ECOLOGY OF PATHOGENIC *E. coli*

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Contents

Summary

Escherichia coli is a ubiquitous bacterial species. It is part of the intestinal microflora of humans and warm-blooded animals and, at the same time, is among the most diffuse bacterial species in the environment. The pivotal aspect of this success is represented by an exceptional genomic plasticity. As a matter of fact, *E. coli* is capable to exchange genetic material with other bacteria, often belonging to different species, through horizontal gene transfer, a process involving the action of mobile genetic elements, such as bacteriophages, transposons, pathogenicity islands and plasmids.

The relationships between *E. coli* and the human host are generally based on commensalism and yet this species exerts a beneficial effect in the gut, however, some strains evolved the capability to harm and cause disease either involving the gastro-intestinal tract or in other organism's districts.
Most of the pathogenic *E. coli* have a human reservoir and an inter-human circulation. These strains cause infections mainly in the developing area of the world where hygienic conditions are generally poor. Some pathogenic *E. coli* have an animal reservoir and are conversely more diffuse in the industrialized countries where intensive farming fostered the circulation of these pathogens.

The complexity of *E. coli* as a pathogen reflects its success as ubiquitous bacterial species. In fact, the ecology of *E. coli* is characterized by a bi-phasic lifestyle, which allows its survival either in a host-associated niche or in the open environment. As a

consequence, the pathogenic *E. coli* survive in soil, manure, and water, including marine water, increasing the possibility to encounter other bacterial species and thus amplifying the sources of new genetic material to be evaluated for suitability at each replication cycle.

The astonishing ductility of *E. coli* is also supported by a sensor system used to gather information from the environment, including the number of species and individuals resident in a given niche at a given time, in order to modulate the genes' expression and properly face the challenge of overcoming the competition.

1. Introduction

Bacteria belonging to *Escherichia coli* species represent an important component of the microbiome, which is defined as the totality of microbes, their genomes and environmental interactions in both human and animal host. This bacterial species colonizes the gastrointestinal tract during the first phases of the life establishing mutual beneficial relationships with the host and playing an important role in maintaining the equilibrium between the numerous other bacterial species constituting the gut microflora. At the same time, *E. coli* is one of the most diffuse bacterial species in the environment, being present in almost all the niches including water and soil. The exceptional capability of *E. coli* strains to colonize a wide range of hosts and environments is linked to their ability of establishing successful relationships with the other microorganisms and it is largely due to their extraordinary genomic plasticity. This is the capability to exchange genetic material with other bacteria, often belonging to other species, through horizontal gene transfer (HGT). This mechanism is mediated by the action of mobile genetic elements (MGE), DNA molecules capable of integrating themselves in the bacterial chromosome, such as bacteriophages, transposons and pathogenicity islands, or autonomously replicating in the bacterial cell cytoplasm such as the plasmids. These MGE are capable of self-mobilization and can move part of the genetic information of the host into a new bacterial cell by following cycles of integration-excision-integration.

HGT represents the key for the success of *E. coli* as ubiquitous bacterial species. It provides the genomic substrate that is probed at each replication cycle in order to select the individuals more adapted to colonize any new ecological niche and to win the "struggle for life".

In the "human environment", generally, *E. coli* are commensals and exert a beneficial effect to the host, however, some strains evolved the capability to harm and cause disease. These strains are grouped into pathogenic groups or pathogroups.

There are two main groups of pathogenic *E. coli* defined on the basis of the district of the organism they colonize: The Extra-intestinal Pathogenic *E. coli* (ExPEC) and the Diarrhoeagenic *E. coli* (DEC) (Nataro and Kaper, 1998). These large *E. coli* pathogroups are then subdivided into more homogeneous sub-groups identified by the mechanisms of pathogenesis or the presence of virulence factors and termed pathotypes. ExPEC include Uropathogenic *E. coli* (UPEC), which are the etiological agents of urinary tract infections, and the neonatal meningitis-associated *E. coli*. ExPEC also

include strains causing septicaemia with bacteraemia and thereof systemic infections in both human and animals.

DEC cause gastroenteritis in humans and animals and are responsible for a range of diarrhoeal diseases and syndromes. They include the following pathotypes: Verocytotoxin-producing *E. coli* (VTEC), also termed Shiga toxin-producing *E. coli* (STEC), enteropathogenic *E. coli*, (EPEC), enteroinvasive *E. coli* (EIEC), enterotoxigenic *E. coli* (ETEC), enteroaggregative *E. coli* (EAEC), diffusely adherent *E. coli* (DAEC).

DEC constitute the *E. coli* pathogroup with the main impact on the public health worldwide; therefore the different DEC pathotypes will be discussed more in detail in the following sections. Each pathotype description will include the virulence features and notes on the epidemiology of the infections. Due to the minor burden of infections, ExPEC will not be broken down into the different pathotypes but will be discussed as whole concerning their ecology in the relevant parts of this work.

2. Pathogenic *E. coli* types

2.1. Verocytotoxin-producing E. coli (VTEC)

VTEC are a heterogeneous pathotype of *E. coli* including strains either non-pathogenic or capable of causing illness to humans. The disease induced by the pathogenic VTEC is characterized by a wide range of symptoms form mild diarrhoea to more severe forms such as Haemorrhagic Colitis (HC) and the life-threatening Hemolytic Uremic Syndrome (HUS). The latter represents the most severe form of VTEC infection, characterized by the following symptoms: microangiopathic anemia, kidney damage, renal failure and thrombotic thrombocytopenic purpura (TTP). HUS patients can sometimes have neurological complications due to ischemic lesions caused by the action of the VT on the endothelium of the brain vessels (Karch et al, 2005).

Administration of antibiotics is not recommended for the treatment of VTEC infections, since their use seems to favor the progression towards the most severe forms of the disease and the management of infection is mainly supportive including rehydration and dialysis for the treatment of HUS.

VTEC elaborate potent cytotoxins termed Verocytotoxins (VTs), which cause cell death by blocking the protein synthesis. VTs are also termed Shiga toxins because of their structural similarity to the Shiga-toxin produced by *Shigella dysenteriae* type I. VTs are a family of toxins including two main antigenically distinct types, VT1 and VT2 (Scotland et al, 1985) and numerous subtypes and variants based on differences in the DNA sequence of the coding genes. Three subtypes of VT1 (VT1a, VT1c and VT1d) and seven subtypes of VT2 (VT2a, VT2b, VT2c, VT2d, VT2e, VT2f and VT2g) have been described so far (Persson et al, 2007). A single VTEC strain can produce either VT1 or VT2 alone or both in any combination type/subtype. The VT-coding genes (*vtx*) are vehiculated by bacteriophages of the lambda (λ) family maintained in a lysogenic state in the bacterial chromosome. Excision of the *vtx*-converting phages and formation of new phage particles capable to infect an indicator *E. coli* strain can be efficiently

obtained by treating a VTEC strain with UV light or the antimicrobial mytomicin, indicating that these bacteriophages are still capable to mobilize and to infect new *E. coli* strains. This observation, suggests that re-infection cycles occur in the natural reservoir ensuring the preservation of the phage population.

The presence of the *vtx*-converting bacteriophages seems to be not sufficient for *E. coli* strains to cause the disease, at least the most severe forms. In fact, pathogenic VTEC associated with bloody diarrhoea or HUS produce additional virulence factors that are involved in colonization and, generally, in the pathogenicity. Most of these strains cause a typical hystopathological lesion to the enterocyte termed "attaching and effacing" (A/E). Such lesion is characterized by the effacement of the microvilli brush border and the intimate attachment of the bacterium to the plasma membrane. The ability to form the A/E lesion is conferred by a pathogenicity island termed the Locus of Enterocyte Effacement (LEE), which harbors the genes encoding a type III secretion system (TTSS), a complex molecular structure allowing the injection of bacterial effectors directly into the host cell. Beside the TTSS, the LEE encodes a number of effectors delivered via the TTSS, an outer membrane protein called the intimin, which mediates direct binding of the bacterium to the host cell surface and encoded by the *eaeA* gene, and its translocated receptor, Tir (Morabito et al, 2003).

VTEC strains associated with severe human disease usually belong to a restricted number of serogroups such as O157, O26, O111, O145, O103 (European Food Safety Authority, 2011) and typically carry the LEE locus. These strains, also known as Enterohemorragic *E. coli* (EHEC), are characterized by the presence of additional virulence genes vehiculated by mobile genetic elements such as the enterohaemolysin-coding operon, carried by a large virulence plasmid, and a pathogencity island, termed O-I 122, which carries a gene, *efa1/lifA*, encoding a factor involved in the colonization of the host by increasing the adhesivity of the strain and by inhibiting the host lymphocyte activation (Karmali et al, 2003b; Klapproth et al, 2000; Morabito et al, 2003). EHEC are thus a sub-group of VTEC with distinct genotypic and phenotypic features and associated to severe disease in humans. VTEC serotype O157:H7 is considered as the prototype of EHEC. It was first isolated in 1982 in the US during an outbreak of HC (Riley et al, 1983) and has become one of the most important pathogenic *E. coli* responsible for sporadic disease and large food and water-borne outbreaks worldwide, since then. EHEC O157 causes the majority of HUS cases in the US and the UK and is responsible of about 50% of HUS in continental Europe. Taken together the five EHEC serogroups (the so called "Top Five") are responsible for about 90% of HUS cases worldwide (European Food Safety Authority, 2011).

VTEC are zoonotic pathogens and ruminants, particularly cattle, are considered their natural reservoirs. The transmission to humans occurs mainly via the consumption of contaminated food and water but other sources such as contact with infected animals and person-to-person contacts are also reported. Initially, VTEC infections were associated to food of animal origin and particularly with undercooked beef burgers. Nowadays, meat of bovine origin still remain one important source of infection, but other vehicles such as unpasteurized milk, dairy products, water and contaminated fresh produce (e.g. sprouted seeds, lettuce, and spinach) are increasingly reported (see Section 5.4).

2.2. Enteropathogenic E. coli (EPEC)

In the mid 20th century EPEC infections were very common and caused large outbreaks with mortality rates up to 50% in Europe and the US. Nowadays, these infections are only sporadically reported in industrialized countries due to the improvement of general standards of hygiene, while this pathotype still remains one of the leading causes of diarrhoea in low-income countries. In the past, the *E. coli* serogroups associated with diarrheal diseases were globally termed as EPEC (Neter et al, 1955). These organisms were in fact identified in routine diagnosis by determining their O serogroups, corresponding to the bacterial lipopolysaccharide surface antigen. At that time all the different DEC pathotypes were not known but it became soon clear that the serogroup-based classification was not efficacious and indeed there was no agreement on which *E. coli* O-antigens should be considered as identifying the true EPEC strains. To settle the subject, the World Health Organization convened a meeting in 1987 and reached the consensus on the definition of the following EPEC serogroups: O26, O55, O86, O111, O114, O119, O125, O126, O127, O128, O142, and O158 (WHO, 1987). This scheme worked for a while, however, in the following years it was recognized that the serogroup was not sufficient to precisely identify EPEC and that the same serogroup could refer to more than one *E. coli* pathotype. In 1995, during an International symposium on EPEC, in Sao Paulo, Brazil, it was decided the subdivision of EPEC into two main distinct groups: typical EPEC (tEPEC) and atypical EPEC (aEPEC) based on the molecular characteristics of the isolates.

Similarly to EHEC and other bacterial species pathogenic to human and animals, such as *Hafnia alvei* and *Citrobacter rodentium* (formerly *C. freundii* biotype 4280), EPEC possess the LEE locus and cause the attaching and effacing (A/E) lesion to the enterocytes (see previous section). The A/E phenotype of EPEC is indistinguishable from that induced by EHEC although with a few differences. In fact, differently from EHEC, the initial stage of adhesion in EPEC is mediated by a bundle-forming pilus (BFP) encoded by the *bfpA* gene localized on a 50-70 MDa plasmid named EPEC adherence factor (EAF) (Kaper et al, 1997).

At the Brazil meeting, the tEPEC have been defined as *E. coli* strains, which cause the A/E lesion but do not produce VTs, possess the EAF plasmid and whose infections are characterized by the inter-human transmission *via* the oral-faecal route. On the other hand, aEPEC lack the EAF plasmid and share with EHEC the presence of additional virulence genes, such as the one encoding the enterohaemolysin (*E-hly)*, as well as a possible animal reservoir. As a matter of fact, aEPEC have been isolated from a wide range of diarrheic and healthy animal species (Aidar et al, 2000; Aktan et al, 2004; Blanco et al, 2005;, Ishii et al, 2007; Krause et al, 2005; Leomil et al, 2005; Stephan et al, 2004; Wani et al, 2007; Yuste et al, 2006) and may therefore be transmitted to humans by contaminated food of animal origin.

The different ecology of the two EPEC groups is likely to explain the differences in the epidemiology and the geographic distribution of the infections. In fact, tEPEC are a leading cause of acute diarrhoea in children less than one year in the developing countries while aEPEC have been reported as agents of persistent diarrhoea in adults

and children aged more than five years and causing either sporadic cases or outbreaks in the industrialized countries (Viljanen et al, 1990; Yatsuyanagi et al, 2003).

2.3. Enterotoxigenic E. coli (ETEC)

Disease induced by ETEC follows ingestion of contaminated food or water and is characterized by profuse watery diarrhoea lasting for several days with little or no fever, a condition that often leads to dehydration and malnutrition in young children. Raw vegetables and soft cheeses are included among the foodstuffs implicated in ETEC infections.

ETEC are a major cause of diarrhoea among infants in developing countries, where hygiene is poor and water sanitation procedures are only rarely in place. Moreover, they represent one of the leading causes of travelers' diarrhoea that affects individuals from industrialized countries traveling to developing regions of the world (Black, 1990; Northey et al, 2007). Travelers' diarrhoea causes an estimated 10 million cases worldwide each year. It has been proposed that individuals genetically predisposed to produce high levels of interleukin-10 (IL-10) are more likely to experience symptomatic ETEC travelers' diarrhoea (Flores et al, 2008). In the developing countries, these strains are the most commonly isolated enteric pathogen bacteria in children below 5 years of age, and account for several hundred million cases of diarrhoea with thousand deaths each year (Black, 1993).

ETEC colonize the gastro intestinal tract of the host by attaching to specific receptors on the enterocytes in the intestinal lumen by the action of hair-like fimbriae. More than 20 types of fimbrial antigens, called *E. coli* surface antigens or colonization factor antigens (CFAs) have been described, which define strain-specific antigenicity (Isidean et al, 2011).

A common feature of ETEC is the ability to express one or more heat-stable (ST) or heat-labile (LT) enterotoxins. One of these toxins (LTI) is plasmid encoded and resembles the cholera toxin produced by *Vibrio cholerae* strains. Another heat labile toxin (LTII) is chromosomally encoded and yet is similar to LTI both in mode of action and in structure. The plasmid-encoded heat-stable enterotoxins belong to two groups, STa/STI and STb/STII (Isidean et al, 2011).

2.4. Enteroinvasive E. coli (EIEC)

EIEC were first described in 1944 as a bacterium called "paracolon bacillus", but later on, a prototype EIEC was identified as *E. coli* belonging to serogroup O124. In the 1950s, while carrying out the Serény-test, an assay intended to assess the invasivity of bacterial strains, a researchers' team identified a group of *E. coli* able to cause experimental keraconjuntivitis in guinea pig, which was initially classified under the species *Shigella* as *Shigella manolovi*, *S. sofia*, *Shigella* strain 13, and *S. metadysenteriae.* These strains were later placed in the *E. coli* subgroup EIEC as *E. coli* O164 (Bando et al, 1998; Monolov, 1959; Rowe et al, 1977). Indeed, EIEC and *Shigella* spp. are remarkably similar either at phenotypic or genetic level and many of these similarities may be attributed to the mechanism of invasion. In fact both these

organisms spend much of their lifetime within eukaryotic cells and therefore have developed a system to use the nutrients coming form the cellular environment, different from the free-living *E. coli* strains (Lan and Reeves, 2002).

EIEC are non-motile, are not able to decarboxylate lysine and approximately 70% of EIEC strains do not ferment lactose. These features are atypical when compared to the majority of *E. coli* strains but are shared by most of the strains belonging to the genus *Shigella* although some strains of *S. sonnei* are able to slowly ferment lactose.

EIEC infection causes a gastroenteric disease identical to Shigellosis. The illness is characterized by abdominal cramps, diarrhea, vomiting, fever, chills, and a generalized malaise. The symptoms appear 12-72 hours after the ingestion of the pathogen depending on the dose assumed. Dysentery caused by this organism is generally self-limiting with no known complications. The site of infection is predominantly the colon and the ability of EIEC to invade and destroy colonic tissue is associated with the presence of virulence factors such as the invasion antigens named as IpaA to IpaH, whose coding genes are harbored by a high molecular weight (140 MDa) plasmid (pINV) which is also present in the *Shigella* strains (Lan et al, 2004). Common symptoms include watery diarrhoea that may precede the production of stools containing blood and mucus. Ulceration of the bowel can also occur in severe cases.

EIEC are human pathogens and infected humans appear to be the primary source of infection as there are no known animal reservoirs. Infection occurs *via* the faecal-oral route by person-to-person transmission. Sometimes, food and water may be implicated as vehicles of infection, but they are secondary contaminated by a human source.

2.5. Enteroaggregative E. coli (EAEC)

The definition of this *E. coli* pathotype has been proposed at the end of the 80s, when the studies on the interactions between *E. coli* strains and cultured HEp-2 cells monolayers allowed the diversification of enteropathogenic *E. coli* on the basis of their pattern of adhesion. The investigators could observe that while EPEC showed the typical Localized Adhesion (LA), other *E. coli* strains displayed a more diffuse adherence phenotype later diversified into “true Diffuse Adherence” (DA) and “Aggregative Adherence” (AA) (Nataro et al, 1987). Despite the late identification, EAEC are supposed to be agents of diarrheal disease at least since 1920 based on the retrospective analysis of the symptoms typically associated to the infections.

The clinical features of EAEC-induced illness include watery, mucoid, secretory diarrhoea with low-grade fever and little or no vomiting (Bhan et al, 1989; Paul et al, 1994). About 30% of patients with EAEC diarrhoea have blood in the stools (Cravioto et al, 1991). The duration of EAEC diarrhoea is its most striking feature, which has been determined to be up to 17 days, longer than that associated with any other pathogen (Bhan et al, 1989). Although observations exist suggesting that EAEC infection may be accompanied by mucosal inflammation; most patients lack of any clinical evidence. Pathogenesis of EAEC infections includes the colonization of the gut by a strong adherence where the bacteria are embedded in a mucus-containing biofilm, whose formation may contribute to its ability to cause long-lasting colonization (see Section 6).

EAEC produce a range of putative pathogenic determinants that are involved in disease. These include the plasmid-encoded aggregative adhesion fimbriae (AAF/I and AAF/II), the main determinant intervening in the AA pattern of adhesion (also termed 'stacked-brick'), whose coding genes are controlled by the transcriptional regulator AggR, a key factor in EAEC pathogenesis.

About half of EAEC strains elaborate toxins, comprising the Enteroaggregative heat-stable toxin-1 (EAST-1), the plasmid encoded Pet toxin and the *Shigella* enterotoxin-1 (ShET1) (Nataro and Kaper, 1998).

The epidemiological features of EAEC infections, including the likely sources, the reservoirs, and the routes of transmission are largely unknown.

At present EAEC is a leading cause of gastroenteritis in developing countries, but EAEC infections are reported also in industrialized countries as causing sporadic cases and outbreaks, sometimes involving a large number of cases. A large outbreak of EAEC diarrhoea occurred in Gifu Prefecture, Japan, in 1993, when 2,697 children in 16 schools became ill after consuming suspected contaminated school lunches. A single EAEC strain was implicated, but the organism was not found in any of the foods served in the implicated lunch (Itoh et al, 1997). Four outbreaks of EAEC-related diarrhoea occurred in the United Kingdom in 1994 (Smith et al, 1997). In two of the outbreaks a single EAEC strain was implicated. Also in this case no single vehicles could be identified, although each of these outbreaks was epidemiologically linked to the consumption of a meal.

An outbreak episode possibly associated to contaminated cheese occurred in Italy in 2008. An EAEC strain of serotype O92:H33 was isolated from six participants to a banquet as well as from one member of the restaurant's staff. A retrospective cohort study indicated a pecorino cheese made with unpasteurized sheep milk as the possible source. However, although samples of the suspected cheese had *E. coli* counts higher than 10^6 c.f.u. g^{-1}, the outbreak strain could not be isolated (Scavia et al, 2008).

Occasionally, EAEC acquire additional virulence genes by horizontal gene transfer originating new *E. coli* pathotypes of great impact in terms of public health. Such a mechanism is supposed to be responsible for the emergence of the *vtx*-producing EAEC O111:H2 that caused a small HUS outbreak in France in the 90s and the *vtx*-producing EAEC O104:H4, which caused a huge outbreak of HUS in Germany in 2011 involving more than 4000 cases of illness and 50 deaths (see Section 8).

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Biographical Sketch

Stefano Morabito is senior scientist at the Unit of Food-borne Zoonoses of the Department of Veterinary Public Health and Food Safety of the Istituto Superiore di Sanità in Rome. The Unit acts as the National Reference Laboratory for *E. coli* for clinical and veterinary aspects and, since 2006, it has been designated European Union Reference Laboratory (EU RL) for *E. coli* by the European Commission. He is deputy director of the EU RL as with the responsibility of the methods development and research activities related to verocytotoxin-producing *Escherichia coli* (VTEC). His main research areas include the molecular bases of virulence in VTEC O157 and other VTEC, with particular emphasis on the genomic asset of the strains causing severe disease in humans. His area of expertise include the mobile genetic elements vehiculating the virulence determinants and their evolution and distribution in the different *E. coli* pathogroups. Other research areas include emergence and phylogenesis of the different VTEC clones as well as studies on the characterization of bacterial toxins and on their capability to disseminate among the different *E. coli* groups.
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