

**Animal and human pathogenic *Escherichia coli* strains share common genetic backgrounds**

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1 **Abstract**

2

3 *Escherichia coli* is a versatile species encompassing both commensals of the digestive  
4 tracts of many vertebrates, including humans, and pathogenic strains causing various intra-  
5 and extraintestinal infections. Despite extensive gene flow between strains, the *E. coli* species  
6 has a globally clonal population structure, consisting of distinct phylogenetic groups. Little is  
7 known about the relationships between phylogenetic groups and host specificity. We therefore  
8 used multi-locus sequence typing (MLST) to investigate phylogenetic relationships and  
9 evaluated the virulence gene content of 35 *E. coli* strains representative of the diverse diseases  
10 encountered in domestic animals and compared these strains with a panel of 100 human  
11 pathogenic and 99 non human and human commensal strains representative of the  
12 phylogenetic and pathovar diversity of this species. A global factorial analysis of  
13 correspondence indicated that extraintestinal infections were caused mostly by phylogenetic  
14 group B2 strains, whereas intractintestinal infections were caused mostly by phylogenetic group  
15 A/B1/E strains, with each group having specific virulence factors. It was not possible to  
16 distinguish between strains of human and animal origin. A detailed analysis showed that some  
17 pathogenic human and animal strains belonged to the same clonal complexes, with a number  
18 of virulence genes in common. However, a set of specific adhesins was identified in animal  
19 non-B2 group strains whatever the *E. coli* pathotype. In conclusion, human and animal  
20 pathogenic strains share common genetic backgrounds, but in non-B2 phylogenetic group  
21 strains, seem to have a different set of adhesins according to their origin.

## 1 **Introduction**

2

3 *Escherichia coli* is one of the most versatile bacterial species. It alternates between its  
4 primary habitat, the gut of vertebrates, where it lives as a commensal (50), and its secondary  
5 habitat, water and sediment (49). It may also function as an intra- and extraintestinal pathogen  
6 in humans and many other animal species (30). This diversity of lifestyles is achieved through  
7 a high degree of genome plasticity, with gains, through horizontal transfer, and losses of  
8 genes (45, 51). This species has a core genome of less than 2,000 genes, but the total number  
9 of genes in the species exceeds 10,000 (45, 51). Thus, the diverse phenotypes observed result  
10 principally from a large number of different gene combinations. Despite the high degree of  
11 gene flow, the structure of this species remains mostly clonal (51), with the clear delineation  
12 of at least six principal phylogenetic groups (A, B1, B2, D, E and F) (25, 50). It has been  
13 shown that genetic background plays a role in the acquisition, retention and expression of  
14 foreign DNA (14).

15 It has been suggested that some group B1 strains with the *hly* gene may be specific for  
16 animals (15) and that some group B2 strains of the O81 serogroup may be specific for  
17 humans (11). However, little is known about the relationships between phylogenetic groups  
18 and host specificity. In this context, the extent to which bacterial strains from infected humans  
19 and animals are phylogenetically related is unclear. Several studies based on serotyping,  
20 multilocus enzyme electrophoresis, outer membrane protein profiles, pulsed-field gel  
21 electrophoresis, ribotyping, randomly amplified polymorphic DNA, phylogenetic group  
22 affiliation and virulence gene content have shown close relationships between human and  
23 animal isolates (1, 6, 7, 16, 19, 20, 27, 29, 34, 35, 39, 44, 53). More recently, multilocus  
24 sequence typing (MLST) has been used to study the phylogenetic relationships between  
25 strains in more detail. These studies have focused principally on extraintestinal pathogenic *E.*

1 *coli* (ExPEC) [including avian pathogenic *E. coli* (APEC) in particular] (37-39) and  
2 enterohemorrhagic *E. coli* (EHEC) (17, 40) strains. A large body of data has accumulated, but  
3 these data are scattered and difficult to compare, as these studies used different typing  
4 approaches and non-redundant sets of strains. Consequently, no overview of the global  
5 relationships between animal and human pathogenic strains in the framework of the  
6 phylogeny of the whole *E. coli* species has yet been produced.

7         The aim of this work was to study the phylogenetic relationships, by MLST, and  
8 virulence gene content of 35 *E. coli* strains representative of the diverse diseases encountered  
9 in domestic animals, comparing these strains with a panel of 100 human pathogenic and 99  
10 non human and human commensal strains representative of the phylogenetic diversity of the  
11 species and including well characterized archetypal strains.

12

## 13 **Materials and Methods**

14

15 **Bacterial strains.** We studied 234 *E. coli* strains and one strain of *Escherichia fergusonii*, the  
16 closest relative of *E. coli* (31) (Table S1). Five groups of *E. coli* strains were represented: (i) a  
17 panel of 35 pathogenic animal strains representative of the various diseases encountered in  
18 domestic animal species (from 8 birds and 27 mammals) comprising 15 ExPEC/APEC and 20  
19 intractintestinal pathogenic *E. coli* (InPEC) [8 enterotoxigenic *E. coli* (ETEC), 4  
20 enteropathogenic *E. coli* (EPEC), 5 Shiga toxin-producing *E. coli* (STEC)/EHEC and 3  
21 unclassified InPEC] strains (Table 1), (ii) a panel of 92 pathogenic human strains comprising  
22 43 ExPEC [29, 8 and 6 involved in urinary tract infection (UTI), newborn meningitis (NBM),  
23 septicemia and miscellaneous infections, respectively] and 49 InPEC [7 ETEC, 6 EPEC, 8  
24 EHEC, 10 enteroaggregative *E. coli* (EAEC), 1 enteroinvasive *E. coli* (EIEC), 16 diffusely  
25 adherent *E. coli* (DAEC) and 1 unclassified InPEC], (iii) 45 non human mammalian

1 commensal strains, (iv) 54 human commensal strains and (v) 8 human InPEC strains for  
2 which the complete genome was available (42, 45), these strains being typed *in silico* in the  
3 present work. The strains from groups (ii), (iii) and (iv) in this list originated mostly from  
4 three published collections (14, 32, 41) and may be considered representative of the  
5 phylogenetic and pathovar diversity of *E. coli*. They encompass archetypal strains for various  
6 diseases, and complete genome sequences are available for some.

7

8 **Virulence factor (VF) screening.** We tested for the presence of virulence factors involved in  
9 extraintestinal (*neuC*, *kpsE*, *sfa/foc*, *ironN*, *aer*, *iha*, *papC*, *papGI*, *papGII*, *papGIII*, *hly*, *cnf1*,  
10 *hra*, *sat*, *ire*, *usp*, chromosomal *ompT*, *ibeA*, *malX*, *irp2*, *fyuA* and *traT*) (12, 26) and  
11 intrainestinal [*afaD*, *ipaH*, *stx1*, *stx2*, *eltB* (LT), *estA* (ST), *bfpA*, *eae*, *aaiC* and *aatA*] (Table  
12 S2) infections by PCR, as previously described (14). We also used PCR, as previously  
13 described (2, 4, 13, 18, 24), to check for the presence of adhesins classically associated with  
14 animal-specific pathogenic strains of *E. coli*: K99 (*fanA*), K88 (*faeG*), F17 (*f17A*), F18 (*fedA*)  
15 and Afr2 (*afr2G*).

16

17 **PCR O-typing.** O-type was determined by an allele-specific PCR (10) using the primers  
18 given in Table S3. We assessed 28 O-types with this assay, based on the O types previously  
19 determined by the classical serological method for the other strains of the collection.

20

21 **MLST.** MLST was performed with partial *dinB*, *icdA*, *pabB*, *polB*, *putP*, *trpA*, *trpB*, and *uidA*  
22 sequences (25). Allele sequences and sequence types (STs) are available from Institut  
23 Pasteur's MLST website, at [www.pasteur.fr/mlst](http://www.pasteur.fr/mlst). Phylogenetic analysis was performed with  
24 the concatenated sequences of the eight genes, by the maximum likelihood method, as  
25 implemented in the PHYML program (23), with *E. fergusonii* as the outgroup.

1

2 **Factorial analysis of correspondence (FAC).** FAC was used to describe associations  
3 between the different data sets. FAC uses a covariance matrix based on Chi squared distances  
4 (22). This computation method determines a plane defined by two principal axes of the  
5 analysis. The first axis (F1) accounts for most of the variance, and the second axis (F2),  
6 orthogonal to F1, accounts for the largest part of the variance not accounted for by F1. FAC  
7 was conducted with SPAD.N 4.5 software (Cisia, Saint Mandé, France), based on a two-way  
8 table. This table had 234 rows, one for each *E. coli* strain, and 35 columns, corresponding to  
9 35 variables: human/animal origin, commensal, ExPEC and InPEC characters, seven  
10 phylogenetic groups corresponding to A, B1, B2, C, D, E, F and ungrouped (UG) strains  
11 according to the MLST data (14, 25), and 23 VFs (*neuC*, *sfa/foc*, *iroN*, *papC*, *papGI*, *papGII*,  
12 *papGIII*, *hlyC*, *cnf1*, *hra*, *fyuA*, “animal adhesins” (AnAd), *afaD*, *ipaH*, *stx1*, *stx2*, *estA*, *eltB*,  
13 *bfpA*, *eae*, *aaiC* and *aatA*). The data in this table were attributed a binary code: "1" for present  
14 and "0" for absent. The loading score for each variable on the plane of the variables (factors  
15 F1 and F2, respectively) can be inferred from the coordinates of the X and Y variables on the  
16 F1/F2 plane. Moreover, the data corresponding to active variables were calculated directly in  
17 the FAC, whereas the illustrative variables were only projected on the plane.

18

## 19 **Results and Discussion**

20

### 21 **Multidimensional analysis**

22

23 We assessed the global relationships between the phylogenetic groups, the VF content  
24 and origin (human versus animal and commensal versus pathogenic) of the strains, by  
25 carrying out a FAC with phylogenetic groups and VFs as active variables and the

1 animal/human, commensal, ExPEC and InPEC variables as illustrative variables. Strains were  
2 assigned to phylogenetic groups on the basis of MLST data. Seven groups were considered:  
3 the six previously recognized groups (A, B1, B2, D, E and F) and the C group plus ungrouped  
4 strains (see below for the definition of the C group and ungrouped strains). On the F1/F2  
5 plane, which accounted for 29.37 % of the total variance, the variables *papGI*, *papGIII*, *cnf1*,  
6 *sfa/sfoc*, *hlyC*, *iroN*, *hra*, *papC*, *neuC*, B2 group and ExPEC were projected onto the positive  
7 values of the F1 axis, whereas the variables *stx2*, E group, *eae*, *stx1*, *bfpA*, *eltB*, *estA*, B1  
8 group, InPEC, AnAd, *ipaH*, *aatA* and A group were projected onto the negative values of this  
9 axis. The variables F group, *papGII*, D group, *aer*, *afaD* and *aaiC* were projected onto the  
10 negative values of the F2 axis. The variables human, animal and commensal were grouped  
11 around the origin of the axes and it was therefore not possible to differentiate between them in  
12 this FAC (Fig. 1). Most of the ExPEC had positive F1 coordinates (Fig. 2A), whereas the  
13 InPEC strains had negative coordinates on this axis (Fig. 2B). Moreover, it was not possible  
14 to distinguish between ExPEC and InPEC strains of human and animal origin in this FAC  
15 (Fig. 2).

16 This global analysis suggests that extraintestinal infections are caused principally by  
17 B2 strains, which have many extraintestinal VFs, whereas intraintestinal infections are caused  
18 mostly by A/B1/E strains with specific intestinal VFs. It was not possible to distinguish  
19 between strains of human and animal origin.

20

### 21 **Fine-scale phylogenetic analysis**

22

23 We investigated the relationships between human and animal pathogenic strains in  
24 more detail, at the clonal level and within the species as a whole, by carrying out MLST  
25 analysis on the 234 strains. Three main MLST schemes are currently available for *E. coli* (25,

1 46, 52), using each a different combination of genes. The data are usually studied in two  
2 ways, with the alleles at different loci providing an allelic profile, which defines the ST. No  
3 weighting is given to take into account the number of nucleotide differences between the  
4 alleles. Alternatively, nucleotide sequences may be used for phylogenetic reconstructions  
5 (50). The results obtained with these three schemes are highly correlated (21), suggesting that  
6 the clonal structure of the species is robust.

7 We chose to use the set of genes described in a previous study (25), as we have  
8 previously used this MLST scheme to characterize a unique set of strains representative of the  
9 phylogenetic and lifestyle diversity of the species. A considerable amount of data is available  
10 for these strains. The phylogenetic tree reconstructed from the concatenated sequences (Fig.  
11 3) showed the major phylogenetic groups previously described (50): A, B1, B2, D, E, F. An  
12 additional group, closely related to the B1 group but identified as A by PCR triplex  
13 phylogrouping (9), was also identified and called C (14, 36). Only six strains (ECOR31,  
14 ECOR42, 126A, DAEC5, DEC9a and 101-1) were not included in these groups and were  
15 classified as ungrouped. The F and B2 groups are the most basal, having emerged first,  
16 whereas the A and B1/C groups diverged more recently. Furthermore, a clear genetic  
17 structure was identified within the B2 phylogenetic group, with at least nine subgroups (I to  
18 IX) in addition to the EPEC1 cluster commonly represented by the O127:H6 strain E2348/69  
19 (32). The A and B1 groups contained three (I to III) and five (I to V) subgroups, respectively  
20 (Fig. 3). However, with the exception of subgroup II of group A, which was delineated by a  
21 high bootstrap value (84%) and encompassed an A<sub>0</sub> genotype with the PCR triplex typing  
22 method (9, 21), these subgroups were not particularly robust phylogenetically. A minimum  
23 spanning tree analysis based on allelic profile data gave similar results for the shallow  
24 phylogenetic grouping (data not shown).



1           With this phylogenetic approach, we clearly identified human and animal strains  
2 belonging to the same phylogenetic subgroups or clonal complexes.

3           Both human and animal pathogenic strains were found in all but three of the  
4 subgroups of the B2 phylogenetic group (I, V and VIII). The most highly represented  
5 subgroup was subgroup IX, which corresponds to ST95 of the Achtman scheme (52), ST29 of  
6 the Whittam scheme (40), and the B2-1 group of a previous study (38). This subgroup  
7 encompasses the APEC strains of serogroup O1, O2 and O18 (28, 37, 38) and the archetypal  
8 human strains UTI89 (5), a strain causing urinary tract infection, and RS218, an isolate from a  
9 neonate with meningitis (54), both of serotype O18:K1:H7. Non-human mammalian ExPEC  
10 strains were also found in subgroup II (O2-type strain, archetypal UTI human strain CFT073  
11 of O6-type), subgroup IV (O2-type strain, archetypal UTI human strain IAI74 of O2-type)  
12 and subgroup VII (O75-type strain, archetypal human UTI strain IH11128 of O75-type).  
13 Furthermore, strains of serotype O6:H31, which have been reported in dog urinary tract  
14 infections (7, 27), belong to subgroup III (archetypal human UTI strain 536 of O6-type) and  
15 O4:H5 dog strains belong to subgroup VI (archetypal human UTI strain J96 of O4-type) (32)  
16 (data not shown). As expected, no pathogenic animal strain belonged to subgroup VIII, as this  
17 clone has been described as strictly commensal in humans (11). Similarly only human EPEC  
18 strains testing positive for *bfpA* by PCR were present in the EPEC 1 cluster.

19           Non B2 ExPEC animal strains of the O78-type belong to phylogenetic groups D (one  
20 APEC strain) and C (2 APEC and 2 bovine ExPEC strains). Group D is diverse, and no close  
21 relationship was identified between group D strains and human strains, although numerous  
22 human ExPEC strains belong to this group (3, 43). Conversely, the group C is clonal. Thus,  
23 the numerous APEC strains of the O78-type assigned to phylogroup A by the triplex PCR  
24 method (16 , 29) probably belong to this clone. One of the group C strains tested, the bovine  
25 ExPEC strain 1404, which carries the Vir plasmid, has been shown to be closely related to

1 avian and human ExPEC strains, as it belongs to esterase electrophoretic type 2 (6). The  
2 group C strains in our collection also include one human ExPEC strain, ECOR72, and animal  
3 and human InPEC strains. A case of neonatal meningitis due to a group C strain has recently  
4 been reported (36). This strain was shown to be virulent in a mouse model of septicemia (43).  
5 One ExPEC strain from pig and another from sheep, these strains belonging to the B1  
6 subgroups III and V, respectively, clustered together with human InPEC strains.

7 Animal InPEC strains were found to belong to phylogenetic groups A, B1, C and E.  
8 Group E, which includes human and animal EHEC O157:H7 strains, is clonal, whereas  
9 groups A and B1 are more diverse, with at least three (I to III) and five (I to V) subgroups,  
10 respectively (Fig. 3). All animal InPEC (ETEC and EHEC) strains of phylogenetic group A  
11 belong to subgroup I, which also contains human InPEC (DAEC and EAEC with the  
12 archetypal EAEC strain JM221) and ExPEC strains (Fig. 3). Four of the five B1 subgroups  
13 include animal InPEC strains. Within the B1 subgroups, bovine O26-type EHEC and EPEC,  
14 together with O45b-type and O15-type EPEC strains, are closely related to human O26-type  
15 and O111-type EHEC strains. The swine ETEC 987 strain belongs to B1 subgroup III, which  
16 encompasses the human EAEC, EPEC and ETEC strains. The rabbit EPEC strain E22 (O103-  
17 type) is very closely related to the human O103-type and O111-type EHEC strains and to the  
18 EPEC 2 strain B171 (45), in B1 subgroup V. Animal ETEC and EHAC strains were found in  
19 group C, together with human InPEC and animal and human ExPEC strains, as stated above  
20 (Table 1).

21

## 22 **Fine-scale VF pattern analysis**

23

24 Animal ExPEC strains have a variable pattern of extraintestinal VFs, whether  
25 considered individually or in the pathogenicity island (PAI) context, even within a clonal

1 lineage (Table 1). Different patterns were also observed between human and animal strains of  
2 the same lineage (for example, within B2 subgroups II, VII and IX; Tables 1 and S1).  
3 Furthermore, no particular pattern of VFs was found to be specific to a particular type of  
4 extraintestinal disease (e.g. UTI, NBM, septicemia or avian colibacillosis). The intrainestinal  
5 VFs studied were pathotype-specific and therefore did not distinguish between human and  
6 animal strains. In a very small number of strains in our collection, we did not find the  
7 expected pathotype-defining VFs (ST, LT, Bfp). However, in these cases, the VFs were  
8 plasmid-encoded and the plasmid was probably lost during the many subcultures of the strains  
9 (Table 1).

10 By contrast, adhesins, which are usually described in animal strains (see above), were  
11 found in 17 animal strains but only four human strains: the F111 adhesin in 1 DAEC strain, 1  
12 EAEC strain and 1 ExPEC strain, and the F17c/GAF adhesin in a ExPEC strain (Tables 1 and  
13 S1). The expression of the F17c/GAF adhesin, which is specific for terminal N-acetyl-D-  
14 glucosamine and blood group M antigen, by human strains (particularly those isolated from  
15 cases of urinary tract infection) is well documented, although the reported incidence is low  
16 (47). By contrast, to our knowledge, the presence of genes encoding F111 adhesins has not  
17 previously been reported in an *E. coli* strain pathogenic in humans. As suggested by the FAC  
18 (Fig. 1), these adhesins were found almost exclusively in non-B2 strains, as only three of the  
19 strains concerned belonged to phylogenetic group B2.

20

## 21 **Concluding remarks**

22

23 Our data for a representative set of animal pathogenic strains analyzed within the  
24 context of the overall phylogeny of the *E. coli* species clearly show that human and animal  
25 pathogenic strains share common genetic backgrounds, with a large overlap between the two

1 populations. Human and animal strains causing the same disease in different host share a  
2 common pool of virulence genes, but a set of specific adhesins was identified in animal non-  
3 B2 strains whatever the *E. coli* pathotype.

4         It has been suggested that, on many occasions, strains derived from a common recent  
5 ancestor have become specialized for a particular host through subtle genetic changes (48).  
6 The recent identification of genes or single nucleotide polymorphisms in O157:H7 EHEC  
7 strains specific for humans or cattle (8, 53) and of specific expression patterns of genes from  
8 B2 phylogenetic group APEC and human urinary tract infection strains in chicken and UTI  
9 mouse models (55) is consistent with this hypothesis. Complete genome sequences for many  
10 animal and human pathogenic strains, generated by “next-generation” sequencing  
11 technologies (33), will facilitate identification of the genetic elements involved in host  
12 specialization.

13

1

2 **Acknowledgments**

3

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1 Table 1. Main characteristics of the 35 pathogenic animal *E. coli* strains studied

Strain ID	Host	Country origin	Pathogenicity	Phylogenetic group and subgroup	O-type	<i>neuC</i>	<i>kpsE</i>	<i>sfa/foc</i>	<i>iroN</i>	<i>aer</i>	<i>papC</i>	<i>papG I</i>	<i>papG II</i>	<i>papG III</i>	<i>hlyC</i>	<i>cnfI</i>	<i>hra</i>	<i>fyuA</i>	"Animal" adhesin	<i>afaD</i>	<i>ipaH</i>	<i>stx1</i>	<i>stx2</i>	<i>e/txB (LT)</i>	<i>estA (ST)</i>	<i>bfpA</i>	<i>eae</i>	<i>aaiC</i>	<i>aataA</i>
25KH9	<i>Bos taurus</i>	Belgium	ETEC	A I	101	-	-	-	+	-	+	-	-	-	-	-	+	-	F17a	-	-	-	-	-	-	-	-	-	-
S1191	<i>Sus scrofa</i>	USA	STEC	A I	139	-	-	-	-	-	-	-	-	-	-	-	-	-	F18	-	-	-	+	-	+	-	-	-	-
510	<i>Bos taurus</i>	Belgium	ETEC	A I	101	-	-	-	-	-	-	-	-	-	-	-	+	-	K99	-	-	-	-	-	+	-	-	-	-
431	<i>Sus scrofa</i>	USA	ETEC	A I	101	-	-	-	-	-	-	-	-	-	-	-	+	-	K99	-	-	-	-	-	+	-	-	-	-
262KH89	<i>Bos taurus</i>	Belgium	Diarrhea	A I	26	-	-	-	-	-	-	-	-	-	+	-	-	-	K88	-	-	-	-	-	-	-	-	+	-
255/1-1	<i>Bos taurus</i>	France	EHEC	A I	Unknown	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-
126A	<i>Bos taurus</i>	Belgium	ETEC	UG	8	-	-	-	-	-	-	-	-	-	-	-	+	-	K99	-	-	-	-	-	+	-	-	-	-
86-1390	<i>Sus scrofa</i>	Canada	EPEC	B1 I	45b	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-
193	<i>Bos taurus</i>	USA	EHEC	B1 I	26	-	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	+	-	-
C/15333	<i>Bos taurus</i>	Northern Ireland	EPEC	B1 I	26	-	-	-	-	-	-	-	-	-	+	-	-	+	-	-	-	-	-	-	-	-	+	-	-
RDEC-1	<i>Oryctolagus cuniculus</i>	USA	EPEC	B1 I	15	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-
5131	<i>Sus scrofa</i>	Canada	ExPEC	B1 III	115	-	-	+	+	+	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
987	<i>Sus scrofa</i>	USA	ETEC	B1 III	9	-	-	-	-	-	-	-	-	-	-	-	-	-	K99	-	-	-	-	-	+	-	-	-	-
31A	<i>Bos taurus</i>	France	Diarrhea	B1 IV	153	-	-	-	-	+	+	-	-	-	-	-	+	+	F17c/GAF	-	-	-	-	-	-	-	-	-	-
211	<i>Bos taurus</i>	Belgium	ETEC	B1 V	Rough	+	+	-	-	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
S5	<i>Ovis aries</i>	UK	ExPEC	B1 V	15	+	+	-	-	-	-	-	-	-	+	-	-	+	F17b	-	-	-	-	-	-	-	-	-	-
E22	<i>Oryctolagus cuniculus</i>	France	EPEC	B1 V	103	-	-	-	-	-	-	-	-	-	-	-	-	-	AFR2	-	-	-	-	-	-	-	-	+	-
111KH86	<i>Bos taurus</i>	Belgium	Diarrhea	B1 V	Unknown	-	-	-	-	-	-	-	-	-	-	-	+	-	F111	-	-	-	-	-	-	-	-	-	-
789	<i>Gallus gallus</i>	Israël	APEC	C	78	-	-	+	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-
BEN 0265	<i>Gallus gallus</i>	France	APEC	C	78	-	-	-	+	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-
1404	<i>Bos taurus</i>	France	ExPEC	C	78	-	-	-	-	-	-	-	-	-	+	-	-	+	F17b	-	-	-	-	-	-	-	-	-	-
239KH89	<i>Bos taurus</i>	Belgium	ExPEC	C	Unknown	-	-	-	-	+	+	-	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-
DEC7a	<i>Sus scrofa</i>	USA	ETEC	C	157	-	-	-	-	-	-	-	-	-	+	-	-	+	K88	-	-	-	-	+	+	-	-	-	-



1

## 2 **Legend of Figures**

3

4 **Fig. 1.** Factorial analysis of correspondence (FAC) of the 234 *E. coli* strains. Projections of  
5 the 35 variables: human/animal origin, commensal, ExPEC and InPEC characters, 7  
6 phylogenetic groups (A, B1, B2, C, D, E and F) and ungrouped strains (UG) and 23 VFs (see  
7 materials and methods) projected onto the F1/F2 plane calculated in the FAC (22). AnAd:  
8 Animal adhesin.

9

10 **Fig. 2.** Factorial analysis of correspondence (FAC) of the 234 *E. coli* strains. Projections of  
11 (A) the ExPEC strains (black symbols) of animal (triangle) and human (square) origin and of  
12 the non ExPEC strains (white symbols) and (B) the InPEC strains of animal (triangle) and  
13 human (square) origin and of the non InPEC strains (white symbols) on the F1/F2 plane  
14 calculated in the FAC (22).

15

16 **Fig. 3.** Phylogenetic tree of the 234 *E. coli* strains studied, reconstructed from the partial  
17 sequences of 8 housekeeping genes ([www.pasteur.fr/mlst](http://www.pasteur.fr/mlst)) by PHYML (23) and rooted on *E.*  
18 *fergusonii*. Bootstrap values are indicated at the corresponding nodes only when they exceed  
19 70%. Pathogenic strains are indicated in red, whereas commensal strains are shown in black.  
20 The name of the strain is followed by H (for human origin) or A (for animal origin). The  
21 colors of the terminal branches of the tree are as follows: orange (F group), red (B2 group),  
22 violet (E group), light blue (C group), blue (A group) and green (B1 group).



1 **Supplementary online material**

2

3 Table S1. List and principal characteristics of the *E. coli* strains studied.

4 Table S2. List of the oligonucleotides used for the detection of the intrainestinal virulence  
5 factors.

6 Table S3. List of the oligonucleotides used in the O-typing PCR assay.

7

1 **References**

2

3

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Factor 2

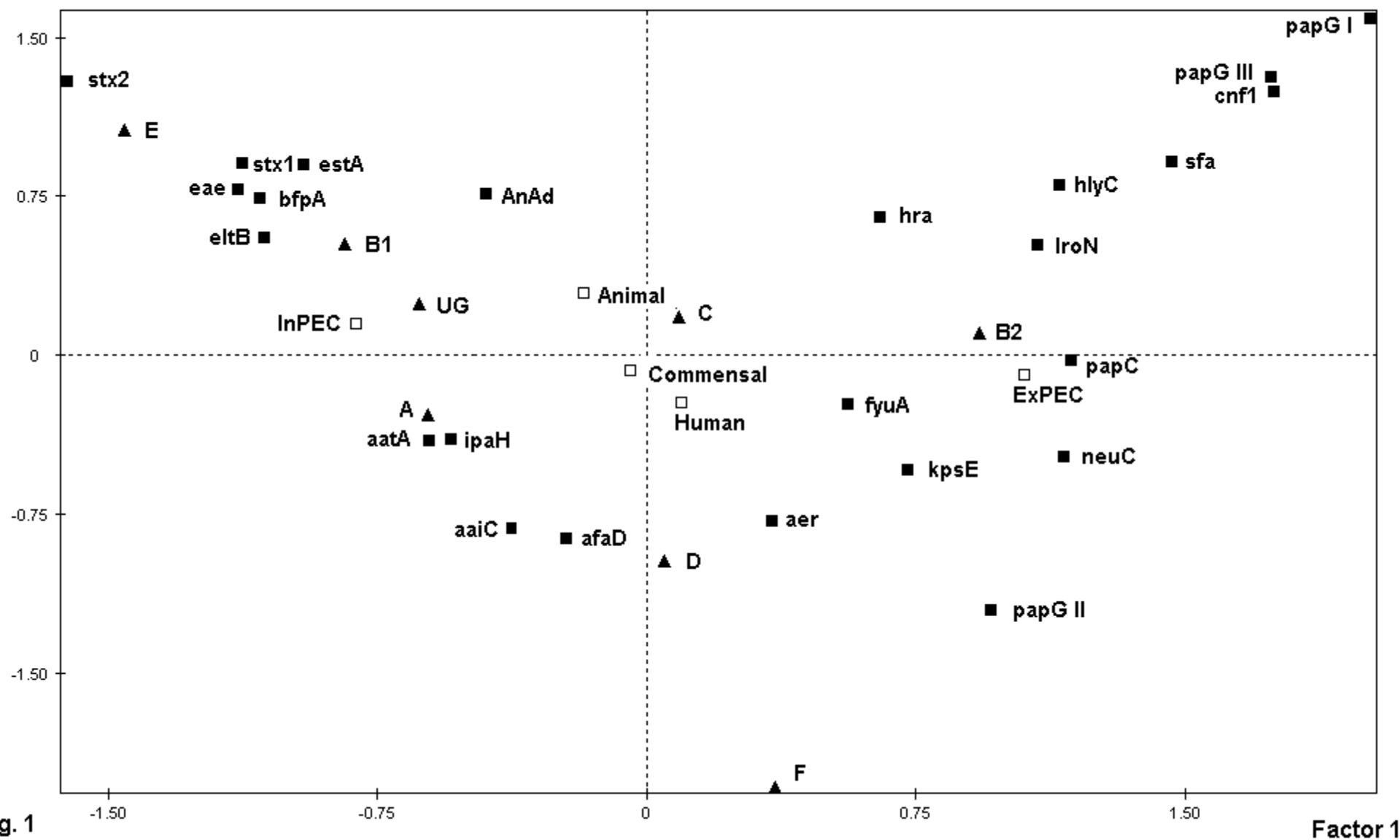
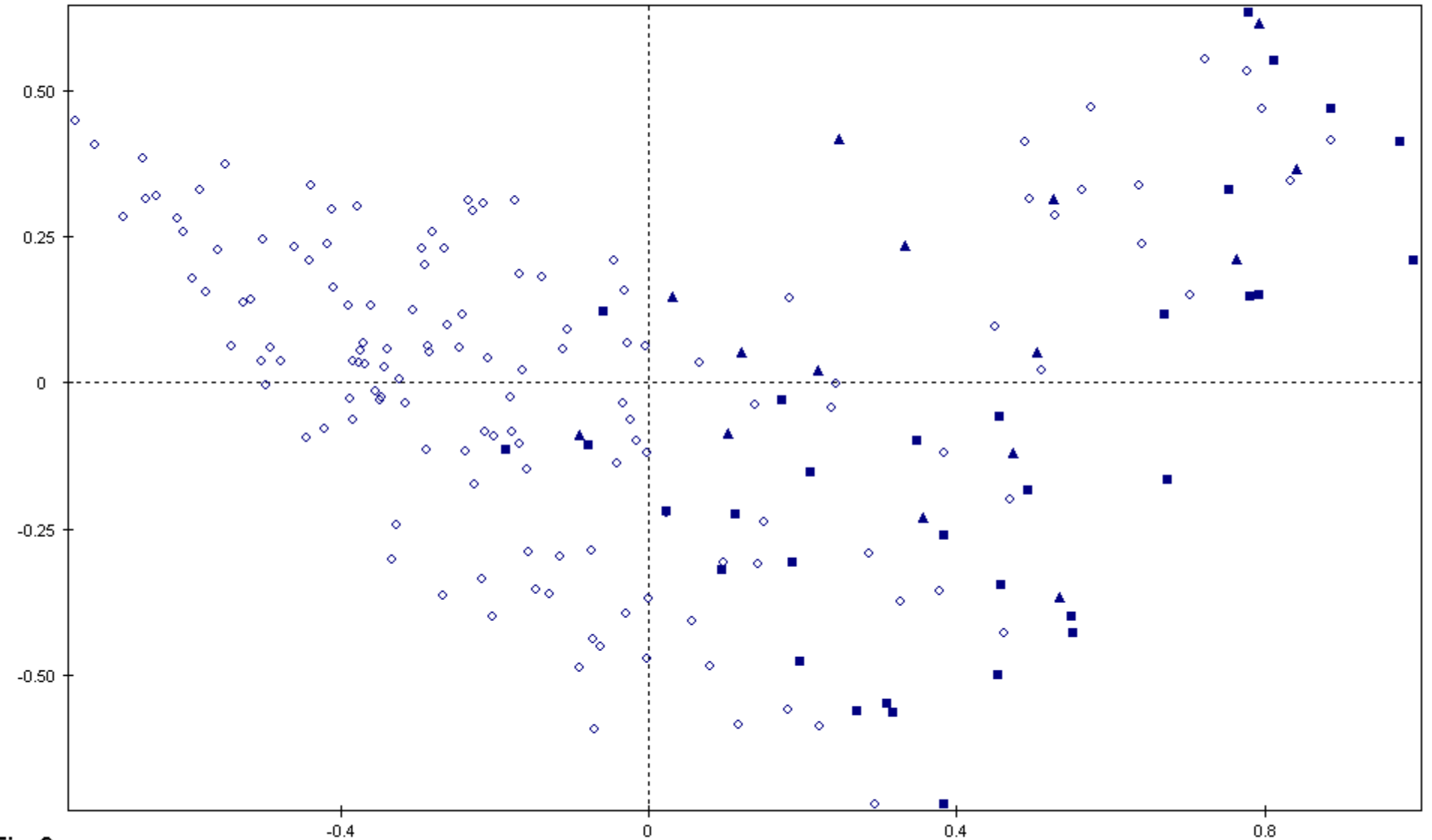


Fig. 1

Factor 1

**A**

Factor 2

**Fig. 2**

Factor 1



**B**

Factor 2

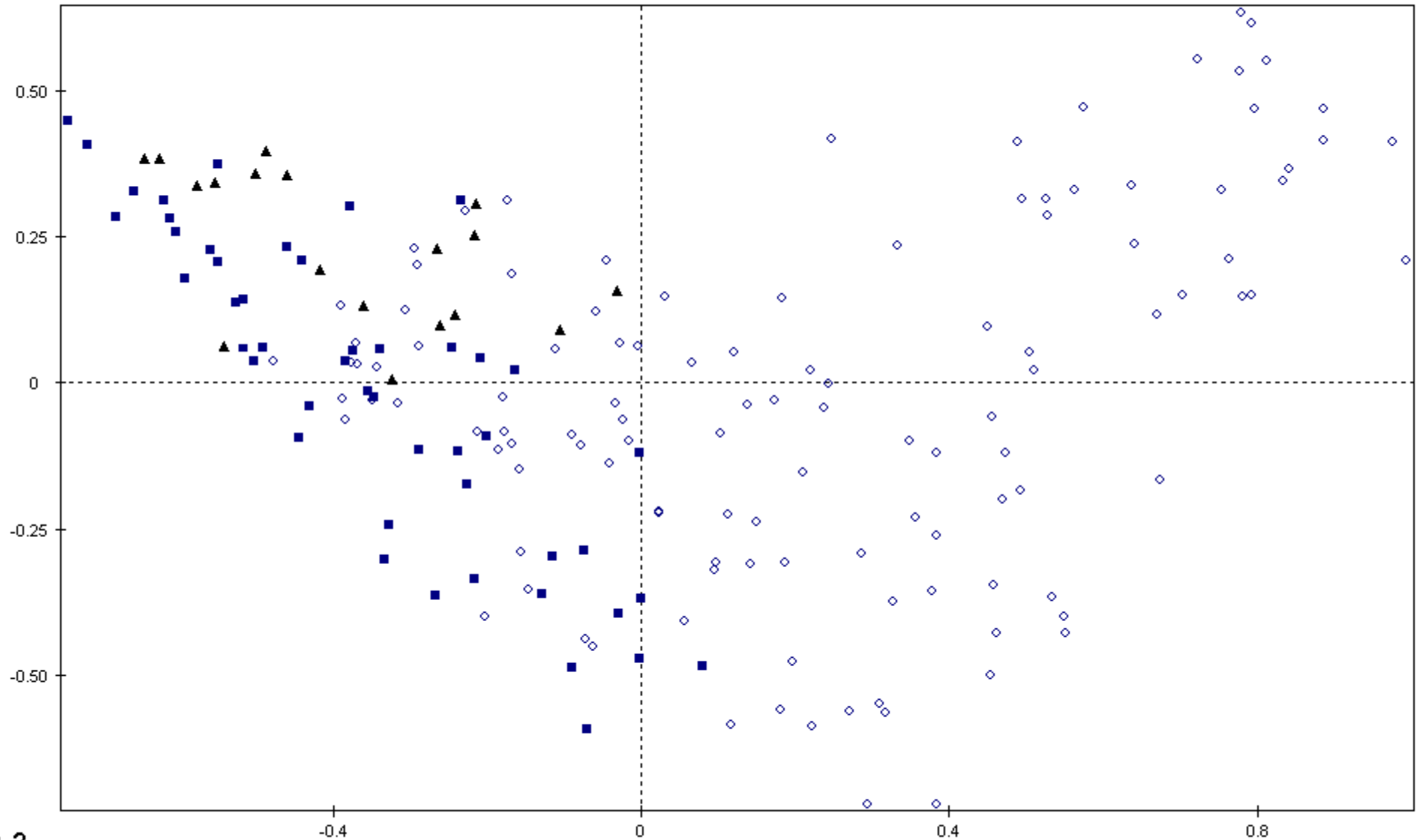


Fig. 2

Factor 1

