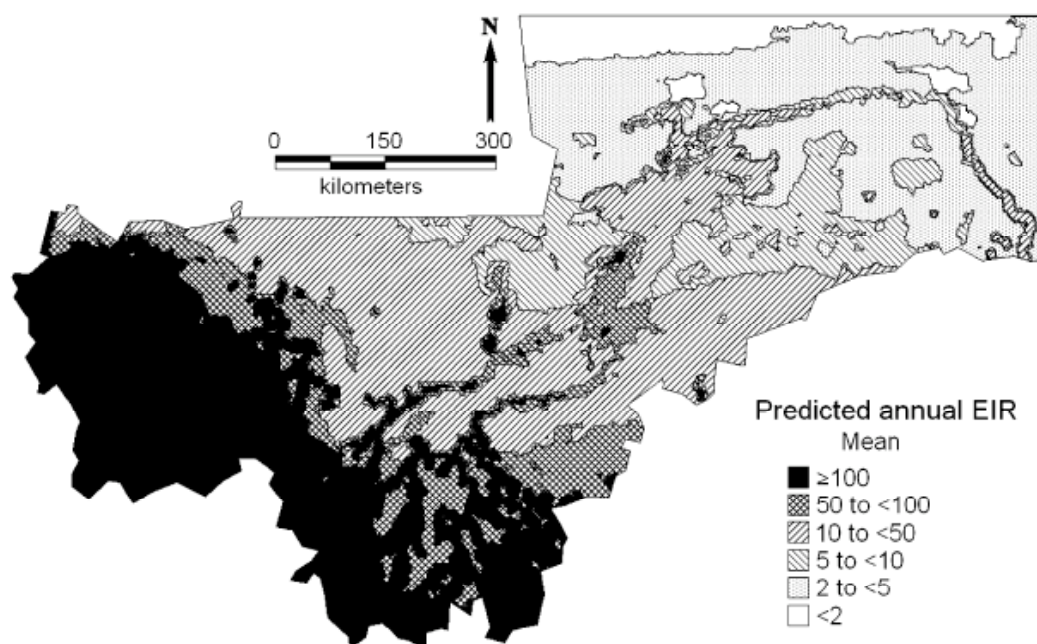


FIGURE III.6: Predicted malaria prevalence <5 years of age (source: Gemperli et al., 2006)

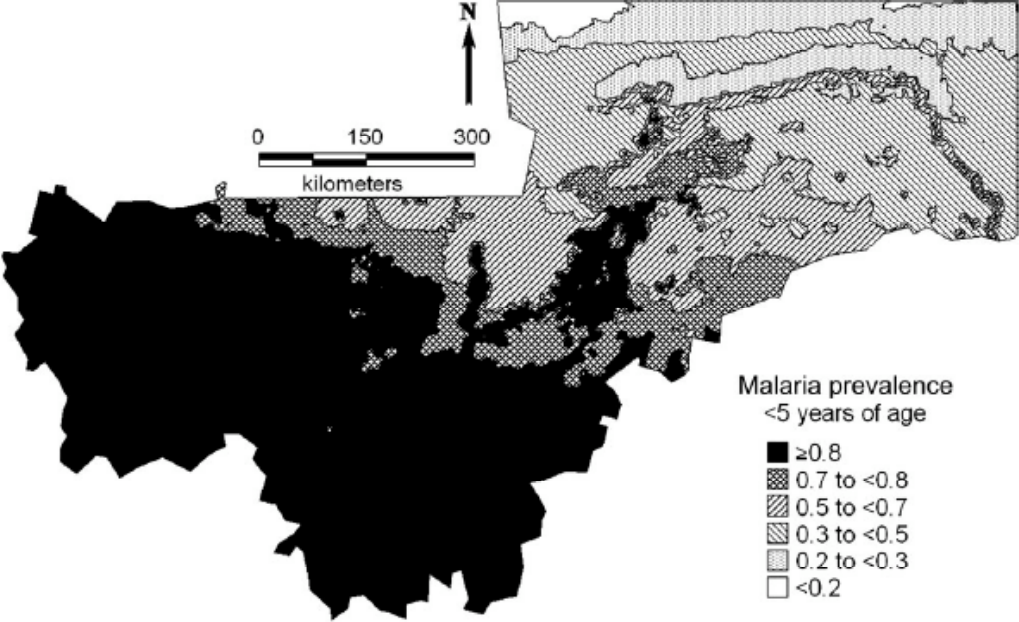


FIGURE III.7: Predicted malaria prevalence 2-10 years of age (source: Gemperli et al., 2006)

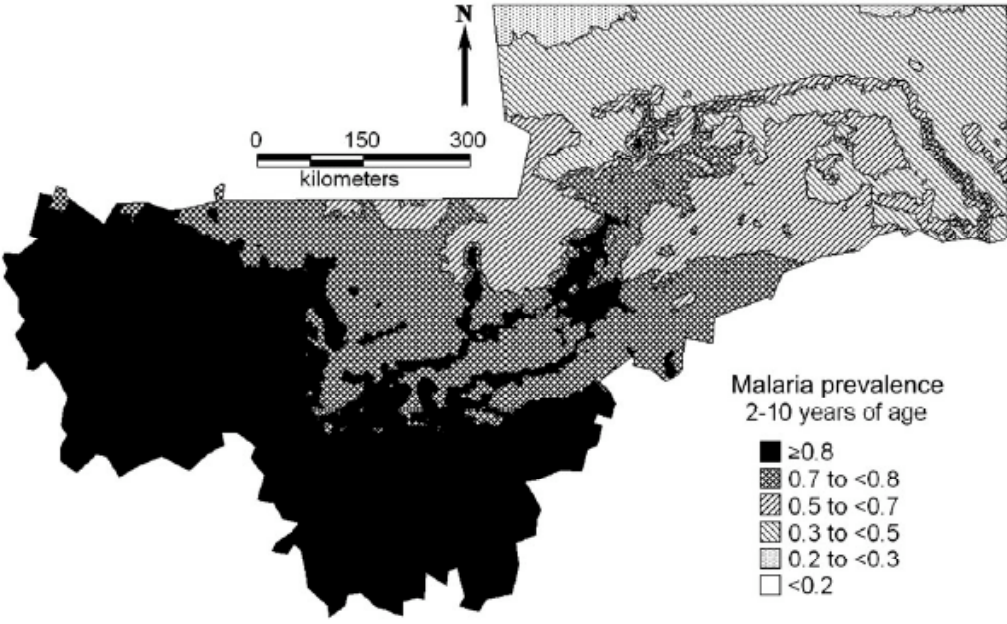


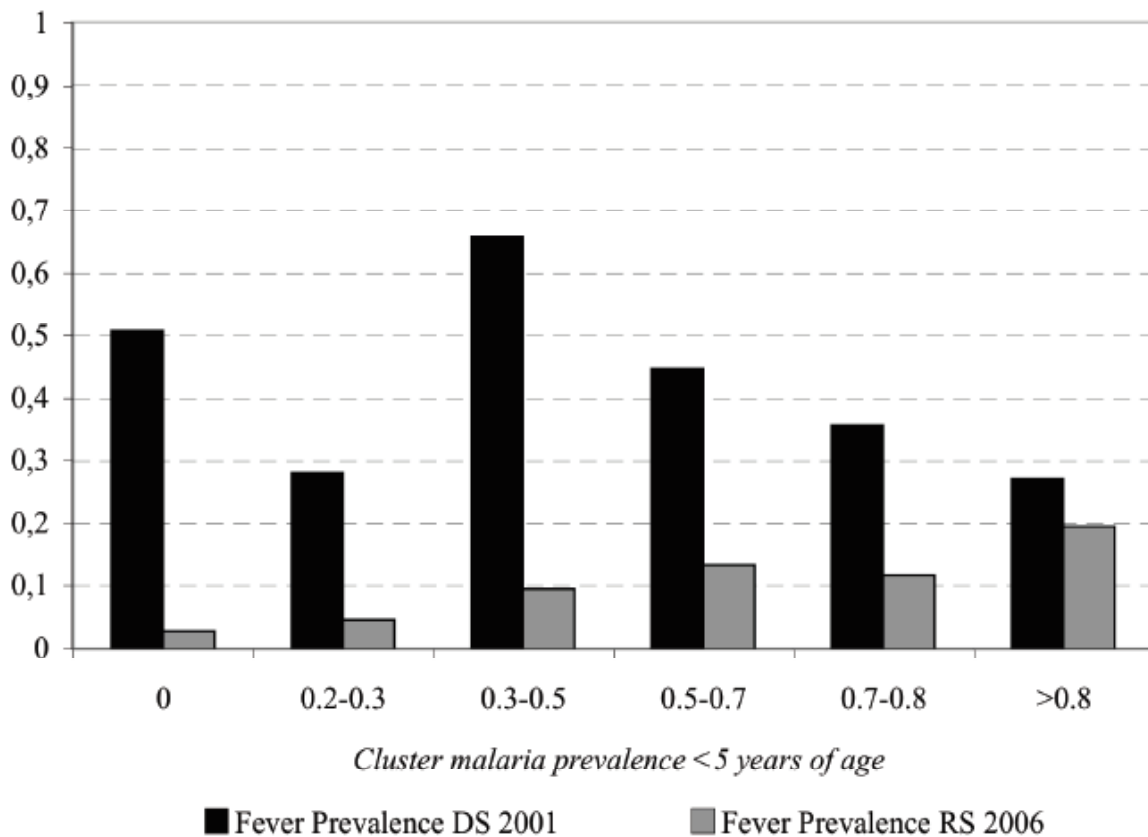
FIGURE III.8: Under-5 Fever Prevalence and Under-5 malaria Prevalence (*source: author*)


TABLE III.1: Pairwise correlation between Fever Prevalence and Spatial Malariological Measures

	Cluster Malaria Prevalence <5 years of age	Cluster Malaria Prevalence 2-10 years of age	Cluster Entomological Inoculation Rate	Cluster Prop of Fever <5 years of age
2001 MDHS				
Cluster Malaria Prevalence <5 years of age	1.0000			
Cluster Malaria Prevalence 2-10 years of age	0.8949	1.0000		
Cluster Entomological Inoculation Rate	0.8034	0.8927	1.0000	
Cluster Prop of Fever <5 years of age	-0.3674	-0.3371	-0.3439	1.0000
2006 MDHS				
Cluster Malaria Prevalence <5 years of age	1.0000			
Cluster Malaria Prevalence 2-10 years of age	0.9268	1.0000		
Cluster Entomological Inoculation Rate	0.8547	0.9132	1.0000	
Cluster Prop of Fever <5 years of age	0.3996	0.4250	0.4426	1.0000

4. Measuring other Health Status Components

We used the DHS children's recode files to create other Health indicators at the cluster level as some proxies of the general health conditions in the cluster. We created five health indicators:

- (i) Cluster proportion of children under-5-years severely underweight: weight for age is based on physical measurements taken by interviewers using a standard protocol and is provided for children less than five years in the DHS children's recode database. We calculated the proportion of children in the cluster whose weight for age z-score was less than -3.0 standard deviations below the NCHS/CDC/WHO international reference median. Child weight for age was preferred to child height for age because it is more likely to represent a short-term measure of undernourishment that vary very quickly with environmental influences (Boyle et al., 2006).
- (ii) Cluster prevalence of diarrhea: the questionnaire asks to the mother whether the child had diarrhea in the 2 weeks prior the interview. Therefore, the cluster diarrhea variable is the percent of children less than 59 months in the cluster who were reported to have diarrhea in the 2 weeks prior the interview.
- (iii) Cluster proportion of children with respiratory infection symptoms. This variable was calculated from two questions asked to the mother: whether the child had cough in the last two weeks and whether the child breaths were rapid and short.
- (iv) Cluster proportion of children under-5-years with severe anaemia: haemoglobin level adjusted by altitude in g/dl is provided for children under-5-years. Levels below 7.0g/dl are considered as severe anaemia and in case of severe anaemia, respondents are asked whether this information can be given to a doctor for follow up. The proportion of children with severe anaemia was then computed by cluster.
- (v) Cluster proportion of households having at least one mosquito net as a proxy for malaria control in the community.

D. Measuring Educational Outputs

Another methodological question deals with the measure of educational outputs. It is generally admitted that national statistics on school outputs and particularly school performances are not reliable in Mali (Dumestre, 2000). Here, we used self-reported data of school attendance status to measure educational achievement. Data from the household schedule and members characteristics are available in the DHS Household Member Recode files. This questionnaire includes some questions about enrolment

for the current and previous school years, educational level during current and previous school years, school attendance status (advanced, repeating, dropout) during current year. As the main objective of this study is to assess the impact of malaria on school performances, the DHS Household Member Recode files were used to generate the Cluster primary repetition rates during current school year. This educational output was defined as the number of individuals in a cluster, declared as repeaters according to the DHS school attendance status divided by the total number of individuals in the same cluster that are attending formal primary school during current school year. Cluster progression to secondary (number of new entrants to the first grade of formal secondary education, expressed as a percentage of the number of pupils in the final grade of primary school in the previous year) was also generated but the number of observation was insufficient to interpret the results.

It would have been interesting to use representative standardized tests scores to have different measures of school achievement. The PASEC (“Programme d’Analyse des Systèmes Educatifs de la CONFEMEN”) provides this kind of data for Mali in 2001/2002. Unfortunately, the PASEC dataset does not allow localising geographically the school selected in the survey and does not provide data about malaria prevalence in the community. Repetition rates have been frequently used to compare school performances across countries or regions in international monitoring reports. Recall that we are not interested in testing the impact of malaria on enrolment or dropout because we want to assess specifically the effects of malaria on school achievement.

E. Measuring Socio-Economic Status

A relative index of households socio-economic status (SES) was derived based on 12 dichotomous variables using principal components analysis (see Appendix 2). Long run wealth is supposed to explain the maximum variance and covariance in the asset variables (Filmer & Pritchett, 2001). An average level of the wealth index was constructed for regressions at the cluster level. Results of this analysis at the household level are given in Table III.2 and the cluster mean wealth index is provided in Table III.2.

TABLE III.2: Results of Principal Component Analysis of Household Socioeconomic Status

	All Mali			Impact on PCA score	Means	
	Scoring Factor	Means	SD		Poorest Quintile	Richest Quintile
2001 MDHS						
Floor is made of finish surface	0.360	0.160	0.367	0.980	0.000	0.702
Cooking fuel is electricity / Gas / Kerosene / Charcoal	0.260	0.105	0.306	0.849	0.000	0.370
House has electricity	0.400	0.108	0.311	1.286	0.000	0.516
Source of water is covered well or piped wate	0.261	0.392	0.488	0.534	0.000	0.828
Toilet type is flush Toilet / Pit Toilet	0.177	0.737	0.440	0.402	0.080	0.986
<i>Household owns at least one:</i>						
Radio	0.163	0.677	0.467	0.349	0.401	0.897
Refrigerator	0.388	0.051	0.220	1.763	0.000	0.254
Television	0.393	0.128	0.335	1.173	0.000	0.600
Bicycle	0.020	0.448	0.497	0.040	0.285	0.424
Motorcycle	0.189	0.188	0.391	0.483	0.000	0.457
Car	0.284	0.041	0.198	1.434	0.000	0.397
Telephone	0.302	0.023	0.151	2.000	0.000	0.116
Household Wealth Index	.	0.000	1.975	.	-1.402	3.150
2006 MDHS						
Floor is made of finish surface	0.370	0.225	0.418	0.885	0	0.773
Cooking fuel is electricity / Gas / Kerosene / Charcoal	0.112	0.005	0.073	1.534	0	0.024
House has electricity	0.413	0.166	0.372	1.110	0	0.761
Source of water is covered well or piped wate	0.340	0.248	0.432	0.787	0	0.754
Toilet type is flush Toilet / Pit Toilet	0.215	0.741	0.437	0.491	0.034	0.992
<i>Household owns at least one:</i>						
Radio	0.167	0.696	0.459	0.363	0.471	0.892
Refrigerator	0.331	0.041	0.199	1.663	0	0.207
Television	0.395	0.218	0.413	0.163	0	0.814
Bicycle	-0.027	0.433	0.495	-0.054	0.328	0.337
Motorcycle	0.236	0.294	0.455	0.518	0	0.633
Car	0.270	0.040	0.198	1.363	0	0.178
Telephone	0.304	0.042	0.201	1.512	0	0.206
Household Wealth Index	.	0.000	1.915	.	-1.559	3.237

Notes:

1. Each variable takes the value 1 if true, 0 otherwise.
2. Scoring factor is the "weight" assigned to each variable (normalized by its mean and standard deviation) in the linear combination of the variables that constitute the first principal component.
3. The percentage of the covariance explained by the first principal component is 32% for 2001 MDHS and 30% for 2006 MDHS. The first eigenvalue is 3.90 for 2001 MDHS and 3.66 for 2006 MDHS; the second eigenvalue is 1.44 for 2001 MDHS and 1.38 for 2006 MDHS.
4. Impact on PCA score is the impact of a change from 0 to 1 for each variable (= Scoring factor / SD)

F. Other cluster characteristic

The other advantage of using DHS is that the survey provides an extensive community questionnaire about the availability of facilities at the cluster level and other household characteristics. Thus, we created variables that allow controlling for a set of theoretical relevant determinants of school achievements. This includes pupils' mean characteristics (median age, number of primary school children in the cluster), parental characteristics (cluster median education years) and dummies for regional and rural location.

We expect that the higher the level of education in the cluster is, the higher the children's performance and the lower the repetition rates will be, other things being equal. Note that including average cluster wealth and median cluster education years in the models is all the more necessary, as they are potential channels other than malaria through which our identifying variables could affect repetition rates (see econometric strategy). When the school is located in the cluster, school results are expected to be higher because travel costs linked with school attendance may be lower and because this variable can reflect community involvement in children's education. Rural location is a potential influential factor of primary repetition. Indeed, in urban areas, school results are expected to be higher because children can have better access to educational inputs. The cluster median age of primary school children and the number of primary school children in the cluster can be important determinants of school and regulation policies that could affect primary repetition rates.

Descriptive statistics about variables included in the multivariate analysis are provided in Table III.3. Because there were no available data about school resources, school enrolment fees or pupils/teachers ratio in the DHS questionnaire, we did not introduce these variables in our models. However, 95.7% of the schools in the data were either governmental schools or community schools, and a great number of studies find no support for the effects of school resources on school achievement (Al-Samarai, 2006). Therefore, we do not think that our conclusions are affected by this lack of data.

II. Econometric strategy

Data were analysed using Stata Version 9.2 (Stata Corporation, Austin, TX, USA). As a panel of data was not available, the method used first is Ordinary Least Squares

(OLS) regressions (Table III.4). We include a variety of controls for potentially confounding characteristics. Second, the malaria indices are exogenous in a linear regression framework provided that they are not correlated with the error term. They can be endogenous either because of an omitted variable, measurement error or simultaneity.

Measurement errors in an explanatory variable can lead to attenuation bias if the measurement error is uncorrelated with the unobserved explanatory variable. IV procedure can be used to solve the so called “classical measurement error problem” if the instrumental variable of the observed variable is uncorrelated with the measurement error. Standard methods for correcting for measurement error bias, such as instrumental variables estimation, are valid when errors are classical and the underlying model is linear, but not in general. Moreover, many econometric estimators are consistent but may be biased in finite samples. Using iterative procedure, bootstrap method will not fully eliminate the bias but one may use them to obtain an estimate of this bias and compute robustness checks. There are actually two uses for bootstrapping techniques. The first one is the estimation of sampling distribution and standard errors. The empirical standard deviation of a series of bootstrap replications of the estimator can be used to approximate the standard error of this estimator. The second one is related to correction of biases. The intuition of bootstrapping methods is rather simple. We suppose that the observed distribution we have of the data is the best estimate of the real probability distribution of the data. We use the empirical distribution of our data to generate (with a nonparametric bootstrap procedure) a large number of new dataset (2000 replications). We apply our OLS estimator to each dataset, which provides us an approximation of the sampling distribution of the estimator and bias-corrected confidence intervals.

Indeed, the proportion of fevers can clearly suffer from measurement errors as a proxy of malaria or because of sampling errors. The geographic based malariological measures can also suffer from measurement errors because they are based on mathematical models that are simplifications of the reality. As we said, the maps are adjusted and smoothed for environmental and climatic covariates (average maximum temperature from march to may, the length of the rainy season defined as the number of months with more than 60 mm of rainfall, the distance from the nearest water source and the normalized difference vegetation index). The main simplification made in Gemperli et al. 2006 concerns clustering of inoculations and vectorial capacity in the transmission season. Authors assumed a constant vectorial capacity for each location. They argue that this simplification will lead to underestimate the true EIR in Mali but malaria prevalence will be less affected (p°295). Comparison of the observed prevalence data with the model-based

prevalence estimates gives a mean difference of -0.03 (not significantly different from 0 at the 5% level) for both children <5 years and children aged 2-10 years (Gemperli et al., 2006; p^o293 and 295).

We cannot exclude that low education performances have a negative impact on malaria eradication directly or through other variables like income levels or general health conditions, as discussed in Chapter 2. Finding a good instrument variable for malaria is not an easy task. The availability of health facilities is generally a good candidate as instrumental variable for endogenous health indicators. As we are specifically interested by the impact of malaria we will use the percent of households with at least one mosquito net in the cluster. As we mentioned in Chapter 2, we also used the Entomological Inoculation Rates (number of infective bites per person per year) provided by Gemperli et al. (2006). This index was calculated on a highly disaggregated level. Because it is built upon smoothed ecological characteristics, it is supposed to be exogenous to economic conditions and public health interventions and thus can serve as an instrumental variable in econometric regressions. Indeed, there is no reason to think that the number of infective bites per person per year of malaria mosquito vectors is a cause of bad educational outcomes apart from the direct influence of malaria. As we said in Chapter 2, a similar instrument was already defined at the macroeconomic level by Kiszewski et al. (2004) and used by Sachs & Gallup (2001). As we have two instruments, we can test the validity of each instrument. We estimated these linear equations using Two Stage Least Squares regression models (Table III.6).

TABLE III.3: Descriptive Statistics

Variables	Descriptive Statistics				
	Obs	Means	Standard Deviations	Min	Max
2001 MDHS					
Cluster Primary Repetition Rate	343	0.139	0.176	0	1
Cluster Malaria Prevalence <5 years of age	403	4.451	1.108	0	5
Cluster Malaria Prevalence 2-10 years of age	403	3.342	0.907	0	5
Cluster Entomological Inoculation Rate	403	3.632	1.175	0	5
Cluster Prop of Fever	402	0.311	0.177	0	0.875
Cluster Prop of Respiratory Infections	402	0.117	0.103	0	0.625
Cluster Prop of Diarrhea	402	0.201	0.126	0	0.692
Cluster Prop of Severe Underweighth	398	0.106	0.087	0	0.555
Cluster Prop of Severe Anaemia	380	0.105	0.169	0	1
Cluster Wealth Index	402	0.252	1.816	-1.498	6.297
Primary School in the Cluster	388	0.912	0.283	0	1
Cluster Number of Primary School Children	402	16.174	13.228	0	59
Cluster Median Education Years (15-59 years)	402	0.256	0.893	0	6.5
Cluster Median Age of Primary School Children	350	9.872	1.250	7	14
Cluster Proportion of Households with Bednets	402	0.563	0.285	0	1
Rural Area	388	0.708	0.454	0	1
Kayes	403	0.143	0.351	0	1
Koulikoro	403	0.138	0.346	0	1
Sikasso	403	0.141	0.348	0	1
Segou	403	0.143	0.351	0	1
Mopti	403	0.156	0.363	0	1
Tombouctou	403	0.042	0.201	0	1
Gao	403	0.042	0.201	0	1
Kidal	403	0.027	0.163	0	1
Bamako	403	0.163	0.370	0	1
2006 MDHS					
Cluster Primary Repetition Rate	389	0.092	0.128	0	1
Cluster Malaria Prevalence <5 years of age	406	4.206	1.271	0	5
Cluster Malaria Prevalence 2-10 years of age	406	3.172	1.027	0	4
Cluster Entomological Inoculation Rate	406	3.482	1.289	0	5
Cluster Prop of Fever	407	0.163	0.108	0	0.465
Cluster Prop of Respiratory Infections	407	0.056	0.061	0	.4
Cluster Prop of Diarrhea	407	0.113	0.098	0	.75
Cluster Prop of Severe Underweighth	406	0.103	0.089	0	.6
Cluster Prop of Severe Anaemia	404	0.073	0.104	0	.5
Cluster Wealth Index	407	0.104	1.594	-1.699	5.212
Primary School in the Cluster	407	0.894	0.307	0	1
Cluster Number of Primary School Children	407	20.213	12.320	0	61
Cluster Median Education Years (15-59 years)	407	0.218	0.873	0	8
Cluster Median Age of Primary School Children	391	9.957	1.300	7	16
Cluster Proportion of Households with Bednets	407	0.669	0.216	0	1
Rural Area	407	0.638	0.480	0	1
Kayes	407	0.113	0.317	0	1
Koulikoro	407	0.113	0.317	0	1
Sikasso	407	0.167	0.373	0	1
Segou	407	0.113	0.317	0	1
Mopti	407	0.184	0.388	0	1
Tombouctou	407	0.078	0.269	0	1
Gao	407	0.076	0.265	0	1
Kidal	407	0.027	0.162	0	1
Bamako	407	0.127	0.334	0	1

III. Estimation results

Table III.4 and III.5 reports the OLS and bootstrap OLS multivariate regression analysis. Table III.6 provides the corresponding two stages least squares (2SLS) regression models.

We first provide a discussion in support to the validity of the ecology-based instrumental variables (IVs) used first by Sachs & Gallup (2001) and next by other authors (Bleakley, 2007). The partial *R square* of IVs in the first stage equation confirms their relevance, and the *F-test* of weak identification of IVs largely exceeds the threshold of 10 recommended by Staiger & Stock (1997) in all regressions. The Hansen *J* test of the validity of instrumental variables is provided in Table III.6. A rejection of the null hypothesis implies that the instruments are not satisfying the orthogonality conditions required for their employment, which is not the case here (see Appendix III.1 for more details on our instrumentation and identification strategy). Except for regression (1), (3) and (5) of Table III.6, the Durbin-Wu-Hausman test does not reject the null hypothesis that the OLS estimators are consistent and that the differences between the OLS and 2SLS coefficients are random. In regressions (1), (3) and (5) of Table III.6, we failed to reject the null hypothesis certainly because of a lack of power. Moreover, the test is not robust in regression (6), (8) and (10) so that results suggest that the treatment of malariological measures as endogenous is not useful in this case.

It is also possible that the maps used to compute the spatial malariological measures are not uniformly reliable and some geographic areas will thus need further investigation. The prediction error could be correlated with the real level of malaria, as field surveys are sparse in areas free of malaria (Sahara Desert for instance). Firstly, this will lead to an underestimation of the real burden of malaria, as discussed in Gemperli et al. 2006. Secondly, this is another argument in favor of good OLS properties. Indeed, if measurement error in the population is correlated with the true unobserved variable, the CEV (Classical Errors-in-Variables) assumption does not hold. It follows that OLS estimation with the observed variable in place of the unobserved variable produces a consistent estimator of the coefficient. Measurement error will just increase the variance of the coefficient i.e. the variance will be larger than if we observe malaria prevalence directly. The use of bootstrapping methods is also justified by the fact that we have a finite sample and we require more reliable standard errors for our estimators.

Therefore, we will only focus on Tables III.4 and III.5 to draw our conclusions. The impact of fever on cluster primary repetition rates is only significant and positive in

the OLS regression models with 2001 MDHS data. On the contrary, clusters with high malaria prevalence (for children under five years or for children from two to ten years of age) have primary repetition rates significantly higher than clusters with no malaria prevalence in both regressions for 2001 and 2006. The malaria prevalence among children from two to ten years of age appears to be a better predictor of malaria for schoolchildren. This effect seems to be relatively stable across seasons and is around 14%. The relationship between school performance and under five years prevalence seems to follow a U-shaped curve whereas the effect of two-to-ten years of age prevalence on school performance grows with malaria intensity. Results are not modified when we use bootstrap techniques to correct for possible finite sample bias. Smoothed spatial malariological variables seem to be better measures of malaria prevalence and risk in an area across seasons.

Other health variables have no significant stable effects on the primary school output. The cluster wealth index reaches the critical value of significance in all the regressions. Therefore clusters with a higher average long-term income seem to have lower primary repetition rates. This result suggests that wealthier clusters are more able to provide a better education quality to children. The coefficient for the cluster median age of primary school children is significant and positive in 2006 regressions only. Differences between the 2001 and 2006 OLS results can also be explained by seasonal variation in the educational output. Indeed, the 2006 MDHS dataset has been collected during two different school years (2005-2006 and 2006-2007) and as a result the school outputs have been mainly collected during school holidays.

TABLE III.4: OLS Multivariate Analysis at the cluster level (2001 and 2006 MDHS)

	Independent variable is Cluster Primary Repetition Rate 2001					Independent variable is Cluster Primary Repetition Rate 2006				
	(1)	(2)	OLS		(5)	(6)	(7)	OLS		(10)
Cluster Prop of Fever <5 years of age	0.217*	.	.	0.230**	0.208*	0.033	.	.	0.004	-0.022
	(0.115)			(0.111)	(0.113)	(0.083)			(0.080)	(0.079)
Spatial Malariological measures										
Cluster Malaria Prevalence <5 years of age										
0.2-0.3	.	0.126	.	0.133	.	.	0.062*	.	0.061*	.
		(0.099)		(0.109)			(0.034)		(0.034)	
0.3-0.5	.	-0.044	.	-0.093	.	.	0.043	.	0.043	.
		(0.062)		(0.067)			(0.035)		(0.035)	
0.5-0.7	.	0.122	.	0.096	.	.	0.054	.	0.054	.
		(0.079)		(0.084)			(0.036)		(0.035)	
0.7-0.8	.	0.107	.	0.087	.	.	0.040	.	0.040	.
		(0.081)		(0.085)			(0.031)		(0.031)	
0.8-1	.	0.196*	.	0.180*	.	.	0.075*	.	0.074*	.
		(0.103)		(0.105)			(0.041)		(0.040)	
Cluster Malaria Prevalence 2-10 years of age										
0.2-0.3	.	.	<i>no obs</i>	.	<i>no obs</i>	.	.	<i>no obs</i>	.	<i>no obs</i>
0.3-0.5	.	.	0.058	.	0.044	.	.	0.045	.	0.046
			(0.080)		(0.090)			(0.037)		(0.037)
0.5-0.7	.	.	0.117	.	0.090	.	.	0.029	.	0.030
			(0.099)		(0.102)			(0.025)		(0.024)
0.7-0.8	.	.	0.130*	.	0.114	.	.	0.088*	.	0.089**
			(0.076)		(0.080)			(0.045)		(0.044)
0.8-1	.	.	0.165*	.	0.149*	.	.	0.145***	.	0.147***
			(0.085)		(0.086)			(0.051)		(0.049)
Other Health Controls										
Cluster Prop of Respiratory Infections	0.080	0.247*	0.229*	0.113	0.107	-0.032	-0.033	-0.040	-0.035	-0.027
	(0.134)	(0.135)	(0.137)	(0.127)	(0.130)	(0.130)	(0.119)	(0.126)	(0.131)	(0.135)
Cluster Prop of Diarrhea	-0.107	-0.014	-0.026	-0.111	-0.116	0.109	0.110	0.092	0.108	0.099
	(0.133)	(0.115)	(0.117)	(0.129)	(0.132)	(0.083)	(0.078)	(0.077)	(0.082)	(0.081)
Cluster Prop of Severe Underweight	-0.095	-0.091	-0.087	-0.077	-0.078	-0.032	-0.044	-0.071	-0.044	-0.072
	(0.139)	(0.145)	(0.139)	(0.141)	(0.137)	(0.081)	(0.081)	(0.078)	(0.082)	(0.079)
Cluster Prop of Severe Anaemia	0.057	0.068	0.078	0.048	0.062	0.079	0.077	0.064	0.077	0.066
	(0.088)	(0.088)	(0.092)	(0.084)	(0.088)	(0.066)	(0.065)	(0.064)	(0.066)	(0.064)
Other Controls										
Cluster Wealth Index	-0.016*	-0.024**	-0.021**	-0.021**	-0.017*	-0.017**	-0.018**	-0.021**	-0.018**	-0.021**
	(0.009)	(0.010)	(0.010)	(0.010)	(0.010)	(0.009)	(0.009)	(0.008)	(0.009)	(0.008)
Primary School in the Cluster	0.037	0.038	0.033	0.043	0.037	0.002	0.003	-0.006	0.003	-0.006
	(0.066)	(0.067)	(0.068)	(0.067)	(0.067)	(0.044)	(0.043)	(0.042)	(0.043)	(0.042)
Cluster Number of Primary School Children	-0.000	-0.000	-0.000	-0.000	-0.000	0.000	-0.000	0.000	-0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Cluster Median Education Years (15-59 years)	0.001	0.006	0.002	0.004	0.000	-0.000	-0.002	-0.001	-0.002	-0.001
	(0.009)	(0.008)	(0.008)	(0.008)	(0.008)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)
Cluster Median Age of Primary School Children	-0.007	-0.008	-0.007	-0.009	-0.008	0.015**	0.016**	0.014*	0.016**	0.014*
	(0.011)	(0.012)	(0.012)	(0.011)	(0.012)	(0.008)	(0.008)	(0.007)	(0.008)	(0.008)
Rural Area	-0.011	-0.022	-0.031	-0.014	-0.025	-0.032	-0.032	-0.033	-0.031	-0.034
	(0.033)	(0.033)	(0.035)	(0.032)	(0.033)	(0.021)	(0.021)	(0.021)	(0.021)	(0.021)
Intercept	0.200	0.052	0.085	0.033	0.071	-0.079	-0.152	-0.185*	-0.152	-0.183*
	(0.167)	(0.198)	(0.198)	(0.197)	(0.198)	(0.091)	(0.101)	(0.098)	(0.101)	(0.099)
<i>Observations</i>	309	309	309	309	309	385	385	385	385	385
<i>R-squared</i>	0.086	0.101	0.083	0.120	0.099	0.056	0.065	0.096	0.065	0.096

Notes:

1. Robust standard errors, adjusted for heteroscedasticity in parenthesis.
2. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
3. Reference category for Malaria prevalence is no malaria prevalence (ie 0%).
4. Regressions also include regional dummies.

TABLE III.5: Bootstrap OLS Regressions at the cluster level (2001 and 2006 MDHS)

	Independent variable is Cluster Primary Repetition Rate 2001					Independent variable is Cluster Primary Repetition Rate 2006				
	Bootstrap OLS					Bootstrap OLS				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Cluster Prop of Fever	0.217* (0.115) {-0.005}	.	.	0.230** (0.111) {0.003}	0.208* (0.111) {-0.000}	0.033 (0.082) {-0.003}	.	.	0.004 (0.080) {-0.004}	-0.022 (0.081) {-0.006}
Spatial Malariological measures										
Cluster Malaria Prevalence <5 years of age										
0.2-0.3	.	0.126 (0.112) {-0.005}	.	0.133 (0.123) {-0.007}	.	.	0.062 (0.038) {-0.001}	.	0.061 (0.038) {-0.001}	.
0.3-0.5	.	-0.044 (0.063) {0.003}	.	-0.093 (0.075) {0.010}	.	.	0.043 (0.036) {0.000}	.	0.043 (0.035) {0.000}	.
0.5-0.7	.	0.122 (0.085) {-0.000}	.	0.096 (0.090) {-0.002}	.	.	0.054 (0.037) {-0.000}	.	0.054 (0.035) {-0.000}	.
0.7-0.8	.	0.107 (0.089) {0.000}	.	0.087 (0.092) {-0.004}	.	.	0.040 (0.033) {0.000}	.	0.040 (0.031) {0.000}	.
0.8-1	.	0.196* (0.107) {-0.002}	.	0.180* (0.110) {-0.007}	.	.	0.075* (0.042) {-0.000}	.	0.074* (0.040) {-0.000}	.
Cluster Malaria Prevalence 2-10 years of age										
0.2-0.3	.	.	<i>no obs</i>	.	<i>no obs</i>	.	.	<i>no obs</i>	.	<i>no obs</i>
0.3-0.5	.	.	0.058 (0.088) {-0.001}	.	0.044 (0.101) {-0.004}	.	.	0.045 (0.037) {-0.002}	.	0.046 (0.039) {-0.000}
0.5-0.7	.	.	0.117 (0.104) {-0.004}	.	0.090 (0.108) {-0.004}	.	.	0.029 (0.025) {-0.000}	.	0.030 (0.026) {-0.000}
0.7-0.8	.	.	0.130 (0.082) {-0.001}	.	0.114 (0.083) {-0.002}	.	.	0.088* (0.045) {-0.002}	.	0.089** (0.044) {-0.002}
0.8-1	.	.	0.165* (0.089) {-0.002}	.	0.149* (0.089) {-0.003}	.	.	0.145*** (0.050) {-0.001}	.	0.147*** (0.049) {-0.002}
Other Health Controls										
Cluster Prop of Respiratory Infections	0.080 (0.136)	0.247* (0.137)	0.229* (0.135)	0.113 (0.126)	0.107 (0.134)	-0.032 (0.133)	-0.033 (0.122)	-0.040 (0.129)	-0.035 (0.133)	-0.027 (0.134)
Cluster Prop of Diarrhea	-0.107 (0.131)	-0.014 (0.114)	-0.026 (0.119)	-0.111 (0.131)	-0.116 (0.131)	0.109 (0.084)	0.110 (0.079)	0.092 (0.079)	0.108 (0.084)	0.099 (0.084)
Cluster Prop of Severe Underweighth	-0.095 (0.139)	-0.091 (0.144)	-0.087 (0.141)	-0.077 (0.141)	-0.078 (0.142)	-0.032 (0.083)	-0.044 (0.081)	-0.071 (0.079)	-0.044 (0.082)	-0.072 (0.079)
Cluster Prop of Severe Anaemia	0.057 (0.090)	0.068 (0.088)	0.078 (0.091)	0.048 (0.086)	0.062 (0.090)	0.079 (0.068)	0.077 (0.066)	0.064 (0.064)	0.077 (0.065)	0.066 (0.067)
Other Controls										
Cluster Wealth Index	-0.016 (0.010)	-0.024** (0.010)	-0.021* (0.011)	-0.021** (0.010)	-0.017* (0.010)	-0.017* (0.009)	-0.018** (0.009)	-0.021** (0.008)	-0.018* (0.009)	-0.021** (0.009)
Primary School in the Cluster	0.037 (0.066)	0.038 (0.066)	0.033 (0.068)	0.043 (0.067)	0.037 (0.067)	0.002 (0.047)	0.003 (0.044)	-0.006 (0.043)	0.003 (0.043)	-0.006 (0.043)
Cluster Number of Primary School Children	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	0.000 (0.001)	-0.000 (0.001)	0.000 (0.001)	-0.000 (0.001)	0.000 (0.001)
Cluster Median Education Years (15-59 years)	0.001 (0.009)	0.006 (0.009)	0.002 (0.009)	0.004 (0.008)	0.000 (0.009)	-0.000 (0.006)	-0.002 (0.006)	-0.001 (0.006)	-0.002 (0.006)	-0.001 (0.006)
Cluster Median Age of Primary School Children	-0.007 (0.011)	-0.008 (0.011)	-0.007 (0.012)	-0.009 (0.011)	-0.008 (0.012)	0.015** (0.008)	0.016** (0.008)	0.014* (0.007)	0.016** (0.008)	0.014* (0.008)
Rural Area	-0.011 (0.033)	-0.022 (0.032)	-0.031 (0.034)	-0.014 (0.032)	-0.025 (0.034)	-0.032 (0.022)	-0.032 (0.021)	-0.033 (0.021)	-0.031 (0.022)	-0.034 (0.021)
Intercept	0.200 (0.166)	0.052 (0.195)	0.085 (0.192)	0.033 (0.197)	0.071 (0.196)	-0.079 (0.093)	-0.152 (0.103)	-0.185* (0.097)	-0.152 (0.100)	-0.183* (0.101)
Observations	309	309	309	309	309	385	385	385	385	385
R-squared	0.086	0.101	0.083	0.120	0.099	0.056	0.065	0.096	0.065	0.096
Replications	2000	2000	2000	2000	2000	2000	2000	2000	2000	2000

Notes:

1. Bootstrap standard errors in parenthesis. Bootstrap estimates of bias into brackets for (malaria and fever coefficients only).
2. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
3. Reference category for Malaria prevalence is no malaria prevalence (ie 0%).
4. Regressions also include regional dummies.

TABLE III.6: 2SLS Multivariate Analysis at the cluster level (2001 and 2006 MDHS)

	Independent variable is Cluster Primary Repetition Rate 2001					Independent variable is Cluster Primary Repetition Rate 2006				
	(1)	(2)	2SLS (3)	(4)	(5)	(6)	2SLS (7)	(8)	(9)	(10)
Cluster Prop of Fever	0.733 (0.486)	.	.	0.170 (0.126)	0.198* (0.113)	0.543 (0.339)	.	.	-0.044 (0.090)	-0.023 (0.076)
Spatial Malariological measures										
Cluster Malaria Prevalence <5 years of age										
0.2-0.3	.	-0.972 (0.819)	.	-0.805 (0.771)	.	.	-0.877 (1.140)	.	-0.861 (1.138)	.
0.3-0.5	.	0.110 (0.469)	.	0.037 (0.408)	.	.	0.105 (0.149)	.	0.107 (0.147)	.
0.5-0.7	.	-0.038 (0.222)	.	-0.028 (0.209)	.	.	-0.099 (0.184)	.	-0.093 (0.185)	.
0.7-0.8	.	-0.199 (0.213)	.	-0.182 (0.198)	.	.	-0.047 (0.146)	.	-0.044 (0.145)	.
0.8-1	.	-0.084 (0.215)	.	-0.064 (0.202)	.	.	0.055 (0.169)	.	0.063 (0.170)	.
Cluster Malaria Prevalence 2-10 years of age										
0.2-0.3	.	.	<i>no obs</i>	.	<i>no obs</i>	.	.	<i>no obs</i>	.	<i>no obs</i>
0.3-0.5	.	.	-0.116 (0.082)	.	-0.141* (0.086)	.	.	0.046 (0.045)	.	0.048 (0.046)
0.5-0.7	.	.	0.124 (0.130)	.	0.095 (0.135)	.	.	0.043 (0.028)	.	0.044 (0.027)
0.7-0.8	.	.	0.024 (0.094)	.	0.003 (0.099)	.	.	0.064 (0.045)	.	0.065 (0.044)
0.8-1	.	.	0.081 (0.107)	.	0.061 (0.109)	.	.	0.125** (0.054)	.	0.128** (0.052)
Other Health Controls										
Cluster Prop of Respiratory Infections	-0.208 (0.321)	0.092 (0.182)	0.197 (0.142)	0.018 (0.168)	0.077 (0.133)	-0.330 (0.234)	-0.036 (0.135)	-0.051 (0.123)	-0.014 (0.140)	-0.039 (0.132)
Cluster Prop of Diarrhea	-0.332 (0.252)	-0.090 (0.137)	-0.035 (0.117)	-0.152 (0.149)	-0.121 (0.133)	-0.053 (0.128)	0.170 (0.116)	0.102 (0.074)	0.183* (0.110)	0.109 (0.077)
Cluster Prop of Severe Underweight	-0.070 (0.149)	-0.056 (0.143)	-0.031 (0.133)	-0.046 (0.137)	-0.020 (0.130)	-0.030 (0.082)	-0.010 (0.096)	-0.068 (0.076)	-0.011 (0.096)	-0.069 (0.076)
Cluster Prop of Severe Anaemia	0.018 (0.093)	0.105 (0.096)	0.080 (0.092)	0.086 (0.092)	0.064 (0.088)	0.030 (0.071)	0.094 (0.085)	0.071 (0.063)	0.098 (0.083)	0.073 (0.064)
Other Controls										
Cluster Wealth Index	-0.006 (0.013)	-0.004 (0.015)	-0.019* (0.011)	-0.005 (0.015)	-0.015 (0.010)	-0.012 (0.010)	-0.006 (0.023)	-0.020** (0.008)	-0.007 (0.023)	-0.020** (0.008)
Primary School in the Cluster	0.047 (0.067)	0.024 (0.064)	0.035 (0.065)	0.030 (0.063)	0.039 (0.064)	0.004 (0.041)	-0.025 (0.060)	-0.004 (0.042)	-0.025 (0.060)	-0.004 (0.042)
Cluster Number of Primary School Children	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	-0.000 (0.001)	0.002 (0.002)	0.000 (0.001)	0.002 (0.002)	0.000 (0.001)
Cluster Median Education Years (15-59 years)	-0.005 (0.011)	-0.007 (0.010)	0.001 (0.008)	-0.007 (0.010)	-0.001 (0.007)	0.004 (0.006)	0.003 (0.012)	-0.001 (0.006)	0.003 (0.012)	-0.001 (0.006)
Cluster Median Age of Primary School Children	-0.008 (0.011)	-0.003 (0.013)	-0.006 (0.012)	-0.004 (0.012)	-0.006 (0.011)	0.017** (0.008)	0.014 (0.009)	0.014** (0.007)	0.014 (0.009)	0.014** (0.007)
Rural Area	0.001 (0.038)	-0.023 (0.037)	-0.024 (0.035)	-0.017 (0.035)	-0.018 (0.033)	-0.021 (0.025)	-0.019 (0.025)	-0.035* (0.020)	-0.020 (0.025)	-0.035* (0.020)
Intercept	0.122 (0.192)	0.318 (0.248)	0.143 (0.205)	0.271 (0.244)	0.133 (0.204)	-0.152 (0.109)	-0.133 (0.190)	-0.170* (0.097)	-0.135 (0.189)	-0.170* (0.098)
Observations	309	309	309	309	309	385	385	385	385	385
R-squared	-0.014	-0.198	0.058	-0.104	0.072	-0.049	-0.476	0.091	-0.463	0.091
Hansen J Test p value	0.325	0.149	0.209	0.192	0.285	0.268	0.296	0.106	0.343	0.120
Durbin Wu Hausman Test	2.892*	8.490	9.437*	7.625	9.373*	2.414	8.266	1.499	8.406	1.504

Notes:

1. Robust standard errors, adjusted for heteroscedasticity in parenthesis.

2. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

3. Reference category for Malaria prevalence is no malaria prevalence (ie 0%).

4. Regressions also include regional dummies.

5. Instrumental variables are Cluster proportion of households with at least one bednet and the spatial Entomological Inoculation Rate.

IV. Conclusion

This analysis of two representative samples of Malian population investigated the effects of fever and malaria on school performances measured by repetition rates in primary education at the cluster level. The results differ from the past literature by taking into account the specific effects of fever and malaria. This chapter takes into account other factors like family and community characteristics such as school location, cluster wealth and education level in the cluster. The importance of group-level socioeconomic and health factors in determining the individual's education is stressed here.

In summary, the main result of this Chapter is that high malaria prevalence is a significant determinant of cluster primary repetition rate in Mali. Health investments and particularly malaria control programs are likely to have important benefits for both wellbeing and educational attainment. Confirmatory multidisciplinary studies on the link between malaria and school achievement are still needed to shed more light on the effects of malaria on the scholastic achievement and cognitive capacity of children. One of the reasons why malaria control strategies are not integrated into broader school health programmes is, perhaps, because the impact of the disease for schoolchildren and the benefits from interventions have not been studied intensively.

The second main result of this analysis is that fever is not a reliable proxy for malaria. This result is obviously not new, but to our knowledge, it is the first time that it is established at a national scale in Africa using large-scale surveys. Bouvier et al. (1997) already emphasised this result in an endemic area in Mali with 9 days' follow-up of 1- to 12-year-old children during two time periods. The authors conclude that most children with high parasite density do not develop fever subsequently. The association between parasite density and fever varies according to age and season. However and despite these results, DHS reports before 2008 commonly used fever as synonymous of malaria (see for instance the Nigeria DHS 2003, *Chap 10 Malaria – Treatment of Children* p°148; or Mali DHS 2006, *Chap 10 Paludisme – Traitement du paludisme* p°153). This variable has to be used carefully in public policy research, taking into account season, transmission intensity and population age. Our conclusion is even more relevant for malaria control programs in Africa. Even if malaria remains a main cause of fever in Mali, malaria is not fever. As underlined in General Introduction, it is truly alarming to observe that specialised agencies in the control of malaria, such as the GFATM are still using *fever* as an official measure of the burden of malaria in 2009, maintaining total confusion on this topic. Instead, we recommend the use of spatial measures of malaria incidence, spleen rate or

biomarkers for future research and programs evaluation. In other words, it is time to invest in reliable surveillance systems in SSA. It does not mean that efforts made by the AMFm are unnecessary but they should be accompanied by credible measures of the burden of malaria. Recall that we do not even know the real number of malaria cases or deaths in SSA. Without reliable data, how can we trust or assess these malaria control policies? One has to be very naive to believe that malaria control programs will succeed without any reinforcement of epidemiologic surveillance structures in endemic countries. Current outpatient diagnostic practice in sub-Saharan Africa is based largely on symptom-based or ‘presumptive’ treatment of fever with anti-malarial drugs. Malaria microscopy, currently considered as the “gold standard”, requires technical skill and equipment that are usually unavailable and inaccessible in most malarious areas. The Rapid Diagnostic Tests (RDTs) offer opportunities for early diagnosis of malarial infection: malaria RDTs can assist in making a rapid, accurate diagnosis of malaria in circumstances where demonstration of parasitaemia has previously been impossible. Of course, variations occur among malaria RDT products and costs, though the principles of the tests are similar. Some malaria RDTs detect specific antigens (proteins) produced by malaria parasites, which are present in the blood of infected or recently infected individuals. Other RDTs detect antibodies and are used for screening blood for evidence of recent infection. Some RDTs can detect only one species (*Plasmodium falciparum*), some detect one or more of the other three species of human malaria parasites (*P. vivax*, *P. malariae* and *P. ovale*). Blood for the test is commonly obtained from a finger-prick (WHO Meeting Report, 2003). Problems with sensitivity of these tests have resulted in considerable uncertainty as to the place of RDTs in the health systems of endemic countries. As many African countries have no monitoring system to assess RDTs accuracy, the effectiveness of RDT implementation is not well documented

The accuracy (sensitivity and specificity) of an RDT (Paracheck-Pf®) for the detection of clinical malaria and the presence of malarial parasites has recently been evaluated in a high-transmission area of southern Mali (Willcox et al., 2009). Sensitivity is the ability of a test to correctly identify individuals who have a given disease or disorder. Specificity is the ability of a test to correctly exclude individuals who do not have a given disease or disorder. Paracheck-Pf® is a test for the detection of *P. falciparum* malaria that utilizes the principle of immunochromatography. A “Test-and-Treat” strategy was compared to a “Treat-all” strategy. In the detection of clinical malaria, authors found that RDT is 95.2% sensitive and 57.4% specific. RDT was found to be 80.2% sensitive compared with the detection of parasites by microscopy. For children aged 0-5 years, use of the RDT was not cost-effective compared with the presumptive treatment of malaria with an ACT. However,

parasite-based diagnosis by microscopy or RDTs must be viewed in the context of diagnosis of febrile illness, not just malaria. Early exclusion of malaria has the potential to enhance early diagnosis and appropriate management of other, often severe, causes of fever. The costs and benefits of demonstrating parasites as a basis for anti-malarial therapy therefore vary not just with prevalence (or frequency of parasitaemia amongst patients with fever) and with the costs of anti-malarial drugs, but also with the costs of non-malarial febrile illness. Reduction of malaria morbidity and drug resistance intensity plus the associated economic loss of these two factors require urgent scaling up of both the use and the quality of parasite-based diagnostic methods (Wongsrichanalai et al, 2007).

As a discussion, we identify some limitations of this study. Firstly, future research on the subject based on panel datasets could improve the analysis of the malaria-schooling nexus. Our results based on cross-sectional retrospective data can only inform us as to the nature and extent of the burden at one point in time. A dynamic process would be best investigated with longitudinal surveys.

Secondly, higher-quality individual data on malaria and school outputs are necessary to improve microeconomic investigations in this field research. The new malaria indicator surveys included in the most recent DHS could be one solution to this problem, as they include biomarkers testing for anaemia and malaria for pregnant women and children under five years. Unlike the DHS, which are carried out at various times during the year, the malaria indicator surveys are usually timed to correspond with the high malaria transmission season. This is essential if the malaria indicator surveys include biomarker testing for malaria. However, these surveys do not provide biomarkers for school-age children because they are too demanding and too costly. Moreover, the malaria indicator surveys are new surveys. To date, the malaria indicator surveys have been carried out in Zambia, Angola, and Senegal. Additional surveys are underway in Tanzania and Uganda. Only some of them (Angola and Tanzania) include biomarkers, depending on the needs of the country. This study confirms the need to include new malaria indicators in the most recent DHS. Our result on the use of fever as an indicator of malaria also justifies directly the analysis presented in Chapter 4. We cannot conclude to any *causal* relationship between malaria and education performance without reliable malaria indicators.

Thirdly, it is possible that the other health control variables have no significant impact on repetition rates because they are seasonal measures of health, because they are not good proxies of child health conditions or because they are not stable over time. The prevalence of diarrhoea, the proportion of children with respiratory

infection and the prevalence of fever are also highly correlated, which can lead to increase the variance of the unbiased estimators.

Last, we tried to correct errors-in-variables bias and finite sample bias by using different estimation techniques, but it is still possible that our results are underestimated. The advantage of using an adjusted and smoothed index to study the impact of malaria on school performance is that the spatial and smoothed index is not year-specific or season-specific, which provides a better general measure of the burden of malaria in an area and of annual prevalence. Repetition rates are the result of one school year of work (and other events) and not only of one season. The fact that we find similar results with two different years (2001 and 2006) for Malaria prevalence 2-10 years of age supports this hypothesis.

CHAPTER IV: MALARIA AND PRIMARY EDUCATION IN
MALI: A LONGITUDINAL STUDY IN THE VILLAGE OF
DONEGUEBOUGOU¹³

¹³ This chapter is based on a study under revision in *Social Science & Medicine* (with M.S Sissoko, O.B Touré, P. Kamaté, M.B Niambélé, B. Kamaté, O. Guindo, A. Balam, J.C Berthélemy and O.K Doumbo).

In Mali, malaria is endemic among more than 90% of the total population. Children under five represent 34.9 % of all consultations for malaria. At the Gabriel Touré Paediatric Hospital in Bamako, malaria ranks high among causes of childhood mortality, is the most common cause of paediatric consultations with approximately 40% of total consultations (Keita, Diakite & Diarra, 2004), and is the most common cause of febrile convulsions in children between the ages of sixteen months and ten years (Sagara, Sangare, Dolo, Guindo, Sissoko, Sogoba et al., 2002). In addition, children who survive to school age continue to be vulnerable to malaria (Kazadi, Sexton, Bigonsa, W'Okanga & Way, 2004; Clarke, Brooker, Njagi, Njau, Estambale, Muchiri et al., 2004).

Three factors motivate the study of the quality of education and associated factors at the individual level:

- (i) Cross-country and Within-Country studies have found that malaria could have a significant impact on aggregate human capital accumulation (see previous chapters and General Introduction). Consequently, research on this relation needs more microeconomic foundations.
- (ii) Accumulated evidence indicates that early test scores are correlated with education, earnings, and future labour participation (Leibowitz, 1974; Robertson & Symons, 1990; Cameron & Heckman, 1998; Currie & Duncan, 1999).
- (iii) Despite the significant impact of malaria on school-age children, there is a lack of evidence in the social science and medical literature about the link between malaria and school achievements. Confirmatory studies are needed.

This chapter assesses the role of malaria and some social determinants on primary education and especially on school achievements in Donéguébougou, a small village located in a malaria-endemic area near Bamako, Mali. As discussed earlier, two main limitations of Chapters 2 and 3 were the quality of data and the lack of time-dimension in the data. We suggested in General Introduction that it was possible to collect appropriate data on malaria and education. Therefore, field data were collected in collaboration with the Malaria Research and Training Center (MRTC) between November 2007 and June 2008 on 227 schoolchildren living in Donéguébougou. The research protocol was designed specifically to assess the effects of malaria on school achievement at the children level. Various malaria indicators and different econometric models were used to explain the variation in cognitive abilities, teachers' evaluation scores, school progression and absenteeism. **In this**

chapter we test a very precise and original hypothesis: the direct negative effects of the presence of *falciparum* malaria parasites *per se* on school achievement, all other things being equal. Until now, the focus has been limited to the indirect effects of exposure in utero on birth outcomes (Holding & Kitsao-Wekulo, 2004) and the limited number of investigations among school age children has focused on the effect of parasite clearance (Clarke et al., 2008). However, the randomized control trials may have affected the educational outcomes or may have omitted other confounding factors correlated with both parasitization and school achievement in the post-intervention analysis. The mechanism could include a “*toxicity effect, leading to biochemical changes in the central nervous system (CNS); excitation of the immune system, leading to changes in behaviours related to appetite and reaction time; and physiological effects such as discomfort and disturbed sleep, leading to reductions in activity levels or causing behavioural change*” (Holding & Snow, 2001). Neurotoxin released from infected red blood cells may also damage the cortical areas of the brain (Kihara et al., 2006). The relative contribution of clinical and asymptomatic malaria on cognitive achievement is discussed and we try to address the time-course of the effects through the introduction of lagged variables in the model. Finally, as there is a wide range of potential pathways to impaired educational development and because combinations between the different sources of variability in outcomes are plausible, we also control for several confounding factors and analyse the relative contribution of different pathways (mainly splenomegaly as an indicator of chronic infection and malarial anaemia).

This study is different from studies already presented (General Introduction) in a number of ways. First, we used a different study design (panel dataset) and specific econometric models for panel data to assess the education production function and compare the incidence of malaria with other factors. We address the possibility of omitted variables bias by estimating within-child fixed effects models. Second, the strength of the data lies in the careful assessment of parasitaemia, clinical malaria and other biological predictor variables. Third, we used careful assessments of socio-economic variables and a range of school outputs. Fourth, the time period under study is one full school year.

This Chapter is structured as follows. Section 1 describes study site, research protocol and methodology used for data collection. We do not recall the different pathways and mechanisms through which malaria may affect children cognitive achievement but the reader should refer to sections 2 and 3 of General Introduction to understand our empirical specification provided in section 2. Section 3 presents our results. Robustness analysis is given in Section 4. Section 5 concludes and

provides a discussion about the external validity of the results and the causal mechanisms involved in the impact of malaria on cognitive achievement.

I. Data and methods

A. Study site

The village of Donéguébougou is a setting of high malaria transmission intensity (annual Entomologic Inoculation Rate > 100 infective bites per person). According to Kamaté (2002), transmission is assured principally by *An. gambiae s.l* (91.6% versus 8.4% by *An. funestus* in 2000). It was found that incidence rates (number of malaria episodes/person/time) decreased significantly with increasing age during two transmission seasons (1999 and 2000) but remained stable between years (Dicko et al., 2007).

Donéguébougou is a village of approximately 1390 people (a full census of the residents was undertaken before the study in 2007 by the MRTC/DEAP). The village has had a primary school since 1994. From 1994 to 1997, the school was managed by the community. Next, it became a Malian public school in 1998. Data on school characteristics by grade are given in Table IV.A2. As the teacher in Grade 6 (who is also the school director) has not changed since 1998, Grade 6 results are a reliable indicator to compare school performance since 1998 (Table IV.A4). Students were exposed to similar facilities in the school. The quality of teaching can be controlled with specific estimation techniques or by introducing grade dummies. None of the children had attended preschools or nurseries previously. In this study, we only focus on *P. falciparum* malaria, which is far more severe than the other types of malaria. *P. ovale* and *P. malariae* malaria cases were insignificant in comparison with *P. falciparum* malaria cases.

B. Ethical Clearance

The study protocol was reviewed and approved by the Ethics Committee of the National School of Medicine and Pharmacy of Mali. Treatment of malaria and other illnesses detected during the course of the study was provided to the study population at no cost to participants. Pupils not participating in the study were also treated free of charge. Community informed consent was obtained before the beginning of the study. A meeting was conducted in the village with traditional leaders and the village head to explain the objective and method of the study. School written informed consent was also obtained from the school director and the school parents'

association. Last, after a meeting held in the school to explain the objectives and methods of the study, individual written informed consent was obtained for every pupil from parents or legal guardians (see Appendix IV.5). Among the 229 schoolchildren, 227 students (81 girls and 146 boys) were included in the study. One legal guardian refused to take responsibility for the consent for one child (Grade 5) because the parents were living outside the village. One child (Grade 1) arrived after the beginning of the study.

C. Survey Methods

The data were collected during the whole school year from November 2007 to June 2008. Once informed consents were obtained, all enrolled students received monthly clinical and laboratory exams (active monthly follow-ups). The time period between two active monthly follow-ups was approximately constant. Cognitive tests were administered the same day (just after the clinical exams) directly in the school. All students presenting with clinical symptoms or physical exam findings were treated according to the national standard of care. Those presenting with clinical signs of uncomplicated malaria had their thick and thin smears read immediately to ensure timely diagnosis and treatment.

The active surveillance was supplemented by continuous passive case surveillance. Students were passively followed by the village health centre in collaboration with the schoolteachers and the school director. Every day, teachers identified and recorded the names of any children who were ill or absent to maximise case detection. Any child falling sick during school days, weekends or holidays was immediately referred to the village health centre, where he received a complete clinical exam. Again, all students presenting with clinical symptoms or physical exam findings were treated according to the national standard of care, and those presenting with clinical signs of uncomplicated malaria had their thick and thin smears read immediately to ensure timely diagnosis and treatment.

To assess home inputs and other socio-economic household characteristics, a questionnaire was designed and interviews were conducted with the head of each student household.

The active monthly follow-up, socio-economic questionnaires, and cognitive test data were checked for internal validity at the end of each cross-sectional survey by one investigator and corrected on-site, if necessary. The data were entered by double keyboarding and validated using Microsoft Access© (Microsoft Corporation, Redmond, Washington DC, USA) and Epi Info© (Center for Disease Control and

Prevention, Atlanta, GA, USA). All of the data were analysed using Stata© statistical software (Stata Corporation, Austin, TX, USA).

D. Health Status

We were specifically interested in *P. falciparum* malaria. Nevertheless, different sets of data were collected in the field with respect to health by the medical team under the supervision of three doctors.

1. Anthropometric indices and haemoglobin level.

The weight of each child was measured using an electronic scale, and their height was measured using a stadiometer. Blood was obtained via a finger prick under aseptic conditions, and the haemoglobin level was determined using a haemoglobin analyser in the field (Hemacue©, Labnet International, Buckley WA). The age of each child was calculated in years and months using the date of birth given on his birth certificate maintained by school and confirmed, if necessary, by the village census. The weight and height of the children and their haemoglobin level were measured monthly during the cross-sectional survey. The evaluation of growth attainment requires the use of a reference population that allows for standardised variation at any age, as recommended by the World Health Organization. In order to compare children of different ages by sex, the anthropometric measurements of this study were converted into two indices, height-for-age and weight-for-age, using the 2000 CDC (Centre for Disease Control and prevention) growth charts. The growth attainment of each child was then expressed as a z-score. The z-score measures the degree to which a child's measurements deviate from those expected based on a reference population. As there was no significant variation between mean z-score from one month to another and from the first and last month, annual mean height-for-age and mean weight-for-age z-scores were used in the analysis. Indeed, the changes in z-scores over time for the same child are likely to reflect some measurement error rather than changes in nutritional status.

2. Malaria indices.

The measurement of malaria is key for our analysis. As mentioned before, each participant underwent a full monthly clinical examination during which auxiliary temperature was measured using an electronic thermometer. Malaria finger pricks were performed monthly on all participants and whenever symptoms or signs of malaria were present during the study. Parasitaemia (parasites / μL) was determined using a Giemsa-stained thick blood film (asexual forms of each *plasmodium* sp were

counted on 300 leukocytes, assuming an average leukocyte count of 7500/ μ L of blood). During the study, clinical malaria was diagnosed if a child had at least one clinical symptom compatible with malaria (elevated body temperature > 37.5 degrees centigrade, cephalgia, vomiting, stomach ache, diarrhoea) together with a malaria-positive smear for *P. falciparum* parasitaemia. From these parasitological and clinical examinations, different indices of malaria were used in this study:

- The absolute number of new clinical malaria episodes per student per month was used as a measure of malaria incidence. A minimum period of seven days between two consecutive positive malaria smears for the same species of parasite was required to count the second positive smear as a new symptomatic episode.
- Three Malaria Risk Indices were computed to avoid some measurement bias in the health status. The use of a “pyrogenic threshold” has been proposed as the best definition of malaria in endemic areas (Trape et al., 1985). In our study, it was considered that subjects, feverish or not feverish, with a level of parasitaemia over 500 *P. falciparum* trophozoites/ μ L (Clarke et al., 2004) might be suspected either of having a malaria attack or of having had one recently. This variable is a dummy equal to one if the child has a parasitaemia > 500 *P. falciparum* trophozoites/ μ L and zero otherwise. Like Audibert et al. (1999), we also considered other thresholds (1000 and 1200) in order to test the sensitivity of the results.
- Annual and monthly geometric means of *P. falciparum* parasitaemia were also used in some regressions to assess the impact of parasitaemia on school outputs.
- Asymptomatic malaria was defined as a malaria-positive smear for *P. falciparum* parasitaemia associated with no clinical symptoms.
- The prevalence of anaemia among school age children is generally defined as a haemoglobin concentration of less than 11.0 g/dL. To account for other threshold effects, malarial anaemia was defined *a priori* as an ordinal variable following Ong'Echa et al. (2006):
 - 0 = No malarial anaemia. Children with a malaria-positive smear for *P. falciparum* parasitaemia and absence of anaemia (i.e., Hb > 11.0 g/dL)
 - 1 = Mild malarial anaemia - Children with a malaria-positive smear for *P. falciparum* parasitaemia and Hb between 8.0 and 10.9 g/dL.
 - 2 = Moderate malarial anaemia - Children with a malaria-positive smear for *P. falciparum* parasitaemia and Hb of 6.1–7.9 g/dL.
 - 3 = Severe malarial anaemia - Children with a malaria-positive smear for asexual *P. falciparum* parasitaemia and Hb \leq 6.0 g/dL.

- Splenomegaly was assessed via spleen measurement using the Hackett classification (Hackett, 1944). Splenomegaly is an enlargement of the spleen resulting from an abnormal immune response to repeated attacks of malaria. The maximum value taken by the splenic index in this study is 1. Many of the mechanisms leading to an enlarged spleen are exaggerated forms of normal spleen function, and a wide variety of diseases (schistosomiasis, post-necrotic cirrhosis, thalassemia, leukemia, lymphoma, myelofibrosis) are associated with enlargement of the spleen. The role of the spleen during *Plasmodium falciparum* malaria in humans remains unclear (Nacher et al., 2001).
- Past convulsions were a final indicator of the severe malaria history of the child. This variable was defined on the basis of a questionnaire administered to the head of household and questions asked to the child's mother.
- The probability of sleeping under a mosquito net the night before the interview was defined on the basis of the questionnaire noted above. This variable is the number of children in the household who slept under a mosquito net the night before the interview divided by the number of children who actually slept in the household the night before the interview. This variable provides a better idea of the use of bednet in the household than the rough possession of bednet variable. We chose to use the probability of sleeping under a mosquito net the night before the interview for three main reasons. Firstly, when the household holds a bednet, it can be exclusively used by parents. Secondly, it was too time-consuming and too demanding (see Appendix IV.5) to ask for every child whether or not this particular child slept under a mosquito net the night before the interview. Thirdly, some children were not sleeping in the household the night before the interview. Parents told us that the child was usually sleeping in the household but did not sleep in the household this particular night. We did not ask the reason why.

E. Socio-economic data about households

Socio-economic data were collected in December 2007 using a questionnaire administered by researchers to the head of household in every pupil's family. Another questionnaire was administered quarterly in December 2007, March 2008, and July 2008 in order to account for seasonal variations.

1. Home input measures.

It is very difficult to estimate incomes and economic levels in rural areas. We chose several types of resources to estimate income level and socio-economic status. We distinguish four types of socio-economic indices (Audibert et al., 2003):

- “Convenience Property”: a relative index of household socio-economic status (SES) was derived based on dichotomous variables (durable goods such as TV set, radio set, motorcycle, housing infrastructure, etc) using principal components analysis (PCA). Long-run wealth is supposed to explain the maximum variance and covariance in the asset variables (Filmer & Pritchett, 2001).
- “Store-of-value property”: through an indicator of livestock possession.
- “Productive capital”: through an indicator of agricultural equipment or land area pertaining to the household.
- “Monetary Income”: to evaluate monetary income from agricultural or non-agricultural activities, we tried to estimate average amount received per month and the source of income using quarterly interviews. Proceeding this way is very simplistic, but it was not possible to use a more demanding procedure.

2. The borrowing constraint hypothesis

The borrowing constraint hypothesis states that credit access could play a role in children’s human capital investments (Jacoby & Skoufias, 1997). Therefore, we solicited information about formal or informal access to credit in the interviews.

Household socioeconomic characteristics are given in Table IV.1. Quintiles were defined on the basis of the convenience property PCA score. Nobody had access to formal credit in our sample.

3. Human Capital factors.

Age, literacy and education of the head of household and the mother and father of the child were used as measured human capital factors. Questions about child labour were also included in the interviews. The mean age of the head of household is 44.67 (standard deviation = 12.46). Only 5.13% of the heads of household could read or write. 86.34% of the 227 children worked the week before the interview, and 40.91% of them worked more than one hour. These household or productive tasks were never paid. A total of 20.80% of the schoolchildren had school homework to do in the week before the interview. Among them, 33.33% dedicated more than one hour per week to schoolwork at home. Only 12.77% of schoolchildren had somebody (generally one sister or brother) who could help them do their homework.

TABLE IV.1: Household socio-economic characteristics

	All Households				Means	
	Scoring Factor	Means	SD	Impact on PCA score	Poorest Quintile	Richest Quintile
Variables used for Convenience Property Index:						
House characteristics:						
Floor is made of finish surface	0.267	0.230	0.423	0.631	0.043	0.583
Roof material is corrugated iron	0.282	0.863	0.345	0.817	0.478	1.000
Number of buildings	0.156	2.564	1.385	0.112	2.347	3.208
Number of rooms	0.232	4.358	2.328	0.099	3.565	5.833
Other place of residence	0.124	0.051	0.221	0.561	0.000	0.083
Household has Pit Toilet	0.111	0.452	0.499	0.222	0.260	0.541
Household owns at least one:						
Radio	0.272	0.641	0.481	0.565	0.217	0.875
Watch	0.261	0.512	0.501	0.520	0.130	0.750
Television	0.157	0.170	0.378	0.415	0.043	0.291
Bicycle	0.219	0.658	0.476	0.460	0.260	0.875
Motorcycle	0.379	0.555	0.499	0.759	0.086	0.916
Car	0.163	0.008	0.092	1.771	0.000	0.041
Mobile Phone	0.244	0.111	0.315	0.774	0.000	0.333
Cart	0.324	0.376	0.486	0.666	0.043	0.791
Plough	0.266	0.128	0.335	0.794	0.000	0.458
Donkey	0.345	0.230	0.423	0.815	0.000	0.666
Socio-economic Indices:						
Convenience Property Score	.	0.000	1.698	.	-2.253	2.399
Store-of-value Property (CFA Francs)	.	209350.44	386351.76	.	91268.1	370302.06
Productive Capital Property:						
Land Area property dedicated to agriculture (Hectares)	.	2.243	2.546	.	1.833	2.368
Electric Pump to irrigate	.	0.487	0.500	.	0.289	0.652
Monetary Income (CFA Francs)	.	49966.52	78002.22	.	11224.64	82791.67
Savings (dummy variable)	.	0.165	0.275	.	0.086	0.208
Access to Informal credit (dummy variable)	.	0.854	0.271	.	0.898	0.833

Notes. - Except for the number of buildings and the number of rooms, each variable takes the value 1 if true, 0 otherwise. Scoring factor is the "weight" assigned to each variable (normalized by its mean and standard deviation) in the linear combination of the variables that constitute the first principal component. Impact on PCA score is the impact of a change of an increase one unit for each variable (= Scoring factor / SD). The percentage of the covariance explained by the first principal component is 16%. The first eigenvalue is 2.89; the second eigenvalue is 2.30. Quintiles were defined on the basis of the Convenience Property PCA score. There was no formal access to credit.

F. Educational outcomes

1. Assessments

We focused on a set of outcomes that are intended to capture the child's human capital accumulation. These include cognitive function (specific tests), educational attainments (routine school notes), school attainment, and absenteeism. A set of cognitive tests was administered monthly to participants the day of the active monthly medical follow-up. Tests were grade-specific, but not time-specific for two main reasons. First, children get used to the procedure quickly, and second, it is possible to account for learning progression. Specific cognitive tests were developed with the team of Donéguébougou teachers. They were derived from validated questions already used in other studies (Jukes et al., 2006) and adapted to the Malian context. This was a multi-step process: first, new questions or statements were developed based on the format of the original test questions. Next, the content of the assessment was modified, but the basic format remained the same. The teachers assessed the suitability of the content to the age and grade-level programs and

defined four categories: knowledge and comprehension (crystallised intelligence), writing, visual memory, and inductive reasoning (fluid intelligence). A Malian adaptation (in Bambara and French) of the Mill Hill vocabulary test was used as the measure of knowledge and comprehension. Writing abilities were tested by asking to the students to reproduce some words or paragraphs. Visual memory was tested with a visual search test, assessing short-term memory for target pictures among distracters. Inductive reasoning was tested with mathematics exercises where children had to find different combinations of numbers. All tests were administered by the teachers in one session lasting around 45 minutes in the school. Cronbach's alpha statistic was derived for the scale formed from the four variables defined above. Internal reliability was high (cronbach $\alpha = 0.72$). Academic performance can be assessed using school progress reports and the teacher's judgement (Nokes et al., 1991). Routine evaluations were given by teachers according to the national education program (first grade children have no evaluations). Absences and causes of absenteeism were also routinely registered each morning and afternoon by the teachers. Teachers gave the causes of absenteeism according to their own knowledge of the child and village events. If the cause reported was illness, it was confirmed by analyzing the health centre registry and then attributed to a specific disease.

2. Output Variables.

First, all cognitive variables were standardised by class, but not by cross-sectional follow-up to take into account the evolution of scores over time (Figure A1). A principal component analysis was then performed on the four cognitive function variables to compute a cognitive function score (Bartlett 1937; Jukes et al., 2006). Test-retest reliability, assessed through the correlation matrix between cognitive function scores from the eight active monthly follow-ups, was acceptable (mean = 0.740; min = 0.503; max = 0.870; standard deviation = 0.116). The validity of the cognitive function score as a measure of primary school performance is illustrated by the high and significant correlation between the annual average standardised academic school notes and the annual cognitive mean function score ($r = 0.815$). Therefore, the cognitive function score seems to be a good measure of educational achievement. Summary statistics are provided in Table IV.A1. Condensing the information contained in our original variables into one dimension with a minimum loss of information is justified by the fact that we do not want to assess merely the impact of malaria on particular cognitive skills but also on the common underlying process involved in knowing, learning, and understanding things. Each skill has not the same explanatory power to explain the global capacity to know, learn and understand. However, additional analysis for each of the four variables included in

the cognitive score is provided in Table IV.6. Second, mean standardised routine school notes were used in order to test the robustness of the results. Third, a school attainment variable was constructed following the Mook & Leslie (1986) procedure. We first run an OLS regression of school grade on age in a constant elasticity model:

$$\text{Log}(G_i) = \alpha + \beta \log(a_i) + \varepsilon_i \quad (2)$$

where G_i is the observed primary education grade level and a_i is the age of child i . Then we subtract the predicted values of the logarithm of G_i from the child's observed grade level. This provides the dependant variable GA_i , primary education grade level given age.

$$GA_i = G_i - e^{\text{pred}(\log G_i)} \quad (3)$$

GA_i is positive (negative) if the child is ahead (behind) in school relative to other children of the same age.

G. School absenteeism and bivariate analysis

Table IV.2 shows comparisons of the cognitive function mean scores between different sub-samples and provides a useful insight about the effect of our different malaria measures on cognitive achievement. During active follow-up, 42% of the children had asymptomatic malaria, and only 1.56% had clinical malaria. Nevertheless, these results do not account for several confounding factors: individual specific effects, age, grade, socioeconomic status, lagged realisation of the variables, etc. They neither allow to identify mediation effects and to test our main hypothesis. The econometric models described in the next section are useful to control for these factors and complete the analysis.

The results for absenteeism are given in Table IV.3 Malaria is the most common cause of absenteeism (28.57% of total absenteeism and 38.23% of school absenteeism for medical reason). However, in absolute terms, our figures are lower than other results found in the literature (Fernando et al., 2003a). In our study, schoolchildren have received specific attention and early treatment. Therefore, it is possible that absenteeism due to malaria or to health in general has been significantly reduced in comparison with other situations. Because of the limited number of days lost and children concerned, it was not appropriate to undertake a multivariate analysis. Table IV.3 also provides instructive descriptive results on the relative impact of malaria on absenteeism. Because absenteeism due to malaria was very low, it seems very

unrealistic to suppose that malaria could have an impact on cognitive function through absenteeism in this study.

TABLE IV.2: Cognitive performance of study children in relation to health status.

Variables		Obs (%)	Cognitive function Mean score	SD	Student's <i>t</i> -test and Anova <i>F</i> -test <i>P</i> <i>value</i>
<i>Active Follow-up</i>					
Clinical Malaria	No	1766 (98.43)	0.008	1.417	0.033**
	Yes	28 (1.56)	-0.562	0.942	
<i>P. falciparum</i> Parasitemia	<500	1491 (83.11)	0.072	1.394	<0.001***
	500-1000	108 (6.02)	-0.035	1.426	
	1000-1200	19 (1.05)	-0.080	1.475	
	>1200	176 (9.81)	-0.588	1.428	
Asymptomatic Malaria	No	1037 (57.80)	0.064	1.356	0.023**
	Yes	757 (42.19)	-0.088	1.484	
Splenomegaly	No	1702 (94.87)	0.006	1.418	0.428
	Yes	92 (5.12)	-0.113	1.316	
Non malarial Anaemia	No	828 (46.15)	0.099	1.320	0.503
	Mild	177 (9.86)	0.016	1.547	
	Moderate	4 (0.22)	-0.541	1.159	
	Severe	<i>no obs</i>	<i>no obs</i>	<i>no obs</i>	
Malarial Anaemia	No	587 (32.72)	0.010	1.497	<0.001***
	Mild	191 (10.64)	-0.439	1.339	
	Moderate	7 (0.39)	-0.749	1.171	
	Severe	<i>no obs</i>	<i>no obs</i>	<i>no obs</i>	
Respiratory infections	No	1773 (98.82)	0.001	1.415	0.665
	Yes	21 (1.17)	-0.132	1.189	
Helminth Infections	No	1788 (99.66)	-0.002	1.412	0.146
	Yes	6 (0.33)	0.836	1.545	
<i>Passive Follow-up</i>					
Clinical Malaria between two active follow-up	No	1666 (92.86)	0.027	1.424	0.003***
	Yes	128 (7.13)	-0.355	1.200	
Respiratory infections between two active follow-up	No	1759 (98.04)	-0.004	1.419	0.356
	Yes	35 (1.95)	0.174	1.033	
Helminth Infections between two active follow-up	No	1785 (99.49)	-0.003	1.413	0.192
	Yes	9 (0.50)	0.612	1.292	
<i>Active and Passive follow-up</i>					
Repeated Malaria between two active follow-up	0	1666 (92.86)	0.027	1.424	0.012**
	1	123 (6.85)	-0.348	1.222	
	2	5 (0.27)	-0.534	0.399	
<i>Interviews</i>					
Childhood Past Convulsion	No	204 (89.86)	0.011	1.044	0.347
	Yes	23 (10.13)	-0.205	1.046	
Childhood Hospitalization	No	211 (92.95)	-0.016	1.069	0.836
	Yes	14 (6.16)	0.043	0.681	
Childhood Malnutrition	No	212 (93.39)	-0.030	1.045	0.636
	Yes	10 (4.40)	0.129	1.006	

Notes. - *** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.3: Absenteeism by cause

Cause of absenteeism	Number of lost days
Health	34.5 (75.82)
Confirmed Malaria Cases	12.5 (27.47)
Malaria Coinfections	2 (4.39)
Pregnancy	9 (19.78)
Respiratory Infections	2.5 (5.49)
Headache	1.5 (3.29)
Wounds/Burn/Whitlow	2 (4.39)
Helminth Infections	0.5 (1.09)
Dysentery/Food Poisoning	1 (2.19)
Caries	0.5 (1.09)
Sent to the health center by the teacher but not ill	2 (4.39)
Sent to the health center by the teacher but did not come	1 (2.19)
Exclusion for economic reason (subscription fees not payed)	3 (6.59)
Unknown	8 (17.58)
Total	45.5 (100)

Notes. - Percent of total lost days in parenthesis. Some children had malaria coinfections. Two children had malaria and respiratory infection (1 day lost) and one child had malaria and wound (1 day lost).

II. Empirical specification

Before running estimates of education quality functions designed to assess the marginal impact of health status on the school achievement of children, a preliminary step is required. This step deals with the conditions necessary for pooling time-series and cross-section data. Our data are from a single cohort of children, but the educational outputs have different time dimensions (panel dimension for cognitive assessments and academic measures; cross-sectional dimension for school attainment). The impact of health on learning and achievement was addressed using the different multiple regression techniques presented in Currie & Stabile (2006) and Fletcher & Wolfe (2008). The authors use cohort data sets and fixed effects models to understand the influence of Attention Deficit Hyperactivity Disorder on education, grade repetition, and test scores in two developed countries (United States and Canada). Our modelling approach is built on these studies in the particular context of a developing country. For the needs of our study, we define the general linear model as:

$$E_{i,t} = \alpha_1 X_{i,t} + \alpha_2 X_{i,t-1} + \dots + \alpha_t X_{i,1} + \beta_t \mu_{i,0} + \rho_1 V_{i,t} + \rho_2 V_{i,t-1} + \dots + \rho_t V_{i,1} + \varepsilon_{i,t} \quad (4)$$

where $E_{i,t}$ is the education output for child i at time t , $X_{i,t}$ represents observed inputs and $v_{i,t}$ unobserved inputs at time t , $\mu_{i,o}$ denotes the child's endowment of mental capacity, and $\varepsilon_{i,t}$ is measurement error. We use different estimation techniques to estimate the education production function.

First, we use the contemporaneous cumulative specification with orthogonal endowments and omitted inputs:

$$E_{i,t} = \alpha_1 X_{i,t} + \alpha_2 X_{i,t-1} + e_{i,t} \quad (5)$$

where $e_{i,t}$ is a residual term that includes the effect of any omitted inputs, endowments, and measurement error. Observable lagged inputs are included, but omitted factors are supposed to be orthogonal to the included input measures. In particular, the time-course and magnitude of the effects of malaria on achievement is addressed through the use of one-period-lagged malaria indices. This model was estimated using OLS regression techniques.

Second, the fixed-effect specification (within child) was preferred to the random-effect model on the basis of the Hausman specification test (Hausman 1978) and because within variation was higher than between variation among variables.:

$$E_{i,t} = \alpha_1 X_{i,t} + \alpha_2 X_{i,t-1} + b_i + e_{i,t} \quad (6)$$

where b_i are individual fixed effects.

A comparison of (5) and (6) indicates whether the OLS estimates are driven by omitted variables at the child level. We did not estimate family fixed effects models because main family-level factors are supposed to be captured by child fixed effects. Measurement error of the health variables is a potentially important problem. However, the research protocol already described and the use of well-measured malaria indicators lower the risk of measurement error.

III. Results and implication

Table IV.4 presents our baseline OLS estimates of the effect of malaria on child outcomes and Table IV.5 the corresponding fixed effects estimates. Other results

regarding the control variables included in the OLS regressions of Table IV.4 are presented in Table IV.A3 (Appendix IV.2.)

- *Asymptomatic malaria and Clinical Malaria.* First, regressions (1) of Tables IV.4 and IV.5 address the question of the relative contribution of asymptomatic malaria and clinical malaria. Results of regressions (1) of Tables IV.4 and IV.5 are rather similar for asymptomatic malaria and clinical malaria. Children who had experienced a clinical attack of malaria between two active follow-ups or during active follow-up score from 0.368 to 0.542 less than children who had had no attack, controlling for other factors. We also confirm a previous result found in the literature about the impact of asymptomatic malaria on cognitive results (Clarke et al., 2008). Asymptomatic infection significantly affects (p -value=13% in regression (1) of Table IV.4) the cognitive performance of the child, but this impact (from -0.108 to -0.186) is smaller than the effect of clinical malaria. We suggested that one explanation should be through the effect of *falciparum* malaria parasites *by itself* on cognitive functions. This hypothesis is tested in regressions (2) to (5) of Tables IV.4 and IV.5.
- *Direct effects of the presence of falciparum malaria parasites per se on school achievement, all other things being equal.* All parasitaemia variables have a significant and negative effect on the cognition function PCA score, controlling for other factors. Another interesting result is about the cumulative effect of malaria parasitaemia. The higher parasitaemia is, the lower is the cognitive score, as regressions (3), (4), and (5) tend to highlight. For instance, children with a *P. falciparum* malaria parasitaemia ≥ 1200 parasites / μL (controlling for clinical malaria, splenomegaly and malaria anaemia) have an average cognitive score 0.354 less than children with a *P. falciparum* malaria parasitaemia < 1200 parasites / μL (regression (5) of Table IV.5). Lagged variables coefficients of Table IV.5 show that effects of the presence of the parasite persist at least one month later but decrease with time.
- *Other hypothesized mediation effects.* Once associated with other variables, the effect of anaemia and splenomegaly is unclear. The effect of the splenic index on cognitive score appears to be statistically significant and negative in Table IV.5 only. This can be due to the fact that the fixed effects model manages to capture other invariant confounding factors determining splenomegaly. Indeed, splenomegaly is not only caused by malaria, but is instead multifactorial. The effect of the convulsion history of the child in Table IV.4 is not statistically significant. This variable is based on a question asked to the family and not on reliable observations. Other studies have already proven the long-term effects of cerebral malaria on cognitive achievement (John et al., 2008). Children with mild

malarial anaemia have lower scores than children with no malarial anaemia but the effect of moderate malaria and the associated lagged anaemia variable is ambiguous. It is possible that the coefficient of moderate malarial anaemia is positive and significant because of the very small number of moderate malarial anaemia cases (see Table IV.3) and because of the definition of thresholds taken *a priori* in the literature. The role of splenomegaly and anaemia in depressing the cognitive performance of the child needs confirmatory studies.

- *Effects of other variables.* Other variables of interest that had significant positive effects on outcomes were head of household literacy, age dummies (reference category was age equal to six years), male dummy, time spent doing homework, and access to informal credit. Variables that had a significant negative effect on outcomes were grade dummies (reference category was first grade) and productive capital variables. Results suggest that effective malaria interventions could also be a valuable addition to school health programs, as the probability of sleeping under a bed net the night before the interview is significantly (at the 1% level) and positively associated with cognitive performances in all regressions. However it is also possible that this variable is rather a proxy for the way parents are taking care of their children. This will be discussed in conclusion. Last, the effect of malaria is also large relative to the effect of other variables. One way to think about the size of these effects is to compare them with the effect of the height-for-age z-score, which has consistently significant effects. For example, a one-point increase in the height-for-age z-score is associated with a 0.55 increase in the cognitive function score. As we said, in this study, the height-for-age z-score does not change significantly for the same child during one year. A one-point increase (or decrease) in the z-score is a rather rare event, particularly for children aged between seven and seventeen, whereas malaria is a common illness in this area. We can interpret the coefficients of anthropometric measures as the long-term effects of childhood nutritional status.

TABLE IV.4: The contemporaneous cumulative specification with orthogonal endowments and omitted inputs.

Variables	Dependant variable is cognitive function PCA score				
	OLS models				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up)	-0.108 (0.072)				
Lagged Asymptomatic Malaria (active follow-up)	-0.128* (0.075)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.024** (0.011)			
Lagged Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.005 (0.008)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)			-0.176* (0.092)		
500			-0.083 (0.084)		
Lagged 500				-0.224** (0.112)	
1000				-0.153 (0.094)	
Lagged 1000					-0.221* (0.114)
1200					-0.149 (0.098)
Lagged 1200					
<i>Clinical Malaria control Dummies</i>					
Clinical Malaria (between two active follow-up or during active follow-up)	-0.386*** (0.100)	-0.392*** (0.100)	-0.385*** (0.100)	-0.369*** (0.100)	-0.368*** (0.101)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.210** (0.093)	-0.170* (0.095)	-0.188** (0.094)	-0.171* (0.094)	-0.172* (0.094)
<i>Chronic Malaria and Malaria Anaemia control Variables</i>					
Splenomegaly	-0.000 (0.142)	-0.008 (0.144)	0.009 (0.140)	0.001 (0.140)	0.004 (0.141)
Lagged Splenomegaly	0.135 (0.129)	0.121 (0.131)	0.111 (0.130)	0.112 (0.129)	0.113 (0.130)
Malarial Anaemia					
Mild	-0.114 (0.127)	-0.175 (0.121)	-0.147 (0.122)	-0.148 (0.122)	-0.158 (0.121)
Moderate	0.021 (0.322)	0.019 (0.317)	0.126 (0.354)	0.068 (0.350)	0.063 (0.349)
Lagged Malarial Anaemia					
Mild	0.020 (0.100)	-0.060 (0.094)	-0.026 (0.097)	-0.016 (0.096)	-0.025 (0.095)
Moderate	-0.697*** (0.256)	-0.714*** (0.257)	-0.681** (0.270)	-0.678** (0.270)	-0.675** (0.270)
<i>Malaria control Variables - Interviews</i>					
Childhood Past Convulsion	-0.089 (0.105)	-0.086 (0.105)	-0.088 (0.105)	-0.087 (0.105)	-0.087 (0.105)
Probability of sleeping under mosquito net the night before the interview	0.442*** (0.104)	0.410*** (0.104)	0.421*** (0.104)	0.405*** (0.104)	0.412*** (0.104)
<i>Health control</i>					
Haemoglobin concentration	-0.063** (0.029)	-0.066** (0.029)	-0.064** (0.029)	-0.064** (0.029)	-0.065** (0.029)
Weight for age z-score	0.127*** (0.039)	0.131*** (0.039)	0.125*** (0.039)	0.124*** (0.039)	0.126*** (0.039)
Height for age z-score	0.549*** (0.145)	0.549*** (0.146)	0.553*** (0.146)	0.551*** (0.146)	0.557*** (0.146)
Intercept	-3.165*** (0.880)	-3.028*** (0.887)	-3.129*** (0.886)	-3.066*** (0.886)	-3.089*** (0.887)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.226	0.223	0.224	0.225	0.225

Notes. - Huber White standard errors are reported in parenthesis. Results are not changed when standard errors are clustered at the household level (not presented here). Reference category for Malarial Anaemia is no malarial anaemia. Other control variables are: mother alive, father alive, age7-age17, grade, sex, religion, ethnicity, convenience property, store-of-value property, capital property, monetary income, savings, informal access to credit, age of the head of household, literacy of the head of household, child work hours, child spent time in homework, child has somebody who helps him for homework. Please refer to Table B3.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.5: Fixed-effects (within child) estimations of the cognitive PCA score.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up)	-0.186*** (0.056)				
Lagged Asymptomatic Malaria (active follow-up)	-0.109** (0.051)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.032*** (0.010)			
Lagged Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.019** (0.009)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500			-0.277*** (0.063)		
Lagged 500			-0.202*** (0.055)		
1000				-0.341*** (0.076)	
Lagged 1000				-0.251*** (0.062)	
1200					-0.354*** (0.077)
Lagged 1200					-0.259*** (0.065)
<i>Clinical Malaria control Dummies</i>					
Clinical Malaria (between two active follow-up or during active follow-up)	-0.535*** (0.070)	-0.542*** (0.070)	-0.524*** (0.070)	-0.519*** (0.069)	-0.515*** (0.069)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.230*** (0.058)	-0.205*** (0.061)	-0.224*** (0.058)	-0.212*** (0.058)	-0.213*** (0.058)
<i>Chronic Malaria and Malaria Anaemia control Variables</i>					
Splenomegaly	-0.301*** (0.116)	-0.250** (0.119)	-0.235** (0.114)	-0.239** (0.113)	-0.234** (0.113)
Lagged Splenomegaly	-0.221** (0.103)	-0.173* (0.104)	-0.201** (0.102)	-0.193* (0.102)	-0.196* (0.102)
<i>Malarial Anaemia</i>					
Mild	-0.136 (0.091)	-0.199** (0.089)	-0.170* (0.087)	-0.165* (0.089)	-0.175** (0.088)
Moderate	0.479*** (0.173)	0.497*** (0.167)	0.675*** (0.157)	0.591*** (0.160)	0.583*** (0.161)
<i>Lagged Malarial Anaemia</i>					
Mild	-0.142* (0.076)	-0.183** (0.072)	-0.140* (0.073)	-0.137* (0.073)	-0.144** (0.073)
Moderate	-0.313* (0.188)	-0.318* (0.180)	-0.253 (0.180)	-0.236 (0.180)	-0.231 (0.180)
<i>Health control</i>					
Haemoglobin concentration	-0.024 (0.024)	-0.028 (0.024)	-0.027 (0.024)	-0.027 (0.024)	-0.030 (0.024)
Intercept	0.784*** (0.300)	0.750** (0.298)	0.780*** (0.296)	0.757*** (0.293)	0.793*** (0.295)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.104	0.103	0.116	0.118	0.119
Breush Pagan <i>F</i> -test	15.34***	15.28***	15.69***	15.69***	15.63***
Hausman specification test	170.56***	257.35***	259.85***	306.52***	312.10***

Notes. - Huber White standard errors are reported in parenthesis. Results are not changed when standard errors are clustered at the household level (not presented here). Reference category for Malarial Anaemia is no malarial anaemia. Breush Pagan Lagrangian multiplier test for the presence of individual specific effects. The Hausman specification test compares the fixed versus random effects under the null hypothesis that the individual effects are uncorrelated with the other regressors in the model (Hausman, 1978). If correlated (H_0 is rejected), a random effect model produces biased estimators, violating one of the Gauss-Markov assumptions; So a fixed effect model is preferred.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

IV. Robustness Checks

A. Factors associated with other cognitive scores

1. Fixed-Effects regressions for every test score

A summary of the results for each of the standardized assessments conducted in the study is presented in Table IV.6. Models used are the same fixed-effects models as in Table IV.5 but only the main coefficients of malaria measures (asymptomatic malaria, parasitaemia and clinical malaria) have been reported here with the corresponding regression numbers and variables.

TABLE IV.6: Fixed-effects (within child) estimations with other dependant variables for cognitive achievement

Corresponding Regression Number and variables as in Table 6	Fixed-effects models (within child)			
	Dependant variable is:			
	Writing Skills	Visual Memory	Knowledge and Comprehension	Inductive Reasoning
<i>Results for regression (1)</i>				
Asymptomatic Malaria (active follow-up)	-0.139** (0.057)	-0.032 (0.036)	-0.113* (0.058)	-0.134*** (0.048)
Lagged Asymptomatic Malaria (active follow-up)	-0.068 (0.051)	-0.016 (0.035)	-0.095* (0.057)	-0.052 (0.044)
Clinical Malaria (between two active follow-up or during active follow-up)	-0.453*** (0.070)	-0.170*** (0.056)	-0.245*** (0.090)	-0.302*** (0.062)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.241*** (0.061)	-0.034 (0.039)	-0.087 (0.083)	-0.080 (0.049)
<i>Results for regression (2)</i>				
Monthly Geom Mean P. falciparum Parasitaemia (/1000)	-0.024** (0.010)	-0.015* (0.008)	-0.046*** (0.016)	-0.019* (0.010)
Lagged Monthly Geom Mean P. falciparum Parasitaemia (/1000)	-0.018* (0.010)	-0.002 (0.006)	-0.029*** (0.010)	-0.036*** (0.011)
Clinical Malaria (between two active follow-up or during active follow-up)	-0.451*** (0.071)	-0.169*** (0.056)	-0.245*** (0.090)	-0.293*** (0.062)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.222*** (0.062)	-0.025 (0.041)	-0.072 (0.082)	-0.073 (0.050)
<i>Results for regression (3)</i>				
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)				
500	-0.132** (0.057)	-0.060 (0.042)	-0.279*** (0.065)	-0.171*** (0.056)
Lagged 500	-0.162*** (0.054)	-0.066* (0.039)	-0.088 (0.060)	-0.119** (0.050)
Clinical Malaria (between two active follow-up or during active follow-up)	-0.434*** (0.070)	-0.161*** (0.056)	-0.239*** (0.090)	-0.288*** (0.063)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.227*** (0.061)	-0.032 (0.039)	-0.087 (0.081)	-0.080 (0.049)
<i>Results for regression (4)</i>				
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)				
1000	-0.231*** (0.065)	-0.099* (0.053)	-0.238*** (0.084)	-0.197*** (0.073)
Lagged 1000	-0.211*** (0.063)	-0.037 (0.043)	-0.152** (0.066)	-0.164*** (0.062)
Clinical Malaria (between two active follow-up or during active follow-up)	-0.435*** (0.070)	-0.165*** (0.056)	-0.237*** (0.091)	-0.285*** (0.062)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.227*** (0.061)	-0.031 (0.040)	-0.078 (0.082)	-0.072 (0.049)
<i>Results for regression (5)</i>				
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)				
1200	-0.204*** (0.067)	-0.116** (0.056)	-0.313*** (0.085)	-0.187*** (0.072)
Lagged 1200	-0.221*** (0.062)	-0.019 (0.046)	-0.176** (0.069)	-0.167*** (0.063)
Clinical Malaria (between two active follow-up or during active follow-up)	-0.442*** (0.070)	-0.167*** (0.056)	-0.232*** (0.091)	-0.287*** (0.063)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.237*** (0.062)	-0.032 (0.040)	-0.079 (0.081)	-0.074 (0.049)

Notes. - Huber White standard errors are reported in parenthesis.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

Main conclusions are unchanged. However, clinical malaria seems to affect particularly writing skills whereas parasitaemia seems to affect rather knowledge and comprehension skills.

2. Proportionnal odds models

Proportional odds models were used as alternative models to quantify the association between the intensity of malaria infection and poor performance on our cognitive variables (Table IV.7). Following Jardim-Botelho (2008), the distribution of scores on each cognitive test as well as the normal distribution of the cognitive function PCA score were divided into four ordinal groups by quartiles.

TABLE IV.7: Ordinal Logit Regressions for Cognitive Achievement Scores

Variables	Proportional odds models - Ordered log-odds (logit) regression coefficients presented				
	Dependent variable is:				
	Cognitive Function Factor Score (1)	Writing Skills (2)	Visual Memory (3)	Knowledge and Comprehension (4)	Inductive Reasoning (5)
<i>Malaria Parasitaemia</i>					
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)	-0.084*** (0.0236) {0.919}	-0.104*** (0.0264) {0.901}	-0.062*** (0.0201) {0.939}	-0.051* (0.0267) {0.950}	-0.041 (0.0256) {0.959}
<i>Clinical Malaria control Dummies</i>					
Clinical Malaria (between two active follow-up or during active follow-up)	-0.348** (0.156) {0.705}	0.0483 (0.159) {1.049}	-0.569*** (0.161) {0.566}	-0.200 (0.190) {0.818}	-0.102 (0.182) {0.869}
<i>Chronic Malaria and Malaria Anaemia control Variables</i>					
Splenomegaly	0.247 (0.196)	0.0952 (0.199)	-0.00380 (0.199)	0.250 (0.253)	0.143 (0.228)
Malarial Anaemia	-0.396** (0.161)	-0.0729 (0.162)	-0.174 (0.161)	-0.291 (0.188)	-0.242 (0.181)
<i>Malaria Control Variables - Interviews</i>					
Childhood Past Convulsion	-0.399*** (0.146)	-0.323** (0.145)	-0.254* (0.146)	-0.0902 (0.168)	-0.772*** (0.170)
Probability of sleeping under mosquito net the night before the interview	0.110 (0.147)	0.161 (0.148)	0.292** (0.147)	0.169 (0.174)	0.143 (0.180)
<i>Health Control Variables</i>					
Haemoglobin concentration	-0.0405 (0.0390)	0.0460 (0.0393)	-0.0670* (0.0390)	-0.00418 (0.0439)	-0.127*** (0.0445)
Weight for age z-score	-0.0333 (0.0490)	-0.0143 (0.0500)	-0.0543 (0.0491)	0.000567 (0.0567)	-0.0263 (0.0554)
Height for age z-score	0.811*** (0.199)	0.593*** (0.203)	0.443** (0.200)	0.237 (0.227)	0.481** (0.224)
<i>Socioeconomic Control Variables</i>					
Convenience Property Score	-0.0450* (0.0271)	0.0147 (0.0279)	-0.0676** (0.0272)	-0.0508 (0.0312)	-0.0628** (0.0316)
Head of Household literate (can read or write)	0.411** (0.208)	-0.125 (0.213)	0.203 (0.222)	0.214 (0.246)	0.252 (0.231)
Number of observations	1794	1794	1794	1324	1324
Number of Children	227	227	227	158	158
Likelihood Ratio Chi-Square	238.71***	148.46***	259.10***	133.79***	124.22***

Notes. - Standard errors are reported in parenthesis. Reference category for Malarial Anaemia is no malarial anaemia. Models also include age dummies. Coefficients are the ordered log-odds (logit) regression coefficients. For a one unit increase in the predictor, the response variable level is expected to change by its respective regression coefficient in the ordered log-odds scale while the other variables in the model are held constant. Proportional odds ratios for the ordered logit model are provided into brackets for malaria parasitaemia and clinical malaria between two active follow-ups. **In regression (5), the p-value of the coefficient on malaria parasitaemia is 0.11.**

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

The quartile categories allow for the comparison of children in the study relative to their peers. Ordinal regressions can be more valid than the OLS regressions when the distribution of scores is highly non-normal, as occurs when the majority of respondents score at the very bottom or top of the scale. Note that our cognitive function PCA score follows a normal distribution and therefore, the OLS regressions or the fixed-effects models are certainly more relevant for this particular variable.

Regressions do not contain lagged variables because including them reduces the sample size and may create difficulties to run the model if any cells of cross-categorical variables are empty or extremely small. Therefore the models presented are a reduced form of the models given in Table IV.4 in order to satisfy proportional odds model assumptions. In an ordinal regression model, odds ratios for each predictor is taken to be constant across all possible collapsings of the response variable. In a cumulative sense, we are comparing pupils who are in groups greater than k versus pupils who are in groups less than or equal to k , where k is the level of the response variable. Hence, one strong assumption is that the relationship between each pair of outcome groups is the same. Brant tests of the parallel regression assumption for every regression suggest that this assumption is not violated in all regressions except in regression (5).

Note that the ordered log-odds regression coefficients are presented in Table IV.7. Proportional odds ratios are presented into brackets for malaria parasitaemia and clinical malaria only. In regression (1), the proportional odd ratio is significantly different from 1 and equal to 0.91. Hence, for a one unit increase in the monthly geometric mean malaria parasitaemia (/1000) – this variable has a mean of 0.589 and a standard deviation of 2.460 –, the odds of high score versus the combined three lowest categories are 0.91 times lower given the other variables in the model are held constant. Likewise, for a one unit increase in the monthly geometric mean malaria parasitaemia (/1000), the odds of the combined two highest categories versus the two lowest categories is 0.91 times lower. Using standard deviation as a measure of variation, for a one standard deviation increase in the monthly geometric mean malaria parasitaemia (the standard deviation of the monthly geometric mean malaria parasitaemia is approximately 2460) odds of high score versus the combined three lowest categories are 0.814 times lower given the other variables in the model are held constant. Hence, using proportional odds models, malaria infection remains a significant predictor of poor performance on the cognitive scores.

B. Factors associated with academic performance and school attainment

Routine school results were not collected by the authors. In this study in general, and more specifically for these particular outcomes, we tried to minimise the influence of the intervention on the normal operation of the school. Hence, routine school assessments were directly performed by the teachers according to their judgement and were provided to the authors at the end of the school year. The robustness of the results presented in Table IV.3, IV.4, and IV.5 is tested with the random-effects model and fixed-effect models applied to routine school notes in Table IV.8. The variable of school attainment has no temporal dimension. The analysis of school attainment is therefore conducted using OLS regressions following Moock & Leslie (1986). As children in grade 1 cannot repeat (we suppose that this is a kind of incentive to keep them at school) and have no examinations, they were excluded from this analysis.

Annual geometric mean parasitaemia is the measure of malaria during the year in these regressions, as it is the most synthetic and relevant variable for this time-period. Like our panel estimations, we find an impact of geometric mean malaria parasitaemia on academic school notes and school attainment. The coefficients are always significant except for regressions (7) and (9). This can be explained by a significant zero-order correlation ($r = -0.29$) at the 1% level between the annual geometric mean malaria parasitaemia and the weight-for-age z-score. In contrast, there is no significant zero-order correlation between annual geometric mean malaria parasitaemia and the height-for-age z-score in the sample ($r = -0.10$). The negative effects of annual geometric mean parasitaemia on teachers' evaluation scores and school progression suggest that malaria may have long-term effects on school achievement.

TABLE IV.8: Factors associated with Academic Performance and School Attainment

Variables	Dependant variable is standardized academic school notes					Dependant variable is school attainment				
	Fixed-effects model (within child)	Random-effects models				OLS regressions				
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Malaria Index										
Geom Mean <i>P. falciparum</i> Parasitemia (/1000)	-0.128* (0.074)	-0.125*** (0.041)	-0.127*** (0.041)	-0.120*** (0.043)	-0.122*** (0.043)	-1.758** (0.855)	-0.943 (0.697)	-1.494** (0.636)	-0.770 (0.732)	-1.153* (0.655)
Probability of sleeping under mosquito net the night before the interview	.	0.040 (0.219)	0.042 (0.217)	0.141 (0.232)	0.153 (0.232)	.	0.828*** (0.244)	0.768*** (0.252)	0.636** (0.292)	0.604** (0.283)
Health control										
Weight for Age z-score	.	0.068 (0.089)	.	0.089 (0.092)	.	.	0.440*** (0.106)	.	0.441*** (0.110)	.
Height for Age z-score	.	.	0.035 (0.313)	.	0.290 (0.329)	.	.	1.890*** (0.233)	.	1.846*** (0.234)
Other Controls										
Age (years)	.	0.278*** (0.054)	0.263*** (0.052)	0.267*** (0.059)	0.273*** (0.060)
Male	.	.	.	-0.076 (0.161)	-0.132 (0.151)	.	.	.	0.462** (0.201)	0.201 (0.142)
Muslim	.	.	.	0.180 (0.137)	0.183 (0.138)	.	.	.	-0.020 (0.182)	-0.065 (0.170)
Bambara	.	.	.	-0.280 (0.355)	-0.286 (0.350)	.	.	.	-0.090 (0.398)	-0.213 (0.234)
Age of the head of household	.	.	.	0.006 (0.008)	0.005 (0.008)	.	.	.	-0.021*** (0.007)	-0.022*** (0.006)
Head of Household literate (can read or write)	.	.	.	0.369 (0.285)	0.401 (0.294)	.	.	.	0.572* (0.298)	0.665*** (0.219)
Convenience Property score	.	.	.	-0.055 (0.041)	-0.048 (0.040)	.	.	.	0.044 (0.049)	0.072 (0.048)
Time per week dedicated to do homeworks	.	.	.	0.509*** (0.164)	0.514*** (0.168)	.	.	.	0.081 (0.234)	0.239 (0.211)
Father alive	.	.	.	-0.114 (0.253)	-0.149 (0.247)	.	.	.	0.204 (0.330)	-0.013 (0.237)
Mother alive	.	.	.	0.512 (0.364)	0.514 (0.376)	.	.	.	0.576 (0.370)	0.678** (0.309)
Intercept	0.021 (0.024)	-2.658*** (0.499)	-2.738* (1.564)	-3.072*** (0.832)	-4.305** (1.862)	0.408*** (0.102)	0.669* (0.359)	-8.117*** (1.067)	0.448 (1.120)	-8.432*** (1.376)
Observations	320	320	320	320	320	167	167	167	167	167
Number of Children	167	167	167	167	167	167	167	167	167	167
R squared	0.038	0.177	0.173	0.261	0.260	0.036	0.213	0.336	0.295	0.429
Breush Pagan F-test	14.52***	12.08***	12.11***	9.61***	9.67***
Hausman specification test	0.92	0.11	0.02	0.91	0.49

Notes. - Huber White standard errors are reported in parenthesis. Results are not changed when standard errors are clustered at the household level (not presented here). R squared given for the fixed effects regression (1) is within R squared. For random effects models (2), (3), (4), (5), between R squared is presented. Other control variables are grade dummies.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

C. Health Expenses

Although all treatments were provided at no cost for all schoolchildren, estimates of health costs for primary school children are given in Table IV.9. The cost estimates are based on prices provided by the pharmacy of the national hospital (Hôpital du Point G, Bamako, August 2008) and national tariffs for artemisinin-based combination therapies (ACT). The costs provided in Table IV.9 do not include the cost of the infrastructure, as they are calculated from the patient's point of view. Moreover, as the health centre is inside the village, there were no travelling costs for the families. All malaria cases were uncomplicated malaria cases, and costs may be considered as direct costs for uncomplicated malaria cases seeking care at formal-sector facilities in the village. These figures complete the economic assessment of the impact of malaria on primary education in Donéguébougou. Despite the fact that average cost per consultation for malaria is lower than average expenses per consultation, malaria is the most costly disease, representing 44.05% of total health expenses.

TABLE IV.9: Health cost estimates by illness (CFA Francs) during the whole school year

Most frequent Infections/Illnesses	Obs (%)	Mean Cost per Consultation	Total cost (%)
Confirmed Malaria Cases	122 (44.36)	1199.631 [1161.601-1237.661]	146355 (44.05)
Respiratory Infections	28 (10.18)	1352.089 [1215-1489.178]	37858.5 (11.37)
Wounds/Burn/Whitlow	26 (9.45)	1518.481 [1372.287-1664.674]	39480.5 (11.86)
Headache	13 (4.72)	622 [622-622]	8086 (2.42)
Helminth Infections	8 (2.90)	952.375 [665.923-1238.826]	7619 (2.28)
Dysentery/Food Poisoning	7 (2.54)	916.142 [825.751-1006.534]	6413 (1.92)
Decayed tooth	5 (1.81)	897.1 [596.232-1197.967]	4485.5 (1.34)
Other (Pyodermite for instance)	40 (14.54)	1300.175 [545.446-2054.903]	52007 (15.62)
Malaria Coinfections:			
Malaria + Respiratory Infection	6 (2.18)	1733.167 [1507.863-1958.47]	10399 (3.12)
Malaria + Helminth Infection	1 (0.36)	1168.5	1168.5 (0.35)
Total (CFA Francs)	275 (100)	1210.271 [1097.411-1323.131]	332824.5 (100)
Total (Euros)	275 (100)	1.845 [1.672-2.017]	507.385 (100)
Total (Dollars)	275 (100)	2.727 [2.473-2.982]	750.162 (100)

Notes. - 95% confidence intervals into brackets and total percentages in parenthesis. Figures given here do not account for coinfections to avoid estimation biases (except for Malaria coinfections). Exchange rate (15/08/08): 1 euro = 655.96 CFA Francs, \$1 = 443.67 CFA Francs. As the active monthly follow-up is an extraordinary cost, only passive follow-up costs were taken into account to assess health expenses. There were no travelling costs as the health center is just near the school.

The average cost per consultation for malaria is 2.70 US dollars, and the mean cost of malaria per child during the entire school year is approximately 1.45 US dollars. In addition to the compulsory scholar material (ruler, pen, slate, notebook which

amount from 500 to 800 CFA Francs per child per year), subscription fees are 2500 CFA Francs per child per year (i.e., a total of 7.43 US dollars per children per year). Books and other learning supports are provided by the public school at no cost for the students. Hence, malaria ranks high among the school budget items for the children's families. Health, and especially malaria, can be perceived as a major financial risk in the children's human capital investment decisions. The total health cost per child during one school year has been estimated to approximately 3.30 US dollars, taking into account that all infrastructures were available on site before the beginning of the study.

D. Other robustness checks and results

1. Interaction terms between malaria indices and the convenience property score

It is useful to think about the estimation biases that the effects of malaria treatment might create. Recall that all the children had received early treatment at no cost. First, it is possible that the absolute value of the effect of malaria is underestimated, as the treatment is supposed to be effective and to improve education achievement (Clarke et al., 2008). Second, this could generate socioeconomic differences or inequalities in educational outcomes, with high-income children more likely to be treated and more likely to perform better than low-income children. This issue has not been examined thoroughly in this chapter because the study was not designed to answer this particular question. However, we estimate models that include interaction terms between malaria indices and the convenience property score to investigate whether higher long-term incomes tend to mitigate the effect of malaria. The same five specifications of the fixed effects models, adding the interaction term, are presented in Table IV.A5 of Appendix IV.3. Only few of them are statistically significant. Other results (not presented in the table) are globally unchanged. As we are using permanent family socio-economic indices, the interaction terms are identified by the fact that malaria indices vary within child. Being in a higher income family offers little protection against the negative effects of malaria on test scores. One explanation could be that the Malaria Research and Training Centre has offered improved treatment at low costs in the village since the early 1990's, thereby reducing the long-term effects of income or income inequalities on access to malaria treatments.

2. Difference between boys and girls

Table IV.A6 asks whether the impacts differ for boys and girls by adding the interactions of a male dummy and malaria indices. It appears that girls suffer much more than boys from the effects of malaria.

3. Interaction terms between malaria and anthropometric indices

One probable pathway identified in Figure 9 was through the co-effects of nutrition status associated with malaria. In order to address this question, we included interaction terms between the weight-for-age z-score, the height-for-age z-score and our malaria indices. This procedure allows investigating whether better nutritional status tends to mitigate the effects of malaria just as in the two previous subsections. The same five specifications of the fixed effects models, adding the interaction term for the weight-for-age z-score, are presented in Table IV.A7 of Appendix IV.3. Only one of them are statistically significant and negative. Having a higher nutritional status offers little protection against the negative effects of malaria on test scores. We obtain similar results with the interaction between the height-for-age z-score and our malaria indices. Other results are globally unchanged.

4. Controlling for other disease or handicap

We have also estimated models excluding children who were declared by their parents to have a known disease or who had a physical handicap at the beginning of the study (sickle-cell anaemia, asthma, rheumatism, one child was one-eyed, etc). The fixed effects estimates presented in Table IV.A8 are globally unchanged.

We next focused on the question of the influence of other infections detected during active or passive follow-ups (mainly helminth infections and respiratory infections) on cognitive achievement to think about the magnitude of these effects compared to the effect of malaria. We address this question by adding other health control variables for these infections. Using interacted variables of clinical malaria with other infections would not have been very useful in this analysis as the number of comorbidity cases was very low. Table IV.A9 shows that, in fixed effects models, having been diagnosed with other health problems is not predictive of poorer outcomes and does not change the effect and significance of malaria variables. These results suggest that, on average, malaria has a greater impact on cognitive function than other health problems.

5. Other controls (not presented)

Other robustness checks have been made. We included a seasonal dummy for dry (=0 from December to may) and rainy season (=1 from June to December). The results are not affected by the inclusion of this variable. However the rainy season dummy is negatively and significantly associated to cognitive performance, which confirms our results.

We also allowed observations to be clustered into families to take into account the probable correlation of observations within families and to control for possible dependence of residuals within families. Once again, results remain unchanged¹⁴.

V. Conclusion

In this chapter, we mainly addressed the problems of malaria measurement, adequate econometric models, and study design to correctly assess the link between malaria and education. On the one hand, empirical economic studies on malaria generally use poor malariological measures and unsuitable medical protocol designs. On the other hand, medical studies in this field of research do not use adequate econometric models to control for potential socioeconomic confounding factors. Our multidisciplinary approach bridges this gap. The main result of this study is that *falciparum* malaria parasites *per se* affect school performance.

A. Mechanisms

We find that children with clinical malaria and asymptomatic malaria suffer large negative consequences in terms of achievement test scores. Clinical malaria is also the most common cause of school absenteeism. The short-term impact of malaria seems to be a more important determinant of reduced human capital accumulation than other health determinants.

The effect of asymptomatic malaria on cognitive abilities has been shown to be less than the effects of clinical malaria. The main pathway identified for the effects of asymptomatic malaria is the presence of *falciparum* malaria parasites *per se*. This is

¹⁴ Other estimation techniques have been used to test the robustness of the results presented in Tables IV.4 ad IV.5: the Random-effects model, the Hausman-Taylor model and the difference-in-differences model. Our results (not presented here) for the effect of malaria parasitaemia are stable and robust, which suggests that malaria parasitaemia could be uncorrelated with individual effects.

an important result. It means that *falciparum* malaria parasitaemia has an effect by itself in addition to its effect through clinical malaria or other forms of malaria. The role of splenomegaly and anaemia in depressing the cognitive performance of the child needs additional examination.

In addition, the results provide evidence of a lasting effect of malaria on academic performance and school attainment. The effects of malaria parasitaemia, asymptomatic malaria and clinical malaria on cognitive outputs persist but decrease with time. The effects of parasitaemia increase with the intensity of infection, which suggests a cumulative effect.

Last, cross-sectional and panel results suggest that the use of bed nets may improve schooling attainment and cognitive and educational achievement. Fighting malaria could have a large payoff in terms of improving the primary education of many children in developing countries (for a discussion on the feasibility of school-based malaria control programs, see Okabayashi et al., 2006). This supposition has to be taken prudently however. As we said, it is possible that the probability to sleep under a mosquito net is rather a proxy for the way parents are taking care of their children, as we controlled for malaria parasitaemia. Nevertheless, the discussion of this variable is relevant because it leads to the question of causality. One main confounding factor that could have an effect both on cognition and parasitaemia could be precisely the way parents are taking care of their children. Should this happen, the introduction of this variable in Table IV.4 or the use of fixed effects models in Table IV.5 should considerably reduce the effect of parasitaemia, which is not the case.

B. Is the effect causal?

The above analysis suggests that *falciparum* malaria parasites *per se* have a negative effect on cognitive performance, but it cannot prove causality. Indeed, one benefit of panel data is that unobserved variables that do not change over time can be differenced out of the regression and thus need not to be measured. However, fixed-effects models assume that the unobserved variables do not change over time and do not interact with variables that change over time (Glewwe & Miguel, 2008).

Fixed effects instrumental variables estimators were not used in this study for three main reasons. First, our malariological measures (especially malaria parasitaemia) during active monthly follow-ups were taken just before the cognitive tests and measured with high precision. Second, a reverse effect of cognitive performance on our malaria indices is unlikely. Third, taking into account the study design, the only relevant variable candidate to instrument for malaria was an

entomological variable (number of infective bits for instance). Such a variable is practically impossible to collect by child and month. Moreover, the non-experimental study design was intentionally preferred to an experimental procedure principally for ethical reasons.

However all the following arguments are combined to infer a causal relationship: our results match with other independent studies, biological evidences of the hypothesis are plausible and have been described in the medical literature, the temporal sequence between cause and effect is appropriate, there is a clear relationship between infection intensity and the magnitude of the effect (the higher is parasitaemia, the higher is the negative effect on cognitive score as regressions (3), (4), and (5) of Tables IV.4 and IV.5 tend to highlight).

C. External validity of the results

First, because the gross enrolment rate in the village is very high (>90%) and children dropping out are generally scarce in this school, we only addressed the effects of malaria on enrolled children.

Second, one limitation of this study is that the results are from one specific village in Mali in which the longitudinal study has been conducted. The Malaria Research and Training Centre has offered improved treatment at low costs in the village since the early 1990's. Therefore, it is possible that the absolute value of the effect of malaria is underestimated, as the treatment is supposed to be effective and to improve education achievement (Clarke et al. 2008). Future investigations should try to go further and test the robustness of our results by following children during a larger time period at a larger scale (including several villages, for instance).

Third, our study is a kind of intervention, even if we tried to interfere as quietly as possible in the normal operation of the school. However this intervention should lead to underestimate the impact of malaria, as children received early treatment. Other descriptive results seem to indicate that malaria prevention has a positive effect for Donéguébougou schoolchildren but these results are just descriptive results. Table IV.A4 in Appendix IV.1 gives the percent of successful candidates at national entry grade 7 examinations since 1998. Before 2007-2008, the school had never achieved 100% successful candidates, and there was no trend indicating that such a result should be expected in 2007-2008. We take this result as a testimony that the improved health surveillance system, especially giving more attention to malaria, could improve the school performances of the children. However we should not overinterpret this result as it is not a *ceteris paribus* conclusion. Randomized controlled trials are still needed to study such causal relationship.

Fourth, the definition of cognitive impairment varies considerably from one study to another and there are also considerable differences in the tools of assessment. This is due to the necessary adaptation of the tests to local circumstances. However, the main objective of this study was to assess the effects of malaria on school achievement and the cognitive function score used in our study is highly correlated with routine school notes given by teachers according to the national education program.

From our results, and particularly from the results dealing with asymptomatic malaria and the effect of parasitaemia *per se*, we suggest that malaria has to be considered one of the most common chronic mental health problems among school children in endemic areas. This unrecognised and underestimated disorder has to be taken into account more seriously by policy makers in national education programs and school health interventions.

GENERAL CONCLUSION

This thesis proposes one possible explanation of the bidirectional relationship between health and economic development through the analysis of the specific case of malaria and education achievement. This approach emphasises the relevance of taking health into account to understand economic development.

Main Results.

Firstly, communities or higher administrative areas may be trapped in any self-reinforcing mechanism that causes a disease to persist, leading to dramatic costs for States, communities and individuals and long-run barriers for economic development and disease control. The combination of diseases traps, poverty traps and possibly other cumulative mechanisms may explain part of the development dynamics.

The first chapter of this thesis theoretically motivates this assumption for malaria, a vector borne disease. One main consequence of this theoretical assumption for public health is that it is not possible to get out off the disease trap without exogenous public policy interventions or exogenous change in climate or epidemiologic conditions. Public policies will allow one community to jump out off the disease trap, (characterised by a high and stable level of disease prevalence) to reach a lower and still stable equilibrium (characterised by a lower level of disease prevalence) by modifying the initial conditions.

Indeed, in our model, preventive measures are not used optimally by individuals - with respect to society and to the control of the disease - because of social externalities of agents' decisions. The model shows that the resulting "prevalence elastic behaviour" associated with some specifications of the parameters (epidemiologic conditions and specific functional form of the opportunity cost function) may lead to multiple equilibria. However, the important fact is that we do not know how to eradicate nor to control the disease. Other dynamic externalities or social inputs due to initial individual or collective characteristics may help to understand the consequences of such "disease traps" and explain how bifurcations at critical points may be released to escape the trap. We have seen in Chapter 4, that being in a higher income family offers little protection against the negative effects of asymptomatic malaria on education in Donéguébougou. This result is probably one of the most interesting. Increasing income level, other things being equal, will have no effects on the possible negative effects of malaria on education. Moreover, in our study, parasitaemia is not statistically associated with income levels. Malaria is often said to be associated with poor income level in regional or national comparisons

whereas the pure effect of poverty on malaria is not proved. If a community is trapped in a high and stable level of malaria prevalence, malaria may be considered as exogenous in this particular community and thus could have huge effects on long-term economic development through education. If people think that there is no hope to escape malaria, the opportunity cost of prevention will be modified. And if education also affects the opportunity cost of protection, thus malaria could generate by itself the socioeconomic barriers to its control.

Another possible handicap endured by endemic communities could be through the bidirectional relationship between health and education that reinforce each other. When education level is initially low and under a critical threshold, cumulative effects of low education levels on health will lead to a long-run cumulative disadvantage process because of the reverse effects of health on education. It follows from this a vicious circle between education and health where the growth rate of the disease will increase with the decrease of education. This could lead the disease to persist at a high and stable equilibrium and impede an economic take-off. The cumulative disadvantage theory applied to education and health provides good support to this hypothesis. Reversely, when education level is initially high and above a critical threshold, a long-run cumulative virtuous circle could follow between health and education. We took the example of education as a key state variable to explain the direction of the cumulative process with respect to a given threshold. However, it is also possible that a vicious cumulative process arises rather from an interaction between initial education and health conditions. This is obviously not the only mechanism as we suggested in Conclusion of Chapter 1. There are certainly different explanations to disease and poverty traps.

For the specific case of malaria, the effect of education on preventive and treatment behaviours is well known, especially the role played by mother's education and knowledge. However, the reverse effects of malaria on knowledge accumulation have never been demonstrated and quantified precisely. That is why we focused on the estimation of the effects of malaria on child cognitive achievement, which could explain part of the direct and indirect effects of malaria on economic development. Direct effect will be through short-term loss of educational outputs and long-term generational productive loss. Indirect effect would be through cumulative mechanisms leading communities and countries to poverty traps.

In chapters 2, 3 and 4, we tried to quantify this burden from the microeconomic to the macroeconomic levels to provide an exhaustive view of the impact. This allows 1) to validate findings at different level of study by comparing different level effects 2) to give microeconomic foundations to macroeconomic findings; 3) to bridge the gap

between microeconomic and macroeconomic studies i.e. identifying possible externalities and general equilibrium effects.

The main empirical results of this thesis suggest that the effects of malaria on primary education and human capital accumulation are strong, robust and causal. First, at the macroeconomic level, we find that countries with intensive malaria have primary repetition rates from 11% to 18% higher than countries without malaria. We also find that countries with intensive malaria have primary completion rates from 48% to 54% less than countries free of malaria in our baseline 2SLS cross-country regressions. Second, at the community level, we find that villages located in areas where malaria prevalence among children aged from 2 to 10 years is more than 80% have repetition rates from 14 to 16% higher than malaria free villages. Third, at the individual level, our results show that children with asymptomatic malaria and clinical malaria suffer large negative consequences in terms of achievement test scores. The effect of asymptomatic malaria is less than the effect of clinical malaria. The main pathway identified for the effects of asymptomatic malaria is the presence of *falciparum* malaria parasites *per se*. This is an important result all the more that the proportion of asymptomatic malaria cases is higher than clinical malaria cases. The effects of parasitaemia increase with the intensity of infection, which suggest a cumulative effect but decrease with time. Even if the effects of malaria are not strictly comparable because methodologies and data used in the Chapters are very different, an attempt to compare the effects is provided in Table GC.1.

TABLE GC.1: Comparison of Results (Beta coefficients)

<i>Malaria Indices</i>	<i>Dependant variables</i>		
Macroeconomic level	Repetition Rates	Completion Rates	
Sachs & Gallup Malaria index 1994	0.810	-0.762	
DALY rates due to malaria 2002	0.628	-0.470	
MAP malaria Index 2007	0.786	-1.864	
MAP malaria Index 2007 - Stable area	0.764	-1.709	
Community Level	Repetition Rates (2001)	Repetition Rates (2006)	
Malaria Prevalence (<5 years of age) >0.8	0.435	0.277	
Malaria Prevalence (2-10 years of age) >0.8	0.407	0.572	
Individual level	Cognitive Function PCA Score	Academic Score	School Attainment
Asymptomatic Malaria	-0.042	.	.
Geometric mean <i>Pf.</i> Parasitaemia	-0.035	-0.285	-0.125
Clinical Malaria	-0.087	.	.

This table provides some results of the standardized regressions coefficients (beta coefficients) for the malaria indices at the different levels of study. Beta coefficients are all measured in standard deviations, instead of the unit of the variables (Beta coefficients are the coefficients obtained if the outcome and predictor variables were all transformed into standard scores before running the regressions). The effects of malaria seem to increase with the level of study.

In addition to these theoretical and empirical issues, our thesis tried to tackle a series of methodological limitations identified in the economic and medical literature: 1) the generally poor quality of malaria measures in the economic literature; 2) the treatment of socioeconomic effects with inappropriate estimation strategies in the medical literature on malaria and education. Our multidisciplinary approach tried to fill these gaps.

Causal effect or mere association?

The question of causality as defined by recent developments in empirical economics is not without ambiguity and far from the plural definition of causality defined by Aristotle (*causa formalis* – the formal conditions of an event such as nature’s laws, *causa materialis* – the material conditions of an event; *causa efficiens* – the conditions of the effect; *causa finalis* – the purpose of an action) or even from more recent definitions given by Kant. The new natural and randomized experiments’ paradigm leads to some kind of *unilateral causality* that may conduce some researchers to omit *causa efficiens* (the conditions of possibility). This paradigm limits the concept of *cause* in human sciences to the interpretation of mechanical invariant successive events and results. This notion implies that a determined effect is only the product of a treatment other things being equal and that the only way to prove causality is to isolate treatment effects from other confounding factors. This may be true in biomedical sciences in some circumstances but it seems incorrect in human sciences. Instead and according to us, experimental frameworks applied to human sciences identify occasional cause that does not suppose any intrinsic link between a cause and its effect. In other words, we obviously need experimental studies but we also need more than experimental studies to establish a causal relation. Some have argued that conducting multiple experiments on a variety of settings will solve the question of causality and heterogenous causal effects. However, the mere repetition of resembling constant conjunctions is not sufficient to establish a necessary and universally valid causal relationship: “*Appearances certainly provide cases from which a rule is possible in accordance with which something usually happens, but never that the succession is necessary; therefore, a dignity pertains to the synthesis of cause and effect that cannot be empirically expressed at*

all, namely, that the effect does not merely follow upon the cause but is posited through it and follows from it." (Kant, Critique of Pure Reason). Hence, the scepticism about the ability of econometrics and empirical analyses to resolve this kind of issues is not new. Here, we would like to stress some key issues in this debate.

Two recent papers (Deaton, 2009 and Heckman & Urzua, 2009) argue against an excessive and inappropriate use of instrumental variables, natural experiments and randomized experiments. Macro econometrics and micro econometrics are both concerned by this scepticism. Heckman & Urzua (2009) state: "*a valid instrument is not guaranteed to identify parameters of economic interest when responses to choices vary among individuals, and these variations influence choices taken. Different valid instruments answer different questions. The sign of the IV estimator can be different from that of the true causal effect*". This issue is dealing with heterogeneity whereas exogeneity is also often misunderstood. Deaton (2009) argues that "*a great deal of imagination is often exercised to find an instrument*", but imagination does not make an economic theory nor a theory at all. "*Exogeneity*" has to be differentiated from "*externality*". According to Deaton, most of the instruments we find in the literature, such as the mortality of colonial settlers (Acemoglu, Johnson and Robinson, 2001), are actually external but not exogenous. The main issue is not about terminology. An instrument will be external to a system if it is not caused by the dependent variable nor by the endogenous independent variable. But this instrument will not be exogenous if it has an effect on the dependent variable other than through the endogenous independent variable, as will almost be the case. "*The absence of simultaneity does not guarantee exogeneity; exogeneity requires the absence of simultaneity but is not implied by it*" (Deaton, 2009). Of course, this is well known in econometrics theory but it is not always applied in practice. In our thesis, if the malaria entomological rate (Chapter 3) or the distribution of malaria vectors (Chapter 2) can have an effect on our explained education variables other than through the effect of malaria, they will be invalid instruments. The difference in our work is that these instruments are not just the product of our imagination but are justified by the fact that malaria is directly affected by entomological and ecological factors, which provides a credible source of exogenous variation in malaria risks i.e. a credible source of exogenous variation and heterogeneity in potential causes.

These issues are related to the central question stated in General Introduction, in Chapter 1 and also highlighted by Marshall (1898) and Deaton (2009): *how and why things work?*" *What are the mechanisms at work?* Randomized experiments are obviously necessary but they serve specific purposes. Besides, they are not always possible. Issues such as externalities, general equilibrium effects, ethical questions, social interactions or contagion can complicate causal inference. If randomized

experiments are clearly one of the most remarkable tools in economic evaluation, applying mechanically instrumental procedures, natural or randomized experiments will not allow understanding the mechanisms at work. In a response to Deaton and Heckman & Urzua, Imbens (2009) emphasizes the benefits of randomized experiments and instrumental variables. He writes, “*Randomization allows the researcher to precisely quantify the uncertainty associated with the evidence for an effect of a treatment*”. Imbens (2009) admits that the current trend toward randomization “*may lead researchers to avoid questions where randomization is difficult, or even conceptually impossible*”. Logical analyses or general equilibrium models help to answer these questions.

Firstly, in our analysis, the combination of mechanisms and context allows to tackle part of the problem. The choice of our “top-down” empirical approach (Chapter 2, 3 & 4) has been made in answer to a particular theoretical economic question motivated by a literature survey (General Introduction and Chapter 1). Uncertainty and randomness generating variability among causes is probably not the same at the macroeconomic level or at more disaggregated levels. If going from macroeconomic analysis to microeconomic field does not give any additional solution to causal inference, it asks the question of how economic development effectively takes place. Recall that we started from the relationship between economic development and malaria. We wanted to assess the causal economic effects of malaria and understand why some countries or individuals are possibly blocked in human development traps. However, it was not possible to provide directly a credible answer to this answer because of endogeneity matters. Randomized experiments alone are unable to answer to these questions and are conceptually impossible. The three causal relationships at different levels analysed through our empirical works provides an idea of the possible mechanism: the effects of malaria on education can be cumulative in time and space and generate large externalities.

Secondly, two kinds of mechanisms interact each other in this thesis: biomedical and socioeconomic. As emphasised by Le Port et al., (2008), individuals harbouring parasites without clinical signs are numerous in areas of high transmission of malaria. However, the consequences and significance of such asymptomatic infections are not clear. Long-term asymptomatic carriage may represent a form of tolerance to the parasite in children building up their immune response. In this way, asymptomatic carriage would protect these children from developing either a mild or a more severe malaria attack, by keeping their immunity effective. Conversely, asymptomatic carriage may represent a mode of entry to symptomatic malaria, especially in young children. Moreover, persistent parasitemic individuals serve to infect anopheline population and perpetuate the transmission cycle, particularly

when transmission is seasonal. Asymptomatic malaria will be critical for the control of malaria in endemic regions. If asymptomatic carriage has long-term underestimated negative effect that counterbalance its positive hypothetical effects on the immune response, it is crucial to increase the knowledge of these effects. The risk/benefit ratio of asymptomatic carriage could determine future malaria control programs. The use of RDTs to detect asymptomatic infections is at the heart of this debate. So is the use of IPTs and their consequences on the development of immunity.

Lastly, it is improbable that the effect of malaria is the same for all children, all communities and all countries. For instance, we have seen that girls suffer much more than boys from the effects of malaria on education. However we have no explanation of this result. Therefore, it would be dangerous to conclude and make inferences without improving our knowledge of the relationship between malaria and education. In other words, the external validity of our results needs to be examined in more details by other researchers, and not only with experimental economic studies or clinical trials. Social scientists or historians could certainly help to answer this kind of questions. Therefore, this thesis can be seen as a first step toward better apprehension of the effects of malaria on child development and more generally on economic development. The question of causality between malaria and educational performance is still far from resolved and providing a systematic answer to this question will need very demanding procedures. The limitations of every chapter have already been presented. Instead, we propose here the following ideas to complete the discussion and improve future research.

Propositions for future research.

First, more experimental studies are needed on this relationship. For instance, the effect of bednet prevention on school achievement has never been studied.

Second, it will be interesting to follow children in a longitudinal framework during a larger time period at a larger scale (including more villages and more schools). This will enable to estimate the effects of malaria in a multilevel regression framework and to better estimate long-run effects of the disease.

Third, future cross-sectional studies should use national surveys with better measurement of both educational outcomes and malaria. This could be achieved in Mali by developing a partnership with the PASEC (Programme d'Analyse des Systèmes Educatifs de la Confemen) that provides representative international tests scores. With this type of data, it will also be interesting to investigate concentration effects in addition to level effects in order to describe the impact of malaria on

education. Level effects are just a first step in the full comprehension of how diseases may affect human capital accumulation.

Last, Ding et al.(2009) have shown that the natural variation within genetic markers across individuals have good statistical properties to identify the effect of a disease on educational performance and thus are promising instrumental variables candidates. For the exclusion restriction assumption not to be violated (i.e the instrumental variables have to be uncorrelated with the error term and can not appear in the regression), the genetic markers will need to be specifically linked to malaria and not to other unobservable factors that influence the education production process. Malaria parasite-human genome interactions have produced and maintained protective genetic variants against malaria for the human race such as sickle cell trait. These genetic variants could be promising instrumental variables to account for the possible endogeneity of malaria. The increasing number of studies that are trying to identify several gene variants, which modulate susceptibility to malaria and its progression, could be exploited.

APPENDIX

APPENDIX 1: MALARIA – DEFINITION, BIOLOGY AND EPIDEMIOLOGY

Malaria is prevalent throughout most of the tropical world, producing a situation in which 40% of the world's population is at risk for acquiring this disease. There were an estimated 247 million malaria cases among 3.3 billion people at risk in 2006, causing nearly a million deaths, mostly of children under 5 years. 109 countries were endemic for malaria in 2008, 45 within the WHO African region (World Malaria Report, 2008). Malaria is considered one of the most common infectious diseases and the most important of the parasitic diseases. Infections in humans are caused by four different species of the genus *Plasmodium*. *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium vivax*, and *Plasmodium ovale* are associated with different clinical presentations, progression, prevalence, and antimalarial resistance patterns. *P. falciparum* infection results in the highest morbidity and mortality, accounting for almost all of the over 1 million deaths caused by malaria annually, most of them in young children in Africa.

- **Life Cycle of Malaria (source: CDC)**

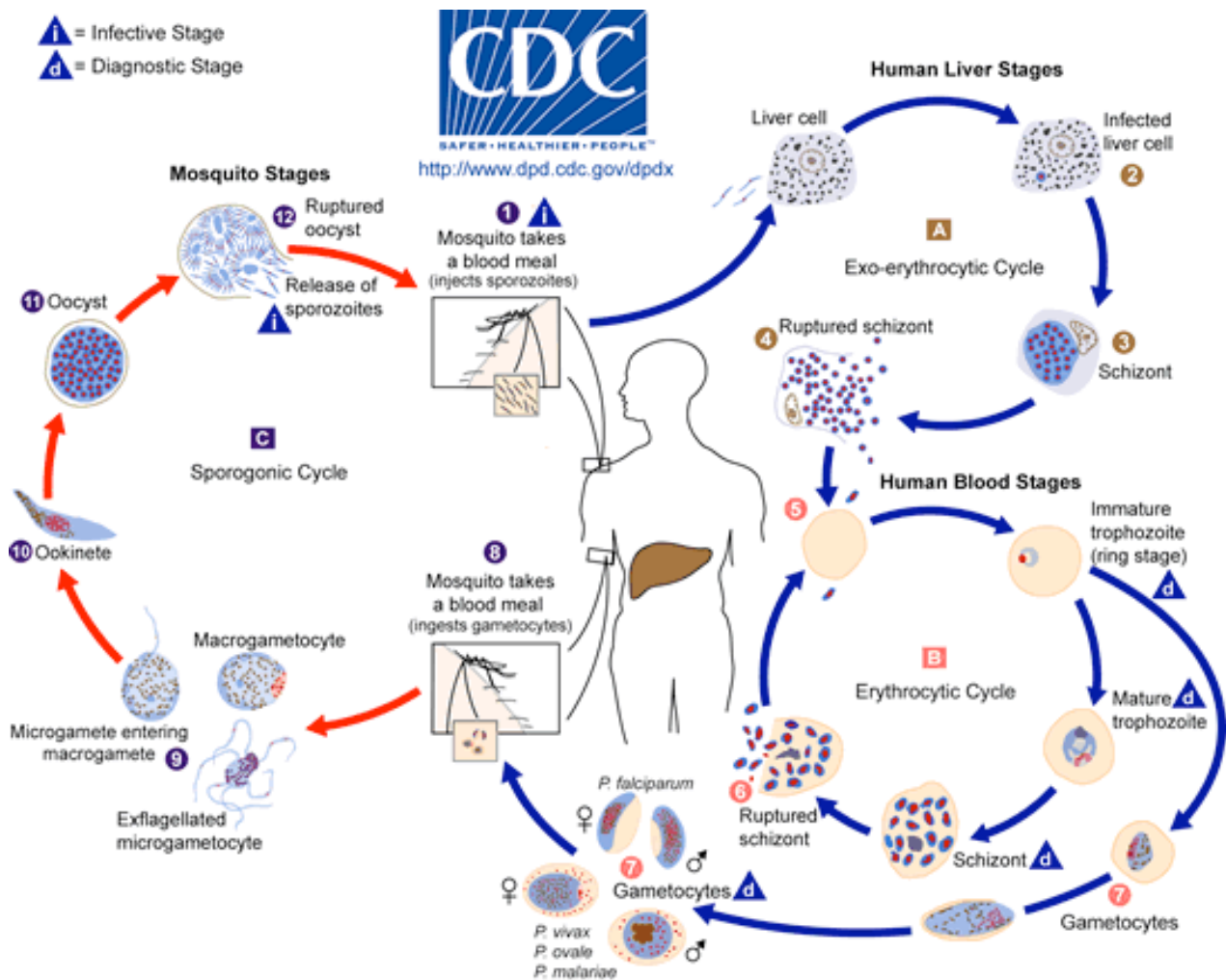
The biologic life cycle of the parasite is described in Figure A.1.

- **Human Hosts and symptoms (source: CDC and Gentilini et al., 1986, Gentilini, 1999)**

Humans infected with malaria parasites can develop a wide range of symptoms. These vary from asymptomatic infections (no apparent illness), to the classic symptoms of malaria (fever, chills, sweating, headaches, muscle pains), to severe complications (cerebral malaria, anemia, kidney failure) that can result in death. The severity of the symptoms depends on several factors, such as the species (type) of infecting parasite and the human's acquired immunity and genetic background.

All the typical clinical symptomatology and severe disease pathology associated with malaria is caused by the asexual erythrocytic or blood stage parasites. Malaria disease can be categorized as uncomplicated or severe (complicated). In general, malaria is a curable disease if diagnosed and treated promptly and correctly.

FIGURE A.1: The Life Cycle of Malaria (source: CDC)



The malaria parasite life cycle involves two hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host (1). Sporozoites infect liver cells (2) and mature into schizonts (3), which rupture and release merozoites (4). Of note, in *P. vivax* and *P. ovale* a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years later. After this initial replication in the liver (**exo-erythrocytic schizogony A**), the parasites undergo asexual multiplication in the erythrocytes (**erythrocytic schizogony B**). Merozoites infect red blood cells (5). The ring stage trophozoites mature into schizonts, which rupture releasing merozoites (6). Some parasites differentiate into sexual erythrocytic stages (gametocytes) (7). Blood stage parasites are responsible for the clinical manifestations of the disease.

The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal (8). The parasites' multiplication in the mosquito is known as the **sporogonic cycle C**. While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes (9). The zygotes in turn become motile and elongated (ookinetes) (10), which invade the midgut wall of the mosquito where they develop into oocysts (11). The oocysts grow, rupture, and release sporozoites (12), which make their way to the mosquito's salivary glands. Inoculation of the sporozoites (1) into a new human host perpetuates the malaria life cycle.

Incubation

Following the infective bite by the *Anopheles* mosquito a period of time (the "incubation period") goes by before the first symptoms appear. The incubation period in most cases varies from 7 to 30 days. The shorter periods are observed most frequently with *P. falciparum* and the longer ones with *P. malariae*.

Antimalarial drugs taken for prophylaxis by travelers can delay the appearance of malaria symptoms by weeks or months, long after the traveler has left the malaria-endemic area. (This can happen particularly with *P. vivax* and *P. ovale*, both of which can produce dormant liver stage parasites; the liver stages may reactivate and cause disease months after the infective mosquito bite; For *falciparum* malaria this delay does not generally exceed two months).

Such long delays between exposure and development of symptoms can result in misdiagnosis or delayed diagnosis because of reduced clinical suspicion by the health-care provider.

Uncomplicated Malaria

A malaria attack lasts 6-10 hours. It consists of:

- A cold stage (sensation of cold, shivering)
- A hot stage (fever, headaches, vomiting; seizures in young children)
- And finally a sweating stage (sweats, return to normal temperature, tiredness)

Classically, the attacks occur every second day with the "tertian" parasites (*P. falciparum*, *P. vivax*, and *P. ovale*) and every third day with the "quartan" parasite (*P. malariae*).

More commonly, the patient presents with a combination of the following symptoms:

- Fever
- Chills
- Sweats
- Headaches
- Nausea and vomiting
- Body aches
- General malaise

In countries where cases of malaria are infrequent, these symptoms may be attributed to influenza, a cold, or other common infections, especially if malaria is not suspected. Conversely, in countries where malaria is frequent, residents often recognize the symptoms as malaria and treat themselves without seeking diagnostic confirmation ("presumptive treatment"). Physical findings may include:

- Elevated temperature
- Perspiration

- Weakness
- Enlarged spleen.

In *P. falciparum* malaria, additional findings may include:

- Mild jaundice
- Enlargement of the liver
- Increased respiratory rate.

Diagnosis of malaria depends on the demonstration of parasites on a blood smear examined under a microscope. In *P. falciparum* malaria, additional laboratory findings may include mild anemia, mild decrease in blood platelets (thrombocytopenia), elevation of bilirubin, elevation of aminotransferases, albuminuria, and the presence of abnormal bodies in the urine (urinary "casts").

Severe Malaria

Severe malaria occurs when *P. falciparum* infections are complicated by serious organ failures or abnormalities in the patient's blood or metabolism. The manifestations of severe malaria include:

- Cerebral malaria, with abnormal behavior, impairment of consciousness, seizures, coma, or other neurologic abnormalities
- Severe anemia due to hemolysis (destruction of the red blood cells)
- Hemoglobinuria (hemoglobin in the urine) due to hemolysis
- Pulmonary edema (fluid buildup in the lungs) or acute respiratory distress syndrome (ARDS), which may occur even after the parasite counts have decreased in response to treatment
- Abnormalities in blood coagulation and thrombocytopenia (decrease in blood platelets)
- Cardiovascular collapse and shock
- Other manifestations that should raise concern are:
 - Acute kidney failure
 - Hyperparasitemia, where more than 5% of the red blood cells are infected by malaria parasites
 - Metabolic acidosis (excessive acidity in the blood and tissue fluids), often in association with hypoglycemia
 - Hypoglycemia (low blood glucose). Hypoglycaemia may also occur in pregnant women with uncomplicated malaria, or after treatment with quinine.

Severe malaria occurs most often in persons who have no immunity to malaria or whose immunity has decreased. These include all residents of areas with low or no malaria transmission, and young children and pregnant women in areas with high

transmission. In all areas, severe malaria is a medical emergency and should be treated urgently and aggressively.

Malaria Relapses

In *P. vivax* and *P. ovale* infections, patients having recovered from the first episode of illness may suffer several additional attacks ("relapses") after months or even years without symptoms. Relapses occur because *P. vivax* and *P. ovale* have dormant liver stage parasites (« hypnozoites ») that may reactivate. Treatment to reduce the chance of such relapses is available and should follow treatment of the first attack.

Other Manifestations of Malaria

- Neurologic defects may occasionally persist following cerebral malaria, especially in children. Such defects include troubles with movements (ataxia), palsies, speech difficulties, deafness, and blindness.
- Recurrent infections with *P. falciparum* may result in severe anemia. This occurs especially in young children in tropical Africa with frequent infections that are inadequately treated.
- Malaria during pregnancy (especially *P. falciparum*) may cause severe disease in the mother, and may lead to premature delivery or delivery of a low-birth-weight baby.
- On rare occasions, *P. vivax* malaria can cause rupture of the spleen or acute respiratory distress syndrome (ARDS).
- Nephrotic syndrome (a chronic, severe kidney disease) can result from chronic or repeated infections with *P. malariae*.
- Hyperreactive malarial splenomegaly (also called "tropical splenomegaly syndrome") occurs and is attributed to an abnormal immune response to repeated malarial infections. The disease is marked by a very enlarged spleen and liver, abnormal immunologic findings, anemia, and a susceptibility to other infections (such as skin or respiratory infections).

Immune responses to malaria

Acquired factors are not present at birth but are those characteristics, which people can adaptively develop later, such as the development of acquired immunity. People residing in malaria-endemic regions acquire immunity to malaria through natural exposure to malaria parasites. This naturally acquired malarial immunity that is protective against parasites and clinical disease results only after continued exposure from multiple infections with malaria parasites over time. Clinical immunity generally provides protection against severe effects of malaria but fails to provide

strong protection against infection with malaria parasites, and generally develops first. After several years of continued exposure people develop immunity that limits high-density parasitemia; however it does not lead to sterile protection.

The transmission intensity influences the course of development of both clinical and parasitic immunity. Where malaria transmission is intense, young children bear the brunt of the disease, but as they grow older, they build up an acquired immunity and are relatively protected against disease and blood stage parasites. In areas of low malaria endemicity both children and adults suffer disease and high parasitemia since exposure is less.

Two other characteristics of the immunity acquired against malaria is that the maintenance of this non-sterile state of immune protection requires continued exposure to malaria infection and a functioning spleen. Splenectomy makes an otherwise immune protected animal or human fully susceptible again to infection and disease. Likewise, when immune individuals leave a malaria endemic area and reside for several years in a malaria-free area often become susceptible to infection and clinical symptoms if they return to a malarious area.

- **Genetic Factors that influence malaria (source: CDC and Gentilini et al., 1986, 1999)**

Innate factors are those specific characteristics of a host that are present from birth. Several innate influence malaria infection. For example, persons who carry the sickle cell trait (heterozygotes for the abnormal hemoglobin gene HbS) will be relatively protected against severe disease and death caused by *Plasmodium falciparum* malaria. In general, the prevalence of hemoglobin-related disorders and other blood cell dyscrasias, such as Hemoglobin C, the thalassemias and G6PD deficiency, are more prevalent in malaria endemic areas and are thought to provide protection from malarial disease. Another example of a genetic factor involves persons who do not have the Duffy blood on their erythrocytes. These Duffy negative individuals have red blood cells that are refractory to infection by *P. vivax*. Most of the people in West Africa and much of East Africa do not have this receptor and they are protected from *P. vivax* infection.

- **Anopheles Mosquitoes (source: CDC and Gentilini et al., 1986, 1999)**

Malaria is transmitted among humans by female mosquitoes of the genus *Anopheles*. Female mosquitoes take blood meals to carry out egg production, and such blood meals are the link between the human and the mosquito hosts in the parasite life cycle. Of the approximately 430 known species of *Anopheles*, only 30-50 transmit malaria in nature. The successful development of the malaria parasite in the

mosquito (from the "gametocyte" stage to the "sporozoite" stage) depends on several factors. The most important is ambient temperature and humidity (higher temperatures accelerate the parasite growth in the mosquito) and whether the *Anopheles* survives long enough to allow the parasite to complete its cycle in the mosquito host ("sporogonic" or "extrinsic" cycle, duration 10 to 18 days). Differently from the human host, the mosquito host does not suffer noticeably from the presence of the parasites. What types of *Anopheles* are present in an area at a given time will influence the intensity of malaria transmission. Not all *Anopheles* are equally good "vectors" for transmitting malaria from one person to another. Different *Anopheles* species may differ in selected behavior traits, with important consequences on their abilities as malaria vectors. In some species the females prefer to get their blood meals from humans ("anthropophilic") while in others they prefer animals ("zoophilic"). Some species prefer to bite indoors ("endophagic"), and others prefer outdoor biting ("exophagic"). All other factors being equal, the anthropophilic, endophagic species will have more frequent contacts with humans and thus will be more effective malaria vectors. Some species prefer to rest inside the dwellings where they have just obtained their blood meals ("endophilic") while others prefer to rest outdoors ("exophilic"). An important biologic factor is insecticide resistance. If the mosquitoes are resistant to the insecticide(s) used locally for spraying or for treating bed nets, these measures will be ineffective in curtailing transmission.

- **Parasites (source: CDC and Gentilini et al., 1986, 1999)**

Characteristics of the malaria parasite can influence the occurrence of malaria and its impact on human populations:

- Areas where *P. falciparum* predominates (such as SSA) will suffer more disease and death than areas where other species predominate.
- *P. vivax* and *P. ovale* have stages ("hypnozoites") that can remain dormant in the liver cells for extended periods of time (months to years) before reactivating and invading the blood. Such relapses can result in resumption of transmission after apparently successful control efforts, or can introduce malaria in an area that was malaria-free
- Various strains of parasites can occur within the same species. Variant strains of *P. falciparum* are considered one cause for repeated episodes of malaria because immunity built against one strain is not necessarily protective against another.
- *P. falciparum* (and to a lesser extent *P. vivax*) have developed strains that are resistant to antimalarial drugs. Such strains are not uniformly distributed.

Constant monitoring of the susceptibility of these two parasite species to drugs used locally is critical to ensure effective treatment and successful control efforts.

• **Clasification of Malaria risk (Hay et al., 2008; WHO malaria terminology, 1953; Saade, 2005)**

Some epidemiologic indices:

Different criterias can be used to define malaria endemicity:

- The Spleen Rate (SR): the proportion of a sampled population with palpable enlargement of the spleen.
- Annual Parasite Incidence (API): the proportion of population with parasites (of any type) in their blood (expressed per 1000 of the population in the administrative area per year). The API is deemed valid if the annual blood examination rate (% of the target population examined is >10%).
- Slide Positive Rate (SPR): the percentage of examined slides found positive in a population.
- *Pf* Parasite Rate (*Pf* PR): the proportion of population with *P.falciparum* parasites in their blood at one point in time.
- The Basic Reproductive Rate (R_0): the number of secondary cases following introduction of a single infected individual into a susceptible population. This is an indicator of the intensity of transmission.
- Entomological Inoculation Rate (EIR): the average number of infective bits per people per time.
- The sporozoite index: the percent of female *Anopheles* with sporozoites in their salivary glands.
- The anthropophilic index or human blood ratio: the proportion of freshly fed *Anopheles* giving a positive precipiting reaction for human blood for the particular conditions in which the capture has been made.
- Transmission patterns: duration, seasonality, intensity, plurimodality, etc.

The Stable/Unstable classification:

The average number of feeds that a mosquito takes on a human being during its life determines the stability of malaria. Other classifications may be subordinate to this one (Hay et al., 2008):

- Stable: vector-based index described above >2.5
- Unstable: vector-based index described above <0.5
- Intermediate stability: vector-based index described above between 0.5 and 2.5.

The Epidemic/Endemic classification:

Malaria endemicity can be defined as follow:

- Holoendemic: SR: > 75%, PR > 75%
- Hyperendemic: SR: 51-75%, PR: 51-75%
- Mesoendemic: SR: 11-50%, PR: 26-50%
- Hypoendemic: SR: 0-10%, PR < 25%
- Non-endemic or epidemic: API < 0.01%
- Free: API = 0%

Eco-epidemiological facies:

In Africa:

- Subequatorial facies
- Tropical facies
- High-altitude tropical facies
- Subdesert facies

In Mali:

- Soudano-Guinean (regions: Sikasso)
- North-soudanian and Sahel (regions: Ségou, Koulikoro, Mopti)
- Saharian (regions: Gao, Kidal, Tombouctou)
- Dams (regions: Sélingué, Manantali, Markala)
- Urban (regions: Bamako, Mopti city)

- **Control and Prevention (source: CDC)**

Prevention:

Prevention of malaria can aim at either:

- Preventing infection, by avoiding bites by parasite-carrying mosquitoes.
- Preventing disease, by using antimalarial drugs prophylactically. The drugs do not prevent initial infection through a mosquito bite, but they prevent the development of malaria parasites in the blood, which are the forms that cause disease. This type of prevention is also called "suppression."

Prevention is achieved through:

- Vector control.
- Personal protection measures.
- Preventive treatment with antimalarial drugs of vulnerable groups.

Treatment:

Most drugs used in treatment are or were active against the parasite forms in the blood:

- Chloroquine
- Sulfadoxine-pyrimethamine
- Mefloquine
- Amodiaquine
- Atovaquone-proguanil
- Quinine
- Doxycycline
- Artemisin derivatives

Resistance depends on the parasite species.

Resistance (WHO, Guidelines for the treatment of Malaria, 2006)

In *P. falciparum*, resistance has been observed to almost all currently used antimalarials (amodiaquine, chloroquine, mefloquine, quinine and sulfadoxine–pyrimethamine) except for artemisinin and its derivatives. The geographical distributions and rates of spread have varied considerably.

P. vivax has developed resistance rapidly to sulfadoxine–pyrimethamine in many areas. Chloroquine resistance is confined largely to Indonesia, East Timor, Papua New Guinea and other parts of Oceania. There are also documented reports from Peru. *P. vivax* remains sensitive to chloroquine in South-East Asia, the Indian subcontinent, the Korean peninsula, the Middle East, north-east Africa, and most of South and Central America.

Malaria control in endemic areas:

The goal of malaria control in malaria-endemic countries is to reduce as much as possible the health impact of malaria on a population, using the resources available, and taking into account other health priorities. Malaria control does not aim to eliminate malaria totally. Complete elimination of the malaria parasite (and thus the disease) would constitute eradication. While eradication is more desirable, it is not currently a realistic goal for most of the countries where malaria is endemic. Malaria control is carried out through the following interventions, which are often combined:

- Case management (diagnosis and treatment) of patients suffering from malaria
- Prevention of infection

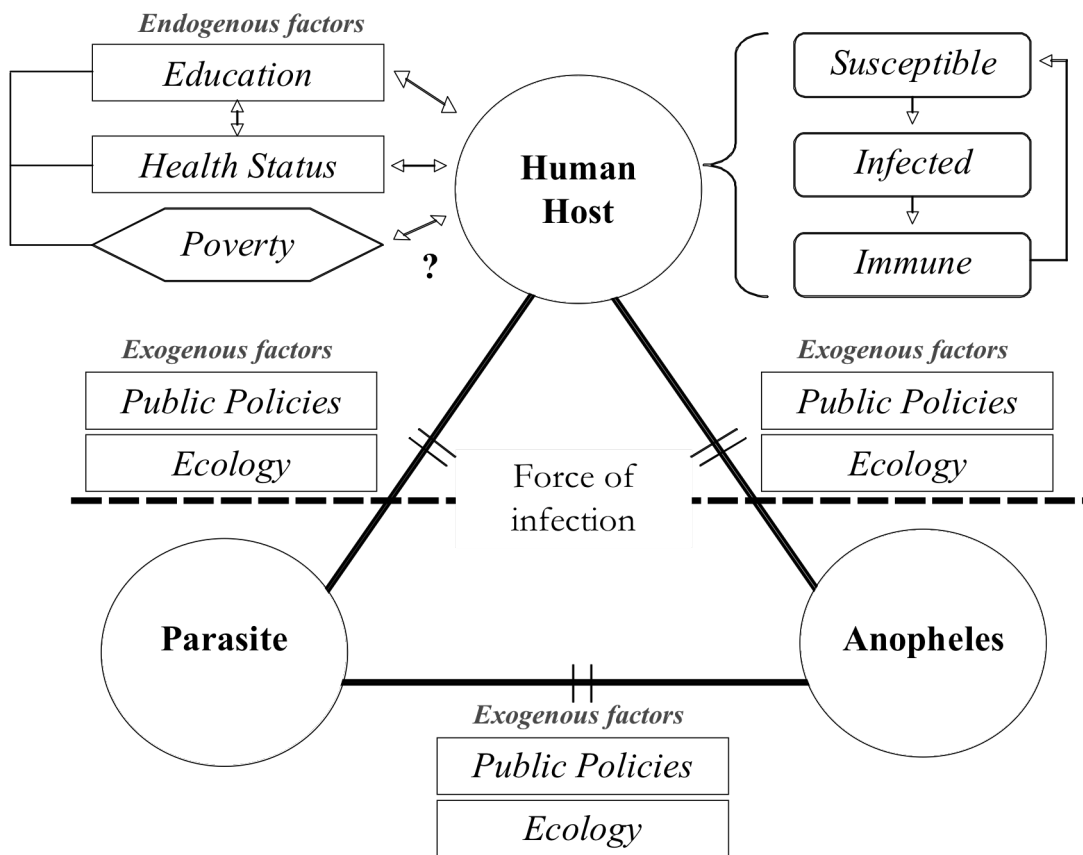
-
- Prevention of disease by administration of antimalarial drugs to particularly vulnerable population groups such as pregnant women and infants.

- **The Malaria cycle: socioeconomic and institutional factors**

As suggested in Chapter 1, many other factors are involved in the risk of malaria for individuals and communities. In particular, human behavior, often dictated by social and economic reasons, can influence this risk. For example:

- Poor rural populations often cannot afford the housing and bed nets that would protect them from exposure to mosquitoes.
- These persons often lack the knowledge to recognize malaria and to treat it promptly and correctly. Often, cultural beliefs result in use of traditional, ineffective methods of treatment.
- Even when people know what to do, they often cannot do it because of financial reasons (no money to buy drugs) or physical impossibility (the nearest health post is a 3-day walk).
- Human activities can create breeding sites for larvae (standing water, burrow pits).
- Agricultural work such as harvesting (also influenced by climate) may force increased nighttime exposure to mosquito bites.
- Raising domestic animals near the household may provide alternate sources of blood meals for *Anopheles* mosquitoes and thus decrease human exposure.
- War, migrations (voluntary or forced) and tourism may expose non-immune individuals to an environment with high malaria transmission.
- Human behavior in endemic countries also determines in part how successful malaria control activities will be in their efforts to decrease transmission. The governments of malaria-endemic countries often lack financial resources. As a consequence, health workers in the public sector are often underpaid and overworked. They lack equipment, drugs, training, and supervision. The local populations are aware of such situations when they occur, and cease relying on the public sector health facilities. Conversely, the private sector suffers from its own problems. Regulatory measures often do not exist or are not enforced. This encourages private consultations by unlicensed, costly health providers, and the anarchic prescription and sale of drugs (some of which are counterfeit products). Correcting this situation is a tremendous challenge that must be addressed if malaria control is to be successful.

FIGURE A.2: The “Human, Anopheles, Parasite” Tryptique (*source: author*)



APPENDIX 2: PCA, CRONBACH'S ALPHA AND TEST-RETEST RELIABILITY

PRINCIPAL COMPONENT ANALYSIS:

Principal Component Analysis (PCA) is a statistical technique for data reduction. It is one of the oldest but still most used techniques of multivariate analysis. The basic idea of this method is to try to describe the variation of the variables in a set of multivariate data as parsimoniously as possible using a set of derived uncorrelated variables, each of which is a particular linear combination of those in the original data. In other words, PCA is a transformation from the observed y_1, \dots, y_m normalized variables to new variables z_1, \dots, z_p , called principal components, where:

$$\begin{aligned} z_1 &= a_{1,1}y_1 + a_{1,2}y_2 + \dots + a_{1,m}y_m \\ z_2 &= a_{2,1}y_1 + a_{2,2}y_2 + \dots + a_{2,m}y_m \\ &\vdots = \quad \vdots \quad + \quad \vdots \quad + \dots + \quad \vdots \\ z_p &= a_{p,1}y_1 + a_{p,2}y_2 + \dots + a_{p,m}y_m \end{aligned} \tag{A1}$$

The new variables are derived in decreasing order of importance. One restriction is often placed on the coefficients i.e. that their sum of squares is one in order to avoid the variance of z to increase indefinitely. The coefficient $a_{1,1}$ to $a_{1,m}$, for the first principal component are derived so that the sample variance of z_1 is as large as possible. The coefficients defining the second principal component z_2 are determined to maximise its sample variance subject to the constraint that the sample correlation between z_1 and z_2 is 0. The other components are defined similarly by requiring that they are uncorrelated with all previous principal components.

In practice, components can be derived from different methods and criterias (Tenenhaus, 2006):

- The inertia criteria is the oldest method (Pearson, 1901)
- The correlation and the variance criteria (Hotelling, 1933)

In this thesis, principal components have been derived from the variance criteria (Hotelling, 1933) and from the correlation matrix, which corresponds, by simplification, to the Pearson approach. We maximise the following criteria:

$$\sum_{h=1}^p Var(z_h) \tag{A2}$$

It can be shown that the required coefficients a_1 to a_m are given by the eigenvectors of the sample correlation matrix, S , of y_1, \dots, y_m and the corresponding eigenvalues are the variance of each principal component, z_h . The principal components are orthogonal and can be written as:

$$z_h = \sum_{j=1}^m \mu_{hj} y_j \quad (\text{A3})$$

As the observed y_1, \dots, y_m are normalized, the variance of each principal component, λ_h , is also the eigenvalue i.e. the inertia of all the points projected on a one dimension graph defined by μ_h , the corresponding eigenvector of the correlation matrix between the observable variables, which maximizes λ_h .

The maximum of (A2) is:

$$\sum_{h=1}^p \lambda_h \quad (\text{A4})$$

The principal components can be interpreted by inspecting the eigenvectors defining them. It can be demonstrated that multiplying the eigenvector by the square roots of the corresponding eigenvalue provides exactly the correlation coefficients between an observable normalized variable and a component. As the eigenvectors are returned in orthonormal form, the proportion of variance explained by the principal component, z_h , is λ_h/p .

An informal rule for choosing the number of components to represent a set of correlations is to use only those components with eigenvalues greater than one (Kaiser criteria). Another commonly used criteria is the scree plot, a plot of the eigenvalues against their rank.

Scores for each individual on the first principal component can be used as a way of summarizing the original data. Filmer & Pritchett (1998; 2001) were some of the first authors to use PCA to estimate wealth and long-run household economic status without expenditure data. They argue that the PCA approach is a “second best” in response to data constraint problems and that PCA is useful to catch information common to all of the variables included in the analysis. This method is now commonly used by DHS to create a wealth index in their surveys. The assumption is that household long-run wealth explains the maximum variance and covariance in the asset variables.

CRONBACH’S ALPHA STATISTIC:

The quantity of interest in the last Chapter of this thesis is the Cognitive function of children. However this quantity is impossible to measure explicitly. Instead, we have made a series of tests and we combined the results to these tests into a single numerical value by using Principal Component Analysis. When different Items are used to form a scale, they need to have internal consistency (Bland & Altman, 1997).

In our case, the tests should all measure the same thing i.e. children educational achievement and all tests should be correlated with one another.

Cronbach's Alpha statistic is commonly used as a measure of the internal consistency of psychometric instruments (Cronbach, 1951). Cronbach's α measures how well a set of variables or items measures a single, unidimensional latent construct. The basic idea of the Cronbach's α statistic is that the variance of the sum of a group of independent variables is the sum of their variances. The formula is:

$$\alpha = \frac{k}{k-1} \left(1 - \frac{\sum s_i^2}{s_T^2} \right) \quad (\text{A5})$$

Where k is the number of items, s_i^2 is the variance of the i^{th} and s_T^2 is the variance of the total score formed by summing all items.

TEST-RETEST RELIABILITY:

The test-retest reliability of a survey instrument, like a psychological test, is estimated by performing the same survey with the same respondents at different moments of time. The closer the results are, the higher is the test-retest reliability of the survey instruments. The correlation coefficient between such two sets of responses is often used as a quantitative measure of the test-retest reliability. In the ideal case, both scores coincide for each respondent and, hence, the correlation coefficient is 1. In practice, this is almost never the case - the scores produced by a respondent would vary if the test were carried out several times. Normally, values of the correlation 0.7...0.8 are considered as satisfactory or good.

APPENDIX II.1: DESCRIPTIVE STATISTICS

TABLE II.A.1: Definitions, Sources and Descriptive Statistics for main Cross-country analysis (1996)

Variables	Definition	Source	Descriptive Statistics				
			Obs	Mean	Std Dev	Min	Max
Primary repetition rate 1996	% of repeaters in primary education, 1996 (some values are for 1995 or 1997)	<i>Al Samarrai S (2006) from UNESCO 2001 & UNESCO EFA 2000</i>	147	0.06	0.08	0.00	0.35
Primary completion rate 1996	Total number of students (of any age) in the last grade of primary school, minus the number of repeaters in that grade, divided by the total number of children of official graduation age, 1996	<i>WDI 2005 WB</i>	79	0.74	0.26	0.13	1.02
Falciparum Malaria index 1994	% of 1995 population at risk of contracting <i>falciparum</i> malaria in a country, 1994	<i>Sachs J & Gallup J.L (2001). Dataset available on the website of The Earth Institute at Columbia University.</i>	167	0.30	0.41	0.00	1.00
Malaria ecology	Ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).	<i>(Kiszewski & al - 2004). Dataset available on the website of The Earth Institute at Columbia University.</i>	173	3.65	6.41	0.00	31.54
Log GNP per capita PPP (\$ constant 95)	Log GNP per capita PPP (\$ constant), 1995	<i>Al Samarrai S (2006) from WDI 2000 WB</i>	176	7.55	1.59	4.69	10.79
SSA	Sub-Saharan Africa Dummy	<i>Author & WB</i>	201	0.23	0.42	0.00	1.00
MENA States	Middle East and North Africa Dummy	<i>Author & OCDE</i>	201	0.11	0.32	0.00	1.00
East Asia	East Asia Dummy	<i>Author & WB</i>	201	0.10	0.30	0.00	1.00
South Asia	South Asia Dummy	<i>Author & WB</i>	201	0.04	0.20	0.00	1.00
Latin America and the Caribbean	Latin America and the Caribbean Dummy	<i>Author & WB</i>	201	0.18	0.39	0.00	1.00
Subcontinents	1 = East asia, 2= Other East Asia, 3= South East Asia, 4 = South West Asia, 5= South Asia, 6 = Oceania, 7 = Eastern Africa, 8 = Middle Africa, 9 = Southern Africa, 10 = Western Africa, 11 = Caribbean, 12 = Middle America, 13 = Temperate South America, 14 = Tropical South America 15 = Arab States, 16 Western Europe, 17 = Eastern Europe, 18 = North America	<i>Author & WB</i>	193	10.87	4.88	1.00	18
Log Urban Pop 1996 (% total pop)	Log Urban Population (% total pop), 1997	<i>Al Samarrai S (2006) from WDI 1998 WB</i>	191	3.85	0.55	1.75	4.6
Log Under 5 Child mortality 1995	Log Mortality Rate, under five years (total per 1000 live births), 1995	<i>WDI 2005 WB</i>	182	3.72	1.13	1.54	5.68
Log Publ Expend in Prim Educ (% GNP) 1996	Log Public current expenditure in primary education as a percentage of GNP, 1996	<i>Al Samarrai S (2006) from UNESCO EFA 2000, WER 2000</i>	136	-4.11	0.6	-5.8	-2.31
Log Primary expenditure per pupil PPP 1996	Log Public current expenditure in primary education per pupil, PPP, 1996	<i>Al Samarrai S (2006) from UNESCO EFA 2000, WER 2000 and WDI 2000</i>	146	6.21	1.39	1.09	8.94
Log Prim School pupil-teacher ratio 90	Log Primary School pupil-teacher ratio 90	<i>Al Samarrai S (2006) from UNESCO 2001, WER 2000</i>	171	3.21	0.47	1.66	4.68
Subtropics (% land area)	Subtropical land area (%)	<i>Sachs J & Gallup J.L (1999) CID geography dataset</i>	150	0.28	0.32	0.00	0.98
Tropics (% land area)	Tropical land area (%)	<i>Sachs J & Gallup J.L (1999) CID</i>	150	0.16	0.25	0.00	1.00
Yellow fever	% of 1995 pop in area with yellow fever, 1996	<i>Sachs J & Gallup J.L (1999) CID geography dataset</i>	163	0.12	0.22	0.00	0.65
Dengue Fever	% of 1995 pop in area with dengue, 1975-1995	<i>Sachs J & Gallup J.L (1999) CID geography dataset</i>	163	0.18	0.25	0.00	0.61
Control of Corruption 96	Extent to which public power is exercised for private gain, including both petty and grand forms of corruption, as well as "capture" of the state by elites and private interests	<i>Aggregate Governance Indicators from Kaufmann D, Kray A & Mastruzzi M (2006)</i>	151	0.00	1.00	-2.19	2.50
Governance effectiveness 96	The quality of public services, the quality of civil services and the degree of its independence from political pressures, the quality of policy formulation and implementation, and the credibility of the government's commitment to such policies	<i>Aggregate Governance Indicators from Kaufmann D, Kray A & Mastruzzi M (2006)</i>	163	0.00	1.00	-1.98	2.23

APPENDIX II.2: MODEL SPECIFICATION

A model specification error can occur when one or more relevant variables are omitted from the model or one or more irrelevant variables are included in the model. Underspecifying the model (i.e. excluding a relevant variable) generally causes the OLS estimators to be biased and inconsistent if the omitted variable is correlated with both included variables and the dependent variable. We tried to account for relevant omitted variables using an instrumentation procedure. However, if irrelevant variables are included in the model, the common variance they share with included relevant variables may be wrongly attributed to them. In other words, overspecifying the model does not affect the unbiasedness of the OLS estimators but has undesirable effects on the variance of the OLS estimators.

The specifications presented in Chapter 2 have been built using a combination of methods: exploratory data analysis, past empirical research survey, forward selection, backward elimination and fragility analysis. Firstly, several explanatory variables candidates were identified from the literature and the exploratory data analysis. Secondly, the forward stepwise selection technique (that is a combination of forward and backward procedures) has been used to select main explanatory variables. The stepwise method is a modification of the forward-selection method. The forward selection technique begins with no variable in the model. For each of the plausible independent variables, the forward method calculates partial F statistics that reflect the variable's contribution to the model. The variable is included if the p -value for the F statistic reaches the 5% significance level for addition to the model. If no F statistic has a significant level greater than 5%, the selection stops. Otherwise, the forward method adds the variable that has the largest F statistic to the model. We next calculate partial F statistics again for the variables still remaining outside the model and the evaluation process is repeated. The selection process stops when no remaining variable produces a significant partial F statistic. The stepwise method is a modification of this forward-selection and differs in that testing is performed after regressor has been entered to see if any of the other regressors in the model can be deleted. Any variable that does not produce a partial F statistic significant at the 10% level is deleted. Only after this check is accomplished can another variable be added to the model. The procedure terminates when none of the variables outside the model has an F statistic significant at the 5% level (for addition to the model) and every variable in the model is significant at the 10% significance level (for removal from the model) or when the variable to be added to the model is the one just deleted from it. The result of this analysis is provided in regressions (1) and (3) of Table II.A.2. The corresponding 2SLS regressions are provided too.

TABLE II.A2 : Results from Stepwise Selection Method

	Dependant Variable is Primary Repetition Rate 1996		Dependant Variable is Primary Completion Rate 1996	
	(1)	(2)	(3)	(4)
	<i>OLS</i>	<i>2SLS</i>	<i>OLS</i>	<i>2SLS</i>
Sachs & Gallup <i>falciparum</i> Malaria index (1994)	0.0661** (0.032)	0.084** (0.0391)	-0.372*** (0.080)	-0.490*** (0.108)
Log Prim School pupil-teacher ratio 1996	0.102*** (0.022)	0.089*** (0.030)	.	.
Log Under 5 Child mortality 1995	.	.	-0.084*** (0.030)	-0.046 (0.037)
Constant	-0.266*** (0.067)	-0.231** (0.088)	1.151*** (0.087)	1.048*** (0.101)
<i>Number of observations</i>	80	80	54	54
<i>Overall R squared</i>	0.533	0.529	0.792	0.780

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
4. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

However, selecting predictors mechanically is not without conceptual problems. The algorithm used does not know our research question nor the literature related to our topic. It cannot distinguish predictors of direct interest from those whose effects we want to control (typically, GNP per capita). Hence, the regression specifications presented in Table II.A.2 have been completed using the existing literature on the subject and some variables have been added taking into account that different models serve different purposes and that we focused on the ceteris paribus interpretation of regressions. The significance and sensitivity of these independent variables were also explored. The resource variables and income per capita have been included directly on the basis of previous results (Al-Samarrai, 2006; Barro & Lee, 2001). The link between public expenditure and educational outcomes is still poorly understood. The nested model has been compared to the full model using an *F*-test. For the model where primary repetition rate 1996 is the dependent variable (regression (3) of Table II.1 compared to regression (1) of Table II.A.2), the change in R^2 ($=+0.066$) was significantly different from 0 at the 10% level. For the model where primary completion rate 1996 is the dependent variable (regression (3) of Table II.2 compared to regression (3) of Table II.A.2), the change in R^2 ($=+0.032$) was not significantly different from 0 at the 10% level.

Moreover, small perturbations in the data can also lead the stepwise selection method to provide different subsets when the variables are highly correlated. Table II.A.3 shows that some variables are highly correlated. In particular GNP per capita is

highly correlated with a number of variables (% of urban population, malaria index, under-five mortality rate) and the under-five mortality rate is highly correlated with the malaria index. The choice of whether or not include these two particular variables in the regression model has been made by analysing the tradeoff between bias and variance.

TABLE II.A.3: Pairwise Correlations

	Primary repetition rate 1996	Primary completion rate 1996	Falciparum Malaria index 1994	Log GNP per capita PPP (\$ constant 95)	Subtropics (% land area)	Tropics (% land area)	Log Urban Pop 1995 (% total pop)	Log Under 5 Child mortality 1995	Log Publ Expend in Prim Educ (% GNP) 1996	Log Primary expenditure per pupil PPP 1996	Log Prim School pupil-teacher ratio 90	Government Effectiveness
Primary repetition rate 1996	1.00											
Primary completion rate 1996	-0.71	1.00										
Falciparum Malaria index 1994	0.54	-0.8	1.00									
Log GNP per capita PPP (\$ constant 95)	-0.42	0.65	-0.67	1.00								
Subtropics (% land area)	0.35	-0.15	0.44	-0.4	1.00							
Tropics (% land area)	0.38	-0.62	0.54	-0.36	0.11	1.00						
Log Urban Pop 1995 (% total pop)	-0.33	0.56	-0.59	0.72	-0.39	-0.3	1.00					
Log Under 5 Child mortality 1995	0.51	-0.77	0.74	-0.91	0.43	0.48	-0.65	1.00				
Log Publ Expend in Prim Educ (% GNP) 1996	-0.15	0.06	-0.09	0.12	-0.12	-0.09	0.05	-0.04	1.00			
Log Primary expenditure per pupil PPP 1996	-0.53	0.63	-0.64	0.88	-0.45	-0.35	0.59	-0.84	0.27	1.00		
Log Prim School pupil-teacher ratio 90	0.62	-0.73	0.73	-0.72	0.46	0.43	-0.58	0.77	-0.07	-0.7	1.00	
Government Effectiveness	-0.26	0.5	-0.5	0.83	-0.37	-0.23	0.48	-0.78	0.1	0.76	-0.52	1.00

Table II.A.4 provides additional collinearity diagnostics for regressions (3) and (6) of Tables II.1 and II.2. The Variance Inflation Factor (VIF) and tolerance are both widely used measures of the degree of multi-collinearity of the i^{th} independent variable with the other independent variables in a regression model. A VIF higher than 10 is often regarded as a sign of severe or serious multi-collinearity.

TABLE II.A4 : Collinearity Diagnostics

	VIF	Tolerance
Sachs & Gallup falciparum Malaria index 1994	3.40	0.294
Log GNP per capita PPP 1996 (\$ constant 95)	13.75	0.072
Subtropics (% land area)	1.59	0.628
Tropics (% land area)	1.59	0.628
Log Urban Pop 1995 (% total pop)	3.01	0.332
Log Under 5 Child mortality 1995	8.07	0.124
Log Publ Expend in Prim Educ (% GNP) 1996	1.39	0.718
Log Primary expenditure per pupil PPP (PPP) 1996	6.67	0.150
Log Prim School pupil-teacher ratio 1996	3.10	0.322
Government Effectiveness 1996	3.82	0.261
Mean	4.64	

The GNP per capita is the only variable with a VIF value greater than 10, which indicates that this variable is possibly redundant and could be considered as a linear combination of other independent variables. However, this variable was not dropped from our main analysis because malaria is correlated to income per capita at the macroeconomic level. Note that our main results are not modified when GNP per capita is removed from the full model (Table II.A.5). Results remain unchanged when the under-five mortality rate is also removed from the regression in addition to GNP per capita.

TABLE II.A5: Regressions (3) and (6) of Tables II.1 and II.2 without GNP per Capita (1996)

	Dependant variable is Primary Repetition Rate (1996)		Dependant variable is Primary Completion Rate (1996)	
	(1) <i>OLS</i>	(2) <i>2SLS</i>	(3) <i>OLS</i>	(4) <i>2SLS</i>
Falciparum Malaria index 1994	0.0737* (0.0452)	0.130* (0.0694)	-0.260** (0.108)	-0.491** (0.223)
Subtropics (% land area)	0.0321 (0.0278)	0.0219 (0.0327)	0.0631 (0.0638)	0.0599 (0.0735)
Tropics (% land area)	0.00526 (0.0425)	-0.0172 (0.0485)	-0.000590 (0.0766)	0.0774 (0.102)
Log Urban Pop (% total pop)	0.0464* (0.0266)	0.0540* (0.0286)	0.0791 (0.0598)	0.0608 (0.0626)
Log Under 5 Child mortality 1995	0.0211 (0.0147)	0.0105 (0.0189)	-0.0523 (0.0521)	-0.00688 (0.0783)
Log Publ Expend in Prim Educ (% GNP) 1996	-0.0179 (0.0143)	-0.0168 (0.0147)	0.0130 (0.0322)	0.00168 (0.0344)
Log Primary expenditure per pupil PPP 1996	0.00719 (0.00789)	0.00663 (0.00855)	0.00936 (0.0262)	0.0197 (0.0339)
Log Prim School pupil-teacher ratio 96	0.0926*** (0.0328)	0.0800** (0.0354)	-0.140 (0.0837)	-0.0516 (0.103)
Government Effectiveness	0.00252 (0.0118)	-0.00505 (0.0149)	-0.0255 (0.0353)	-0.000192 (0.0442)
Intercept	-0.626*** (0.205)	-0.576*** (0.209)	1.126* (0.563)	0.690 (0.792)
<i>Number of observations</i>	80	80	54	54
<i>R squared</i>	0.576	0.559	0.820	0.795

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.
2. Standard errors in parenthesis
3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.
5. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

It is common for the results of cross-country regression analysis to be very sensitive to changes in the specification and the sample of countries used to estimate the model. Sensitivity analysis was used to determine how sensitive our model is to changes in the structure of the model and to the quality of data (see Appendix II.3).

Last we have shown that our main conclusions are not modified by a change in the dataset or year used. Actually, the use of actualised indicators seems to improve the quality of our estimates.

APPENDIX II.3: ROBUSTNESS CHECKS

TABLE II.A6: 2SLS results: Other controls.

Two Stage Least Squares: other controls								
	<i>Dependant variable is Primary Repetition Rate (1996)</i>				<i>Dependant variable is Primary Completion Rate (1996)</i>			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	Geografic Var	Health Var	Resources Var	Governance Var	Geografic Var	Health Var	Resources Var	Governance Var
Falci-parum Malaria index 1994	0.136 (0.090)	0.129** (0.062)	0.093*** (0.037)	0.114*** (0.036)	-0.713*** (0.232)	-0.539*** (0.199)	-0.494*** (0.104)	-0.495*** (0.081)
Log GNP per capita PPP (\$ constant 95)	0.001 (0.006)	0.024** (0.010)	0.019* (0.010)	0.014 (0.013)	0.015 (0.023)	0.011 (0.036)	0.020 (0.027)	0.013 (0.039)
SSA	0.016 (0.066)	-	-	-	0.156 (0.178)	-	-	-
Middle East and North Africa	0.031 (0.019)	-	-	-	-0.059 (0.073)	-	-	-
East Asia	0.005 (0.028)	-	-	-	0.112 (0.071)	-	-	-
South Asia	0.020 (0.032)	-	-	-	-	-	-	-
Latin America and the Caribbean	0.061*** (0.013)	-	-	-	-0.025 (0.048)	-	-	-
Log Under 5 Child mortality 1995	-	0.041** (0.020)	-	-	-	-0.013 (0.074)	-	-
Yellow fever 96	-	-0.078 (0.074)	-	-	-	0.056 (0.202)	-	-
Dengue Fever 75-95	-	0.046 (0.034)	-	-	-	-0.072 (0.096)	-	-
Log Publ Expend in Prim Educ (% GNP) 1996	-	-	-0.006 (0.013)	-	-	-	-0.005 (0.026)	-
Log Primary expenditure per pupil PPP 1996	-	-	-0.012 (0.012)	-	-	-	0.008 (0.022)	-
Log Prim School pupil-teacher ratio 96	-	-	0.100*** (0.030)	-	-	-	-0.033 (0.088)	-
Corruption 96	-	-	-	0.027 (0.018)	-	-	-	-0.041 (0.048)
Government Effectiveness 96	-	-	-	-0.055** (0.021)	-	-	-	0.049 (0.061)
Intercept	0.000 (0.059)	-0.301** (0.136)	-0.363** (0.152)	-0.073 (0.112)	0.794*** (0.193)	0.865 (0.469)	0.770 (0.417)	0.793** (0.316)
<i>Number of observations</i>	117	114	85	108	73	71	56	68
<i>R squared</i>	0.36	0.36	0.55	0.36	0.60	0.64	0.77	0.61

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.

2. Standard errors in parenthesis

3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

5. Instrument used is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

6. The p-value of the coefficient estimate on the falci-parum malaria index 1994 in regression (1) is 0.13.

7. In regression (5), South Asia was dropped because there is no South-Asian country in the sample.

TABLE II.A7: 2SLS with observations clustered within subcontinents.

	Two Stage Least Squares regressions (2SLS) with clusters					
	<i>Dependant variable is Primary Repetition Rate (1996)</i>			<i>Dependant variable is Primary Completion Rate (1996)</i>		
	(1)	(2)	(3)	(4)	(5)	(6)
Falciparum Malaria index 1994	0.180** (0.027)	0.185*** (0.070)	0.111 (0.070)	-0.543*** (0.212)	-0.554** (0.211)	-0.480* (0.274)
Log GNP per capita PPP (\$ constant 95)	0.019 (0.017)	0.030 (0.021)	0.034** (0.017)	0.029 (0.076)	0.002 (0.059)	-0.009 (0.071)
Subtropics (% land area)	0.021 (0.042)	0.009 (0.040)	0.006 (0.027)	0.013 (0.124)	0.047 (0.117)	0.065 (0.118)
Tropics (% land area)	-0.019 (0.044)	-0.025 (0.043)	-0.011 (0.042)	0.069 (0.111)	0.089 (0.119)	0.074 (0.116)
Log Urban Pop (% total pop)	0.042 (0.031)	0.039 (0.036)	0.026 (0.034)	0.042 (0.056)	0.053 (0.060)	0.068 (0.063)
Log Under 5 Child mortality 1995	0.029 (0.026)	0.026 (0.027)	0.025 (0.023)	0.000 (0.123)	0.000 (0.127)	-0.012 (0.136)
Log Publ Expend in Prim Educ (% GNP) 1996	-0.017 (0.016)	-0.012 (0.019)	-0.014 (0.020)	0.001 (0.029)	-0.002 (0.037)	0.002 (0.040)
Log Primary expenditure per pupil PPP 1996	-	-0.010 (0.015)	-0.007 (0.013)	-	0.023 (0.024)	0.022 (0.023)
Log Prim School pupil-teacher ratio 96	-	-	0.083** (0.040)	-	-	-0.055 (0.091)
Government Effectiveness	-0.014 (0.017)	-0.017 (0.016)	-0.017 (0.014)	0.003 (0.048)	0.005 (0.041)	0.002 (0.040)
Intercept	-0.466** (0.209)	-0.432* (0.250)	-0.692 (0.270)	0.497 (0.912)	0.483 (1.088)	0.737 (1.329)
<i>Number of observations</i>	84	82	80	55	54	54
<i>R squared</i>	0.46	0.48	0.58	0.75	0.77	0.79

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.

2. Standard errors in parenthesis

3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

5. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

6. The p.value of the coefficient estimate on the falciparum malaria index 1994 in regression (3) is 0,13.

TABLE II.A8: 2SLS Regressions controlling for outliers.

	Two Stage Least Squares regressions (2SLS) without influential observations					
	<i>Dependant variable is Primary Repetition Rate (1996)</i>			<i>Dependant variable is Primary Completion Rate (1996)</i>		
	(1)	(2)	(3)	(4)	(5)	(6)
Falciparum Malaria index 1994	0.132*** (0.051)	0.132** (0.060)	0.094 (0.083)	-0.633*** (0.213)	-0.631*** (0.202)	-0.585** (0.254)
Log GNP per capita PPP (\$ constant 95)	0.028** (0.011)	0.043** (0.017)	0.049*** (0.017)	0.025 (0.043)	0.003 (0.049)	-0.005 (0.056)
Subtropics (% land area)	0.046** (0.022)	0.034 (0.027)	0.014 (0.027)	-0.024 (0.081)	0.005 (0.080)	-0.003 (0.103)
Tropics (% land area)	0.002 (0.041)	-0.005 (0.048)	0.017 (0.062)	0.079 (0.104)	0.101 (0.105)	0.102 (0.111)
Log Urban Pop (% total pop)	0.014 (0.020)	-0.017 (0.025)	-0.003 (0.029)	0.071 (0.063)	0.071 (0.079)	0.089 (0.079)
Log Under 5 Child mortality 1995	0.040** (0.016)	0.036** (0.017)	0.037** (0.017)	0.039 (0.087)	0.023 (0.069)	0.020 (0.074)
Log Publ Expend in Prim Educ (% GNP) 1996	-0.008 (0.008)	0.000 (0.011)	-0.005 (0.013)	0.018 (0.029)	0.021 (0.022)	0.029 (0.021)
Log Primary expenditure per pupil PPP 1996	-	-0.019 (0.017)	-0.011 (0.021)	-	0.011 (0.020)	0.015 (0.016)
Log Prim School pupil-teacher ratio 96	-	-	0.042* (0.024)	-	-	-0.023 (0.095)
Government Effectiveness	-0.011 (0.013)	-0.011 (0.014)	-0.021 (0.014)	0.041 (0.047)	0.035 (0.039)	0.023 (0.034)
Intercept	-0.422 (0.137)	-0.287** (0.152)	-0.540*** (0.205)	0.393 (0.552)	0.532 (0.446)	0.536 (0.619)
<i>Number of observations</i>	73	71	69	47	46	45
<i>R squared</i>	0.63	0.65	0.64	0.87	0.91	0.91

Notes:

1. Robust standard errors, adjusted for heteroscedasticity are used in all cases.

2. Standard errors in parenthesis

3. *** denotes statistical significance at the 1% level, ** at the 5% level, * at the 10% level.

5. Instrument used in two stage least squares is malaria ecology which is an ecological-based spatial index of the stability of malaria transmission. The index is measured on a highly disaggregated sub-national level (Kiszewski & al - 2004).

6. 11 countries were identified as influential observations for regressions with primary repetition rates. For instance, the 11 observations dropped in regression (1) are: Cambodia, Chad, Congo, Gabon, Kenya, Lesotho, Madagascar, Nicaragua, Papua New Guinea, Uganda, Zambia.

7. From 8 to 9 countries were identified as influential observations for regressions with primary completion rates. For instance the 8 observations dropped in regression (4) are: China, Gabon, Indonesia, Kyrgyz Republic, Morocco, Nicaragua, Saudi Arabia, Switzerland.

APPENDIX II.4: MALARIA ECOLOGY

FIGURE II.A.1: World distribution of *Anopheles* vectors (source: Kizewski et al. 2004)

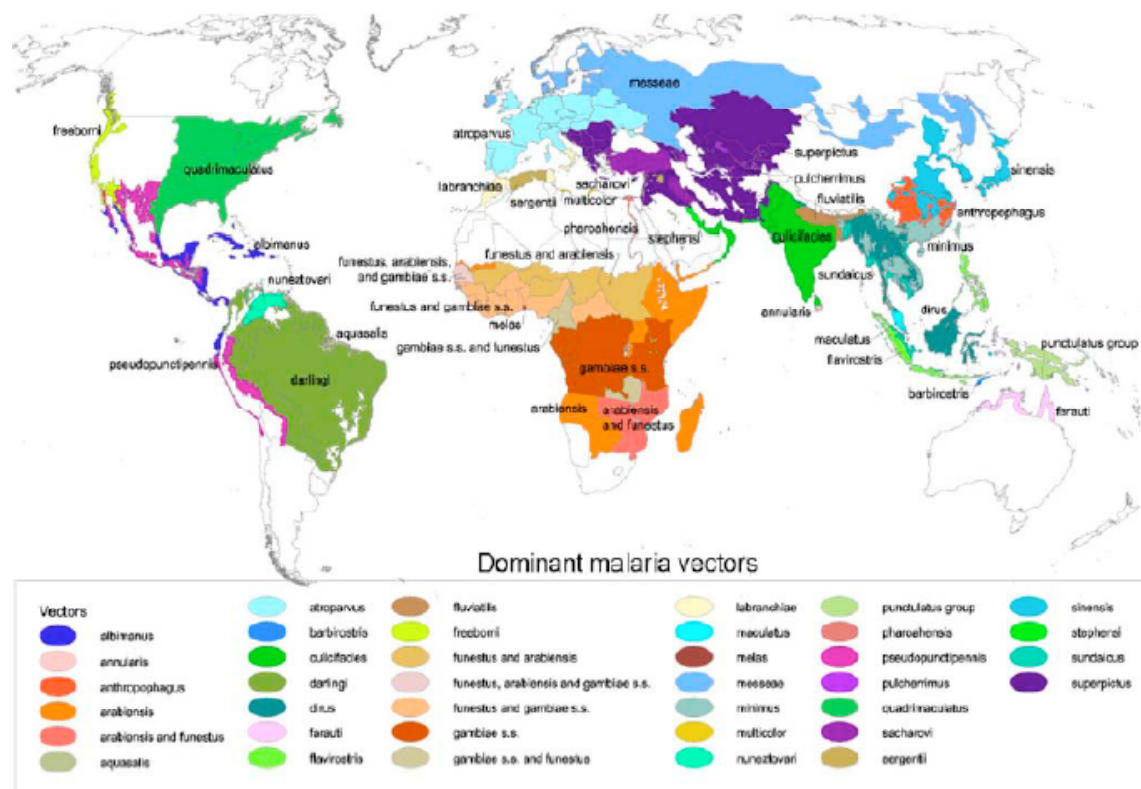
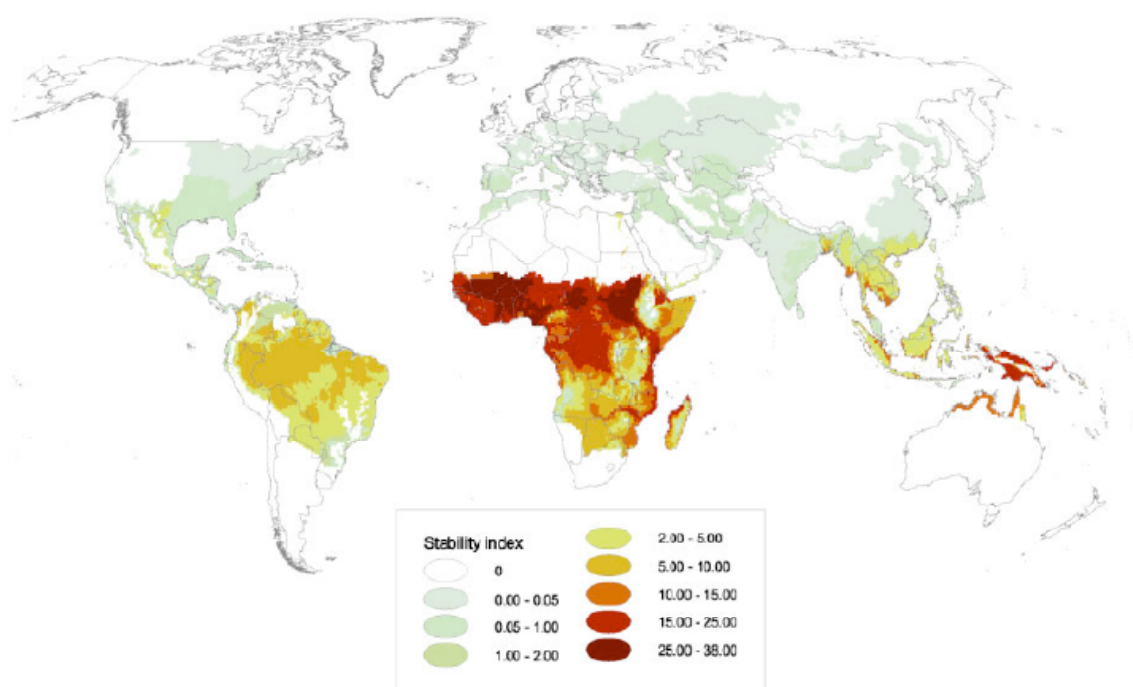


FIGURE II.A.2: Malaria Ecology: distribution of actual and potential stability (source: Kizewski et al. 2004)



APPENDIX III: INSTRUMENTATION STRATEGY

Consider our standard linear model but with only two explanatory variables (to simplify the discussion):

$$\text{Repetition Rates} = \beta_0 + \beta_1 \text{Malaria} + \beta_2 \text{Wealth Index} + u_1 \text{ (error term)} \quad (1)$$

Where we assume that $E(u_1) = 0$ and $\text{cov}(\text{Wealth Index}, u_1) = 0$.

We want to produce an unbiased and consistent estimator of β_1 . Malaria is suspected to be endogenous and Wealth Index is supposed to be exogenous i.e. “Malaria” is suspected to be correlated with u_1 and “Wealth index” is not correlated with u_1 .

We seek an instrumental variable (IV). One candidate is the percent of households with at least one mosquito net in the cluster. A valid instrument is a variable that is: 1) correlated with the observed variables that are likely to have bias problems, 2) uncorrelated with the error term i.e. uncorrelated with all unobserved variables and any measurement errors, 3) not already included as explanatory variables in the model (Wooldridge, 2002; 2003). So the % of households with at least one mosquito net in the cluster has to check:

- 1) $\text{cov}(\text{Malaria}, \% \text{ households with at least one bednet}) \neq 0$ - *this can be tested easily.*
- 2) $\text{cov}(\% \text{ households with at least one bednet}, u_1) = 0$ - *this can not be tested with one instrument variable.*
- 3) % households with at least one bednet is not included in the model - *this holds true.*

The correlation between Malaria and the % households with at least one bednet has to be seen in terms of partial correlation. The first-stage regression will be:

$$\begin{aligned} \text{Malaria} = & \pi_0 + \pi_1 \% \text{ households with at least one bednet} \\ & + \pi_2 \text{Wealth Index} + v_2 \text{ (error term)} \quad (2) \end{aligned}$$

By definition: $E(v_2) = 0$, $\text{cov}(\% \text{ households with at least one bednet}, v_2) = 0$, $\text{cov}(\text{Wealth index}, v_2) = 0$

The key identification conditions are that $\pi_1 \neq 0$ and “% households with at least one bednet” not to be perfectly correlated with “Wealth Index”. In other words after

partialling out “Wealth index”, we need “% households with at least one bednet” and “Malaria” to be correlated. This correlation cannot be 0. And minor additional assumption is that there is no perfect linear relationships among exogenous variables.

In our case “% households with at least one bednet” is not perfectly correlated with “Wealth Index” ($r = 0,10$; $p \text{ value} = 0,04$) and “% households with at least one bednet” is significantly correlated with Malaria i.e. $\pi_1 \neq 0$ (We estimate (2) by OLS and use a t test robust to heteroskedasticity).

In addition to that, as we use two instruments chosen by following a different logic (the second instrument is EIR, Entomological inoculation rate), the first stage equation will be:

$$\begin{aligned} \text{Malaria} = \pi_0 + \pi_1 \% \text{ households with at least one bednet} + \pi_3 \text{EIR} & \quad (3) \\ + \pi_2 \text{Wealth Index} + v_2 (\text{error term}) & \end{aligned}$$

The key identification assumption is then:

$\pi_1 \neq 0$ or $\pi_3 \neq 0$, and the IVs not to be perfectly correlated with “Wealth Index”.

The order condition states that we need at least as many excluded exogenous variables, as there are included endogenous variables, which is the case here. Moreover, we need the rank conditions for identification to hold. When the model is overidentified, overidentifying restrictions are the extra moment conditions that come from having more instrumental variables than endogenous explanatory variables. We have one overidentifying restriction here. As we have several instruments it is possible to test the overidentifying restrictions i.e. whether or not some of the instruments are correlated with the error term provided that at least one instrumental variable is valid (i.e. not correlated with the structural error term) (Wooldridge 2002, Chapters 5 & 6).

In our case, both instruments “% households with at least one bednet” and “EIR” are strong (partial R-squared confirm their relevance in all specifications). The F-stat of weak identification of IVs largely exceeds the threshold of 10 recommended by Staiger and Stock (1997) in all regressions. The Hansen J Test of overidentifying restrictions shows that the instruments are well identified. A rejection of the null hypothesis implies that the instruments are not satisfying the orthogonality conditions required for their employment (i.e. that they are uncorrelated with the error term of the estimated equation), which is not the case here.

Note that we used Stata (Stata Corporation, Austin, TX, USA) 2SLS command in all case rather than explicitly carry out the two-step procedure, as recommended by

Wooldridge (2002). Indeed, the standard errors and test statistics obtained by performing the two stages explicitly are not valid because the error term in the second-stage equation includes ν_2 but the standard errors involve the variance of u_1 only. The variance–covariance was therefore corrected by applying the correct mean squared error in all cases.

APPENDIX IV.1: DESCRIPTIVE STATISTICS

TABLE IV.A1: Descriptive statistics of variables included in the analysis

Variables	Obs	Mean	SD	Min	Max
<i>Active Follow-up variables</i>					
Cognitive function factor score	1794	0.000	1.413	-5.696	4.097
Temperature	1802	36.511	0.436	34	39.9
Positive Blood smear	1802	0.437	0.496	0	1
Weight	1802	28.61795	7.941081	13.9	56.6
Height	1802	132.135	12.6093	105	166
Weight for age z-score	1802	-1.424	0.988	-5.167	1.845
Height for age z-score	1802	3.999	0.345	2.834	5.270
Haemoglobin concentration	1802	12.045	1.391	6.6	16.4
P falciparum parasitaemia (parasites / μ L)	1802	920.482	6427.123	0	183275
Clinical Malaria cases	1802	0.016	0.127	0	1
Asymptomatic Malaria	1802	0.421	0.493	0	1
Splenomegaly	1802	0.051	0.220	0	1
Anaemia	1802	0.218	0.429	0	2
Helminth Infections	1802	0.003	0.057	0	1
Respiratory infections	1802	0.011	0.107	0	1
<i>Passive Follow-up variables</i>					
Clinical Malaria cases	275	0.487	0.500	0	1
Temperature	266	37.534	0.974	36	40.4
Helminth Infections cases	275	0.032	0.178	0	1
Respiratory infections cases	275	0.127	0.333	0	1
<i>Academic School Outcomes</i>					
School Delay	167	0.285	1.227	-3.630	4.079
Standardized Academic school Notes	320	0.000	0.988	-2.117	2.400
<i>Interview variables (117 households, 227 children)</i>					
Muslim	227	0.581	0.494	0	1
Bambara ethnic group	227	0.947	0.224	0	1
Childhood Past Convulsion	227	0.101	0.302	0	1
Childhood Past Hospitalization	225	0.062	0.242	0	1
Childhood Past Malnutrition	222	0.045	0.207	0	1
Mother alive	227	0.995	0.066	0	1
Father alive	227	0.885	0.319	0	1
Head of Household literate (can read or write)	117	0.051	0.221	0	1
Number of wife of the Head of Household	117	1.384	0.505	1	3
Number of Children in the Household	117	5.880	2.871	0	16
Number of persons who slept in the household the night before the interview	116	7.112	3.764	1	22
Number of children who slept in the household the night before the interview	117	4.119	2.930	0	19
Bednet in the household	117	0.512	0.501	0	1
Insecticide treated bednet in the household	117	0.444	0.499	0	1
Number of children who slept under net the day before the interview	117	0.461	1.038	0	5
Use of Insecticides	117	0.786	0.411	0	1

TABLE IV.A2: Data on school characteristics

Grade	Number of Children Per classroom (%)	Children mean age	Children age SD	Cause of Teacher Absenteeism	Teacher Absenteeism in days (% of total)
Grade 1	59 (25.99)	7.716	0.829	Total	20 (27.77)
				Strike	18 (25.00)
				Disease	1 (1.38)
				Other (family, personal)	1 (1.38)
Grade 3	65 (28.63)	9.905	1.265	Total	43 (59.72)
				Strike	18 (25.00)
				Disease	24 (33.33)
				Other (family, personal)	1 (1.38)
Grade 4 and 5	86 (37.89)	12.649	1.679	Total	6 (8.33)
				Strike	3 (4.16)
				Disease	2 (2.77)
				Other (family, personal)	1 (1.38)
Grade 6	17 (7.49)	13.808	2.105	Total	3 (4.16)
				Strike	3 (4.16)
Total	227 (100)	10.667	2.584	Total	72 (100)
				Strike	42 (58.33)
				Disease	27 (37.50)
				Other (family, personal)	3 (4.16)

Notes. - Number of children in Grade 4 is 16 (7.05%) and in Grade 5 is 70 (30.84%). Because of insufficient teachers, Grade 4 and 5 have been grouped in one classroom.

APPENDIX IV.2: OTHER RESULTS

TABLE IV.A3: Factors associated with Cognitive function factor score (Table IV.5)

Variables	Dependant variable is cognitive function PCA score				
	OLS models				
	(1)	(2)	(3)	(4)	(5)
Convenience Property Score	-0.004 (0.024)	-0.008 (0.024)	-0.008 (0.024)	-0.009 (0.024)	-0.008 (0.024)
Monetary Income (CFA Francs)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)	0.000 (0.000)
Store-of-value Property (CFA Francs)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)	-0.000 (0.000)
Land Area property dedicated to agriculture (Hectares)	-0.026*** (0.007)	-0.024*** (0.007)	-0.024*** (0.007)	-0.024*** (0.007)	-0.024*** (0.007)
Saving dummy	0.074 (0.078)	0.073 (0.079)	0.071 (0.078)	0.073 (0.078)	0.076 (0.078)
Access to Informal credit dummy	0.560*** (0.166)	0.540*** (0.167)	0.548*** (0.167)	0.545*** (0.167)	0.545*** (0.167)
Male	0.152** (0.067)	0.143** (0.068)	0.143** (0.068)	0.140** (0.068)	0.141** (0.068)
Muslim	0.078 (0.066)	0.095 (0.066)	0.090 (0.066)	0.090 (0.066)	0.090 (0.066)
Bambara ethnic group	-0.003 (0.129)	-0.030 (0.128)	-0.027 (0.128)	-0.026 (0.129)	-0.028 (0.129)
Age of the Head of Household	0.005 (0.003)	0.004 (0.003)	0.004 (0.003)	0.004 (0.003)	0.004 (0.003)
Head of Household literate (can read or write)	0.409*** (0.128)	0.404*** (0.130)	0.420*** (0.128)	0.408*** (0.130)	0.416*** (0.131)
Labour hours of the child	0.026* (0.015)	0.028* (0.015)	0.029* (0.015)	0.030** (0.015)	0.031** (0.015)
Time per week dedicated to do homeworks	0.472*** (0.103)	0.469*** (0.103)	0.468*** (0.103)	0.468*** (0.103)	0.470*** (0.103)
Somebody can help the child to do homeworks (dummy)	0.132 (0.091)	0.137 (0.092)	0.138 (0.092)	0.134 (0.092)	0.132 (0.092)
Father alive	0.007 (0.097)	0.006 (0.097)	0.003 (0.097)	-0.002 (0.097)	-0.002 (0.097)
Mother Alive	0.313 (0.261)	0.275 (0.261)	0.303 (0.261)	0.279 (0.262)	0.289 (0.261)
Grade 3 dummy	-0.889*** (0.095)	-0.879*** (0.095)	-0.877*** (0.095)	-0.868*** (0.095)	-0.872*** (0.095)
Grade 4 and 5 dummy	-2.104*** (0.157)	-2.111*** (0.158)	-2.100*** (0.158)	-2.092*** (0.159)	-2.097*** (0.158)
Grade 6 dummy	-3.393*** (0.278)	-3.383*** (0.280)	-3.379*** (0.280)	-3.361*** (0.280)	-3.374*** (0.280)
Age 7	0.555*** (0.177)	0.529*** (0.193)	0.531*** (0.190)	0.509*** (0.193)	0.507*** (0.194)
Age 8	1.106*** (0.187)	1.074*** (0.203)	1.089*** (0.198)	1.061*** (0.202)	1.059*** (0.203)
Age 9	1.530*** (0.223)	1.486*** (0.237)	1.495*** (0.233)	1.469*** (0.236)	1.472*** (0.237)
Age 10	1.818*** (0.236)	1.755*** (0.251)	1.764*** (0.246)	1.731*** (0.250)	1.738*** (0.251)
Age 11	2.226*** (0.274)	2.152*** (0.288)	2.168*** (0.284)	2.128*** (0.287)	2.134*** (0.287)
Age 12	2.818*** (0.303)	2.771*** (0.314)	2.775*** (0.310)	2.740*** (0.314)	2.748*** (0.314)
Age 13	3.246*** (0.307)	3.175*** (0.320)	3.187*** (0.316)	3.147*** (0.319)	3.159*** (0.319)
Age 14	3.734*** (0.309)	3.633*** (0.321)	3.643*** (0.316)	3.609*** (0.319)	3.615*** (0.319)
Age 15	3.831*** (0.383)	3.727*** (0.396)	3.742*** (0.391)	3.678*** (0.394)	3.689*** (0.394)
Age 16	4.417*** (0.375)	4.311*** (0.387)	4.326*** (0.384)	4.295*** (0.387)	4.305*** (0.386)
Age 17	4.687*** (0.462)	4.543*** (0.470)	4.587*** (0.466)	4.497*** (0.465)	4.513*** (0.467)

Notes. - Huber White standard errors are reported in parenthesis. Reference category for grade is Grade 1. Reference category for age is Age 6.

*** denotes statistical significance at the 1% level.

** at the 5% level

* at the 10% level.

APPENDIX IV.3: ROBUSTNESS CHECKS

TABLE IV.A4: Grade 6 school exam performance since 1998

School Year	Annual Mean Results	% of successful candidate at national entry Grade 7 examination
1998-1999	5.484 (1.008)	70.59 (0.462)
1999-2000	5.931 (1.113)	39.28 (0.497)
2000-2001	6.585 (1.109)	71.11 (0.458)
2001-2002	6.785 (0.970)	70.37 (0.465)
2002-2003	5.737 (0.792)	88.88 (0.323)
2003-2004	6.208 (0.807)	95.65 (0.208)
2004-2005	5.763 (1.335)	97.05 (0.171)
2005-2006	5.962 (0.823)	91.66 (0.288)
2006-2007	5.935 (1.842)	73.68 (0.452)
2007-2008	6.213 (1.401)	100 (0.000)

Notes. - Standard deviation in parenthesis. Annual Mean results for 1998-2008 is 6.060 and mean standard deviation for 1998-2008 is 1.120.

TABLE IV.A5: Effects of interactive terms for Malaria and Socioeconomic indices on cognitive score.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up) x Convenience property	0.015 (0.030)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000) x Convenience		-0.007 (0.008)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500 x Convenience Property			-0.072** (0.035)		
1000 x Convenience Property				-0.076* (0.042)	
1200 x Convenience Property					-0.090** (0.043)
Intercept	0.788*** (0.301)	0.753** (0.298)	0.761** (0.295)	0.745** (0.292)	0.779*** (0.294)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.104	0.104	0.119	0.121	0.122

Notes. - Huber White standard errors are reported in parenthesis. Reference category for Malarial Anaemia is no malarial anaemia.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.A6: Comparing the effect of malaria for boys and girls.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up) x Male	0.340*				
	(0.204)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000) x Male	.	0.019			
		(0.021)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500 x Male	.	.	0.211*		
			(0.126)		
1000 x Male	.	.	.	0.291*	
				(0.149)	
1200 x Male	0.259*
					(0.151)
Intercept	0.766**	0.752**	0.779***	0.759***	0.790***
	(0.301)	(0.298)	(0.296)	(0.292)	(0.294)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.106	0.104	0.118	0.121	0.121

Notes. - Huber White standard errors are reported in parenthesis. Reference category for Malarial Anaemia is no malarial anaemia.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.A7: Effects of interactive terms for Malaria and Anthropometric indices on cognitive score.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up) x Weigh-for-age z-score	-0.087				
	(0.057)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000) x Weigh-for-age z-	.	0.001			
		(0.024)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500 x Weigh-for-age z-score	.	.	-0.070		
			(0.071)		
1000 x Weigh-for-age z-score	.	.	.	-0.172**	
				(0.084)	
1200 x Weigh-for-age z-score	-0.137
					(0.088)
Intercept	0.780***	0.751**	0.768***	0.735**	0.776***
	(0.300)	(0.298)	(0.296)	(0.291)	(0.293)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.106	0.103	0.117	0.122	0.121

Notes. - Huber White standard errors are reported in parenthesis. Reference category for Malarial Anaemia is no malarial anaemia.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.A8: Fixed-effects (within child) estimations of the cognitive PCA score, excluding children with a known handicap or disease at the beginning of the study.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up)	-0.202*** (0.056)				
Lagged Asymptomatic Malaria (active follow-up)	-0.113** (0.051)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)	.	-0.032*** (0.010)			
Lagged Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)	.	-0.020** (0.009)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500	.	.	-0.272*** (0.063)		
Lagged 500	.	.	-0.207*** (0.055)		
1000	.	.	.	-0.343*** (0.076)	
Lagged 1000	.	.	.	-0.256*** (0.063)	
1200	-0.357*** (0.077)
Lagged 1200	-0.265*** (0.065)
<i>Clinical Malaria control Dummies</i>					
Clinical Malaria (between two active follow-up or during active follow-up)	-0.251** (0.117)	-0.199* (0.119)	-0.190* (0.115)	-0.185* (0.112)	-0.181 (0.112)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.232** (0.106)	-0.178* (0.107)	-0.207** (0.105)	-0.200* (0.105)	-0.202* (0.105)
<i>Chronic Malaria and Malaria Anaemia control Variables</i>					
Splenomegaly	-0.173* (0.091)	-0.238*** (0.089)	-0.208** (0.087)	-0.203** (0.090)	-0.213** (0.089)
Lagged Splenomegaly	0.445*** (0.170)	0.465*** (0.165)	0.643*** (0.157)	0.560*** (0.159)	0.553*** (0.159)
<i>Malarial Anaemia</i>					
Mild	-0.122 (0.077)	-0.164** (0.072)	-0.120 (0.074)	-0.115 (0.073)	-0.122* (0.073)
Moderate	-0.268 (0.188)	-0.275 (0.179)	-0.208 (0.179)	-0.192 (0.178)	-0.186 (0.178)
<i>Lagged Malarial Anaemia</i>					
Mild	-0.535*** (0.072)	-0.542*** (0.072)	-0.522*** (0.072)	-0.517*** (0.071)	-0.514*** (0.071)
Moderate	-0.256*** (0.058)	-0.231*** (0.061)	-0.250*** (0.058)	-0.238*** (0.058)	-0.238*** (0.058)
<i>Health control</i>					
Haemoglobin concentration	-0.030 (0.024)	-0.034 (0.024)	-0.033 (0.024)	-0.033 (0.024)	-0.036 (0.024)
Intercept	0.869*** 1518	0.823*** 1518	0.849*** 1518	0.828*** 1518	0.864*** 1518
Number of observations	0.108	0.106	0.118	0.122	0.122
Number of Children	220	220	220	220	220
R squared					

Notes. - Huber White standard errors are reported in parenthesis. Reference category for Malarial Anaemia is no malarial anaemia.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

TABLE IV.A9: Comparing the effect of malaria with those of other infections.

Variables	Dependant variable is cognitive function PCA score				
	Fixed-effects models (within child)				
	(1)	(2)	(3)	(4)	(5)
<i>Asymptomatic Malaria and Malaria Parasitaemia</i>					
Asymptomatic Malaria (active follow-up)	-0.181*** (0.056)				
Lagged Asymptomatic Malaria (active follow-up)	-0.112** (0.051)				
Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.032*** (0.010)			
Lagged Monthly Geom Mean <i>P. falciparum</i> Parasitaemia (/1000)		-0.020** (0.009)			
<i>P. falciparum</i> Parasitaemia thresholds (active follow-up)					
500			-0.279*** (0.063)		
Lagged 500			-0.202*** (0.055)		
1000				-0.344*** (0.076)	
Lagged 1000				-0.255*** (0.063)	
1200					-0.356*** (0.077)
Lagged 1200					-0.263*** (0.065)
<i>Clinical Malaria control Dummies</i>					
Clinical Malaria (between two active follow-up or during active follow-up)	-0.300** (0.117)	-0.247** (0.119)	-0.233** (0.114)	-0.235** (0.113)	-0.231** (0.113)
Lagged Clinical Malaria (between two active follow-up or during active follow-up)	-0.221** (0.103)	-0.172* (0.104)	-0.202** (0.102)	-0.194* (0.102)	-0.196* (0.102)
<i>Chronic Malaria and Malaria Anaemia control Variables</i>					
Splenomegaly	-0.127 (0.092)	-0.187** (0.090)	-0.156* (0.087)	-0.151* (0.090)	-0.161* (0.089)
Lagged Splenomegaly	0.482*** (0.174)	0.498*** (0.168)	0.677*** (0.157)	0.592*** (0.161)	0.585*** (0.161)
<i>Malarial Anaemia</i>					
Mild	-0.142* (0.076)	-0.184** (0.072)	-0.141* (0.073)	-0.137* (0.073)	-0.145** (0.073)
Moderate	-0.320* (0.188)	-0.326* (0.181)	-0.261 (0.181)	-0.244 (0.181)	-0.239 (0.181)
<i>Lagged Malarial Anaemia</i>					
Mild	-0.534*** (0.070)	-0.540*** (0.070)	-0.522*** (0.070)	-0.516*** (0.069)	-0.513*** (0.069)
Moderate	-0.225*** (0.059)	-0.199*** (0.061)	-0.218*** (0.058)	-0.205*** (0.058)	-0.206*** (0.058)
<i>Health control</i>					
Haemoglobin concentration	-0.023 (0.024)	-0.028 (0.024)	-0.027 (0.024)	-0.027 (0.024)	-0.030 (0.024)
Respiratory Infections (at active follow-up)	0.340 (0.280)	0.359 (0.283)	0.351 (0.284)	0.346 (0.282)	0.343 (0.282)
Helminth Infections (at active follow-up)	-0.035 (0.137)	-0.068 (0.139)	-0.051 (0.145)	-0.087 (0.142)	-0.076 (0.141)
Respiratory Infections between two active follow-ups	0.121 (0.133)	0.142 (0.128)	0.126 (0.127)	0.143 (0.125)	0.147 (0.125)
Helminth Infections between two active follow-ups	-0.137 (0.216)	-0.172 (0.210)	-0.221 (0.210)	-0.234 (0.206)	-0.212 (0.208)
Intercept	0.777*** (0.301)	0.742** (0.298)	0.773*** (0.297)	0.749** (0.294)	0.785*** (0.295)
Number of observations	1567	1567	1567	1567	1567
Number of Children	227	227	227	227	227
R squared	0.106	0.106	0.118	0.121	0.121

Notes. - Huber White standard errors are reported in parenthesis. Results are not changed when standard errors are clustered at the household level (not presented here). Reference category for Malarial Anaemia is no malarial anaemia.

*** denotes statistical significance at the 1% level.

** denotes statistical significance at the 5% level.

* denotes statistical significance at the 10% level.

APPENDIX IV.4: COGNITIVE FUNCTION FACTOR SCORE DISTRIBUTION

FIGURE IV.A.1: Cognitive function factor score evolution over time (*source: author*)

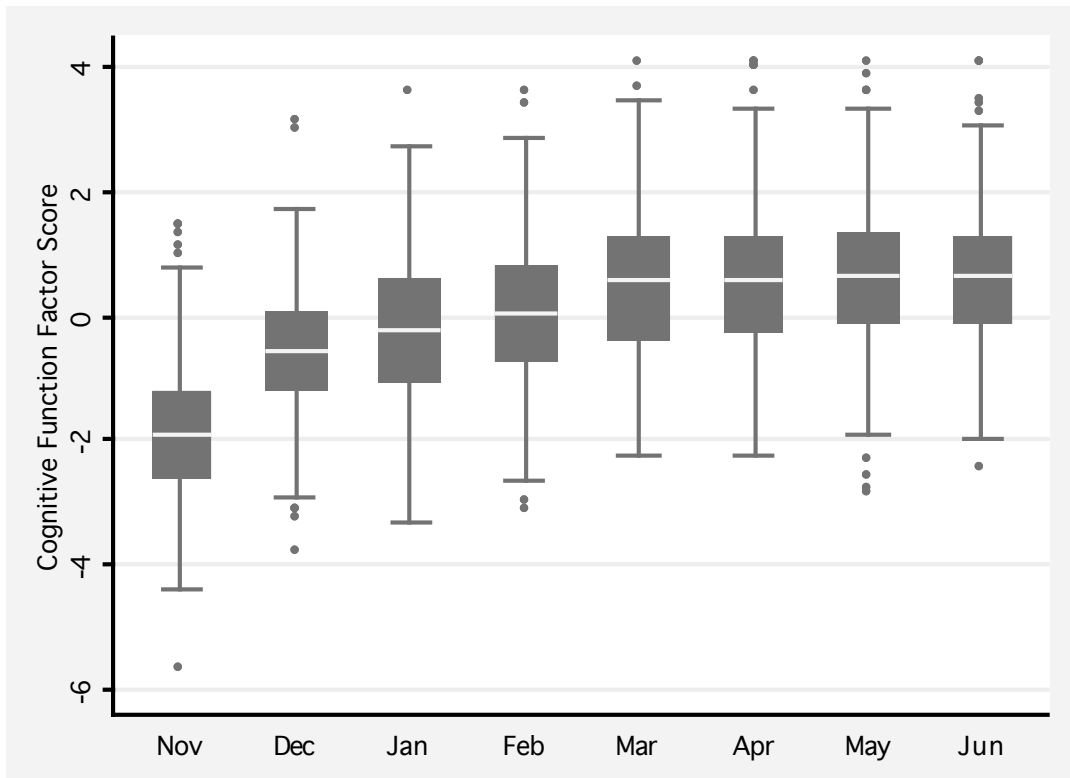


FIGURE IV.A.2: Cognitive function factor score distribution (*source: author*)

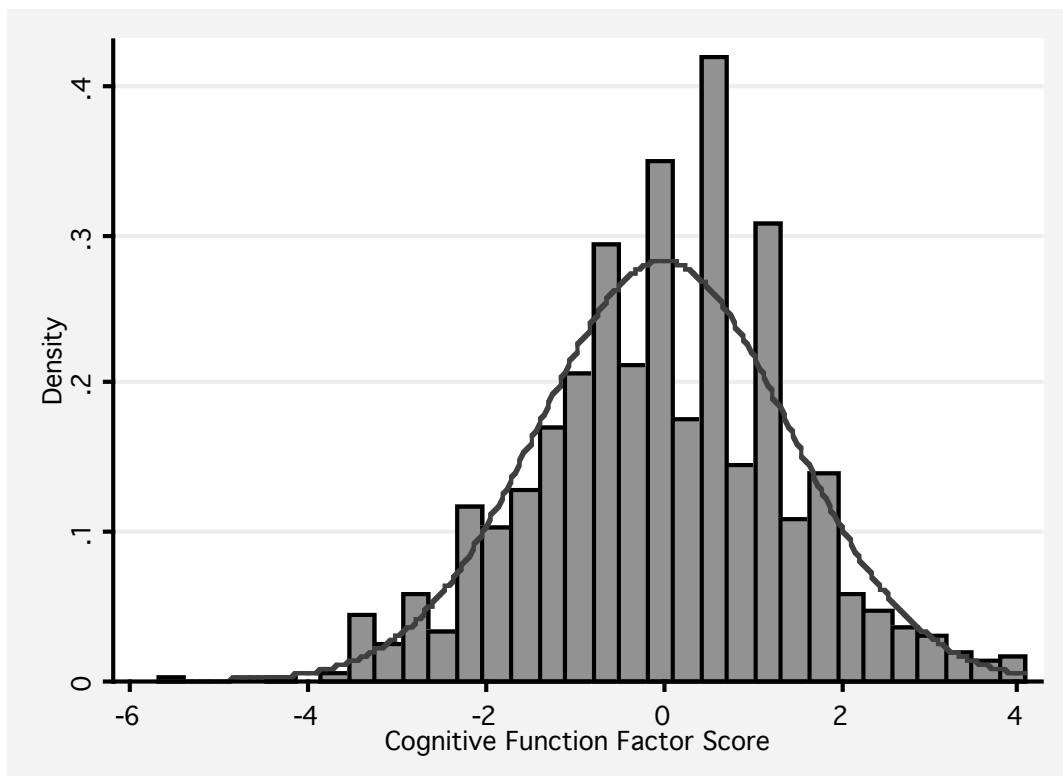


FIGURE IV.A.3: Cognitive function factor score and annual average standardized academic school notes (*source: author*)

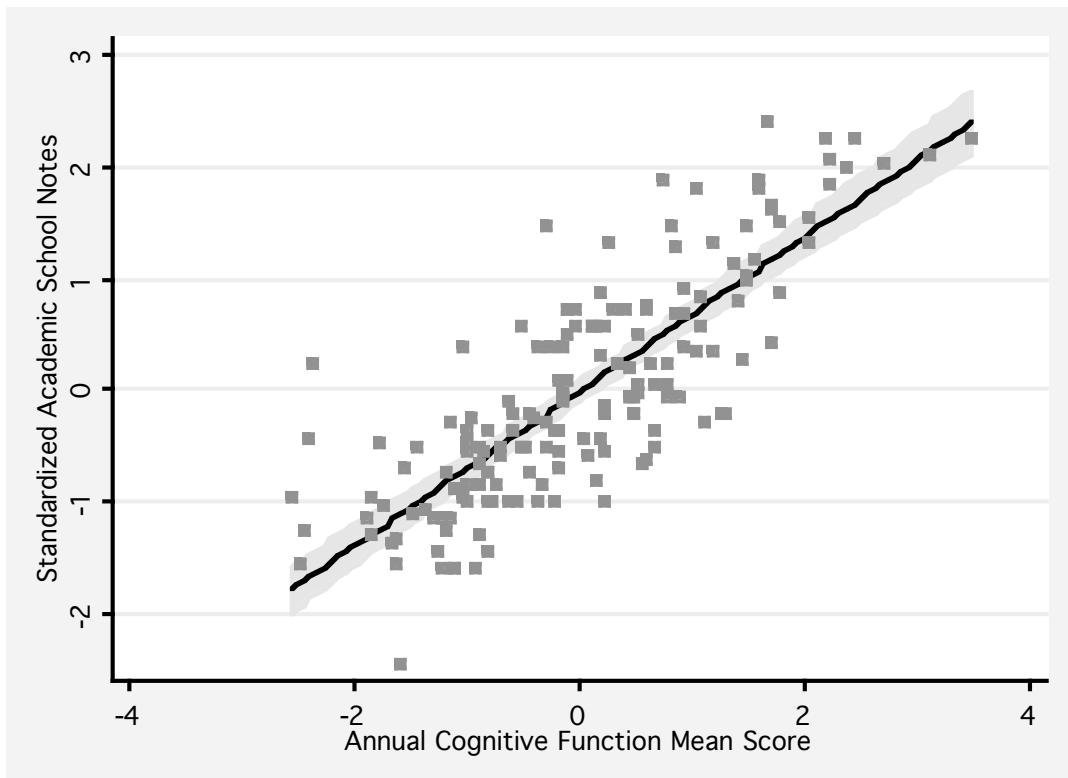
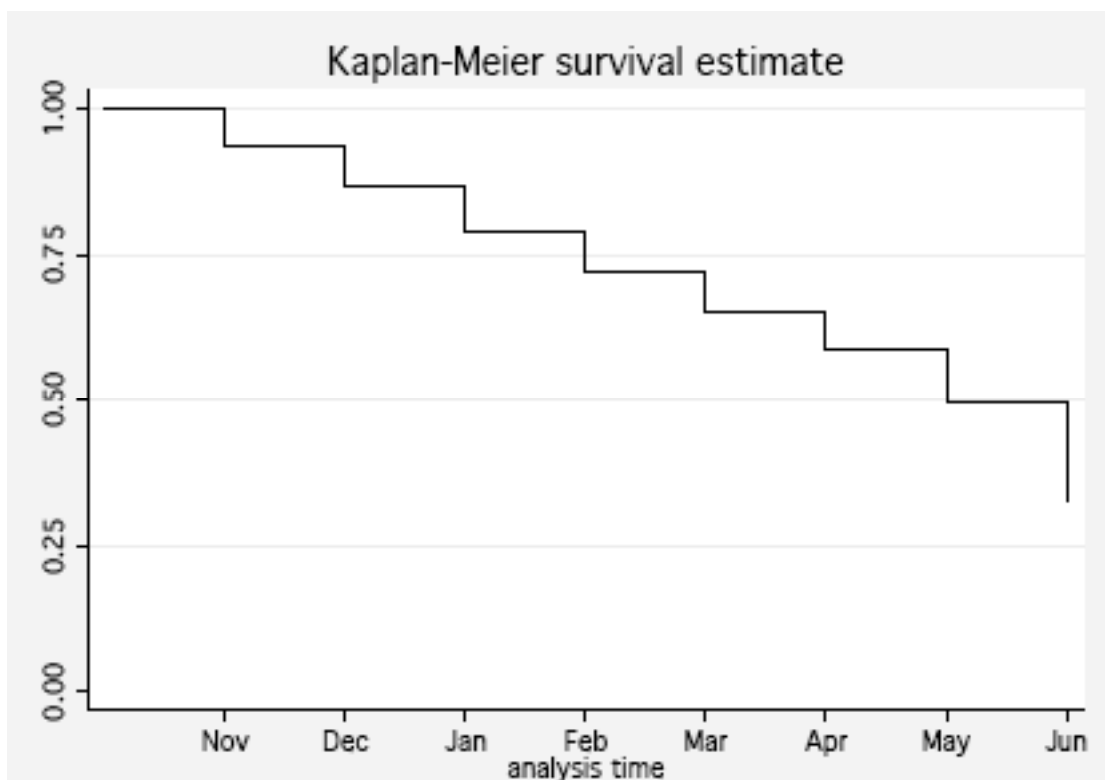


FIGURE IV.A.4: Kaplan Meier Survival Estimate of Asymptomatic malaria during Active Follow-up (*source: author*)



APPENDIX IV.5: QUESTIONNAIRES

FICHE D'ENQUETE
IMPACT DU PALUDISME SUR LA SCOLARITE
 Année scolaire 2007 / 2008

DATE: _ _ _ _ _ _	PASSAGE TRANSVERSAL : _ _ _
NOM ET PRENOM DE L'ELEVE : _____	NUMERO D'ETUDE : _ _ _
_____	ID : _ _ _ _ _ _ _ _

EXAMEN CLINIQUE

1. Température axillaire _____. ____°C
2. Hépatomégalie Oui ___/ Non ___/
3. Splénomégalie Oui ___/ Non ___/
4. Autres signes ou symptômes : _____
5. Diagnostic _____

POIDS :**TAILLE :****EXAMEN BIOLOGIQUE**

1. Taux d'hémoglobine _____. ____ g/dl
 2. Goutte épaisse Positive ___/ Négative ___/
- | | |
|--------------------|---|
| Pf ___/ | Parasitémie _____/mm ³ de sang |
| Pm ___/ | Parasitémie _____/mm ³ de sang |
| Po ___/ | Parasitémie _____/mm ³ de sang |
| Gamétocyte Pf ___/ | Parasitémie _____/mm ³ de sang |

TRAITEMENT : **OUI** **NON**

Si OUI, préciser : _____

- QUESTIONNAIRE DONEGUEBOUGOU -
IMPACT DU PALUDISME SUR LA SCOLARITE
Année scolaire 2007 / 2008

VISITE :

DATE VISITE 1 : |_|_| |_|_| |_|_|

HEURE VISITE 1 : |_|_|

DATE VISITE 2 : |_|_| |_|_| |_|_|

HEURE VISITE 2 : |_|_|

DATE VISITE 3 : |_|_| |_|_| |_|_|

HEURE VISITE 3 : |_|_|

INTERVIEW :

NOM DES ENQUETEURS : _____

LANGUE DE L'INTERVIEW : _____

REPONDANT (sera interviewé le chef du Menage exclusivement) :

NOM et PRENOM DU REPONDANT : _____

ID : |_|_| |_|_| |_|_| |_|_|

AGE DU REPONDANT : |_|_|

NIVEAU D'EDUCATION :

AUCUN	<input type="checkbox"/>	SECONDAIRE	<input type="checkbox"/>	AUTRE (préciser)	<input type="checkbox"/>
1 ^{ère} - 6 ^{ème} ANNEE	<input type="checkbox"/>	SUPERIEUR	<input type="checkbox"/>	_____	
7 ^{ème} - 9 ^{ème} ANNEE	<input type="checkbox"/>	NE SAIT PAS	<input type="checkbox"/>	_____	

SAIT LIRE : OUI ___/ NON ___/

SAIT ECRIRE : OUI ___/ NON ___/

NOMBRE DE FEMMES: |_|_|

NOMBRE D'ENFANTS A CHARGE: |_|_|

***NB: Toutes les réponses aux questions qui vous seront posées
resteront strictement confidentielles.***

		Antécédents médicaux				
		Q1	Q2	Q3	Q4	Q5
NOM et PRENOM de l'enfant scolarisé à Donéguébougou appartenant au ménage	N° Etude	De la naissance à maintenant l'enfant a-t-il été hospitalisé ?	De la naissance à maintenant l'enfant a-t-il convulsé ?	De la naissance à maintenant l'enfant a-t-il souffert de malnutrition ?	L'enfant a-t-il souffert de maladie dans les 30 derniers jours (si oui préciser)?	L'enfant a-t-il une maladie connue (si oui préciser)?
1						
2						
3						
4						
5						
6						
7						
8						
9						
10						
11						
12						

Codification: OUI = 1 NON = 0

		Q6-nformations sur la mère de l'enfant							
		Q6-A	Q6-B	Q6-C	Q6-D	Q6-E	Q6-F	Q6-G	Q6-H
NOM et PRENOM de l'enfant scolarisé à Donéguébougou appartenant au ménage	N° Etude	La mère est-elle toujours en vie ?	Age de la mère	Habite-t-elle à Donéguébougou?	Activité/Travail	Est-elle allée à l'école?	Si OUI quel niveau?	Sait lire?	Sait écrire?
1									
2									
3									
4									
5									
6									
7									
8									
9									
10									
11									
12									

Codification: OUI = 1 NON = 0

		Informations sur le travail de l'enfant et ses devoirs							
		Q8-A	Q8-B	Q8-C	Q8-D	Q8-E	Q8-F	Q8-G	Q8-H
NOM et PRENOM de l'enfant scolarisé à Donéguébougou appartenant au ménage	N° Etude	Au cours de la semaine dernière, l'enfant a-t-il travaillé ?	Si Oui, est ce que ce travail était payé ?	Combien d'heures environs a-t-il travaillé ?	Au cours de la semaine dernière, l'enfant a-t-il eu des devoirs à la maison pour l'école?	Si Oui combien de temps a-t-il consacré par jour ? (Heures)	Y-a-t-il une personne qui l'aide à la maison ?	Si Oui préciser qui (grand frère,...)	Y-a-t-il une autre personne extérieur au ménage qui l'aide pour les études ?
1									
2									
3									
4									
5									
6									
7									
8									
9									
10									
11									
12									

Codification: OUI = 1 NON = 0

	Questions	REPONSES	Codes
Q9	Type de Maison - ENREGISTRER L'OBSERVATION	Maison traditionnelle rectangulaire en terrasse Maison rectangulaire en tôles Maison ronde en paille Autre (préciser) _____	1 2 3 999
Q10	Principal matériau du sol - ENREGISTRER L'OBSERVATION	MATERIAU NATUREL Terre / Banco Bois / Planches / Palme PLANCHER FINI Carreaux Ciment AUTRE (préciser) _____	11 12 21 22 999
Q11	Principal matériau du toit- ENREGISTRER L'OBSERVATION	Paille Bois Banco Tôle Autre (préciser) _____	1 2 3 4 999
Q12	Principal matériau des Murs- ENREGISTRER L'OBSERVATION	Paille Bois Banco Tôle Autre (préciser) _____	1 2 3 4 999
Q13	Nombre de Bâtiments dans le domicile?		
Q14	Nombre de pièces dans le domicile ?		
Q15	Disposez vous d'un autre domicile? (en construction ou déjà construit)	OUI NON	1 2
Q17	Au total, combien de personnes ont dormi dans votre foyer la nuit dernière?		
Q18	Au total, combien d'enfants ont dormi dans votre foyer la nuit dernière?		
Q19	En dehors des enfants scolarisés à Donéguébougou, combien avez-vous d'enfants scolarisés au total? (Préciser le nombre et le niveau atteint pour chaque enfant entre parenthèse) ex: 3 enfants (7ème, 9ème)		
Q20	Dans votre ménage avez vous des moustiquaires?	Non Oui	0 1
Q21	Dans votre ménage avez vous des moustiquaires imprégnées ?	Non Oui	0 1
Q22	Au total, combien d'enfants ont dormi sous la moustiquaire la nuit dernière dans votre foyer?		
Q23	Utilisez vous des insecticides?	Non Oui	0 1

	Questions	REPONSES	Codes
Q24	Dans votre ménage, quel genre de combustible utilisez-vous principalement pour faire la cuisine?	Electricité Gaz Petrole Charbon Bois Paille Autre (préciser) _____	1 2 3 4 5 6 999
Q25	D'où provient principalement l'eau que boivent les membres de votre ménage?	CHATEAU D'EAU MRTC <u>Puit couvert/Forage autre</u> Dans le logement Dans la cour Puit du Village couvert <u>Puit Ouvert/ Non protégé</u> Dans le logement Dans la cour Puit du Village ouvert Eau de Surface Source Fleuve/Rivière Marigo/Lac Canal Eau de Pluie <u>Autre (préciser)</u> _____	11 21 22 23 31 32 33 41 42 43 44 51 999
Q26	Combien de temps faut-il pour aller là-bas, prendre de l'eau et revenir? (en min)		
Q27	Quel genre de toilettes la plupart des membres de votre ménage utilisent?	WC Moderne Fosse/ Latrine En plein air/ rudimentaire Fermée/ Améliorée Pas de toilettes / Nature Autre (préciser) _____	11 21 22 31 999
Q28	Partagez-vous cette installation avec d'autres ménages?	Non Oui	0 1
Q29	Dans votre ménage, avez-vous :	L'électricité / Groupe ? Une radio? Une télévision? Un téléphone? Un réfrigérateur?	1 2 3 4 5
Q30	Dans votre ménage, y-a-t-il quelqu'un qui possède ?	Une montre ? Une bicyclette ? Une Mobiclette/Moto ? Une Voiture/Camionette ? Une Charette ? Une Charue ? Un Cheval ? Un Ane ? Une pompe électrique?	1 2 3 4 5 6 7 8 9

	Questions	REPONSES	Codes
Q31	Combien avez-vous gagné en moyenne (en FCFA) le mois dernier ?		
Q32	D'où proviennent ces revenus?	Salaire mensuel régulier Agriculture Vente ou revente de produits 21 Travaux agricoles pour autrui 22 Elevage Vente de bétail 31 Gardiennage de bétail 32 Autre Commerce A donéguebougou 41 A Kati 42 Autre _____ 999	11 21 22 31 32 41 42 999
Q33	Combien de personnes travaillent (au sens de gagner un revenu) dans votre foyer?		
Q34	En cas d'urgence, disposez vous d'économie ou d'argent de coté ?	OUI NON	1 2
Q35	Si NON, y-a-t-il un autre moyen de revenu en cas d'urgence?	Emprunt à un ami Emprunt à la famille Vente de biens personnels Tontine Crédit à un établissement Autre (préciser) _____	1 2 3 4 999
Q36	Disposez vous de terrains qui vous appartiennent pour cultiver?	Non Oui	0 1
Q37	Si OUI, combien d'hectares environ?		
Q38	Si OUI, sont-ils en culture en ce moment?	Non Oui	0 1
Q39	Disposez vous d'une pompe électrique pour arroser?	Non Oui	0 1
Q40	Quels types de cultures pratiquez vous ce mois-ci ?	— — — —	
Q41	Ces cultures sont-elles destinées à ?	La vente L'autoconsommation Les deux Autre (préciser) _____	1 2 3 999
Q42	Disposez vous de bétail ce mois-ci?	Non Oui	0 1
Q43	Si oui, quel type de bétail et combien de tête par type de bétail?	— — — —	
Q44	Ce bétail est-il destiné à ?	La vente L'autoconsommation Les deux Autre (préciser) _____	1 2 3 999

APPENDIX IV.6: INDIVIDUAL AND COMMUNITY CONSENTS

UNIVERSITE DU MALI
 FACULTE DE MEDECINE DE PHARMACIE
 ET D'ODONTO-STOMATOLOGIE
 DEPARTEMENT D'EPIDEMIOLOGIE
 DES AFFECTIONS PARASITAIRES (DEAP)
 MALARIA RESEARCH AND TRAINING CENTER (MRTC)
 BP 1805 Tél 222 52 77 BAMAKO

REPUBLIQUE DU MALI
 Un Peuple-Un but-Une Foi

**REF. PROJET: ETUDE DE L'IMPACT DU PALUDISME SUR LA SCOLARITE AU
 MALI – Donéguébougou – Année Scolaire 2007/2008**

Titre complet de l'étude : « L'impact du paludisme sur la scolarité au Mali : une étude Cas / Témoins en suivi de cohorte fermée dans l'école de Donéguébougou sur l'année scolaire 2007/2008. »

Consentement Communautaire

Procès verbal de l'approche communautaire :

L'approche communautaire a eu lieu le Samedi 20/10/2007. Etaient présents :

- Le Chef du Village et les deux conseillers du Chef du village
- Le guide
- Le directeur de l'école de Donéguébougou
- Le responsable du centre de santé de Donéguébougou
- L'investigateur principal de l'étude.

Nous souhaitons avant tout remercier le village pour sa bonne participation aux études antérieures.

Le projet et ses objectifs ont été présentés aux dignitaires du village après les salutations protocolaires. L'investigateur principal de l'étude a également été présenté et introduit dans le village. Le paludisme affecte principalement les enfants et peut avoir des effets sur leur développement futur. L'étude que nous souhaitons réaliser consiste à regarder l'impact du paludisme chez les élèves de l'école afin de mieux comprendre ses effets et de mieux les prévenir (via l'école).

Le Chef du Village et ses conseillers ont constaté que les élèves de Donéguébougou souffraient du paludisme et que cela affectait leur scolarité. Ils ont donc accueilli l'étude avec intérêt puisque l'étude servira au village et à la lutte contre le paludisme à Donéguébougou. L'accord a été donné pour le début de l'étude et la nouvelle va être transmise dans les foyers à partir de ce jour afin de faciliter le travail.

Durant la période de suivi tout élève infecté avec un signe sera traité gratuitement pour le paludisme. Du matériel scolaire sera fourni à l'école. Nous rendrons visite aux parents d'élèves dans chaque concession pour réaliser de petits entretiens afin d'évaluer le niveau de vie des habitants.

Président NPE, Donéguébougou

27/10/2007
Date

M Pankoro Nare
Emprunte digitale des Dignitaires

THUILLIER Josselin
Signature de l'Investigateur



UNIVERSITE DU MALI

FACULTE DE MEDECINE DE PHARMACIE
ET D'ODONTO-STOMATOLOGIE
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Je soussigné, Monsieur Bakary Dembélé, Directeur de l'Ecole de DONEGUEBOUGOU, déclare avoir pris connaissance de l'ensemble des conditions de déroulement de l'étude qui se réalisera dans mon école pendant toute l'année scolaire 2007/2008.
Je donne mon autorisation pour le déroulement de cette étude.

21/10/2007, DONÉGUÉBOUGOU

Date

Nom et Signature de Monsieur le Directeur de l'école de Donéguébougou



THUILLIER Josselin

Nom et Signature de l'investigateur

NB : Si vous avez des questions ou des inquiétudes pendant la durée de cette étude, vous pourrez en parler avec un des membres de notre équipe ou demander à votre chef de quartier d'envoyer une lettre au Dr. Mahamadou Soumana Sissoko à la Faculté de Médecine de Pharmacie et d'Odonto-Stomatologie du Point-G à Bamako (BP. 1805 ; Tel/Fax: (223) 222 81 09) ou au Secrétariat permanent du comité d'éthique à la FMPOS (Tél : (223) 222 52 77 Poste 113). Les médecins du centre de Santé vous aideront aussi à contacter les personnes ressources.

NB: The 227 original individual consents have been kept at the Malaria Research and Training Center. The individual consents include a complete description of the study and the rights for each participant. A blank copy of this consent is provided below. The individual consents have been traduced in Bambara for all parents during a one-hour session.

UNIVERSITE DU MALI

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**REPUBLIQUE DU MALI
Un Peuple-Un but-Une Foi**

**REF. PROJET: ETUDE DE L'IMPACT DU PALUDISME SUR LA SCOLARITE AU
MALI – Donéguébougou – Année Scolaire 2007/2008**

**Titre complet de l'étude : « L'impact du paludisme sur la scolarité au Mali : une étude Cas /
Témoins en suivi de cohorte fermée dans l'école de Donéguébougou sur l'année scolaire
2007/2008. »**

Consentement éclairé pour parent ou tuteur

Nom de l'élève..... Numéro de l'élève..... Nom du Parent/Tuteur

1. But

Nous invitons votre enfant à prendre part à une étude portant sur l'impact du paludisme sur la scolarité des enfants. Le paludisme est une maladie causée par un très petit parasite qui peut entrer dans le corps quand un moustique infecté vous pique. La maladie peut entraîner des maux de tête, de la fièvre, des vomissements et beaucoup d'autres malaises. Ces malaises et les conséquences associées au Paludisme peuvent avoir des effets sur le développement de votre enfant et sur sa scolarité. Toutefois, cette maladie peut être traitée et complètement guérie, si elle est diagnostiquée précocement. Le but de ce travail est d'évaluer l'impact de cette maladie sur la scolarité de votre enfant. Notre étude tente de montrer qu'il existe un impact important du paludisme sur la scolarité de votre enfant et sur son absentéisme scolaire durant l'année scolaire 2007/2008. Un total de 230 élèves de l'école de Donéguébougou sera inclus dans cet essai et tous les malades au cours de l'étude seront traités au centre de santé de la localité.

2. Procédure

La participation de votre enfant consistera à lui faire subir un questionnaire, un examen physique (pour savoir s'il n'a pas une autre maladie), la prise de la température et des analyses de sang chaque fin de mois pendant toute l'année scolaire. Chaque participant

subira un prélèvement de l'équivalent de quelques gouttes de sang. Nous procéderons au nettoyage du point de prélèvement au niveau du bras avec de l'alcool et le prélèvement se fera toujours avec des instruments stérilisés et à usage unique. Une analyse de ce sang sera faite pour déterminer la composition du sang et le nombre de parasites dans le sang. L'étude n'aura pas d'influence sur le déroulement de l'année scolaire. En dehors des évaluations normales des enfants par les professeurs, nous donnerons tous les mois un test aux enfants qu'ils devront effectuer. Ce test aura été conçu par les professeurs avec l'équipe de recherche. Ces tests n'auront pas d'influence sur le passage des élèves dans la classe supérieure et n'auront aucune conséquence sur les résultats scolaires de l'élève.

Ces analyses de sang et les tests réalisés par votre enfant permettront de savoir quel est l'impact du paludisme sur ses résultats à l'école et sur l'absentéisme scolaire.

Vous devrez vous aussi répondre à un questionnaire assez court sur l'enfant et sur vous-même. Vous devrez aussi prévenir l'enseignant si l'enfant tombe malade.

3. Participation

La participation de votre enfant est volontaire et vous pouvez mettre fin à cette participation à n'importe quel moment sans qu'il ne perde les avantages liés à celle-ci. Votre enfant sera également traité gratuitement s'il attrape le paludisme et suivi également par l'équipe médicale pour les autres maladies tout au long de l'étude. Une équipe sera installée de façon permanente au centre de santé pendant toute la durée de l'étude.

Si après traitement de votre enfant il est toujours malade, il recevra d'autres soins jusqu'à la guérison. Chaque fois que votre enfant tombera malade, vous pourrez le conduire au centre de santé où un diagnostic sera établi. Vous devrez également prévenir l'enseignant de l'élève.

4. Risques et Bénéfices

Les risques encourus sont minimes. Le prélèvement de sang pourrait causer des désagréments et des malaises au point de prélèvement. Ces tests sont non invasifs et nous n'en connaissons aucun événement adverse. La prise de sang peut causer des malaises et des douleurs occasionnelles au point de prélèvement, et rarement, l'évanouissement. Nous nettoierons le point de prélèvement au niveau du bras de votre enfant avant tout prélèvement de sang et utiliserons de nouvelles aiguilles pour prélever le sang. Les médicaments utilisés pour traiter le paludisme sont ceux déjà utilisés par le Programme de Lutte Antipaludique dans notre pays.

La participation de votre enfant sera bénéfique pour la communauté en aidant les médecins à mieux appréhender les impacts du paludisme sur la communauté et sur l'enfant scolarisé. Elle sera bénéfique pour le Mali en mettant en évidence un problème potentiellement important pour le développement du pays. Un rapport sur cette étude sera déposé aux Ministères de l'Education Nationale et de la Santé du Mali. Tous les tests qu'auront subis les enfants, de même que les médicaments qui leur seront donnés seront gratuits pour le traitement du paludisme. Tous les problèmes potentiels relatifs au traitement seront pris en charge gratuitement par les médecins de notre équipe.

5. Confidentialité

Nous prenons l'engagement que les résultats de ces tests seront confidentiels. Une copie de ce formulaire de consentement contresigné sera placée dans votre dossier au niveau du laboratoire. Les résultats pourraient être publiés dans des revues médicales ou présentés dans des conférences mais votre nom (ou celui de votre enfant) ne sera en aucun cas utilisé dans les rapports. Les informations spécifiques recueillies sur votre enfant ne seront en aucun cas partagées avec d'autres personnes exceptés les investigateurs de l'étude. De même les résultats scolaires des enfants et les résultats aux tests resteront confidentiels et aucun nom ne sera divulgué.

6. Droit au retrait

La participation à cette étude est entièrement volontaire. Vous pouvez refuser que votre enfant y participe ou le retirer de l'étude à tout moment sans aucune sanction ni la perte du droit aux soins requis en cas de diagnostic de paludisme. L'investigateur en charge de ce travail de recherche a aussi le droit de mettre fin à cette participation à tout moment si une raison médicale l'exigeait et dont la nature vous sera précisée, ou pour tout autre motif incompatible avec le bon déroulement de l'étude. Vous serez informés des raisons qui motiveront le retrait de votre enfant par le médecin-chercheur.

7. Coût et Compensation

Tous les soins médicaux incluant les contrôles de santé, les analyses de sang, la prise de la température, l'examen clinique seront faits gratuitement. Que vous décidiez de participer à cette étude ou non, nous procéderons au traitement du paludisme que nous diagnostiquerons chaque fin de mois et lors du suivi continu de l'enfant. Des soins adéquats seront administrés à chaque volontaire indépendamment de son statut à l'étude et selon les bonnes pratiques cliniques. Les compensations en nature ou en espèces ne sont pas prévues pour la participation de votre enfant à cette étude en dehors du traitement reçu. Cependant l'école de

Donéguebougou recevra des fournitures scolaires qui seront distribuées aux enfants en classe.

8. Consentement éclairé

Je confirme avoir reçu toutes les informations concernant la participation de mon enfant à cette étude par Mr J'ai compris ce qu'on demande et ce que subira mon enfant en participant à l'étude. J'ai également compris que je pourrai décider de retirer mon enfant au cas où je le jugerai opportun sans risque de perdre les avantages liés à cette étude.

Si vous avez des questions ou des inquiétudes pour la participation de votre enfant à cette étude, vous pourrez en parler avec un des membres de notre équipe ou demander à votre chef de quartier d'envoyer une lettre au Dr. Mahamadou Soumana Sissoko à la Faculté de Médecine de Pharmacie et d'Odonto-Stomatologie du Point-G à Bamako (BP. 1805 ; Tel/Fax: (223) 222 81 09) ou au Secrétariat permanent du comité d'éthique à la FMPOS (Tél : (223) 222 52 77 Poste 113). Les médecins du centre de Santé vous aideront aussi à contacter les personnes ressources.

Empreinte digitale

ou

Signature du Tuteur

date

Signature de l'investigateur

date

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Summary. This thesis contributes to the literature on the endogenous relationship between the health status of a population and the economic development of a nation. We focus on a wide phenomenon touching many low-income countries: malaria. Firstly, we propose to rethink the economic analysis of malaria by combining economic epidemiology tools with the poverty trap literature. The endogeneity of malaria with respect to households and individuals socioeconomic characteristics and choices remains a particularly relevant and unresolved issue. In spite of massive efforts to generalize efficient prevention (such as ITNs), malaria remains prevalent in many countries. We design a theoretical model of rational preventive behaviors in response to the disease, which includes endogenous externalities and disease characteristics. Two important conclusions emerge from the model. First, agents increase their preventive behaviors when the degree of prevalence of malaria in a society is more severe. This result is consistent with the literature on "prevalence-elastic behavior". Second, we find that multiple equilibria are theoretically possible and so is the existence of malaria traps, defined as any self-reinforcing mechanism, which causes malaria to persist. Two implications discussed in this chapter concern malaria exogeneity for trapped communities and possible mechanisms to get out of the trap (through public policies). For the communities trapped and if the disease affects negatively some economic variables, it is plausible to assume that the disease itself generates the socio-economic obstacles to its control. Secondly, we try to understand the socioeconomic effects of malaria. If malaria deaths and illness occur mostly in young African children, the economic literature on the subject primarily focused on its effects on adult productivity or on private health and public expenditure accumulation related to the disease. The last three chapters of this thesis tackle the question of the economic effects of malaria under a new angle: the effects of the disease on children human capital accumulation, which can potentially have heavy consequences on future generations. A small but growing number of medical studies are drawing an alarming report of the impact of malaria on creative and intellectual development of children. Given the frightening distribution of *falciparum* malaria, any effect of malaria on cognitive function or educational achievement is likely to result in a massive macroeconomic loss. Our macroeconomic results show that performance of educational systems in countries with intensive malaria is lower than in countries not affected by malaria, other things being equal. These results suggest that the macroeconomic effects of malaria on education are not negligible, taking into account the number of malaria clinical cases worldwide. These results are then confronted to two microeconomic analyses. We start with data from Demographic and Health Surveys. We next move to a longitudinal investigation we carried out in a village in a malaria endemic area in Mali in collaboration with the *Malaria Research and Center training*. From a methodological point of view, thanks to a multi-disciplinary approach, we improve the measure of malaria indicators generally used in economic studies. The main finding of this thesis relates to the effects of asymptomatic malaria. Asymptomatic malaria is defined as a malaria-positive smear for *P. falciparum* parasitaemia associated with no clinical symptoms. Asymptomatic malaria affects significantly school performances of the children in endemic area. We also show that higher income offers little protection against the negative effects of asymptomatic malaria on education in the village. Our results can lead to change the social and psychological perception of the risks associated with malaria, thus opening promising prospects as regards to malaria eradication and control programs.

Keywords: Malaria, Multiple Equilibria, Human Capital, Education, Mali.

Résumé. Cette thèse contribue à la littérature sur la relation endogène entre l'état de santé d'une population et le développement économique d'une nation. Nous nous focalisons pour cela sur un phénomène de grande ampleur touchant de nombreux pays en développement: l'endémie palustre. Dans un premier temps, nous reformulons le cadre théorique dans lequel la relation bidirectionnelle entre paludisme et développement économique est généralement analysée. Nous utilisons un modèle d'épidémiologie économique qui place la maladie et les comportements préventifs au centre de l'analyse et débouche sur des comportements dits «prévalence-élastiques». Nous montrons que ce modèle conduit à de possibles équilibres multiples et, par conséquent, à des trappes de santé potentielles du fait des externalités engendrées par les décisions des agents. Une implication forte de ces trappes concerne le caractère exogène de la maladie pour les communautés piégées. Une autre implication importante concerne les politiques de contrôle de la maladie. Si les communautés sont piégées dans de telles trappes et que la maladie affecte négativement certaines variables économiques, il est possible que la maladie engendre, de par sa nature même, des obstacles socioéconomiques à son contrôle. Dans un deuxième temps, nous cherchons donc à mieux comprendre les effets socioéconomiques du paludisme. Si le paludisme touche principalement les enfants, les études économiques sur le sujet se sont essentiellement focalisées sur ses effets sur la productivité chez l'adulte ou l'accumulation des dépenses de santé privées et publiques liées à la maladie. Les trois derniers chapitres de cette thèse abordent la question des effets économiques du paludisme sous un angle nouveau: celui des conséquences de la maladie sur l'accumulation de capital humain chez l'enfant, pouvant avoir des conséquences lourdes sur les générations futures. Certaines études médicales font un constat alarmant de l'impact du paludisme sur les capacités de développement créatif et intellectuel chez l'enfant. Nos résultats montrent que les pays intensivement touchés par le paludisme ont des systèmes éducatifs moins performants par rapport aux pays non touchés. Ces résultats suggèrent des effets non négligeables du paludisme au niveau macroéconomique, compte tenu de l'ampleur de l'endémie palustre. Ils sont ensuite confirmés par deux analyses microéconomiques à partir de données DHS et d'une enquête longitudinale que nous avons réalisée dans un village en zone endémique au Mali en collaboration avec le *Malaria Research and Training Center*. D'un point de vue méthodologique, nous améliorons la mesure des indicateurs de paludisme généralement utilisés dans les études économiques. L'une des principales découvertes de cette thèse concerne les effets du paludisme asymptomatique. Le paludisme asymptomatique à *P. falciparum* affecte significativement les performances scolaires des enfants en zone endémique. Nous montrons également qu'un niveau de richesse plus élevé ne protège pas des effets négatifs de la parasitémie sur l'éducation. Nos résultats peuvent déboucher sur une modification de la perception sociale et psychologique des risques associés à la maladie, ouvrant ainsi des perspectives prometteuses en matière de politique de lutte.

Discipline : Sciences Economiques (05).

Mots-clés : Paludisme, Equilibres Multiples, Capital Humain, Education, Mali.

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