

## Weighing the Evidence

### Part I. A Guide to Thinking about *E coli* O157:H7

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Since the Jack in the Box *E coli* outbreak in 1993 a lot of research and discussion about the bacterial cause, *E. coli* O157:H7, has filled the scientific literature and media. These fact sheets are to help sort the myths from the science, particularly when discussing pre-harvest controls in cattle. Part I is fairly generic and discusses the bacteria, why it is unique, how it was discovered, and where it likes to live. The other two parts are devoted to how we can control specific *E coli* O157:H7 shedding from cattle, a reservoir of these organisms. If you would like more detailed information on how cattle feeding might affect their shedding of *E coli*, go to Part Two of this series. If you are more interested in other cattle control measures used to help reducing shedding, see Part Three.

#### Part I.

There are many, many different kinds of *E coli* but this one in particular has gotten a lot of attention in the last three decades because of its ability to cause diarrhea and kidney failure in people, particularly children, and its appearance in outbreaks of illness. Where did this bug come from? How did we come to know it? What has research on cattle provided in the ways to potentially control food contamination and the risk for human infection? We'll try to answer these questions and a few more from research and investigations funded by a grant from the US Department of Agriculture. For an excellent summary on general *E coli* facts for the public, the American Academy of Microbiology released an FAQ in 2011 entitled: "*E. coli*: Good, Bad, & Deadly" (<http://academy.asm.org/images/stories/documents/EColi.pdf>) that provides answers to the questions the public might have about this group of organisms.

#### How Did Bacteria Like You Get in a Place Like That?

*E coli* – (*Escherichia coli* as its given name) are everywhere and of so many different kinds; some good, and some not so good because they can cause human or animal illness. If we could see all the *E coli* around and in us, it would be astounding. Picture of all the places you've been and all the things you touch and *E coli* is there. Some *E coli* live within us as [commensal bacteria](#) and are part of normal digestive processes. Some live in our gut and may be protecting us from more invasive disease-causing bacteria. Coliform or *E coli*-like bacteria are used to identify fecal contamination of water sources, for example, because of



the large number that we, and other animals, excrete.

Our intestinal tracts are colonized by *E coli* just after birth, by the kinds of *E coli* that our mother has and what's in our environment. As an infant, most of the bacteria within our intestine are *E coli*. By the time we reach adulthood, there are about ten million *E coli* bacteria per gram of feces (but they account for only 0.1% (1 in 1000) of bacteria in our feces) – out of the about 5 billion total bacteria in our intestines. Importantly, we actually need some *E coli* to produce most of our vitamins B<sub>12</sub> and K. The differences between different strains of *E coli* come down to their genetics – what different proteins their DNA is capable of coding for.



But, other *E coli* we do not need nor want; such as the kind that hits the news. The strains of *E coli* causing most of the outbreaks we hear about, *E coli* O157:H7 and other STECs ([Shiga-toxin](#) producing *E coli*), can affect people in different ways and may come from animal or environmental reservoirs. But, different animal species have their own *E coli* problems to worry about and if, for example, cows read the headlines, they'd want to know about a different kind of *E coli*.



For example, within the first five days of a calf's life, they have to worry about an *E coli* called K99 which can result in diarrhea and death in calves but does not affect people and does not affect older calves. Why do we see different *E coli* causing disease in different species and at specific ages? It's all about the receptors for attachment of the *E coli* to the cells lining the gut, the ability of the *E coli* to invade the intestinal cells, or the ability of the immune system to keep the invasion at bay. For example, *E coli* O157:H7 does not cause disease in cattle but does in people... primarily because cattle lack the receptors for binding the toxin (that's [shiga-toxin](#)) in the gut. So, cattle are considered "tolerant" hosts for these bacteria. The problem is that cattle can carry these bacteria – with the usual area of colonization at the recto-anal junction (yes – the very end of the gastro-intestinal tract) – and shed them into their feces. It's not just cattle. Other animals, such as sheep and pigs can shed this organism as well.

The good news is that relatively few cattle actually shed the organism. The bad news is they can spread it to other cattle, or their feces can contaminate food or water that people ingest. However, *E coli* O157:H7 is not the major cause of foodborne disease in people. When it comes to human illness, the [Centers for Disease Control](#) shows that the top five reported causes of foodborne illness in people are: Norovirus (estimated 5 million per year), Salmonella (about 1 million cases per year), Clostridium perfringens (a little less than 1 million per year), Campylobacter (about 800,000 cases per year) and Staphylococcus aureus (about 250,000 cases per year). But, when it comes to the foodborne pathogens that result in hospitalization, the causes are Salmonella (about 20,000 cases), norovirus (about 15,000 cases), Campylobacter (about 8,000 cases) and *E coli* O157 (about 2,000 cases). Our major concern with people, though, is the effect it has on

young children with development of [hemolytic uremia syndrome](#) and kidney failure. Most researchers believe that anything that could reduce the fecal shedding by our largest source of animal protein – cattle – should reduce the numbers of cases that we see each year.

So what is it about cattle that have people so concerned? And, what are the control measures that could reduce shedding? (See Part II.)

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