

Chapter ?? Sent out for review

## ARE SCIENTISTS REALLY UNAWARE COLIFORM BACTERIA ARE KILLERS? ©

[Jim Bynum](#), VP, and Gail Bynum, Ph.D

Help for Sewage Victims

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EPA would have you believe coliform and fecal coliform are some form of benign bacteria that indicates there may be fecal contamination or disease causing microorganisms in food, water or sewage and sewage sludge. It would also have you believe there is some safe level of these microorganisms. While a good immune system will protect most people, it is possible to be infected and not know it. For the past 116 years medical scientists have been documenting that coliform, now called the Enterobacteriaceae family, are potential killers of animals and humans. Currently, they are responsible for approximately 50% of all hospital acquired infections. They can enter the body through cuts and scraps on the skin, nose, mouth, eyes, ears, or sexual organs. These bacteria also create endotoxins, enterotoxins, and exotoxins which may be very deadly. The coliform bacterial endotoxins attack the linings of the stomach and intestines and allow the bacteria to slip through the walls into the blood where the infection is carried to all major organs. Besides diarrhea, they may cause brain damage, strokes, heart attacks, aneurysms, blood clots, arthritis, abscesses in bone joints, cascading organ failure, (kidney, liver, lungs, etc.), shock and death.

In reviewing the large body of literature, it is virtually impossible for anyone to believe regulators and scientists involved in the water and wastewater industry could not be aware coliform bacteria are disease causing organisms with the potential to kill humans as well as animals. On the other hand, some experts give out the impression that coliform is just the name of an EPA approved test. Coliform were originally known as *Bacillus coli* or *Colibacillus* and finally *Escherichia coli*. *E. coli* and similar coli-like-forms of colon bacteria became the family Enterobacteriaceae. These were the first recognized bacteria. They were also the first known cause of disease. *E. coli* is the primary coliform disease causing organism with several strains becoming antibiotic resistant superbugs. *E. coli* O157:H7 is not only antibiotic resistant, drugs given for treatment will actually cause it to be more devastating. While there is a debate about how many individual bacteria are needed to cause disease, the reality is that only one is needed that multiplies every 20 minutes. In the three to 7 day incubation period of a disease, one bacteria becomes billions. The [Statens Serum Institut](#) of Denmark has a strain bank containing over 60,000 clinical *E. coli* strains that can multiply every 20 minutes under the appropriate conditions. The Material Safety Data Sheet (MSDS) furnished to researcher states, "All *E. coli* are Hazard Group 2 bacteriological agents except *E. coli* O157:H7 [which is] (Group 3)". The Biosafety level groups are for laboratory reference only, because biological agents were never expected to be deliberately spread in communities under the guidance of federal agencies.

The most notorious coliform requiring Biosafety level group 3 laboratory practices is [Yersinia pestis](#) which causes the bubonic, pneumonic, and septicemic plagues. It is better known as the Justinian's Plague of the 6th century. It was known as the Black Plague of the middle ages which is estimated to have killed about 100 million people and the China plague of the late 19th and early 20th century which spread around the world killing upward of 12 million people. In the last few years drug resistant strains have surfaced. In 2005 there was an average of 13 plague cases annually in the Western states.

Yet, the Environmental Protection Agency ([EPA](#)) in referring to the aftermath of Katrina flooding in New Orleans states "coliforms generally do not pose a danger to people or animals, but they indicate the presence of other disease-causing bacteria, such as those that cause typhoid, dysentery, [hepatitis A](#), and [cholera](#)." As EPA defines them, "Coliforms are bacteria that live in the intestines of warm-blooded animals (humans, pets, farm animals, and wildlife). Fecal coliform bacteria are a kind of coliform associated with human or animal wastes. *Escherichia coli* (*E. coli*) is part of the group of fecal coliforms." Furthermore, in the basic information for the [Total Coliform Rule](#) EPA states, "When the news media announce a "boil water emergency," reporters often speak of a "total coliform violation." Coliforms are a group of bacteria, most of which are harmless. At first glance, it might seem strange that a harmless group of bacteria such as coliforms could cause such commotion. But like police tape

that a harmless group of bacteria such as coliforms could cause such contamination. But like police tape and chalk outlines, coliform bacteria are often found at the scene of a crime even though they are not themselves criminals."

Since [EPA claims](#), "Protecting human health is an integral part of EPA's mission", we can understand why EPA does not want to cause a national panic while it is promoting the spreading of these disease causing organisms on our food crops in sludge/biosolids, in our water as coliform and on our parks, school grounds and lawns in reclaimed water and compost. Typhoid (now known as enteric fever) is actually caused by the coliform *Salmonella typhimurium*. The case of Typhoid Mary (Mary Mallon) in 1906 was the first indication healthy humans could be carriers of disease causing organisms without any symptoms or illness. Now we know that many of us can carry chronic infections with little outward harm. As noted with the Black Plague, many can walk through the middle of a pandemic with total immunity.

Dysentery is caused by the coliforms *Salmonella* and *E. coli* as well as other bacteria such as *Vibrio Cholera* that don't show up in the coliform test. There are at least 30 clinically important coliform members of the [Enterobacteriaceae](#) family. They all release endotoxins, enterotoxins, and exotoxins which can cause breaches in the stomach lining, destroy stomach cells and cause a cascade of organ failures.

Despite spreading these known disease organisms around the environment, in our water and on our food, the EPA Office of Inspector General stated in a 2002 ["Status Report on Land Application of Biosolids"](#) that "EPA officials said investigating health impacts from biosolids is not an EPA responsibility; rather, they believe it is the responsibility of the National Institute of Occupational Safety and Health, the Centers for Disease Control, and local health departments." Not only that but, EPA's "Compliance and Enforcement" has disinvested from the program." In effect, EPA has refused, and still refuses, to investigate any human health effects caused by these disease organisms.

Yet, EPA allows drinking water treatment plants to fail 5% of the required monthly coliform tests. Moreover, EPA claims sludge/biosolids is safe on agricultural and grazing land if there are less than 2 million *E. coli* colonies at the end of the fecal coliform test. As we documented in the [previous chapter](#), the fecal coliform test has nothing to do with fecal contamination. The fecal coliform test only enumerates a few thermotolerant strains of *E. coli* and *Klebsiella* that show some activity at 112.1°F. Not only that but the coliform and fecal coliform tests suppress the growth of gram negative disease causing bacteria EPA does not want to find.

According to modern textbooks there are only three pathogenic types of *E. coli*. The three pathogen types cause, urinary tract infections (UTI), neonatal meningitis, and intestinal diseases (gastroenteritis). The first two types are caused by the common enteric *E. coli* that finds a way out of the lower intestine and colon where they can also damage other organs. There are five acknowledged types that may be involved in gastroenteritis, enterotoxigenic *E. coli* (ETEC), enteroinvasive *E. coli* (EIEC), enterohemorrhagic *E. coli* (EHEC), enteropathogenic *E. coli* (EPEC), and enteroaggregative *E. coli* (EAEC).

The Food and Drug Administration ([FDA](#)), states, "the term "coliform" was coined to describe this group of enteric bacteria", such as [E. coli](#), [Citrobacter](#), [Klebsiella](#) and [Enterobacter](#). Moreover, they are members of the "family Enterobacteriaceae, which includes many genera, including known pathogens such as [Salmonella](#), [Shigella](#), and [Yersinia](#)." Furthermore, FDA states in Chapter 4 of the Bacteriological Analytical Manual, "Although most strains of [E. coli](#) are not regarded as pathogens, they can be opportunistic pathogens that cause infections in immunocompromised hosts. There are also pathogenic strains of *E. coli* that when ingested, causes gastrointestinal illness in healthy humans. --- Coliform is not a taxonomic classification but rather a working definition used to describe a group of Gram-negative, facultative anaerobic rod-shaped bacteria that ferments lactose to produce acid and gas within 48 hours at 35°C. In 1914, the U.S. Public Health Service adopted the enumeration of coliforms as a more convenient standard of sanitary significance."

Medical experts and microbiologists know coliform members of the Enterobacteriaceae family are disease causing organisms. McGraw-Hill's 2002 Concise Dictionary of Modern Medicine states Enterobacteriaceae are "distributed in nature in plants and animals, and are important pathogens," - "they cause 1/2 of all nosocomial infections in the US, most commonly by [Escherichia](#), [Enterobacter](#)

they cause  $\pm 1/2$  of all nosocomial infections in the US, most commonly by [Escherichia](#), [Enterobacter](#), [Klebsiella](#), [Proteus](#), [Providentia](#), and [Salmonella](#) spp; less pathogenic Enterobacteriaceae include [Citrobacter](#), [Edwardsiella](#), [Erwinia](#), [Hafnia](#), [Serratia](#), [Shigella](#), [Yersinia](#) spp." According to Western Kentucky University's [208 MICROBIOLOGY. SECTION 3. LABORATORY WEEK 12](#), "Members of genera belonging to the Enterobacteriaceae family have earned a reputation placing them among the most pathogenic and most often encountered organisms in clinical microbiology. These Gram-negative rods are usually associated with intestinal infections, but can be found in almost all natural habitats."

However, these are not the only coliform disease causing organisms. Medical studies find the disease causing coliform bacteria: *Averyella*, *Budvicia aquatica*, *Buttiauxella noackiae*, *Calymmatobacterium*, *Cedecea*, *Kluyvera*, *Koserella*, *Leclercia adecarboxylata*, *Leminorella*, *Moellerella wisconsensis*, *Morganella*, *Pantoea*, *Photorhabdus*, *Rahnella aquatilis*, *Tatumella*, *Xenorhabdus* and *Yokenella regensburgei*.

### Historical Studies Showing Coliform Are Disease Causing Organisms

In 1884, Dr. Eugene A. Darling, Harvard Medical School, reported on Escherich's experiment which found that *B. coli* from normal feces was fatal to rabbits and guinea pigs. Large amounts cause the animal to become drowsy, stupid and exhibit diarrhea symptoms. Twenty-four to 48 hours later they became paralyzed, comatose resulting in death. A quick death was generally from a bloodborne infection while some infections resulted in slow paralysis with death in 12 to 49 days. *B. coli* isolated from gastroenteritis was generally more virulent than from feces. It was thought that *B. coli* in low doses were rarely fatal to animals and should not be considered virulent under normal circumstances.

It has taken over 100 years to verify some of the medical observation made in 1899 by Albert G. [Nicholls](#), M.D., Demonstrator of Pathology, McGill University who reviewed the studies on kidney infection. He wrote, "Acute inflammation of the kidneys is now said to be the result of the following conditions":

1. Intoxication, e.g., from bacterial toxins [[exotoxins](#), [endotoxins](#), [enterotoxins](#), [neurotoxins](#), and toxic enzymes], alcohol, [lead](#), [cantharides](#) [[Spanish fly from blisterbeetles](#)], [phosphorus](#), [chlorate of potash](#), [salicylic acid](#), etc.
2. Complication of :—
  - (a). The acute infections, as. scarlatina [[Scarlet fever caused by group A Streptococcus](#)], smallpox [[Variola major or Variola minor viruses](#)], pneumonia [[bacteria](#), [viruses](#), or [fungi](#)], acute rheumatism [[bacterial infection](#), erysipelas [[skin infection](#)], endocarditis [[heart disease](#)], typhoid [[Salmonella infection](#)], diphtheria [upper respiratory tract illness caused by [Corynebacterium](#)], septieoema [[bacteria in the blood](#)], cholera [infection of the small intestine with [Vibrio](#) bacteria], [epidemic cerebro-spinal meningitis](#), [gastro-intestinal disorders](#), etc.
  - (b), Chronic diseases and cachexias [[wasting syndrome](#)]: arterial sclerosis [[hardening of the arteries](#)], [diabetes](#), [syphilis](#), pulmonary phthisis [[tuberculosis](#)], carcinoma [[cancer](#)], etc.

Among the bacteria which have been recently shown to produce acute nephritis [kidney diseases], are the *B. Typhi* [[Salmonella](#)], the diplococcus lanceolatus [former name for [Streptococcus pneumoniae](#)], meningococcus intracellularis [[Neisseria](#)], *B. Friedlanderi* [[Klebsiella pneumoniae](#)], [streptococcus pyogenes](#), [staphylococcus albus](#) and [aureus](#), and the *B. Coli* [[Escherichia coli](#)].

Of 140 cases of acute Bright's disease [kidney diseases], 70 per cent, could be traced to acute infections.

Further, all grades of severity exist, from a mild inflammation up to a true local suppurative [formation or discharge of pus] condition. The infection may be in some cases an 'ascending' one from the bladder, but more commonly a 'descending' one from the blood-stream.

It is usual to teach that the acute cases may become chronic, and that the cirrhotic kidney is an end-stage of the chronic parenchymatous nephritis, or is due to arterial disease, or again, to certain poison, as alcohol, gout, and lead. (The "primare Schrumpfnier" of the Vienna School. )

And again, cirrhotic [degeneration of] kidneys may occur in children, where there could be no question of arterio-sclerosis or chronic intoxications from mineral substances.

Of the 32 acute forms of various kinds, bacteria, generally the specific germs of the disease, were found in 28. The overwhelming proportion of positive results leads me strongly to the conclusion that in the vast majority of cases, if not in all, acute nephritis is due to the presence of specific microbes.

Of these one case, which was associated with Atrophic Cirrhosis of the liver, showed a few well marked minute diplococci with a halo. In two, one an alcoholic kidney, bacilli of doubtful nature were seen.

As to the nature of these diplococcus forms, it may be said that they are identical in appearance and size with the diplococci which Adami has found recently in the liver, associated with progressive portal cirrhosis, and which he has proved to be a variant of the colon bacillus.

He found diplococcus forms in all livers which stained a brownish hue and were probably dead forms, while in atrophic cirrhosis of the liver they were increased in number and stained well.

That the process in chronic nephritis with productive inflammation is due to an embolic [blood] infection, is strongly supported by the histological features in the sections I have studied.

In the great majority of the acute interstitial and acute mixed varieties, the areas of round-celled infiltration are to be found around the glomeruli or around the afferent vessels, and interlobular arterioles exactly as would be expected in an embolic infection. The same holds good for the chronic cases. In the arterio-sclerotic type, that the infiltration and proliferation is mostly confined to vascular districts needs only to be mentioned. In the early stages of the chronic diffuse nephritis one sees the inflammatory exudation in the same way about the afferent blood vessels, associated with connective tissue hyperplasia.

Clinical evidence then strongly supports the view that Chronic Bright's Disease, and indeed Acute, may be a result of some long-standing gastro-intestinal disorder, 50 per cent, of cases giving this history, thirty per cent, of cases are insidious in onset, all the usual causes being absent: such might be called " Cryptogenetic forms." Can these be due to an infection from the intestine ?

Macaigne, (Arch.Gen.de Me"d., Dec, 1896,) has published some important experimental observations. He has found that B. Coli derived from the healthy intestine is harmless in the abdominal cavity, but it becomes virulent if there is some disorder of the intestinal tract as diarrhea, constipation, strangulation, etc. He could produce nephritis in animals by intravenous inoculation with B. Coli but usually obtained a suppurative form.

Anything then which causes a loss of the lining epithelium of the intestine with increased virulence of the germ, provides the starting point

for a systemic infection. That this often happens is beyond doubt. The occurrence of pneumonia due to colon infection is well recognized in strangulated hernia, and in septic peritonitis due to the same germ, the bacillus coli has been found in all the organs of the body including the kidneys.

In the light of the present study we get an entirely new conception of the process at work in the case of Bright's Disease. All cases, acute and chronic, are brought into the category of 'infections.' The nature of the infecting germ varies; in the acute forms it is usually the specific germ causing the primary disease although in some cases it is the colon bacillus. In the chronic cases, in the great majority, it is the colon which is the infective agent, but there is some evidence to favor the view that a few germs like the bacillus Pfeifferi and the diplococcus lanceolatus are capable of producing fibrosis. Two processes are at work, parenchymatous degeneration and productive inflammation. Parenchymatous degeneration alone is not to be regarded as a true nephritis, but is the result of chemical and bacterial toxins, bringing about injury to the secreting epithelium. Whether inflammatory infiltration occurs in addition or not depends on several factors.

1st, the number and size of the infecting germs.

2nd, the degree of virulence.

3rd, their specific qualities.

If the germs are few in number and of small size, they may pass through the glomerular capillaries, and merely produce degeneration and necrosis without further change. If they be sufficiently numerous to block the vessels or get into the capillary endothelium, then we get local inflammatory reaction with acute leucocytic infiltration.

[Veranus A. Moore](#), State Veterinary College, Ithaca, New York, stated in a 1902 Committee report that:

"(1) Many cultures of B. coli communis are fatal to guinea-pigs of 900 grammes weight when inoculated subcutaneously in doses of 1/4 to 1/2 Cc. of a fresh (24-hour) bouillon culture. Other cultures require the peritoneal injection of a like quantity of the virus for fatal results. (2) Some cultures do not produce morbid changes when injected in doses of 1 Cc. of the culture into the peritoneal cavity. (3) Cultures that have been isolated from the intestines of dogs have, in the writer's experience, been more virulent than those obtained from the normal viscera of other animals. (4) Although the colon bacillus appears to have become localized in the digestive tract of living animals, (including man) and to that extent become parasitic, it is not necessarily virulent as determined by animal inoculation."

The report include tables for the reaction of B. coli from different animals their and their fermentation of sugars and milk.

[Joseph Coats](#), Professor Of Pathology In The University Of Glasgow reported in 1900 that while B.coli was nonpathogenic in the colon, when the intestinal wall was penetrated B. coli caused an infection in the thin membrane (peritonitis) that lines the abdominal wall that covers the internal organs. It was also thought the peritonitis might be a result of an infection moving from the vagina and uterus. Coats noted B. coli was often found in the liver and suggested it was one of the causes of cirrhosis.

Dr. [Edgar Macharg](#), Late Senior Assistant Physician, Belvidere Fever Hospital, Glasgow, relates his findings in 57 cases of childbed fever that lasted over 24 hours. He noted that 31 patients died, while only 26 recovered. Many infections were due to rupture and/or injury of tissues during childbirth. Infections included: inner lining of the uterus; infections within blood vessels; Meningitis --inflammation of the membranes covering the brain and spinal cord; the uterus lining, fallopian tubes, or ovaries; inflammation of the small intestine; abscess; tissue that lines the wall of the abdomen and covers the abdominal organs; and connective tissue adjacent to the uterus. [Bacillus coli](#), [streptococcus pyogenes](#), [staphylococcus pyogenes albus](#) were the primary bacteria found

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Dr. [T. Gillman Moorhead](#), Dublin Hospital, reviewed the history of B. coli group of colon bacteria in 1905. He noted that Escherich had described the bacteria in 1884 and shown that B. coli toxemia could be fatal to animals. However, Escherich thought it was harmless to man. Yet, by 1887, other doctors had found that B. coli was pathogenic in wounds. Four years later colon bacteria had been a proven cause of (1) severe gastrointestinal disturbances, diarrhea, inflammation of the colon, and other organ injuries; (2) inflammation of the stomach lining; (3) infected bile ducts; (4) urinary and bladder infections; (5) lung diseases; (6) infection of heart and valves; (7) infections of the brain and its lining; (8) inflammation of bone joints. By 1905, the surgeons list had expanded with the addition of (9) inflammation of the pancreas; (10) inflammation of the mammary gland (humans) or udder (animals); (11) ear infections; (12) infections of the eyes; etc.

By 1906, Bacillus coli communis is acknowledged by the [United States Supreme Court](#) to be an index of fecal contamination in MISSOURI V. ILLINOIS, 200 US 496. Missouri was attempting to stop Chicago from dumping sewage in a water way leading to the Mississippi River. Judge Holmes opinion states that since both Missouri and Illinois dump sewage into the Mississippi River, each is responsible for cleaning up their drinking water supply. Otherwise, states downriver from Missouri would have a suit against Missouri as well. As we documented in the [previous chapter](#), the test for Bacillus coli incubated the sample for 24 - 48 hours at the optimum growth temperature of 37°C (98.6°F).

At about the same time Christiaan Eijkman suggested in 1906 that the only B. coli in feces from warm blooded animals of any sanitary significance would grow when incubated at 46°C (114.8°F). However, the test for the total B. coli and other coli-like-forms of bacteria incubated at 37°C (112.1°F) was adopted by the Public Health Service in 1914 and became known as the coliform test. The high temperature Eijkman test was accepted by the Public Health Service in 1964 with the stipulation the sample would be incubated at a temperature of 44.5°C (112.1°C) and it was renamed the fecal coliform test. This was only to be used as a field confirmatory procedure as some fecal bacteria would be missed ([Salmonella](#), [Shigella](#), etc.) and some nonfecal bacteria ([Klebsiella](#)) would be included. The Public Health Service warned that "it is necessary to consider all fecal coliform organisms as indicative of dangerous contamination." The current confirmatory procedure is to verify the presence of E. coli and disregard all other coli-like-forms as well as all other noncoliform pathogenic bacteria. It would be another 15 years (1979) before scientists discovered that less than 5% of the E. coli would show minimum growth at the higher temperature.

In 1909, Dr. [L. Van Es](#), Agricultural College, ND, read a paper on Colibacillosis at a meeting of the Minnesota Veterinary Medical Association. Colibacillosis was first described in 1799. By 1892, scientists were focusing in on the normal intestinal B. coli as the primary disease causing organism for which a vaccine was created. However, it was also noted that a number of bacteria species could also cause fatal cases of diarrhea in calves. It was understood that B. coli was generally harmless because it could not pass through the normal mucosa membranes that line the stomach and intestine, also called epithelial cells. If there is any damage to this lining, bacteria can pass through causing infection in blood and other organs. Since the mucosa lining is not fully formed in new born humans and animals and there is little formation of gastric acid, bacteria can easily enter the blood, heart and spleen. It was noted that a vaginal infection could be passed on to the calf as well as infection of the udders (e. g. mastitis). It is also possible for the infection to pass through the stump of the umbilical cord. Even boiled milk could effect the mucosa and cause a fatal infection. Once the infection enters the blood the fatality rate is about 80%. Treatment does little good. Surviving calves may suffer a relapse and fail to thrive normally.

The [Lawrence, Kansas Daily World](#) for Oct. 7, 1910 reported a Typhoid outbreak that caused one death and 10 people suspected of having Typhoid fever. The typhoid bacillus Eberth was found in the filth (manure, open toilets, slaughter products, etc.) in the infected ravine. Thirty private drinking water wells were closed as some were within five feet and many were within 10 feet of the ravine. Authorities initiated a cleanup and threatened to pour kerosene in the private wells to prevent their use, after another 20 drinking wells were closed. Karl Joseph Eberth had described the Typhoid bacteria in 1882. Very seldom is the term Typhoid used any more since the name of the bacteria was changed from Eberth to Salmonella typhi.

Lt. [C.E.H. Milner](#), 4th London General Hospital, reported on a meningitis patient wounded in France in 1915. Splinters from a high explosive shell fractured the soldier's skull. While the wound had mostly healed by the time he was brought to England, the soldier developed a high fever and there was a small amount of cerebrospinal fluid still draining out. Milner recovered pure colonies of *B. coli* from the spinal fluid, which he used to create a vaccine. Three days later the soldier's temperature dropped and he had a normal recovery.

[Alexis Thomson](#) and Alexander Miles' 1921 Manual of Surgery states *B. coli* invades any organ or tissue where vitality is lowered. It causes localized lesions, cystitis, peritonitis, peritoneal suppuration (or the formation of pus), appendicitis, also abscesses (localized collection of pus) in kidney, suppuration in bile duct and liver, wounds have a fecal smell from putrefaction. *B. coli* is one of less recognized bacteria that forms pus.

[W.A. Newman Dorland](#) updated "The American Illustrated Medical Dictionary" in 1922 with the following list of diseases for Escherich's bacillus - (Theodor Escherich, German physician, 1857-1911). The Bacillus coli. [*E. coli*]

- colibacillary -- produced by *Bacillus coli*
- colibacillosis -- infection with *Bacillus coli*
- colibacilluria - presence of colon bacillus in urine
- colibacterin -- vaccine made from killed *Bacillus coli* used in treating cystitis, catarrhal jaundice and various local infections.
- colicystitis - cystitis dependant on the presence of the colon bacillus. [inflammation of the bladder]
- colicystopyelitis - inflammation of the bladder and Kidney due to colon bacillus
- coliform -- 1. Cribiform, 2. Resembling the *Bacillus coli*.
- coli-group -- A group of bacteria, including *Bacillus coli*, the paracolon bacillus, typhoid bacillus [e.g. *Salmonella typhi*], paratyphoid bacillus and bacillus of psittacosis -- influenza of parrots that infects humans caused by *E. coli* or streptococcus -- paratyphoid fever -- a continued fever with symptoms identical with those of typhoid fever but with the Widal reaction negative. It is caused by a bacillus intermediate in form [e.g., *Salmonella paratyphi*] between the typhoid bacillus and the colon bacillus -- paracolon bacillus.
- colysin - A lysin formed by *Bacillus coli*. [toxin]
- colipyelitis - Pyelitis due to *Bacillus coli* [Acute inflammation ascending from kidney]
- colisepsis - [blood inflammation] infection with *Bacillus coli*
- colitis - inflammation of the colon
- amebic c. - colitis due to ameba coli
- mucous c. -- a disease of the mucous membrane of the colon, marked by colic, constipation, or diarrhea and the passage of mucous and membranous shreds.
- colitoxemia - Toxemia due to infection of colon bacillus.
- coluria -- Presence of the *Bacillus coli* in the urine

gas or gaseous gangrene -- gangrene in which hydrogen sulphid or other gas is formed in subcutaneous tissue, due to the action of *Bacillus aerogenes* [[Entrobacter/Klebsiella](#)] or *Bacillus perfringens* [[Clostridium perfringens](#)].

In a 1930 paper, Dr. [K. Douglas Wilkinson](#), reported that [Bacillus coli](#) was a common etiological agent of urinary tract infections. Moreover, the symptoms were misleading and difficult to diagnose. There are three stages of infections, acute, subacute, or chronic inflammation. The acute stage may be mistaken for meningitis or pneumonia. The infections are often mistaken for appendicitis or abdominal tuberculosis. He noted the infections could appear in the first week of life with the most fatalities occurring to children under 2 years old. The greatest number of infections in boys and girls was between the ages of 2 and 12. Out of 117 childhood cases studied, 7 of the nine (7.7%) deaths were girls under two years of age. Wilkinson recognized the ascending nature of the bacterial infection into the bladder. More importantly, he found that some cases did not respond to treatment which led to kidney damage and cardio-vascular damage. It is surprising to find that *B. coli* will actually grow at a pH of 9.0. Urinary tract infections are often associated with gastroenteritis and chronically inflamed appendix. Wilkinson was adamant that urinary tract infections were not associated with bloodborne infections which included meningitis, pneumonia, empyema, otitis media, acute infective enteritis and jaundice. In adult women a urinary tract infection is common during pregnancy and especially after

the birth of a child. It is also common during or after a honeymoon as well as retaining urine during a long trip without going to the toilet. It was pointed out during the discussion section of the published paper that while streptococcus and staphylococcus was a major cause of kidney disease, B. coli rarely did. However, it was not rare for young calves to developed B. coli kidney infections.

In 1934 study, [Edith E. Nicholls](#) reported on the nature of hemolytic and nonhemolytic [Bacillus coli](#) found in healthy individuals. Hemolytic means the bacteria break down red blood cells. She found that in appropriate doses, both types of bacteria killed white mice. Greater numbers of hemolytic bacteria were more likely to be found in people with "diarrhea or colitis." However, time and temperature could cause the bacteria to lose the hemolytic capability. According to Nicholls, "Fifty to one hundred per cent of the specimens, from each individual, showed hemolytic Bacillus coli." The specific finding was, "The hemolytic strains of Bacillus coli recovered from stool specimens were found to be only slightly more virulent for white mice than were the nonhemolytic."

According to Anna Dean [Dulaney](#) and I. D. Michelson, Medical School, University of Tennessee, Memphis, An outbreak of neonatal diarrhea occurred at the Memphis General Hospital during the winter of 1933-34 killing 47% of the babies. Symptoms appeared 5 to 9 days after birth. The babies did not respond to any medication available. The predominate organism found was E. coli along with Staphylococcus albus, and pneumococci. Strange colored stools contained slow lactose fermenting bacilli referred to as B. coli mutabile. Three type of colonies were cultured: B. coli colonies; enterococci; and the typhoid-dysentery group. The final observation was that the deaths might be caused by a bacteria yet to be recognized.

According to a 1940 study by Drs. [Byron D. Bowen](#), M.D., AND Ernest Witebsky, Medical Service of the Buffalo General Hospital, they had been involved with four cases of fulminating [Bacillus coli](#) septicemia, (e.g. sudden onset of severe infection in the blood). Three cases involved E. coli infection and death within 3 days of entering the hospital. The fourth case involved a blood infection by Bacillus Aerogenes Capsulatus ([Aerobacter](#) aerogenes, e.g. [Enterobacter](#) aerogenes and later [Klebsiella](#) aerogenes) and [Enterococci](#). According to their study, Septicemia caused by B. coli had only been reported 110 times in the last 50 years. The death rate was 30 to 40 per cent. They noted that while the textbooks described a clinical picture of blood infection by other bacteria such as delirium, stupor and finally coma., they did not describe the same features for E. coli. The theory was that the weak immunity of diabetics allowed a sudden invasion of bacteria from a urinary tract infection. In this study all were obese diabetic subjects.

P. N. [Coleman](#) and S. Taylor investigated the bacteria associated with urinary tract infection in 1949. They found Bacteria aerogenes was resistant to sulphanilamide and to penicillin. They found that six, out of 18 Bacteria aerogenes strains incubated at 44°C did not produce produced gas at 24 hours, but they did produce gas at 48 hours. They also found P. vulgaris, P. morgani and three paracolon anaerogenic strains. Where there was no damage to the urinary tract, E. coli was the predominate organism though there were 12 other organisms involved. Where surgery or some medical condition was involved, E. coli was uncommon and replaced by P. vulgaris, P. morgani, and Bact. aerogenes. When looking at 200 clinical cultures in 1929 from all types of infections 100 involved E. coli, 79 involved [B. aerogenes](#) and some [proteus](#) group. It was opined that E. coli was the most successful bacteria at invading tissue.

In 1954, Dr. R. [Meyer](#) reported on an abscess (pus pocket) in the knee of an 18 year girl. There was no history of the girl being infected by E. coli. Meyer found a pus sac in the knee joint. Pure E. coli cultures were grown from the pus. The E. coli was resistant to streptomycin and pencillin. However, it was sensitive to cholomycetin. He noted the literature suggested that while abscesses, conjunctivitis and invasion of body tissues had been observed, they were unusual.

[Donald G. McKay](#) and associates at the Departments of Pathology, Obstetrics and Gynecology, Harvard Medical School, Boston, and the Pathology Laboratory, Free Hospital for Women, Brookline, Mass. did some of the first work to understand the potential for endotoxins to cause blood clotting in 1958. According to the textbooks, Endotoxins are part of the outer wall of all gram negative bacteria, including those that are not generally considered pathogens. As the bacteria grow, some of the cell wall endotoxins are released into the body as antigens, which may cause inflammatory responses. Endotoxins are less potent than exotoxins which are released by some bacteria when they

die, which could be lethal as they could damage the stomach lining, disrupt nerve and cell functions or kill the cells. The symptoms include fever, inflammation, blood clotting, hemorrhaging and even toxic shock. Since this information was not known in 1958, McKay's work focused on whether endotoxins had a direct or indirect effect on causing blood clotting. He found that as little as 0.005 mg/ml of purified endotoxin shortened the clotting time by 20%. At 1.0 mg/ml the clotting time was cut in half. An unusual aspect of the research was finding that endotoxins did not cause clotting in hemophiliac blood (hereditary non-clotting defect) when the sample was contained in glass, but it did when the sample was contained in a silicone container.

Sidney [Gaines](#) and Joseph G. Tulley, in 1961, investigated the role of the endotoxin called "O" antigen might have on the virulence of the typhoid causing bacteria, Salmonella. They noted the virulence of the "O", like the Vi antigen was well known at that time. They also noted the smooth cell type bacteria with "O" antigen was more virulent than the rough type cell with little or no "O" endotoxin in the typhoid bacteria. This is one of the few studies that identifies antigens as endotoxins.

In 1963, [Claude DE LA Vaissiere](#) and Bernard Goiffon described a procedure to use a fluorescence microscope to quickly diagnose toxic gastroenteritis due to pathogenic colibacillus in infants. They noted that the test used for identifying Escherichia coli enteropathogens usually took 24-48 hours and the diagnoses is not normally received for another 2 days. They did acknowledge some E. coli did not show up in the test.

Clive C. [Gay](#), University Veterinary Hospital, University of Glasgow, discussed Escherichia coli and Neonatal Disease of Calves in a 1965 study. We find that colibacillosis is the term used for "calf scours" and an acute septicemias infection (invasion of the bloodstream) caused by the coliforms Escherichia coli and Salmonella as well as by Diplococcus, Pasteurella, and Streptococcus. Gay said by the time the calf is 24 hours old the gut "flora is comprised mainly of E. coli, Streptococcus spp., and Clostridium perfringens, with Lactobacillus and Bacteroides species" Gay showed that the gut flora of calves failing to receive the globulin fraction of colostrum was over run by E. coli causing their death. Any calves that survived failed to grow normally. However, he acknowledge little was known about how E. coli produced three different types of disease syndromes in calves. One interesting point made was that K antigens (endotoxins) are extremely heat stable. L-type and B-type K antigen take 100°C heat for 1 hour to be destroyed, while A-type K antigens require a temperature of 121°C for 2 1/2 hours. Some E. coli have more than 1 type of K antigen.

One dose of the antibiotic Bacitracin was shown to kill more than 80% of guinea pigs in a 1966 study by W. Edmund [Farrar](#), Jr. and associates at Walter Reed Army Institute of Research. Gram positive bacteria dropped 2,000 fold in the first 12 hours. Gram negative coliform bacteria increased 10 million-fold, from less than 100 per gram to over a billion per gram within 48 hours. Coliform bacteria in the blood was evident in 40% of the animals killed between 72 and 96 hours. The coliforms found were: E. coli, Klebsiella-Aerobacter, and paracolon organisms including [Proteus](#). An unusual finding was that high doses of Bacitracin actually inhibited the growth of coliform for about 24 hours.

Howard University infectious disease experts, [Vinod R. Mody](#) and associates, compared the changing nature of bacterial hospital infections over a 10 year period at Freedmen's teaching hospital in 1968. The based period was for adult infections during November and December 1965 and January 1966 compared to the same period in 1955-56. Over all, there was little change in the total number of infections. There was a 14% increase in gram negative coliform bacterial infections during the 10 period for a total of 56%. There was also a 14% drop in gram positive infections. The biggest drop was in the gram positive [Staphylococci](#) and [Streptococci](#) infections. The largest increase was gram negative E. coli infections. They evaluated the gram negative bacterial infections in 320 patients with the following results.

Organisms	Diabetes %	kidney - ureters %	High blood pressure %	Pregnancy %	abnormal growth of body tissue %	cerebrovascular - arteriosclerotic heart disease %	Pneumonia %	Cirrhosis %	Septicemia %
<a href="#">E. coli</a>	40	69	48	62	53	64	18	75	33

<a href="#">Klebsiella - Enterobacter</a>	28	18	41	19	21	9	27	25	50
<a href="#">Proteus species</a>	28	9	22	19	21	27	55	0	0
<a href="#">Pseudomonas aeruginosa</a>	7	4	1-	0	5	1	0	0	17

### **Modern Studies Confirming the Historical Studies -- After EPA Was Created**

Gram negative coliform bacteria contain inflammatory causing antigens which cause the immune system to produce antibodies in an attempt to clear the foreign substances (e.g., bacteria, fungi, parasites, viruses, toxins and other chemicals). [James W. Smith and Bertil Kaijser](#) published their research on E. coli K and O antigens spread through the blood in 1976. They focused on infections of the kidney and the ureters. They found both local and serum antibody response was much less to K-antigens than it was for O-antigens. Furthermore, K-antigens produced local immune responses in less than half the animals infected. It was noted that antibodies to O-antigens developed in ascending kidney infection, but did not develop in bladder infections. Moreover, the antigens remained in the kidney long after bacteria could no longer be cultured. However, they were not sure how this effected kidney destruction. The unusual finding was that E. coli urinary tract infection contained more K-antigens than stools, or bladder infections, from the same patients.

In 1976, [M.S. Schiffer](#) and associates reported there are 3 types of antigens (O, H, K) produced by E. coli. E. coli with the encapsulated (smooth) K-antigen causes 80% of neonatal meningitis cases and 40% of the bacterial blood infections. They noted that more K-antigen strains were found in kidney tissue than other types. Research also showed that smooth type K-antigen strains more likely to cause a fatal infection than an unencapsulated (rough type) strains that were recognized by the immune system and cleared from the blood. It would take another 27 years before K.J. Kim and associates discovered the smooth type K-antigen strain could be enclosed in a membrane bubble and passed through the blood-brain barrier without being recognized by the immune systems.

Jane Pitt, Columbia University College of Physicians and Surgeons, reviewed 137 cases of adult patients with E. coli in their blood in 1979. She found that the average age of those with the E. coli K1 antigen in their blood was 54, while the average age of those with other antigens in their blood was 61. While none of the people with the E. coli K1 antigen in their blood died, 33% of those with other E. coli antigens in their blood did. Women were the largest majority in both groups.

Dr. Lee W. Riley and associates reported on the first documented outbreak of E. coli 0157:H7 in 1983. There was an unusual aspect to this E. coli strain, it was not invasive or toxigenic by standard tests. They noted the first sporadic case of hemorrhagic colitis was from a 1975 case. While they neglected to mention it, the 1975 case was a Naval Officer from Oakland, CA. Perhaps, it is only a coincidence that the Naval Biosciences Laboratory at Oakland was involved in the controversy over recombinant DNA used to create bacteria never before seen in nature, such as E. coli 0157:H7. This is just one of the antibiotic resistant coliform that will cause Hemolytic uremic syndrome (HUS) and acute kidney failure. It should be mentioned that antibiotic treatment may trigger a release of exotoxins that could cause a cascade of damage to organs and death.

The [Australian Veterans' Entitlements Act](#) of 1986 recognized that antigens are "toxins [e.g., Endotoxins, enterotoxins and exotoxins - which includes neurotoxins and cell killing Cytotoxins] and foreign proteins, or particulate such as bacteria and tissue cells" which causes extrinsic allergic alveolitis (Hypersensitivity pneumonitis) Extrinsic allergic alveolitis is associated with a restrictive pattern of respiratory function tests." Antigens sources of interest here are:

- Detergent powder
- [Sewage sludge contaminated with micro-organisms](#)
- [Sauna water contaminated with micro-organisms](#)
- [Fertilizer contaminated with micro-organisms](#)
- [Compost dust contaminated with micro-organisms](#)

The [Canadian Centre for Occupational Health and Safety](#) fact sheet on extrinsic allergic alveolitis illustrates why it is difficult to prove harm from the disease causing organisms and their toxins in

reclaimed water, sludge/biosolids and sludge/biosolids compost. Examples of different names for Extrinsic Allergic Alveolitis disease caused by antigen toxins: "Sewage sludge disease; Wheat weevil lung; Farmers' lung; Animal handlers' lung; Cheese washers' lung; Bagassosis; Hot tub lung; Air conditioner lung; Bird fanciers' lung; Maltworkers' lung; Maple bark strippers' disease; Mushroom workers' lung; Wood pulp workers' disease; Sequoiosis; and Sequoiosis."

Dr. [Edwin A. Deitch](#) Department of Surgery, Louisiana State University Medical Center, read the paper, "[Endotoxin Promotes the Translocation of Bacteria From the Gut](#)", before the 6TH ANNUAL MEETING OF THE SURGICAL INFECTION SOCIETY, CHICAGO, APRIL 21-22, 1986-PART II. The paper was published in the Archives of Surgery in 1987. This was followed up in 1989 when [Dr. Edwin A. Deitch and associates](#), further documented that endotoxins in the gut allows bacteria to translocate through the stomach wall into the mesenteric lymph nodes. While [E. coli](#) is the primary bacteria found to translocate through the stomach wall to the lymph nodes, [Proteus](#), [Enterococcus](#), [Pseudomonas](#), and [Staphylococcus](#) have also done so. When the mucosal barrier of the stomach wall is breached by bacteria they may enter the blood and travel to other organs leading to sepsis (i.e., acute inflammation reaction throughout the entire body which may cause a cascade of organ failures.). Any amount of E. coli endotoxin above 0.25mg killed 100% of the mice in the tests. E. coli endotoxin injections of 0.05 mg was not lethal, but it allowed the mucosal barrier to be breached by bacteria in 50% of the mice. They documented that a non-lethal dose of E. coli endotoxin altered the mucosal barrier leading to multi-organ failure. Deitch said, "Furthermore, the combination of a nonlethal dose of endotoxin plus a protein-malnourished state or a nonlethal thermal injury results in lethal sepsis -- Endotoxin has a wide range of biologic actions, including the ability to modulate the immune system, increase vascular permeability, impair cellular metabolism and oxygen utilization, initiate disseminated intravascular coagulation, and produce profound hemodynamic changes resulting in hypotension and death."

In the 1989 proposed sludge regulation Part 257 et al. (503) EPA released a list of [25 primary pathogens](#) in sewage sludge. The list was removed four years later from the final regulation. The 1989 proposed sewage sludge regulation only mentioned five bacteria of which three (2-3-4) are coliform:

1. [Campylobacter jejuni](#) ----- Gastroenteritis.
2. [Escherichia coli](#) (pathogenic strains): -----Gastroenteritis.
3. [Salmonella sp](#) ----- Gastroenteritis and enteric fever [Typhoid]
4. [Shigella sp](#) ----- Gastroenteritis.
5. [Vibrio Cholerae](#) -----Cholera

Under the Resource Conservation and Recovery Act ([RCRA](#)) sludge/biosolids containing these infectious bacteria are classified as a hazardous waste because they may "(A) cause, or significantly contribute to an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness; or (B) pose a substantial present or potential hazard to human health or the environment when improperly treated, stored, transported, or disposed of, or otherwise managed."

The average person would accept EPA's implied implication that there is no serious health effects associated with the three coliforms. The implication is that the worse that could happen is an upset stomach or perhaps some diarrhea and associated fever. Research shows that enteric fever is really the benign name for Typhoid fever. If the disease is not properly diagnosed, Typhoid fever will progress to delirium, loss of mental capacity, intestinal hemorrhage, bowel perforation, and death. According to Dr. Arthur Diskin, Vice-President, Global Chief Medical Officer, Royal Caribbean Cruise Lines, gastroenteritis results in an estimated 100 million cases of acute diarrhea in the United States. Diskin states, "210,000 pediatric hospitalizations occur yearly, with as many as 10,000 deaths." Except for some laboratory cultivated strains, all E. coli are potential pathogens if they enter the blood system (blood poisoning -- spreading infections into other organs) or enter the body through the urinary system (UTI), mouth and nose (intestinal diseases -- gastroenteritis). More than 200,000 people die each year from gram negative and positive bacterial sepsis (septic shock) -- the body's exaggerated immune response to infections and foreign chemicals. Other than Typhoid, Salmonella causes many of the same diseases as E. coli. The same is true for Shigella. An unusual aspect of Shigella infection is that it may cause part of the rectum to be pushed out of the body. Permanent loss of bowel control may result.

Dr. [Tessy A. Joseph](#) and associates from Department of Pediatrics, Cook County Children's Hospital reported on the changes in infections of neonates with early-onset E coli infection born between 1982 and 1993. Their 1998 study showed a shift from a slow onset of E. coli ampicillin-sensitive bacterial strains infecting blood (sepsis), to ampicillin-resistant strains that developed quickly, was intense and severe to the point death (septic shock) could follow in the first 24 hours. They opined that this was a result of giving the mother ampicillin for fever during birth to prevent neonatal group B streptococcal disease.

[P. Brouqui](#) and D. Raoult reported in 2001 on "Endocarditis Due to Rare and Fastidious Bacteria." The gram negative bacteria they found were, the HACEK group of bacteria [Haemophilus](#) species (*Haemophilus parainfluenzae*, *Haemophilus aphrophilus*, *Haemophilus paraphrophilus*), [Actinobacillus](#) *actinomycetemcomitans*, [Cardiobacterium hominis](#), [Eikenella corrodens](#), and [Kingella species](#), [Campylobacter fetus](#), [Pasteurella](#) spp., [Brucella](#) spp., [Bordetella](#) spp., [Francisella tularensis](#), [Aeromonas hydrophila](#), [Streptobacillus moniliformis](#) and the coliforms, [Yersinia enterocolitica](#), [Salmonella](#) spp., and [Klebsiella](#) spp. They said, fastidious bacteria include: [Abiotrophia](#), HACEK group bacteria, [Clostridium](#), [Brucella](#), [Legionella](#), [Mycobacterium](#), and [Bartonella](#) spp.. Moreover, rare bacteria associated with heart disease are: [Lactobacillus](#) spp., [Klebsiella](#) spp., [Corynebacterium](#), [Salmonella](#), [Gemella](#), [Campylobacter](#), [Aeromonas](#), [Yersinia](#), [Nocardia](#), [Pasteurella](#), [Listeria](#), or [Erysipelothrix](#) spp.. Furthermore, slow growing bacteria such as the HACEK group, [Abiotrophia](#) spp., [Brucella](#) spp., [Bartonella](#) spp., [Legionella](#) spp., and [Mycoplasma](#) spp. may not show up in standard blood testing. The symptoms may suggest congestive heart failure, stroke caused by emboli clots and bronchopneumonia. Exposure may be oral (including dental), sexual, surgical, animals, employment and pests.

Ines [Niehaus](#) reported on a case of Parkinsonism caused by Lipopolysaccharides [endotoxins] of *Salmonella* Minnesota at the XIV International Congress on Parkinson's Disease in 2001. A 22 year old laboratory worker was exposed to the endotoxin through an open wound on the thumb. This resulted in a sepsis-like inflammatory reaction with flu-like symptoms and vomiting. Over time it progressed to chronic inflammation of the central nervous system such as slowed ability to start and continue movements, and impaired ability to adjust the body's position, rigidity, tremor, stiffness.

[Lei Wang](#) and associates from the School of Molecular and Microbial Biosciences, The University of Sydney, Australia sequenced all of the 53 forms of the E. coli H-antigens in 2002. They supposed the 43 forms contained the *fliC* gene from the common ancestor E.coli/Salmonella. It was opined that the other 10 forms were due to lateral transfer mutations. They note the H and O antigens were the best understood components of E. coli clones and had been used for blood serotyping since the 1930s.

In 2002, The European Council of Applied Sciences and Engineering, an independent non-profit organisation of national academies of Engineering, Applied Sciences and Technology from 21 European countries published a study by Dr. [Wolfram Martens](#) and Prof. Dr. Reinhard Böhm, Universität Hohenheim, on the public health aspects of the pathogens found in sludge. The primary pathogenic coliform were [Salmonella](#) spp.; [Shigella](#) spp.; [Escherichia coli](#); and [Yersinia enterocolitica](#). The secondary pathogenic coliform were [Escherichia coli](#); [Klebsiella](#); [Enterobacter](#); [Serratia](#); [Citrobacter](#); [Proteus](#); [Providencia](#). Most of these are Multiresistant bacteria.

It wasn't until 2003 that [K.J. Kim](#) and associates, Division of Pediatrics Infectious Diseases, The Johns Hopkins University School of Medicine, first demonstrated how, and why, the E. coli bacteria could cross the blood-brain barrier to cause meningitis. Basically, E. coli K antigens as well as *Salmonella*, *Mycobacterium* and *Legionella* are known to survive and multiply within a cell membrane bubble and are not recognized by the immune system. The E. coli K antigens are devastating killers of infants, both humans and animals, but can be cleared from the blood of adults with proper treatment.

Also, In 2003, [R. MICOL](#) and associates reported on 5 cases of native (heart) valve endocarditis (NVE) caused by E. coli. They found that older people could develop acute heart disease from a urinary tract infection which resulted in a high death rate. The E. coli were not a virulent clone, but the normal urosepsis E. coli (group B2, *papG* allele II, siderophores).

Michael D. [Wheeler](#) reported in 2004 on the science of endotoxins and liver disease. Endotoxins are the cell walls of gram negative coliform bacteria. Cirrhosis of the liver has long been blamed on

the cell walls of gram negative commensal bacteria. Symptoms of the liver has long been blamed on excessive alcoholic consumption, but Wheeler shows that when bacteria in the gut die, endotoxins are released which penetrate the walls of the gut and enter the blood stream leading to endotoxemia (i.e., adult respiratory distress syndrome (ARDS), hemorrhages, necrosis of the kidneys, and shock). . When the endotoxins enter the liver, Kupffer cells are activated. The Kupffer cells release chemicals leading to tissue damage in the liver.

Lidwien A.M. [Smit](#) and associates studied the effects of endotoxins on wastewater treatment plant workers who were only exposed 8 hours a day in 2005. They found three types of symptoms: "lower respiratory and skin symptoms"; "flu-like and systemic symptoms"; and "upper respiratory symptoms."

[Yufeng Yao](#) and associates, Division of Pediatric Infectious Diseases, Department of Pediatrics, School of Medicine, Johns Hopkins University, compared the gene grouping of 11 E. coli K1 antigen strains taken from the spinal fluid of neonatal meningitis patients in 2006. Yao said, "Escherichia coli is a major cause of enteric/diarrheal diseases, urinary tract infections, and sepsis." Sepsis is a term doctors don't use much anymore. It is bacterial blood infection that is generally referred to as a "complication" of infections that may start in any part of the body. The blood pressure will drop resulting in a toxic shock to the body interrupting the function of all major organisms and the central nervous system. Yao found that there were two groups within the 11 E. coli K1 antigen strains they investigated that caused meningitis (i.e., E. coli O1, O7, O12, O16, O18 and O45). Each group used a different mechanism. An unusual finding was that while the cytotoxic necrotizing factor 1 (cell killing protein toxin) was known to be involved in meningitis, only two K1 strains investigated included this protein toxin. Yao confirmed that the outer cell membrane of the gram negative Enterobacteriaceae (coliform) contained Lipoprotein. This cell protein causes an immune system proinflammatory cytokine storm which may lead to lethal toxic shock. The study notes mobile genetic islands as well as individual virulence factors can be translocated by lateral transfer to other bacteria.

[Martin Baumgart](#) and associates at Cornell University found in a 2007 study that only about 30% of the human fecal flora organisms could be cultured in the laboratory. The focus of the study was investigating the cause of chronic debilitating colon inflammation known as Crohn's disease. It has been accepted that the disease is restricted to genetically susceptible individuals. A number of bacteria have been implicated in Crohn's disease, but never substantiated. Part of the problem appears to be that different bacteria may inhibit the final section of the small intestine (ileum) and the lower bowel section (colon). Baumgart found that "a novel group of E. coli contains opportunistic pathogens" that may be the cause of Crohn's disease in the ileum. He said, the novel group is similar to "uropathogenic and avian pathogenic E. coli, and pathogenic Enterobacteriaceae" (e.g., coliform). The finding was based on the high numbers of novel invasive pathogenic E. coli found in the inflamed intestinal mucosa lining.

In 2008, [Jeffrey T. Borenstein](#), Director of the Biomedical Engineering Center at Draper Laboratory, reported on new information that bacterial clusters can trigger coagulation in the blood system. He noted that many bacterial infections from [Staphylococcus aureus](#) to [Escherichia coli](#) are known to produce blood clotting factors that can lead to sepsis, septic shock and organ failure. This is a special problem for those with compromised immune systems. His unusual finding was that bacteria evenly distributed in the blood system did not cause blood clotting. However, when the bacteria congregated and acted together, clotting factors were released and blood clots developed. It is interesting to note Borenstien did not review any endotoxin studies such as Dr. McKay's 1958 discussion on the potential for bacterial endotoxins to cause blood clotting.

According to [Isaac P Humphrey](#) and associates (2009), heart valve infections were the result of gram positive bacteria and the more virulent gram negative slow growing bacteria: "[Haemophilus species](#) (Haemophilus parainfluenzae, Haemophilus aphrophilus, Haemophilus paraphrophilus), [Actinobacillus actinomycetemcomitans](#), [Cardiobacterium hominis](#), [Eikenella corrodens](#), and [Kingella species](#)." They state this group was referred to by the first letter of each bacteria, HACEK. They also cause infections of: the mouth and gums, bacterial blood poisoning, lining of the stomach, middle ear, outermost layer of the eye, inflammation of the lung, arthritis, bones, urinary tract, wounds and brain abscesses. Humphrey noted 60 percent of the cases were a result of dental infections and most were subacute. However, the vegetation created by the bacteria may break loose causing blood vessel blockage leading to stroke, heart attack, kidney problems, bowel malfunction and focal pneumonia.

[Karen J. Vigil](#) and associates at the Medical School and School of Public Health, The University of Texas Health Center reported in 2010 that a new type of pus producing E. coli infection of the skeletal muscles was emerging as a serious problem worldwide. This is a fluoroquinolone-resistant, ESBL-positive E. coli. The disease has usually been caused by gram-positive bacteria such as [Staphylococcus aureus](#).

[Tarun Madappa](#), Department of Pulmonary and Critical Care Medicine, Elkhart General Hospital, and Chi Hiong U Go, Department of Internal Medicine, Texas Tech University Health Science Center at Odessa, outline some of the diseases caused by E. coli in a September 2010 study published online. Diseases include: enteric infections, Arrhythmias (Tachycardia), psoas abscess appendicitis, infectious arthritis, abnormally fast breathing (Tachypnea), brain abscess, confusion, diarrhea, viable bacteria in the circulating blood, infection of the biliary tract, inflammatory process of the bone and its structures, Central Nervous System manifestations, enterotoxin, endotoxin disseminated intravascular coagulation, fever, gastroenteritis, congestive heart failure infection of the endocardial surface of the heart, hemorrhagic colitis, hemolytic-uremic syndrome - kidney failure, hypotension, Jaundice, intra-abdominal infections, inflammation of the gallbladder, inflammatory reaction of the intraocular (eye) fluids or tissues, liver abscess, meningitis, maternal peripartum (vaginal) infection, neonatal tetanus, pneumonia, pulmonary embolism, prostatitis, respiratory tract infections, ruptured aneurysm, seizures, shaking chills, toxic shock symptomatic cystitis, inflammation of the sinuses, skin and soft-tissue (necrotizing) infections that kill tissue - interfere with blood flow - break down materials in tissue, inflammatory diseases of the thyroid gland, urinary tract infection, infection-induced inflammation of the urethra, infection of the umbilical stump, death.

In July 2010, Dr. Jae Myung [Park](#), reported on the follow up studies on the Long-term Prognosis of Postinfectious Irritable Bowel Syndrome from the massive 2000 acute gastroenteritis outbreak in Walkerton, Canada. The town's water well was contaminated with Escherichia coli 0157:H7, Campylobacter jejuni, and other pathogens. Over 2300 out of 4315 residents were infected. This resulted in 27 cases of kidney failure and 7 deaths. The authors concluded that acute gastroenteritis could trigger Irritable Bowel Syndrome symptoms which lasted over 8 years for 15.4% of those infected. However, that number may be low because only 53.5% participants returned for the last follow up of the study.

[Sania S. Raza](#) and associates recently (September 2010) reported on the changing nature of bacterial heart disease. In the past gram negative HACEK bacterial endocarditis was most often associated with injected drug use. While rare, but often fatal, the major gram negative bacteria involved were, "[Haemophilus species](#), [Actinobacillus](#), [Cardiobacterium](#), [Eikenella](#) and [Kingella](#)." More recently, [Pseudomonas](#) species and the [Enterobacteriaceae](#) family were also implicated in Non-HACEK bacterial heart disease. Gram positive bacteria actually cause about 80% of the heart valve infections. It was unfortunate but most early studies associated Non-HACEK bacterial heart valves with injected drug use. About 80% of these cases involved [Pseudomonas](#) and [Serratia](#) species. Besides the gram negative Pseudomonas, recent research show members of the gram negative Enterobacteriaceae family such as [Citrobacter](#) species, [Enterobacter](#), [Escherichia coli](#), [Klebsiella](#), [Proteus](#), [Providencia](#), [Salmonella](#) and [Serratia](#) as causative pathogens. These bacteria caused 30% of the infections in immune compromised individuals. Raza and associates also report that urinary tract infections were the major pathway to heart infections for elderly patients.

Brendon [Nafziger](#), DotMed, reported on a survey released 10/22/2010 by Rush University Medical Center and the Cook County Department of Public Health on the prevalence of Klebsiella pneumoniae carbapenemase-producing bacteria in Chicago hospitals. This deadly bacteria is resistant to virtually all antibiotics, except colistin and tigecycline. This strain of Klebsiella pneumoniae first showed up on the East Coast in 1999 and Chicago in 2007. Twenty-six Chicago hospitals reported Klebsiella pneumoniae carbapenemase-producing bacteria in 2009. The number of hospitals increased to 37 in 2010. The average number of infected patients per hospital increased from 4 to 10. Nafziger said it is even worse in New York hospitals where 30% of the hospital patient laboratory cultures are positive. Natural antibiotic sensitive Klebsiella pneumoniae is a coliform which generally causes pneumonia, sepsis (i.e., blood and whole-body inflammatory state) and urinary tract infections.

The [Environmental Protection Agency](#) employs over 17 thousand people with a budget of \$10.020 billion in discretionary budget authority. The focus of the 2011 budget, which started October 1st, is "developing common-sense steps toward clean air, addressing the climate challenge, protecting our nation's waters, cleaning up communities and ecosystems, and strengthening EPA's scientific and enforcement capabilities." A major part of the funding (\$4,896,505,000.00) is for: Protecting Human Health; Protecting Water Quality; and Enhanced Science and Research. There is another \$1,764,384,000.00 is to be used for: Preserving Land; Restoring Land; as well as Enhancing Science and Research, because, "Land is one of America's most valuable resources and cleaning up our communities to create a safe environment for all Americans is a priority for EPA. Hazardous and nonhazardous wastes on the land can migrate to the air, groundwater, and surface water, contaminating drinking water supplies, causing acute illnesses or chronic diseases, and threaten healthy ecosystems in urban, rural, and suburban areas." "Clean water is essential for our quality of life and the health of our communities."

It is self-evident with 17 thousand employees, a 10 billion dollar budget and a Congressional mandate to protect human health that EPA and associated scientist would know all about these studies and others showing coliforms are disease causing organisms in sludge and water. The only conclusion to be drawn is that EPA is more interested in transferring liability from industry and municipalities to farmers and the public at large. EPA's continued claim that coliform bacteria do not cause disease is a disservice to the public and proof that it has no interest in fulfilling its Congressional mandated responsibility to protect public health.

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