

A REPORT FROM
THE AMERICAN ACADEMY
OF MICROBIOLOGY

E. COLI: GOOD, BAD, & DEADLY

FAQ



AMERICAN
SOCIETY FOR
MICROBIOLOGY

“What is true for *E. coli*
is true for the elephant”

FAQ

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FAQ reports are based on the deliberations of 15-20 expert scientists who gather for a day to develop science-based answers to questions the public might have about topics in microbiology. The reports are reviewed by all participants, and by outside experts, and every effort is made to ensure that the information is accurate and complete. However, the report is not intended to advocate any particular position or action, nor to replace the advice of an individual's health care provider.

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If you followed news headlines in the spring/summer of 2011, you may recognize *E. coli* as the agent responsible for outbreaks of serious diarrheal illness in Germany. But this is only one small part of the story of *E. coli*; its relationship to human health and the food we eat is much more complex. Not all *E. coli* are bad - in fact most are not - and some are even beneficial! In this report the larger story of *E. coli* is told: its role in human health, in food, and even in our understanding of our own biology.

What is *E. coli* anyway?



E. coli is a bacterium; a one-celled organism that is too small to see by the naked eye, and is also sometimes referred to as a microorganism or microbe. *E. coli* is an abbreviation of the organism's full scientific name: *Escherichia coli*. Scientists normally use *E. coli* for short; similar to how we call ourselves humans, rather than using our full scientific name *Homo sapiens*. *E. coli* got its first name, *Escherichia*, from the German pediatrician

Theodor Escherich, who discovered the bacteria in 1885. Its second name, coli, means "from the colon," which is the organism's natural habitat. Most *E. coli* live and grow harmlessly in the gastrointestinal tract, or gut, of many animals, including humans.

There are many different types of *E. coli*. Scientists refer to these different types as strains. In a sense, *E. coli* strains are like dog breeds, all strains of *E. coli* are the same type of organism but they may have somewhat different traits. Like dog breeds, different *E. coli* strains can mix with each other to produce new strains with a combination of traits. Scientists sort *E. coli* into different strains according to the particular set of marker compounds they carry on their surfaces (not unlike dogs' different colors and textures of hair). For example, the *E. coli* strain responsible for the outbreaks in Germany is named O104:H4. The letter "O" refers to a marker on *E. coli*'s surface that is found in hundreds

of different shapes, in this case, shape #104. The "H" describes a different marker that is found on the *E. coli*'s flagellum, a tail-like appendage used by *E. coli* to swim around. It is not the "H" or "O" molecules themselves that make *E. coli* lethal. They are simply markers that are easy to detect, allowing us to tell one *E. coli* strain from another.

Unfortunately, the *E. coli* strains people are most familiar with are those that cause disease. Perhaps the most infamous strain is O157:H7, which was responsible for the Jack in the Box hamburger outbreak in 1993 and the more recent spinach outbreak in 2006. Rather than existing harmlessly in our gut, disease-causing strains like O157:H7 disrupt body functions, resulting in diarrhea. The most dangerous strains can also affect the kidneys and nervous systems of victims, causing permanent damage and sometimes even resulting in death.

Yet despite all the attention given to their harmful brethren, most *E. coli* are not harmful to humans, and some are even beneficial. Many of us host a population of *E. coli* in our gut that aids digestion and protects us from other harmful microbes. Scientists have used strains of *E. coli* to study fundamental biological processes, contributing to many important scientific breakthroughs and teaching generations of biology students the rudiments of the scientific method. Other *E. coli* strains are utilized by researchers in industry to produce important compounds we use every day.

How has *E. coli* contributed to our understanding of biology?

The Nobel Prize winning French scientist, Jacques Monod, once said “What is true for *E. coli* is true for the elephant.” Although his statement may seem puzzling, it is perhaps the best way to summarize the contribution of *E. coli* to our understanding of biology.

What Monod meant was that all living organisms operate in a similar manner, whether bacterium or elephant. Many of the same fundamental processes that occur in elephant cells (or human cells for that matter) occur in *E. coli* as well. While humans and other higher organisms are often made of trillions of cells working

together, *E. coli* is composed of a single cell. Thus biological processes that would be difficult or unethical to study in humans or animals can be studied more easily in *E. coli*. This is why scientists often refer to *E. coli* as a “model organism” - it provides them with an easily studied model of biological processes that are shared by living organisms.

One particular strain of *E. coli*, K-12, has been used as a model organism by scientists for decades and has been an important player in many of the keystone experiments that have contributed to our knowledge of biological processes. For example, the discovery of codons, the language of DNA in which all our genes are written, would not have been possible without the use of *E. coli*. Studies in *E. coli* have helped scientists understand how certain sets of our genes are turned on or off, depending on the circumstances. By studying the viruses that infect *E. coli*, scientists have learned much about the form and function of viruses that infect humans, like the influenza virus or HIV.

E. coli is such an important tool to scientists that it was one of the first organisms selected to have its complete set of genes, or genome, sequenced. When techniques were developed to insert genes from other organisms into *E. coli*, it became even more important as a tool to help us learn about gene function and regulation. *E. coli* jumpstarted the biotechnology era because *E. coli*'s genetic flexibility allowed industrial microbiologists to use *E. coli* as a factory to produce many biological compounds we use every day. In the past, insulin used for the treatment of diabetes had been harvested from pigs (or even sharks). By inserting the human gene for insulin into *E. coli*, human insulin can now be produced inexpensively in large quantities. Rennin is a good example of a commercial product produced in *E. coli*. Used in cheese production, rennin was previously harvested from the stomachs of calves. Now it is mostly manufactured in special *E. coli* strains.

It may seem counter-intuitive, but *E. coli* can even be used to make our food and water safer to consume. Diagnostic tests designed to detect all types of *E. coli* (good or bad) can alert food safety microbiologists to the possibility of contamination before food or water is consumed. *E. coli* are excreted in feces, so while it may only signal the presence of harmless *E. coli*, positive results warn regulators that food may have been exposed to unsanitary conditions.

BOX 1: *E. coli* wins record number of Nobel Prizes!



The Nobel Prize is science's highest honor. Each year there are only a few Nobel prizes awarded. These go to scientists whose discoveries “have conferred the greatest benefit on mankind.” Below is a list of the Nobel-worthy discoveries to which *E. coli* has contributed and the year that they received the prize.

- 1958: Bacterial sex, and other ways bacteria can share genes with one another
- 1959: DNA replication, how life copies its genetic code
- 1965: Gene regulation, how genes are turned on or off
- 1968: The genetic code, the language in which our DNA is written
- 1969: Virus replication, how viruses reproduce inside cells
- 1978: Restriction enzymes, cellular “scissors” that allow scientists to cut DNA
- 1980: Recombinant DNA, the creation of the first genetically engineered DNA
- 1989: RNA as an enzyme, additional roles for RNA discovered
- 1997: ATP generation, how cells make ATP, the energy molecule that powers life
- 1999: Signal sequences on proteins, one way that cells organize themselves
- 2008: Green fluorescent protein, a tag scientists use to track cell components

What does the naturally occurring *E. coli* in our GI tract do?

E. coli is just one member of a much larger community of microorganisms that live in our intestines. To give an idea of the size of this microbial community - called the gut microbiome - the average human gut contains about two pounds of microbes (about 90 trillion individual microbes!) that live in an intimate partnership with their human host. For comparison, the average human body is made up of about 10 trillion cells. With 90 trillion microbes associated with you, only about 1 in 10 cells in your body is even human! Despite these amazing numbers, scientists are only just beginning to understand how important the association with this microbial community is for our health.

Humans cannot properly digest some of the compounds we ingest in our food. Microbes in our gut can break down these compounds and release nutrients that would otherwise be inaccessible. These microbes can also produce vitamins like K and B12 that are important for our health. In addition to these functions, the microbes in our gut take up space. While this might not seem important at first, it's vital for our health. The moist warm environment of our gut is a perfect habitat for microbes to grow in; including many that can make us sick. The microorganisms in our gut microbiome can actually protect us by claiming this space and preventing many other harmful organisms from growing there. If you have ever taken a potent antibiotic for an infection, you may have experienced firsthand the importance of our gut microbiome. An unfortunate side-effect of many broad spectrum antibiotics is the disruption of this protective layer of microorganisms. Without that protective layer other harmful microbes can colonize the gut. This is why many people develop diarrhea after taking antibiotics. There is evidence to suggest that our normal micro-



biome may play an even more active role in protection of our health by stimulating our immune system, keeping it primed to fight off other microbes.

E. coli is a minority member of the gut microbiome, but a very important one. Unlike many microbes in the gut, *E. coli* can grow in the presence of oxygen, which is toxic to many other microbes in our gut microbiome. As the *E. coli* consume oxygen from the gut, they help establish a welcoming habitat for the other microorganisms of our microbiome.

Human beings' interactions with their gut microbiome are not a one way street. Our gut provides this microbial community with a protected niche in which to grow and a constant supply of food. Our intimate interaction with the microorganisms in our gut has fueled the fast-growing field of probiotics; products that contain live cultures of the bacteria that naturally live in our gut or compounds that will promote the growth of these organisms to help us maintain a healthy gut.

What is the difference between “good” *E. coli* that inhabits our gut, and “bad” *E. coli* that makes us sick?



The big difference between the “good” and the “bad” *E. coli* strains is all in their DNA.

As all organisms live and grow, they must produce vital cellular components and replace those that wear out. DNA is the biological material that holds the information (or blueprint) that living organisms need to build these new components. Specific areas of DNA called genes contain the information for each individual component. For the most part, the “bad” or pathogenic *E. coli* strains have the same collection of genes, or genome, as the *E. coli* that normally live in our gut. But pathogenic *E. coli* also have a few extra genes that contain the information needed to produce components that make these strains harmful. Most of these pathogenic strains are adapted to life in our gut, but others have armed themselves with genes that can wreak havoc if they grow in other areas of the body like the urinary tract.

An important caveat is that even some of the *E. coli* strains that are normal inhabitants of our gut can cause illness if they get into the wrong place. Our bodies have become acclimated to and even depend on the *E. coli* that naturally reside in our gut, but if they grow elsewhere they can cause problems. Think of a jackhammer – it is a useful tool at a construction site but you would not want it in a china shop! Likewise, some *E. coli* that are harmless when growing in the gut can cause infections if they get into the blood stream, urinary tract infections if they grow in the urethra, or kidney failure if they grow in the kidneys.

How do pathogenic *E. coli* make us sick?

Pathogenic *E. coli* cause illness by disrupting the normal function of the intestines.

There are several ways that *E. coli* can do this, and different strains of *E. coli* may possess one or more of these traits.

Toxins are biological poisons produced by some *E. coli* strains. These poisons can be injected into intestinal cells, or simply secreted into our bodies. Depending on the mode of delivery, toxins can have local or far-reaching effects on intestinal cells. Invasion into the cells lining the intestines also causes disease by disrupting normal cellular functions. Diarrhea results



BOX 2: What are toxins?

The English root for poison, “toxi,” was adapted from the Greek word for arrow poison, “toxicon pharmakon” (τοξικον φαρμακον). In scientific vernacular, toxin refers to a biological poison produced by a living organism. *E. coli* strains that acquire genes for the production of toxins become more dangerous, just like arrows coated with poison.

E. coli toxins are often very potent and can disrupt or kill cells with deleterious effects. The *E. coli* strain linked to the outbreaks of foodborne illness in Germany in spring/summer of 2011 was particularly harmful because it produced Shiga toxin (abbreviated as Stx). There are two major types of Shiga toxins: Stx1 and 2. Both types interfere with our cells’ ability to build new proteins, and particularly target cells lining the blood vessels of certain organs. Disruption of protein building capability leads to cell death. Shiga toxins are released from pathogenic *E. coli* under certain circumstances when they are stressed or die. This can often make treatment complicated as some treatments to kill the harmful *E. coli* will result in a temporary increase in the levels of the toxin in the body. Enterotoxins interfere with the ability of the gut to absorb fluids normally and may even reverse the process so that fluids pour out into the intestine. Endotoxins, another major type of toxin associated with *E. coli*, are actually structures built on the surface of the bacterium that act as an irritant to human cells.

The strategy behind *E. coli* toxin production is not fully understood. Why would *E. coli* produce proteins harmful to their host? Scientists are still debating the reason behind this, but many believe that it may simply be a case of these strains being in the wrong place. Many strains of *E. coli* that are pathogenic in humans are harmless to cattle. It is possible that same toxins that cause illness in humans may help the *E. coli* colonize or survive in the gut of cattle or other hosts. Some recent studies have shown that this may be true of Shiga toxins, in particular. Many toxins even resemble compounds produced by plants to protect themselves from viruses. Only with more research can scientists hope to understand the curious strategy of toxin production.

when the *E. coli* cause cells in the intestine to absorb less water, or release water into the intestine. In some cases *E. coli* can damage small vessel cells that line the intestines resulting in bloody diarrhea.

There are three features that all pathogenic *E. coli* need to cause intestinal illness: they must be able to get into our gut, they must be able to stay there, and they must have the ability to disrupt the normal functions of cells in our intestines.

Pathogenic *E. coli* does not require any special genes to gain entry to the gut. Harmless *E. coli* are also capable of passing through the digestive tract. Food and water are the most common routes of entry for both types of *E. coli*.

Once in the gut, pathogenic *E. coli* must be able to attach to intestinal cells in order to make us sick. Some strains produce hair-like appendages called pili or fimbriae that allow them to stick to the cells lining the intestines. Others produce a compound called intimin that allows them to attach to intestinal cells. Still others are actually capable of invading and getting inside intestinal cells! Different strains of pathogenic *E. coli* may use one or more of these methods to remain in the intestines.

Pathogenic *E. coli* cause illness by disrupting the normal function of the intestines. Diarrheal symptoms result when *E. coli* cause cells in the intestine to absorb less water, or release water into the intestine. There are several ways that *E. coli* can disrupt intestinal function, and different strains of *E. coli* may possess one or more of these traits. Some produce toxins, which are biological poisons that can either be secreted into our bodies, even entering the bloodstream, or injected directly into intestinal cells. Depending on the mode of delivery, toxins can have local or far-reaching effects on intestinal cells. In some instances damage to small blood vessel cells that line the intestines can result in bloody diarrhea. Other *E. coli* strains invade the cells lining the intestines and cause disease by disrupting normal cellular functions.

■ THE ALPHABET SOUP OF PATHOGENIC *E. COLI* STRAINS

What's in a name? For pathogenic *E. coli* strains, a name can provide a good deal of information. In addition to strain names, pathogenic *E. coli* strains can also be characterized more broadly by some key traits, including how they attach to intestinal cells, the toxins they produce, and where they come from. Below is a list of some of the major categories into which pathogenic *E. coli* strains can be divided and the characteristics that define them:

- **ETEC:** (Enterotoxigenic *E. coli*) attaches to the intestines via hair-like appendages called fimbriae, and produces toxins. ETEC causes diarrhea without fever. It is common in infants and is often the cause of travelers' diarrhea (AKA Montezuma's revenge).
- **EIEC:** (Enteroinvasive *E. coli*) invades and destroys cells lining the colon and causes watery, dysentery-like diarrhea. Fever is another common symptom.
- **EPEC:** (Enteropathogenic *E. coli*) attaches to cells in the intestines via an attachment protein called intimin and causes watery, sometimes bloody diarrhea. It is a common cause of infantile diarrhea in underdeveloped countries.
- **EAEC:** (Enteraggregative *E. coli*) attaches to the cells lining the intestines in a distinctive clumping manner and produce enteroaggregative toxin. EAEC strains often cause prolonged diarrhea in children.

ETEC, EIEC, EPEC, and EAEC are all passed in human feces.

- **EHEC:** (Enterohemorrhagic *E. coli*) attaches via intimin protein, but produces a poison called Shiga toxin. EHEC strains cause bloody diarrhea and can sometimes damage the kidneys and progress to the potentially fatal hemolytic uremic syndrome (HUS). EHEC has caused many large food-borne outbreaks worldwide; O157:H7 is the best known strain. This group is also known as STEC (Shiga-toxin producing *E. coli*) and is the only group that is passed in animal feces.



Why does *E. coli* make some people sick and not others?

There are many physiological factors involved in whether or not someone is sickened by exposure to pathogenic *E. coli*.

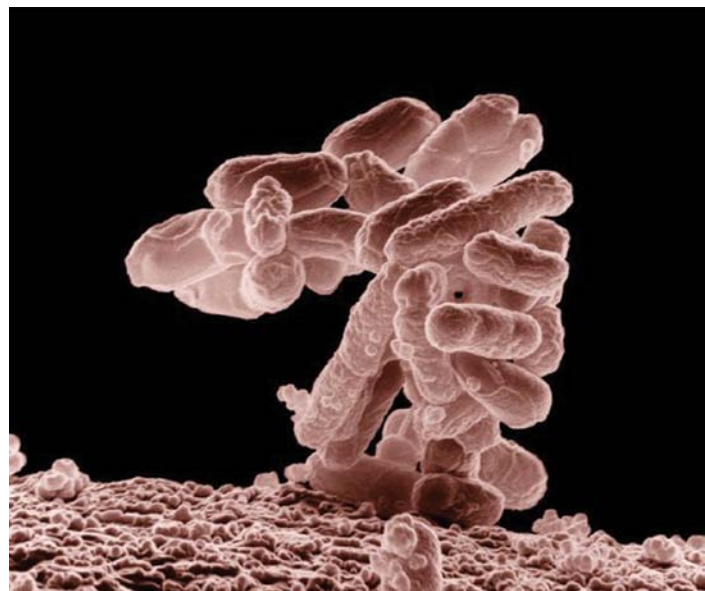
The state of the immune system is one major factor. Individuals with healthy immune systems will be much more likely to fight off pathogenic *E. coli* before symptoms emerge. Other groups may not be so lucky; these include the very young, very old, and people who suffer from compromised immune systems from HIV infection, diabetes, or some cancer and arthritis treatments. Prior exposure to *E. coli* may play a role in the severity of illness as well. Individuals who have been exposed to pathogenic *E. coli* in the past may have retained an immune memory of the pathogen, allowing their immune system to act faster to deal with a second assault. In some *E. coli* illnesses, gender can play a role in susceptibility. Anatomical differences between the sexes make it easier for non-pathogenic *E. coli* to stray into the urinary tract and cause urinary tract infections (UTI's) in women compared to men.

Aside from immunological and anatomical differences, the amount of the *E. coli* or toxins consumed can have an effect on the severity of illness. The saying that "poison is in the dosage" is accurate for illnesses caused by *E. coli*. Following guidelines for proper food preparation can help to cut down or eliminate pathogenic *E. coli* ingested with food and thereby reduce your chances of getting ill. Good hygiene practices, like washing your hands before cooking or eating and keeping raw foods apart from cooked foods, are all useful practices to prevent you from inadvertently contaminating your food, help limit exposure to *E. coli* and decrease your odds of becoming sick.

How does *E. coli* become pathogenic?

E. coli is genetically promiscuous. It can exchange genes with other strains of *E. coli* and even other types of bacteria.

Occasionally this can result in a non-harmful *E. coli* strain becoming pathogenic. There are many different genes that, when acquired, could change a harmless *E. coli* strain into one that makes us sick the most obvious of these being genes for toxins. For example, the *E. coli* strain responsible for the outbreaks in Germany in 2011 was a less harmful strain that acquired genes for production of Shiga toxin. This is something of an abnormality. Generally the conversion of *E. coli* to pathogenicity is not a one-step process but rather involves the acquisition of several genes in a step-wise fashion. While toxin genes are the most obvious culprits for the conversion to pathogenicity, other genes can have similar effects. Acquiring genes that allow an *E. coli* strain to better attach to or invade intestinal cells can also be important steps to developing the ability to cause disease. Other genes, like those that code for antibiotic resistance, can make strains more dangerous by making them more difficult to treat.

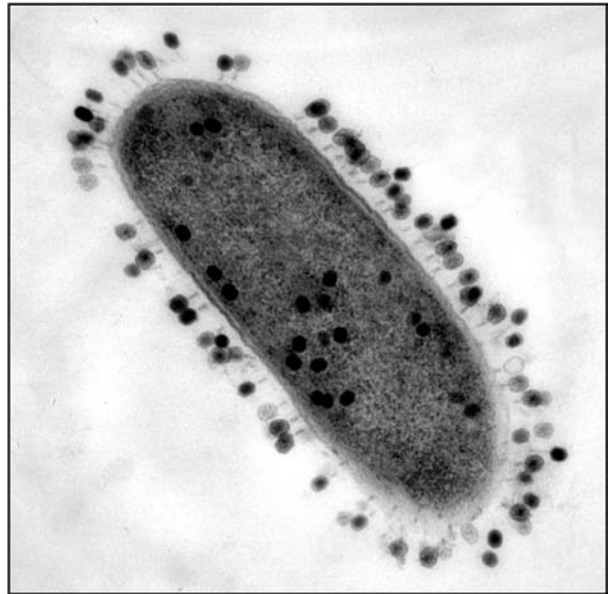


How do *E. coli* get genes from other bacteria?

The most common way that *E. coli* and other bacteria exchange genes is by way of infection with special viruses that target bacteria, called bacteriophages, or phages for short. These special viruses reproduce themselves by injecting their genes into *E. coli* and other bacteria where the viral genes begin a program that hijacks the bacterium's internal machinery, effectively taking over the bacterium. Once this happens the hijacked machinery begins to produce more viruses by replicating the virus genes and outer cover, or capsid, of the virus. Once new viruses have been assembled from these parts, they escape and infect other bacteria.

Occasionally the copying and packaging of the viral genes is sloppy, and some of the bacterium's genes are copied as well. These genes may then be packaged into the viral capsid, and inserted into the next bacterium to be infected. Although the chance of this happening is low, phages are so numerous that these mispackages represent the most common form of gene exchange in bacteria. Some viruses carry the genes for toxins in their genome, making this a particularly potent method of passing toxin genes from one bacterium to another.

Genes can also be transferred from one bacterium to another by conjugation, the bacterial equivalent of sex. To begin the process, one bacterium extends a bridge to another bacterium, establishing a connection between the two, similar to an enclosed walkway between buildings. Once this link is established, genes on DNA structures called plasmids can be transferred from one bacterium to the other. Plasmids are small



circular collections of genes that can copy themselves independently of the bacterial genome. Genes that give bacteria resistance to antibiotics are often found on plasmids, so conjugation is a common way that antibiotic resistance is spread. *E. coli* aren't picky and can conjugate with other strains of *E. coli* as well as with other unrelated bacteria, spreading genes among many different bacteria.

The last method by which *E. coli* can acquire new genes is through the acquisition of naked DNA. When bacteria die they often pop, a process called lysis, and release their contents, including their DNA, into the environment. There is a low, but real chance that this "naked DNA" will be picked up by *E. coli* and become incorporated into its set of genes. This method is less common than the other two processes, but still represents a method of gene transfer.

How does our food become contaminated with *E. coli*?

It all starts with poop.

This may seem flippant or disgusting, but it is quite accurate. Because *E. coli* lives in the gut, transmission of *E. coli* from one organism to another is predominantly from feces to mouth. The source of *E. coli* in almost all food and water contamination events can be traced back to exposure to fecal matter at some point in the food chain; whether it is on the farm, at the processing plant, in transportation, during retail, at the restaurant, or even during preparation in our homes.

One direct route of fecal contamination is from the use of manure as a fertilizer in agriculture. Most manure is devoid of harmful *E. coli*, but a small percentage of cattle carry pathogenic *E. coli* in their gut. These harmful *E. coli* are then shed in the cattle's manure and, if applied to crops without first being composted, can be a source of contamination. If contaminated produce is not properly cleaned or processed, it may contain *E. coli* when it reaches the supermarket. Wildlife may also contaminate food. Deer, birds, and pests native to agricultural areas can deposit feces that may contain pathogenic *E. coli* on plants. Manure can cause contamination problems in food even when not used as fertilizer. Raw milk can be contaminated during the milking process. When cattle are slaughtered, *E. coli* from their feces can occasionally make its way onto beef. When this happens, it is only the surface of the meat that becomes contaminated. For many cuts of beef, like steak, cooking is likely to kill the bacteria. In ground meat however, the grinding process will mix and distribute the contaminated meat throughout the product, which is why it is so important to cook ground

beef thoroughly. Improper food handling is another potential source of fecal contamination. Poor personal hygiene, like failure to wash hands properly after using the toilet, can pass harmful *E. coli* from infected food handlers to food products.

Contamination of food can also come through water. Spraying contaminated water on plants to irrigate, wash, or chill them can contaminate foods. If produce (especially leafy green vegetables) are grown in water that has been contaminated by manure, the *E. coli* can adhere to their surfaces and become extremely difficult to wash off. In some cases, the *E. coli* can even find its way inside the vegetable's cells where washing will have no effect. Water laced with *E. coli* is not only a problem on the farm. Water used in food processing, or even to wash food at the supermarket, if harboring harmful *E. coli*, can also contaminate food.

It is difficult to list all the ways that food could be contaminated with *E. coli* – there are so many possibilities. This is why ensuring food safety, by protecting our food from contamination by *E. coli* and other pathogens, is such a challenging and complex task!

Food is not the only way we can ingest *E. coli*. Pathogenic *E. coli* contamination is a problem in recreational water as well. If not properly cleaned and chlorinated, *E. coli* can survive in swimming pools. Ponds and rivers are other recreational water sources that can potentially be contaminated by *E. coli*. Certain settings where humans are in close contact with animals or their manure, such as farms, petting zoos, or using manure in gardening are a few examples of how harmful *E. coli* strains can gain entry into our bodies.



What steps are being taken to protect our food from contamination by pathogenic *E. coli*? And what can be done?

There are two ways to protect food from contamination with pathogenic *E. coli*: prevent bacteria from getting into food in the first place or treat food products in such a way as to kill or inactivate any bacteria that have slipped past all precautions.

In the modern world, food follows a complicated and sometimes very long path from the farm to the consumer's table. As described in the previous section, contamination can occur anywhere along that path. There are regulations and guidelines in place to reduce the risk of contamination, but no single regulatory agency is responsible for the entire food manufacturing process from raw to finished product.

The U.S. Department of Agriculture (USDA) is responsible for meat, poultry, and eggs, and regularly inspects these manufacturing plants. The U.S. Food and Drug Administration (FDA) is responsible for regulating the safety of all other food including seafood and produce and routinely tests food to ensure the absence of harmful *E. coli* and other bacteria. The FDA has also developed voluntary guidelines for farmers called "Good Agricultural Practices" for ensuring farm worker hygiene, a clean water supply, and appropriate handling of animal waste during production. Food processing plants are required to implement a Hazards Analysis and Critical Control Points (HACCP – pronounced "hassip") plan. Part of a HACCP plan is to examine the entire manufacturing or processing system to identify the most vulnerable points where contamination may occur and develop appropriate risk reduction, monitoring and intervention strategies. Local retail establishments are subject to local public health regulations, which are often based on Federal guidelines. Safe handling and food preparation practices on the part of the consumer are also essential for reducing food borne illness – both the FDA and the USDA provide extensive consumer information on their websites. We cannot rely solely on testing, because it is impossible to test all the food we import and eat, plus food is so diverse and manufacturing is so complex that risk cannot be completely eliminated.

Some risks are considered so substantial that federal and state laws prohibit certain practices. For example, in most states unpasteurized (raw) milk is prohibited or can only be purchased directly from a farmer. Milk that enters into the commercial pipeline must be pasteurized, a process by which milk is sufficiently heated to kill pathogens, like *E. coli* that may be present. Other processed foods and beverages (like juices) can also be pasteurized to reduce health risk. Irradiation is another process that could decrease the incidence of *E. coli* infections. Irradiation involves passing high energy beams through food. The beams can be produced in several ways, but the important point is that they pass completely through the food, damaging the DNA of living organisms so that they can no longer reproduce. Irradiation leaves no radiation or harmful chemicals behind. Depending on the beam dosage, insects, molds, and bacteria can be killed by this treatment, with minimal effects on the appearance and taste of food. As the CDC website on irradiation explains, the process is not a substitute for safe practices from the farm to the table any more than pasteurization can replace responsible milk production and handling (<http://www.cdc.gov/ncidod/dbmd/diseaseinfo/foodirradiation.htm>). Nevertheless, irradiation has the potential to dramatically reduce the number of cases of foodborne illness caused by *E. coli*.

What else could be done to reduce the incidence of disease due to *E. coli*? One intriguing idea is the development of vaccines against the toxin produced by disease-causing *E. coli*. This would protect people from the worst effects of virulent *E. coli* infections, while not harming the benign *E. coli* that live in the human gut. There are also vaccines that could be given to the animals to reduce the number of harmful *E. coli* they carry.

At every step of the path from the farm to the table, there are many ways to reduce the likelihood of *E. coli* from contaminating the food supply, and treating food so that contamination is eliminated before the food reaches the consumer. But the final line of defense – handling and cooking food properly, will remain the responsibility of the cook and consumer.

What types of foods are most commonly associated with *E. coli*, and why do there seem to be more cases of contamination recently?

E. coli live in the gut. If an animal or person is infected with a pathogenic strain of *E. coli*, the bacteria will be shed in manure or feces. Therefore, foods that are likely to come into contact with animal or human fecal waste, therefore, are the most likely to be contaminated, especially if they are not cooked; these include inadequately cooked beef, sprouts, raw cookie dough, raw nuts, raw milk cheeses, and raw fruits and vegetables.

Short of avoiding these foods entirely, it is impossible to reduce the risk of foodborne *E. coli* infection to zero, although safe food handling practices can reduce the risk substantially.

Epidemics caused by pathogenic *E. coli* are extremely newsworthy, which gives the impression that they are becoming more frequent. But surveillance by the CDC and state public health departments actually shows a marked decrease in the number of *E. coli* O157 cases in the last fifteen years (<http://www.cdc.gov/mmwr/pdf/wk/mm60e0607.pdf>). Better surveillance and detection of outbreaks means that more outbreaks are being identified, but the vast majority affect a small number of people. Rapid detection and investigation of outbreaks with better public health surveillance, more microbial testing, and increased regulatory authority to recall products when *E. coli* O157 is detected have been identified as factors that have contributed to the decrease in the number of cases. Still, food borne infections continue to occur and some modern practices have been suggested as potential contributors to an increase in the risk of *E. coli* infections. Food products, including meat and fresh produce, mass produced by large manufacturers, are widely distributed and are increasingly likely to travel long distances from the farm – some are even from other countries where regulations, farm practices, and processing standards may be less stringent. At the same time, an increase in the consumption of ready-to-eat raw, organic, and local produce complicates efforts to guarantee the safety of our food. In addition to these fears of potential increased risk of exposure to *E. coli*, there are concerns that increased travel and trade, increased use of antibiotics, and intensive animal farming practices could contribute to the emergence of new strains of *E. coli* that might be more virulent or resistant to one or more antibiotics.



Most experts agree that food safety is better than ever, but we must remain vigilant to protect our food. *E. coli* has not been domesticated. There are still “wild” strains that have the capacity to cause illness and death and we should expect new strains to emerge that will continue to threaten our health and the safety of our food. Despite this, it is important to remember that these are not new challenges - *E. coli* has accompanied humans and larger animals for millennia. It has become an important part of our gut and, much more recently, a remarkable tool for scientific study. We cannot predict how *E. coli* will impact mankind in the future, but we know that it will always be with us.

FAQ

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