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Extraintestinal Foodborne Pathogens

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Abstract

In general, foodborne diseases present themselves with gastrointestinal symptoms caused by bacterial, viral, and parasitic pathogens well established to be foodborne. These pathogens are also associated with extraintestinal clinical manifestations. Recent studies have suggested that *Escherichia coli* and *Klebsiella pneumoniae*, which both cause common extraintestinal infections such as urinary tract and bloodstream infections, may also be foodborne. The resolution and separation of these organisms into pathotypes versus commensals by modern genotyping methods have led to the identification of key lineages of these organisms causing outbreaks of extraintestinal infections. These epidemiologic observations suggested common- or point-source exposures, such as contaminated food. Here, we describe the spectrum of extraintestinal illnesses caused by recognized enteric pathogens and then review studies that demonstrate the potential role of extraintestinal pathogenic *E. coli* (ExPEC) and *K. pneumoniae* as foodborne pathogens. The impact of global food production and distribution systems on the possible foodborne spread of these pathogens is discussed.

INTRODUCTION

Foodborne diseases make up the most common disease category reported in the United States, according to the infectious disease surveillance systems of the Centers for Disease Control and Prevention (CDC). Gastrointestinal symptoms (diarrhea and vomiting) are the primary clinical events associated with foodborne illnesses. However, most infections with such symptoms do not get reported unless they occur as part of recognized and investigated outbreaks or if a stool sample from a sporadic case of enteric illness is tested and is shown to harbor a reportable pathogen. On the basis of data from active and passive surveillance systems and other sources, the CDC estimates that each year approximately 10 million episodes of foodborne illness in the United States are caused by 31 major pathogens (Scallan et al. 2011b). These pathogens include bacteria, viruses, and parasites. Furthermore, the CDC estimates that nearly 40 million additional annual episodes of foodborne gastrointestinal illness are caused by agents that are not specified (Scallan et al. 2011a).

The above estimates are extrapolated from surveillance data and outbreak investigations of mostly gastrointestinal illnesses caused by recognized pathogens associated with the ingestion of contaminated food. These numbers, however, may grossly underestimate the public health impact and global burden of diseases caused by foodborne pathogens. Foodborne infections are not confined to those associated with gastrointestinal symptoms. Foodborne pathogens can cause extraintestinal infections, and many such infections are well recognized but may or may not get reported in the surveillance systems.

This review first discusses the extraintestinal infections and clinical manifestations caused by recognized foodborne pathogens. The recent applications of molecular epidemiology tools to investigate infectious diseases have revealed that, in addition to the known extraintestinal clinical manifestations associated with recognized foodborne pathogens, there are extraintestinal infections that have not been previously viewed as foodborne diseases caused by pathogens that have not been previously recognized to be foodborne. The second part of this review discusses these diseases and pathogens.

EXTRAINTESTINAL INFECTIONS CAUSED BY RECOGNIZED FOODBORNE PATHOGENS

Extraintestinal infections known to be caused by established foodborne pathogens include bloodstream infections (BSIs), meningitis, and other organ infections caused by *Salmonella* spp. BSIs occur in less than 5% of nontyphoidal salmonella infections in developed countries. In Africa, the estimated annual number of invasive nontyphoidal salmonellosis is approximately 3.4 million, with more than 600,000 deaths (Crump & Heyderman 2015, Haselbeck et al. 2017, Uche et al. 2017). HIV infection, *Plasmodium falciparum* malaria, and sickle cell disease are important risk factors for invasive salmonellosis (Gordon 2011). Invasive nontyphoidal salmonellosis is most often caused by *Salmonella enterica* serotype Enteritidis, *Salmonella* Choleraesuis, and *Salmonella* Dublin (Haselbeck et al. 2017, Stephen et al. 2003). Of particular concern in Africa is a multidrug-resistant strain of serovar *Salmonella* Typhimurium belonging to sequence type (ST) 313, which is more frequently associated with bloodstream than gastrointestinal infections (Graham & English 2009, Hawkey & Livermore 2012, Kingsley et al. 2009). The reservoir of *S.* Typhimurium ST313 is not known, and it has not been established that it is foodborne. Variants of ST313 have been isolated from humans and poultry in Brazil, but they appear to be genetically distinct from those reported from Africa (Almeida et al. 2017).

Other extraintestinal infections or clinical manifestations associated with recognized foodborne pathogens include meningitis caused by *Listeria monocytogenes*; hemolytic uremic syndrome

Table 1 Extraintestinal infections caused by recognized foodborne pathogens

Pathogen	Extraintestinal clinical manifestations	Associated food
Bacteria		
Nontyphoidal *Salmonella* spp.	Bloodstream infection, meningitis, osteomyelitis, spleen infection, postinfectious reactive arthritis	Meat, dairy, vegetables, fruits
Campylobacter jejuni	Guillain-Barré syndrome, postinfectious reactive arthritis	Meat, especially poultry
Listeria monocytogenes	Bloodstream infection, meningitis	Dairy, meat
Enterohemorrhagic *Escherichia coli* (O157:H7)	Hemolytic uremic syndrome (HUS), chronic kidney disease	Meat, dairy, vegetables, fruits
Shigella spp.	HUS, postinfectious reactive arthritis	Restaurant-served food
Yersinia enterocolitica	Bloodstream infection, postinfectious reactive arthritis	Dairy, meat
Clostridium botulinum	Adult botulism: descending muscle paralysis, cranial nerve involvement Infant botulism: descending or global hypotonia	Adult botulism: canned food, fermented tofu, prison brew Infant botulism: raw honey?
Vibrio vulnificus	Bloodstream infection, wound infection	Seafood
Brucella spp.	Systemic symptoms involving multiple organs	Meat, dairy
Parasites		
Taenia solium	Cysticercosis	Pork
Trichinella spiralis	Trichinellosis (muscle involvement)	Pork
Paragonimus westermani	Lung cavitary disease	Freshwater crab, crayfish, snail
Trypanosoma cruzi	Parasitemia, lower extremity edema, lymphadenopathy, rarely cardiomyopathy	Sugarcane juice, açai
Diphyllobothrium latum	Vitamin B12 deficiency, megaloblastic anemia	Freshwater fish
Toxoplasma gondii	Fetal malformation, retinal disease, deafness	Undercooked meat, fruits or vegetables contaminated with feline feces
Angiostrongylus cantonensis	Eosinophilic meningitis	Freshwater fish, shrimp, crab, frog
Others		
Prions	Transmissible spongiform encephalopathy	Beef

(HUS) caused by *Escherichia coli* O157:H7 or other serotypes of Shiga toxin–producing *E. coli* (STEC) such as *E. coli* O104:H4 (Buchholz et al. 2011, Frank et al. 2011); Guillain-Barré syndrome associated with infection with *Campylobacter jejuni* (Allos 1997, Rhodes & Tattersfield 1982); and postinfectious reactive arthritis following recovery from gastrointestinal illness caused by *Campylobacter*, *Salmonella*, *Shigella*, or *Yersinia enterocolitica* (Hill Gaston & Lillicrap 2003, Pope et al. 2007). Exposure to *Vibrio vulnificus* in seafood or seawater can cause severe wound infections (Tacket et al. 1984a,b). The botulinum toxin expressed by *Clostridium botulinum* in food causes descending paralysis in adults; ingestion of *C. botulinum* spores is associated with infant botulism (Arnon et al. 1979, 1981; Arnon & Chin 1979; Chin et al. 1979) (**Table 1**).

In developing countries as well as in some communities of developed countries, food products contaminated with ova, cysts, or larvae of parasitic organisms cause a wide spectrum of extraintestinal illnesses (Dorny et al. 2009). They include neurocysticercosis caused by *Taenia solium* in pork (FAO/WHO 2014, Moazeni et al. 2019); trichinellosis caused by *Trichinella spiralis* acquired through eating undercooked pork (Moorhead et al. 1999); lung cavitary disease caused by *Paragonimus westermani* in freshwater snails, crabs, or crayfish (Chai 2013); orally acquired Chagas'

disease caused by *Trypanosoma cruzi* in triatomine insects or insect feces contaminating sugarcane and tropical fruit such as açaí (Pereira et al. 2009, Shikanai-Yasuda & Carvalho 2012); megaloblastic anemia following vitamin B12 deficiency due to *Diphyllobothrium latum* spp. found in raw or undercooked freshwater fish (Dupouy-Camet & Peduzzi 2004); fetal malformation caused by *Toxoplasma gondii* in meat (Belluco et al. 2018); and eosinophilic meningitis caused by *Angiostrongylus cantonensis* in freshwater fish, shrimp, frogs, and crabs (Serrano-Moliner et al. 2018) (**Table 1**). Finally, proteinaceous infectious particles (prions) in meat, in particular, beef, can cause neurodegenerative diseases in humans collectively called transmissible spongiform encephalopathy (Belay 1999).

Some of these extraintestinal diseases (BSIs, meningitis, HUS) occur concurrently with gastroenteritis, whereas others occur without diarrhea or after resolution of the acute symptoms (Guillain-Barré syndrome, reactive arthritis). Most of the foodborne parasitic diseases are not accompanied by any enteric symptoms and may occur days to weeks to even years after ingestion of the contaminated food. Some of these extraintestinal diseases that occur together with gastrointestinal symptoms are included among disease surveillance systems, such as HUS in the Foodborne Diseases Active Surveillance Network (FoodNet; **https://www.cdc.gov/foodnet/index.html**), whereas other extraintestinal diseases, such as sporadic cases of BSIs or those caused by parasitic organisms may not get reported. Thus, many of these extraintestinal foodborne diseases may not get included as part of foodborne disease estimates. The discussion below focuses on diseases that are not currently included in any of the foodborne disease surveillance systems.

EXTRAINTESTINAL DISEASES CAUSED BY PATHOGENS NOT TRADITIONALLY ASSOCIATED WITH TRANSMISSION BY FOOD

In addition to extraintestinal clinical manifestations caused by established foodborne pathogens, there is now increasing evidence that there are other types of extraintestinal illnesses caused by pathogens that are not traditionally recognized to be associated with contaminated food. If these extraintestinal diseases are caused by such pathogens in food, the global burden of foodborne diseases needs to be reevaluated and reestimated. The remainder of this review discusses new observations that suggest that common human extraintestinal infectious diseases, such as urinary tract infections (UTIs) and BSIs, may be caused by Gram-negative bacterial pathogens not traditionally associated with contaminated food or diarrheal diseases. These organisms are not included among the 31 major pathogens that the CDC reported to cause an estimated 10 million episodes per year of foodborne disease in the United States (Scallan et al. 2011b). Here, we focus on studies that describe *Enterobacteriaceae* organisms, in particular, *Escherichia coli* and *Klebsiella pneumoniae*, that have been newly suggested to be foodborne pathogens.

Pathogenic *E. coli*

E. coli is a member of the normal or commensal intestinal flora of all mammals. However, we know that *E. coli* can cause a variety of enteric diseases as well as extraintestinal infections, including UTIs, BSIs or sepsis, neonatal meningitis, and wound infection. These pathogenic *E. coli* or *E. coli* pathotypes can be divided into two major groups. Strains that cause enteric disease or diarrhea are called diarrheagenic *E. coli* (DEC) or intestinal pathogenic *E. coli* (IPEC) (Kohler & Dobrindt 2011, Nataro & Kaper 1998). *E. coli* that cause extraintestinal infections are referred to as extraintestinal pathogenic *E. coli* (ExPEC) (Russo & Johnson 2000), and they are the most common cause of community-onset (CO) UTIs as well as the most common Gram-negative bacterial cause of BSIs.

IPEC are currently classified into six major subgroups on the basis of their characteristic virulence genes and the clinical symptoms they cause. They include enterotoxigenic *E. coli* (ETEC), enteroinvasive *E. coli* (EIEC), enteropathogenic *E. coli* (EPEC), enteroaggregative *E. coli* (EAEC), diffuse adherent *E. coli* (DAEC), and Shiga toxin–producing *E. coli* (STEC), which includes a subgroup, enterohemorrhagic *E. coli* (EHEC). Although all of these IPEC organisms can cause a wide spectrum of diarrheal manifestations, prototypically, ETEC causes watery or secretory diarrhea, EIEC causes invasive or inflammatory diarrhea, EPEC causes malabsorption diarrhea due to effacement of the intestinal microvilli, and EHEC causes bloody diarrhea or hemorrhagic colitis sometimes accompanied by HUS (Kaper et al. 2004, Nataro & Kaper 1998). An IPEC strain, *E. coli* O104:H4, which caused a large outbreak of gastroenteritis, hemorrhagic colitis, and HUS in Germany in 2011, expresses features of both EHEC and EAEC (Buchholz et al. 2011, Frank et al. 2011). The outbreak was traced to contaminated fenugreek or lentil seed sprouts grown in Saxony, Germany (Buchholz et al. 2011, Frank et al. 2011). The pathogenic features of IPEC subgroups mediated by distinct virulence gene products unique to each of these subgroups are extensively reviewed elsewhere (Kaper et al. 2004, Nataro & Kaper 1998).

Other IPEC subgroups have also been implicated in foodborne outbreaks. EHEC O157:H7 is the most common foodborne STEC in the United States, which is included among pathogens reported by the FoodNet surveillance system (**https://www.cdc.gov/ecoli/outbreaks.html**). Recent ETEC outbreaks have implicated contaminated kimchi in Korea, Christmas buffet scrambled eggs in Norway, and sushi restaurants in Nevada, USA (Jain et al. 2008, MacDonald et al. 2015, Shin et al. 2016). EIEC has caused outbreaks due to contaminated lettuce in the United Kingdom, an unspecified food item served at a kindergarten in China, and cheese in the United States and Europe (Newitt et al. 2016, Olsvik et al. 1991, Zhou et al. 2015). The first recognized foodborne outbreak of EPEC in the United States was caused by imported French cheese in 1971 (Marier et al. 1973).

IPEC, particularly ETEC, EIEC, EPEC, and STEC, can be readily distinguished from *E. coli* organisms that reside in the human intestine as commensal or normal flora based on their distinct virulence genes (Fialho et al. 2013, Gunzburg et al. 1995, Tornieporth et al. 1995, van Ijperen et al. 2002). They transiently colonize the human intestine to cause disease and are not part of the normal flora. Hence, the natural reservoirs of IPEC are located outside of the human colon. It is, however, more complicated to distinguish ExPEC from commensal *E. coli*. A variety of so-called virulence genes or factors (e.g., adhesins, siderophores, protectins, hemolysins, toxins, polysaccharide antigens, invasins, colicins, outer-membrane proteins, and pathogenicity island gene products) have been described in ExPEC isolates from extraintestinal infection sites, but many of these factors are also shared by fecal *E. coli* isolates from healthy individuals (Johnson et al. 2001, 2003; Johnson & Russo 2018; Kohler & Dobrindt 2011; Sarowska et al. 2019). Although certain virulence-associated traits and membership in phylogenetic groups have been proposed to characterize ExPEC (Johnson & Russo 2005, Johnson & Stell 2000), at this time, no set of genes can be used to unequivocally distinguish ExPEC from commensal *E. coli* (Kohler & Dobrindt 2011). For the purpose of this review, *E. coli* strains are considered ExPEC if they have been isolated from patients with clinically diagnosed extraintestinal infections with no underlying chronic or immunosuppressive medical conditions.

An *E. coli* strain that causes extraintestinal infections enters the extraintestinal site from the intestine of the infected host. It can translocate directly into the bloodstream from the colon, or it can enter the bladder from the perineum. An *E. coli* strain that enters the bladder can ascend the ureter to cause pyelonephritis, and from the kidneys, it can enter the bloodstream. However, this does not necessarily mean that such *E. coli* is a commensal. After all, a *Salmonella* strain that causes BSIs must colonize the intestine first before it enters the blood. If ExPEC organisms are

nothing more than commensal *E. coli* that happen to breach a sterile barrier to cause extraintestinal disease, then their natural reservoir is the human intestine. If ExPEC are true pathotypes, then their natural habitat must lie outside of the human intestine. They first must enter the intestine to transiently colonize the intestine and subsequently spread to extraintestinal sites to cause disease. If so, the possibility of ExPEC transmission by food or water or from other external sources must be considered.

Are Extraintestinal Pathogenic *E. coli* Organisms Transmitted by Food?

An investigation of a community outbreak of diarrhea can help to implicate food as a vehicle of the outbreak, which can then lead to the identification of an etiologic agent. The hemorrhagic colitis/HUS outbreak in Germany described above led to the identification of a new subtype of IPEC *E. coli* (EHEC/EAEC) (Buchholz et al. 2011, Frank et al. 2011). EHEC *E. coli* O157:H7 was first identified based on investigations of two distinct outbreaks of hemorrhagic colitis that occurred in the United States in the early 1980s (Riley et al. 1983). However, extraintestinal infections such as CO-UTIs, healthcare-associated BSIs, *E. coli* meningitis, and wound infections are not readily recognized to occur as outbreaks. Hence, food as vehicles of such infections had not been entertained in the past.

However, in the mid-1980s in south London, a large increase in the number of community-acquired extraintestinal infections (UTIs, BSIs, meningitis, endocarditis) caused by a single serotype of *E. coli* (O15:K52:H1) was recognized, and it was suggested that this increase represented an outbreak or epidemic (Phillips et al. 1988). During the same time period, also in south London, a large increase in the number of hospital infections due to *E. coli* serogroup O15 was recognized (Waghorn et al. 1988, Wright & Perinpanayagam 1987). This was the first time that these extraintestinal infections due to *E. coli* were suggested to occur as part of an outbreak or epidemic. The serotype was later shown to belong to multilocus sequence type (MLST) ST393 [MLST based on Achtman's seven-gene scheme (**https://pubmlst.org/mlst/**)]. ST393 has subsequently been identified in several countries in western Europe, as well as in the United States and Asia, causing CO-UTIs as well as BSIs (Johnson et al. 2002; Lee et al. 2010; Olesen et al. 1995, 2009).

What led the investigators in south London to suggest that the increased number of extraintestinal infections represented an outbreak was the *E. coli* serotype (O15:K52:H1) information. In 2001, Manges et al. (2001) reported a possible outbreak of CO-UTIs that occurred at a college community in Northern California caused by a distinct strain of *E. coli* resistant to trimethoprim-sulfamethoxazole (TMP-SMZ) based on another subtyping test—*Enterobacterial* repetitive intergenic consensus sequence (ERIC) polymerase chain reaction (PCR). ERIC-PCR is a genotyping method based on electrophoretic band patterns generated from PCR-amplified sequences located between repetitive DNA sequences distributed across the chromosome of some *Enterobacteriaceae* species. Between October 1999 and January 2000, 255 *E. coli* isolates from 228 women with uncomplicated UTIs were analyzed. Of 55 (22%) TMP-SMZ-resistant isolates, 28 (51%) showed a distinct agarose gel electrophoresis band pattern generated by ERIC-PCR amplicons. Strains exhibiting such a pattern were called clonal group A (CgA). UTI isolates obtained from patients at college campuses in Minnesota and Michigan around the same time period showed that 39% and 38% of TMP-SMZ-resistant isolates, respectively, had the CgA agarose gel electrophoretic band pattern (Manges et al. 2001). These clusters of CO-UTIs caused by a single genotype of ExPEC at three different geographic sites led investigators to suggest that CgA strains may have been disseminated by contaminated food that was nationally distributed (Manges et al. 2001). These CgA strains were subsequently found to belong to ST69, which is now recognized to be globally disseminated (Dias et al. 2009, Johnson et al. 2011, Riley 2014, Tartof et al. 2005).

Table 2 ***Escherichia coli*** **sequence types (STs) isolated from human extraintestinal clinical sources from two or more regions of the world and have also been isolated from food and domesticated, companion, and wild animals**

ST[a]	Number of entries	Food and food-animal sources	Other sources
ST10	6,432	Poultry, bovine, swine, dairy (raw-milk cheese), goat, sheep, fish	Dog, horse, rabbit, sea lion, camel, pigeon, gazelle
ST12	598	Swine, bovine, fish	Dog, cat, horse, mink, raccoon, rat
ST69	1,529	Poultry, bovine, sheep, dairy (raw-milk cheese)	Dog, horse, dolphin, mink, bald eagle, seagull
ST73	1,984	Poultry, swine, bovine	Cat, donkey, duck, horse, giraffe, orangutan, elephant, gorilla, rhesus monkey, ferret, mouse
ST95	1,590	Poultry, bovine, lettuce	Dog, ostrich, swan, rat, gecko, poultry feed
ST117	925	Poultry, bovine, calf, swine	Dog, cat, mink, rabbit, rat, animal feed
ST127	525	Turkey, bovine, celery	Dog, gazelle, rat, horse
ST131	6,574	Poultry, bovine, pork	Dog, cat, rook, horse, seagull, rodent
ST405	646	Bovine, whale	Dog, crow, marmoset

[a]These sequence types are included among multilocus sequence type (MLST) entries in Enterobase MLST site as of June 18, 2019, based on Achtman's seven-gene scheme (**http://enterobase.warwick.ac.uk/**).

Because IPEC organisms, especially *E. coli* O157:H7, can cause multistate foodborne outbreaks (see **https://www.cdc.gov/ecoli/outbreaks.html**), it is not far-fetched to suggest that another group of *E. coli*—i.e., ExPEC—may be spread by contaminated food, especially meat, or food or water contaminated with food-animal manure. Indeed, CgA or ST69 *E. coli* strains have been isolated from a variety of meat and food-animal sources. An *E. coli* CgA isolate from a cow was found by pulsed-field gel electrophoresis (PFGE) to be approximately 95% similar to a CgA isolate from a patient with a UTI in California (Ramchandani et al. 2005). A retail food survey conducted in the Minneapolis-St. Paul area in 1999–2000 found CgA in turkey products (Johnson et al. 2005a). ST69 has been isolated from chicken and pork as well as from vegetables (Campos et al. 2013, Ramchandani et al. 2005, Vincent et al. 2010, Yamaji et al. 2018a) (**Table 2**).

ST69 is one of a small group of ExPEC lineages that are overly represented among UTI and BSI patients from different communities in the world. These so-called pandemic or intercontinental ExPEC lineages include ST69, ST73, ST95, ST131, and ST393 (Riley 2014). They are responsible for more than a third to half of all ExPEC-associated extraintestinal infections all over the world (Adams-Sapper et al. 2013, Bert et al. 2010, Blanco et al. 2011, Croxall et al. 2011, Gibreel et al. 2012, Horner et al. 2014, Lau et al. 2008, Manges et al. 2008, Riley 2014, Salipante et al. 2015, Yamaji et al. 2018b). Other STs, such as ST10, ST12, ST117, ST127, and ST405, are also increasingly recognized in human extraintestinal infections in multiple regions of the world (Bergeron et al. 2012, Hussain et al. 2017, Jones et al. 2008, Manges et al. 2019, Manges & Johnson 2012, Riley 2014). A recent population-based study identified nearly 200 distinct ExPEC STs from 1,188 clinical samples (Liu et al. 2018). Thus, despite the fact that extraintestinal infections can be caused by many different ExPEC genotypes, the observations that fewer than 10 ST lineages account for most of the extraintestinal infections in the world suggest point-source or common-source exposures to these dominant lineages. These observations led to the idea that ExPEC or a subset of ExPEC strains may represent foodborne pathogens.

E. coli Genotypes Shared Between Human Clinical Sources and Food

The idea that ExPEC organisms may be transmitted by food is not new. The modern genotyping tests such as MLST or whole-genome sequencing (WGS) have only helped to provide

further support to this idea. Before MLST or WGS came to be widely applied, *E. coli* strains were distinguished based on multilocus enzyme electrophoresis (MLEE) analysis into so-called *E. coli* reference collection (ECOR) phylogenetic groups (Ochman & Selander 1984). Of the four main phylogenetic groups (A, B1, B2, and D) (Clermont et al. 2000), *E. coli* strains isolated from extraintestinal infections are disproportionally represented among groups B2 and D (Groisman & Ochman 1996; Hacker et al. 1997; Johnson 1991; Johnson et al. 2001, 2005b; Munkhdelger et al. 2017; Nuesch-Inderbinen et al. 2017). Studies from the early 2000s have shown strains from these phylogenetic *E. coli* groups to be isolated from a variety of retail food products, including poultry, beef, pork, and certain ready-to-eat foods (Dezfulian et al. 2003; Jakobsen et al. 2011; Johnson et al. 2003, 2005c; Maynard et al. 2004). Furthermore, *E. coli* isolates from chicken and egg belonging to B2 and D phylogenetic groups have been shown to be capable of causing sepsis, UTIs, and meningitis in rodent models of infection (Mellata et al. 2018).

However, typing by ECOR phylogenetic groups does not achieve the level of resolution needed to convincingly show epidemiologic relatedness between ExPEC and food *E. coli* strains. In contrast, there are more than 7,500 unique *E. coli* STs and 56 *E. coli* clonal complexes (CCs) archived in the Enterobase MLST database (**http://enterobase.warwick.ac.uk/**). The database reveals that most of the internationally observed ExPEC lineages (ST10, ST12, ST69, ST73, ST95, ST117, ST127, ST131, ST405) have each been isolated from feces, intestines and other organs of livestock animals, and animal food products (meat and dairy) as well as from green leafy vegetables (**Table 2**). Although these STs account for approximately 0.001% of all STs, the number of *E. coli* isolates (20,803) represents more than 19% of all *E. coli* entries (109,190; $p < 0.000001$) in the MLST database (as of June 2019). Some of them have also been identified from retail poultry feed, the environment (sewage, river), wild birds (seagull, ostrich, crow, swan, cormorant), and wild animals (rat, gazelle, hedgehog, marmoset, monkey, lizard) as well as companion and domesticated animals (cat, dog, horse) (**Table 2**). Interestingly, of 163 entries of ST393 in the MLST database, none is noted to be isolated from a specific food product. The small number of ST393 entries may be contributing to this lack of association with nonhuman sources.

Among meat products, poultry in particular has been shown to frequently contain *E. coli* genotypes that match the pandemic ExPEC STs. They most often include ST10, ST69, ST95, ST117, and ST131 (Bergeron et al. 2012, Cortes et al. 2010, Hussain et al. 2017, Jakobsen et al. 2010, Johnson et al. 2017, Liu et al. 2018, Leverstein-van Hall et al. 2011, Manges 2016, Mitchell et al. 2015, Mora et al. 2013, Vincent et al. 2010, Yamaji et al. 2018a). In a survey of retail meat products in Northern California conducted concurrently with a study of CO-UTIs in a Northern California college campus, 21% of *E. coli* isolates from suspected cases of UTIs were found to belong to *E. coli* STs found in retail chicken and turkey (Yamaji et al. 2018a). In a 12-month study conducted in Flagstaff, Arizona, Liu et al. (2018) found 76 distinct STs shared by 2,452 meat and 1,188 clinical *E. coli* isolates. ST131 (fimbrial subtype H22) was the most common ST among the clinical *E. coli* isolates and thirteenth among meat isolates; all but two of 27 meat ST131-H22 isolates were from poultry products (Liu et al. 2018). Using PFGE, which is more discriminating than MLST for subtyping *E. coli*, Kluytmans et al. (2013) showed closely matched PFGE patterns of human and chicken meat *E. coli* isolates. In the Netherlands, extended-spectrum β-lactamase (ESBL)-producing strains of *E. coli* ST10, ST69, and ST131 were isolated from both human clinical samples and chicken meat (Leverstein-van Hall et al. 2011). Ciprofloxacin-resistant strains of ST10 were found in Italy from both human clinical sources and chicken (Leverstein-van Hall et al. 2011). During 2005–2007, ST117 was found to be the most common *E. coli* ST isolated from chicken in different regions of Canada (Bergeron et al. 2012).

Jakobsen et al. (2010) have shown that CgA or ST69 isolates from chicken and humans can cause bladder as well as kidney infections in a mouse infection model. The same group later showed

that *E. coli* strains with similar PFGE patterns isolated from human UTI patients and broiler chicken and pork meat sources were found to be similarly virulent in a mouse model of UTIs (Jakobsen et al. 2012). Another more recent study showed that genetically similar *E. coli* strains (not typed by MLST) from human clinical sources, chickens, and eggs were shown to cause disease (UTIs, sepsis, and meningitis) in rodent models of infection (Mellata et al. 2018). These animal studies suggest that food *E. coli* isolates genetically related to human ExPEC isolates are indeed pathogenic, at least in the mouse model, and thus the potential for zoonotic transmission of such *E. coli* strains to humans to cause extraintestinal infection is plausible.

Other studies support the biological plausibility of poultry as an important reservoir of some strains (not all) of ExPEC. *E. coli* strains that cause extraintestinal infections, collectively called colibacillosis, in birds are referred to as avian pathogenic *E. coli* (APEC) (Cordoni et al. 2016; Dho-Moulin & Fairbrother 1999; Johnson et al. 2008; Jorgensen et al. 2019; Kobayashi et al. 2011; Moulin-Schouleur et al. 2007; Rodriguez-Siek et al. 2005a,b; Ronco et al. 2017; Sola-Gines et al. 2015). Many of these APEC strains belong to phylogenetic group B2, and some of the internationally disseminated human ExPEC frequently represented among them include ST10, ST69, ST95, ST117, and ST131. In addition to poultry as described above, they have been identified from a wide variety of wild birds, including pigeon (ST10), seagull (ST69, ST95, ST131), bald eagle (ST69), ostrich (ST95), swan (ST95), rook (ST131), duck (ST73, ST117), and wild turkey (ST117), as annotated on the Enterobase MLST site (**http://enterobase.warwick.ac.uk/**) (**Table 2**).

A recent comparative WGS analysis of 323 APEC and human ExPEC genomes belonging to ST95 revealed that APEC was highly diverse and did not form a phylogenetic branch distinct from ExPEC (Jorgensen et al. 2019). Their phylogenetic analysis showed APEC on multiple branches together with closely related human ExPEC, some of which were nearly identical, which suggests that APEC can be pathogenic for humans and ExPEC can be pathogenic for birds. Interestingly, however, ExPEC ST95 strains separated into those with or without bird virulence–associated plasmids (ColV plasmids), suggesting some ExPEC ST95 strains may be less virulent for birds or that these plasmids are not necessary for causing human infection (Jorgensen et al. 2019). These observations suggest that the intestinal ecosystem of birds may possess unique characteristics that select for *E. coli* strains with increased fitness that contributes to their colonization in the human gut and proliferation in human extraintestinal sites.

Of course, the detection of identical STs or closely related genotypes of *E. coli* isolates from human extraintestinal infections and food or food animals per se does not necessarily demonstrate foodborne transmission of ExPEC. Unlike IPEC infections in which incubation periods of diarrhea are readily ascertainable and thus food exposure could be assessed, incubation periods for ExPEC-associated diseases cannot be determined. If ExPEC is introduced into the intestine via contaminated food, an extraintestinal infection such as a UTI or BSI could occur any time during the intestinal colonization period, which would make it impossible to assess the time of exposure to a contaminated food vehicle. Thus, the standard procedures used to provide evidence for exposure to contaminated food cannot be applied to ExPEC.

Outbreaks of enteric disease caused by newly recognized IPEC organisms such as *E. coli* O157:H7 and *E. coli* O104:H4 were not recognized until the 1980s and 2000s, respectively. They came to be recognized during the period corresponding to profound changes that occurred in human food consumption habits and animal food production practices and distribution systems, including intensification and centralization of food-animal feeding operations, a mass food distribution network, the globalization of food trade, and the global expansion of fast-food restaurant chains. In the United States, the consumption of chicken per capita increased from 28 pounds/year in 1960 to 93.5 pounds/year in 2018; turkey consumption per capita increased from 6.2 to

16.1 pounds/year during the same period (Natl. Chick. Counc. 2018). This increase in consumption of poultry coincides with the period of increased frequency of extraintestinal infections caused by ExPEC STs found in poultry.

As of this writing (June 2019), no human ExPEC ST69 and ST393 entries are included among STs of more than 400 *E. coli* isolates collected before 1980 included in the MLST database, and only one human ST131 entry is included (isolated in 1967) (**http://enterobase.warwick.ac.uk/**). Among 109,190 *E. coli* strain entries currently in the MLST database, ST69 and ST131 make up 1,525 (1.4%) and 6,558 (6%) entries, respectively. Of course, the absence of such STs among these early collections of *E. coli* could be a sampling artifact. Or the human intestinal microbiota composition may have evolved with changes in what we consume, and the human intestinal flora may just be a reflection of the microflora found in food, water, or the environment. If so, the extraintestinal sources of ExPEC still need to be considered, and the notion of human-specific intestinal microbiota may not be meaningful.

Analysis of a larger collection of human *E. coli* isolates from earlier periods is needed to determine whether these STs represent commensal or true pathotypes of *E. coli* or whether the so-called commensal *E. coli* genotypes change over time. *E. coli* genotypic analyses of feces from a large number of healthy persons may also provide a better understanding of the relationship between ExPEC and commensal *E. coli*.

Nevertheless, comparative genotypic analyses of ExPEC to date suggest that ExPEC, at least some of them, may be foodborne. Evidence in support of this idea is (*a*) the geographic and temporal clustering of ExPEC genotypes isolated from patients with extraintestinal infections, which is suggestive of outbreak occurrence or common-source exposures; (*b*) the global distribution of identical ExPEC lineages indicating spread by contaminated globally traded food vehicles; (*c*) the detection of identical genotypes of ExPEC isolated from human infections and *E. coli* isolated from retail food products in concurrent surveys conducted in the same geographic sites; (*d*) the disproportional representation of pandemic or international ExPEC lineages among thousands of ST lineages causing extraintestinal infections in all parts of the world, indicating the biologic fitness advantage of these strains in different food-animal or non-food-animal (e.g., wild birds) reservoirs; and (*e*) the relatively recent appearance of *E. coli* genotypes ST69, ST131, and ST393 as ExPEC, suggesting recent introduction of these genotypes into the human intestinal niche from extraintestinal sources.

We learn a tremendous amount about the epidemiology of IPEC infections from outbreak investigations and surveillance systems. A surveillance system of ExPEC genotypes causing extraintestinal infections could similarly provide opportunities for conducting outbreak and traceback investigations. Such a surveillance system can further elucidate the role of ExPEC as new extraintestinal foodborne pathogens. Unfortunately, such a surveillance system does not currently exist.

Klebsiella pneumoniae as a Pathogen

Although it is often reported as a commensal organism, *K. pneumoniae* is probably an environmental saprophytic organism found in a variety of ecologic niches, especially water-related niches such as sewage, wet soil, surface waters, industrial effluents, vegetation, and even drinking water (Bagley 1985). It is highly adaptive and its ubiquitous environmental occurrence contributes to its frequent colonization of the human gut, which may be viewed as a continuum of the ecological niches of this organism. From the gut, *K. pneumoniae* strains gain entry into other sites to cause extraintestinal infections. It is usually not associated with diarrhea but is frequently recovered from persons with diarrhea.

In most communities, *K. pneumoniae* is the second most common cause of CO-UTIs and second-most common Gram-negative bacterial cause of BSIs after *E. coli*. Although most severe *K. pneumoniae* infections (sepsis, ventilator-associated pneumoniae, meningitis, liver abscess) are healthcare associated, a large proportion of UTIs and BSIs are acquired in community settings or have onset in the community. Even in hospitalized patients, most patients diagnosed with a BSI acquire the infection in the community. In a population-based study of BSIs caused by Gram-negative bacteria diagnosed among hospitalized patients at a large county hospital in San Francisco, Adams-Sapper et al. (2012) showed that in two-thirds of these patients, the blood culture tested positive for Gram-negative bacteria within less than 48 hours of their hospital admission, which, according to the CDC definition, would be considered to represent CO or community-acquired infection. This is not surprising given that a large proportion of BSIs caused by Gram-negative bacteria are preceded by UTIs.

Before the 1990s, *K. pneumoniae* was largely considered an opportunistic pathogen, causing mostly healthcare-associated infections in patients with chronic underlying medical conditions or patients admitted to intensive care units or long-term acute-care hospitals. However, we now know that some lineages of *K. pneumoniae* cause widespread epidemics, both in healthcare settings and in the community.

The emergence of an epidemic-prone genotype of carbapenem-resistant *K. pneumoniae* in the 1990s, starting on the East Coast of the United States, led the CDC in 2013 to declare carbapenem-resistant *Enterobacteriaceae* (CRE) strains "urgent threat" pathogens (Cent. Dis. Control Prev. 2013a). A plasmid-encoded carbapenem-hydrolyzing β-lactamase called KPC was first described in a *K. pneumoniae* isolate recovered from a patient in North Carolina in 1996 (Yigit et al. 2001). KPC (encoded by gene bla_{KPC}) is currently the most common carbapenemase expressed by *Enterobacteriaceae* organisms globally (Bradford et al. 2004; Bratu et al. 2005a,b,c; Chiang et al. 2007; Endimiani et al. 2009a,b; Gupta et al. 2011; Hong et al. 2013; Hrabak et al. 2013; Jain et al. 2013; Lomaestro et al. 2006; Ma et al. 2013; Marchaim et al. 2008; Munoz-Price et al. 2013; Nordmann et al. 2009; Woodford et al. 2004). The most common KPC-expressing *K. pneumoniae* (KPC-Kp) genotypes worldwide belong to CC258 (Temkin et al. 2014, Nordmann et al. 2009). KPC hydrolyzes nearly all β-lactam drugs, and strains carrying the gene expressing KPC often also carry genes that confer resistance to other classes of antimicrobial agents, which renders clinical management of infections caused by these strains extremely challenging.

The strain isolated in North Carolina was ST258, the most common member of CC258. It caused widespread hospital outbreaks in New York City in the 2000s (Bradford et al. 2004; Bratu et al. 2005b,c; Woodford et al. 2004). Mortality exceeding 50% has been reported in some hospital outbreaks with this strain, and it continues to be responsible for the majority of KPC-Kp infections in US hospitals, especially on the East Coast, including long-term acute-care hospitals; it is responsible for more than 77% of all outbreaks in the US and 90% of all KPC-Kp infections in Israel (Cent. Dis. Control Prev. 2013b, Chen et al. 2014a, Endimiani et al. 2009a, Kitchel et al. 2009, Schwaber et al. 2011, Temkin et al. 2014, Woodford et al. 2004).

ST258 is believed to have emerged after a recombination event involving ST11 (a member of the CC258) and a distantly related strain, ST442 (Chen et al. 2014b, Endimiani et al. 2009a). KPC-Kp ST11 is distributed in China and Latin America (Andrade et al. 2014, Chen et al. 2014a, Munoz-Price et al. 2013, Qi et al. 2011). Another KPC-Kp CC258 member ST512 has been found in Colombia, Italy, and Israel (Munoz-Price et al. 2013). In Brazil, the most common KPC-Kp strain isolated from patients in many hospitals belongs to ST437, which is also related to ST11 (Andrade et al. 2011, Martins et al. 2006). Among 226 rectal swab isolates of ESBL-producing *K. pneumoniae*, four CC groups—CG17 in France, CG101 in Italy, CG15 in Spain, and CC147

in Israel—were dominant [based on Pasteur Institute MLST nomenclature (Inst. Pasteur 2019)] (Baraniak et al. 2013, Chen et al. 2014a).

The Pasteur MLST database currently (June 2019) contains 4,044 MLST entries comprising 67 STs for *K. pneumoniae*. The clonal distribution and epidemics caused by key drug-resistant *K. pneumoniae* strains are reminiscent of the international or pandemic ExPEC lineages described above. Various biological bases for the global dissemination of these clonal strains have been suggested, but no mechanism has yet been clearly demonstrated to contribute to such transmission (Paczosa & Mecsas 2016). Their clonal global spread could also be explained epidemiologically via, e.g., their potential widespread distribution by food, following multiple point-source contaminations. The pan-adaptive trait of some strains of *K. pneumoniae* could contribute to their persistence in food products, which could explain their highly successful global dissemination.

Genotype ST258's clustering by time and place, especially in hospital settings in eastern US urban centers, could result from common-source exposures, but it could also be explained by this genotype's enhanced adaptability to hospital environments and efficient colonization of human intestine to mediate medical-device as well as person-to-person transmissions. Community acquisition of *K. pneumoniae* suggests exposures to something harboring the bacteria introduced via the mouth. In either case, the extraintestinal infection in an individual results from infection by *K. pneumoniae* introduced to that person from an external source.

Is *Klebsiella pneumoniae* Transmitted by Food?

In 1998, Sabota et al. (1998) reported a case of foodborne sepsis attributed to *K. pneumoniae* and *E. coli* in a 28-year-old healthy man who ate a hamburger (Sabota et al. 1998). The blood isolates of *K. pneumoniae* and *E. coli* were identical to the isolates from the hamburger in terms of a drug-susceptibility assay, plasmid profile, and toxin gene assay by a DNA hybridization probe (Sabota et al. 1998). Although this was a case report, it is the first and only known report documenting an occurrence of a BSI by *K. pneumoniae* following the ingestion of a food item contaminated with the same *K. pneumoniae* strain.

In 2008, a large cluster of patients was infected by and fecally colonized with *K. pneumoniae* over a period of nine months in multiple hospital wards of a 500-bed acute-care hospital in Barcelona, Spain; this infection was found to be caused by a single PFGE type (Calbo et al. 2011). The time interval between admission and colonization was very short, and no healthcare workers or environmental surfaces in the wards were colonized with this strain, whereas a third of the hospital kitchen surfaces and 14% of food handlers were found to be colonized (Calbo et al. 2011). This led the investigators to conclude that this cluster of infections and colonization represented a foodborne outbreak (Calbo et al. 2011). No specific food item was implicated, but this was the first known report of a possible hospital outbreak of foodborne disease attributed to *K. pneumoniae*.

More recently, Davis et al. (2015) examined 82 *K. pneumoniae* isolates from human clinical and retail meat (chicken, turkey, pork) sources in Flagstaff, Arizona, and found that several of the strains were closely related by WGS phylogenetic analysis and that ST14, ST76, ST188, and ST111 were shared between the two sources (Davis et al. 2015). A mouse sepsis model showed that isolates from both sources were similarly virulent (Davis et al. 2015). Although international clonal lineages such as ST258 were not detected, this is the largest study to date to show shared genotypes of *K. pneumoniae* between human clinical and food sources. Similar studies are needed in regions of the United States (e.g., the eastern states) where ST258 is more prevalent.

K. pneumoniae, of course, can be recovered from a wide variety of environmental as well as food sources (Davis & Price 2016). At this time, there are not yet very many studies that have simultaneously analyzed genotypes of *K. pneumoniae* from clinical sources and food or environmental

sources in the same geographic settings. Nevertheless, the above studies suggest that some strains of *K. pneumoniae*, a member of *Enterobacteriaceae*, like ExPEC, may be foodborne. If ExPEC and pathogenic *K. pneumoniae* organisms are transmitted by foods to cause extraintestinal infections, the global estimates of foodborne diseases may need to be revised. The impact on public health and clinical medicine of these types of extraintestinal infections need to be further explored from the perspectives of intensification of food production, a mass food distribution network, and the globalization of food trade.

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