MYTH # 5

"**there is no evidence that we have failed to protect the public health and environment" (2006)**"
Wesley Carr, Environmental Protection Specialist
Colorado Department of Public Health & Environment
Water Quality Control Division
http://www.epa.gov/region8/water/biosolids/pdf/Biosolids%20Inspection.pdf

"**It's only when a strange alliance of the stars occurs you get an extraordinary event like this," (2011)**
Jim Gorny, a produce safety expert at the FDA.
http://news.yahoo.com/listeria-outbreaks-produce-rare-deadly-225335003.html

A Short History

Coliforms: Dangerous Biological Bioterrorism Agents

By Jim Bynum, vp 11/11/2011
Help for Sewage Victims

Edited by Gail Bynum, Ph.D

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Introduction

This paper examines thirty dangerous coli-like-forms of biological bioterrorism agents called coliforms and the claim that they indicate that food and water might be contaminated with fecal pathogens? State and federal agencies have a rather strange way of explaining away problems they cause by allowing the spreading of dangerous biological bioterrorism agents in our environment. Coliforms, or as Colorado Environmental Protection Specialist, Wesley Carr, calls them, fecal pathogens, are used to indicate pathogens such as Listeria in water or sewage sludge (aka biosolids) used on fruits and vegetables. Federal Food and Drug expert, Jim Gorney, thinks the Colorado cantaloupe out break was caused by a strange alliance of the stars, but that does not explain away the fact that these people have a federal list of specific dangerous biological bioterrorisms agents, including Listeria, known to be in water and sludge.

Seven species of coliforms and their serotypes are on the list of 48 species in the Health and Human Service's regulation 42 C.F.R. § 72.3 listed as dangerous biological bioterrorism agents. Another twenty-three coliforms meet the federal definition. Coliforms are a generic term for the family Enterobacteriaceae, which is the generic term for a group of gram negative pathogenic bacteria that ferment lactose to produce gas and/or acid within 24 – 48 hours when incubated at 95°F. While coliforms infect humans with an internal body temperature of 98.6°F, some scientists claim only thermotolerant coliforms that show some activity when incubated at 112.1°F for 24–48 hours are actually fecal coliform – that is coliform from human feces.

Coliforms cause 40-50% of hospital acquired infections and some have become superbugs. Yet, they are not considered to be a cause of disease by the food and water industry. There are 30 species of pathogenic coliforms (Aerobic Gram-Negative Bacilli: Enterobacteriaceae—Fermentative) that will be addressed in this paper before we get to the real long list of bacteria that can destroy your life, that coliforms are claimed to indicate, such as: Aerobic Gram-Positive Cocci; Aerobic Gram-Negative Cocci; Aerobic Gram-Positive Bacilli; Aerobic Gram-Negative Bacilli: Nonenterobacteriaceae—Fermentative; Aerobic Gram-Negative Bacilli: Nonenterobacteriaceae—Nonfermentative; Aerobic Gram-Negative Fastidious Coccobacilli; Mycoplasma (Pleuropneumonia-Like Organisms [PPLO]); and Treponemataceae (Spiral Organisms).

Any scientist, or writer, who claims coliforms incubated at 95° fahrenheit (F) are just indicators of potential fecal contamination in food or water is clearly uninformed or does not have your best interest in mind. A higher form of deception is practiced when a scientist, or writer, claims raising the

temperature another 17.1°F to heat shock the coliforms incubated at 112.1°F confirms fecal coliform contamination. That would only be true if you were looking for fecal material from desert dwelling animals such as the Mojave Fringe-toed Lizard which does live rather well with an internal body temperature of 112.1°F or the desert iguana whose internal body temperature has been recorded at 113°F.

The deception gets a little deeper. Heat shocking pathogenic coliforms such as Citrobactor, Enterobactor, Salmonella and Shigella at 112.1°F for 24 hours severely stresses the bacteria, which inhibits the biological activity, but does not kill them. Some E. coli (fecal coliforms) will show some minor activity at 112.1°F including: Uropathogenic E. coli (UPEC); Enterotoxigenic E. coli (ETEC); Enteropathogenic E. coli (EPEC); and Shiga-toxin producing E. coli (STEC). Klebsiella pneumoniae and Yersinia pestis (Black Plague) also meets the definition of a fecal coliform. Campylobacter species would meet the temperature requirements but it is not a gram negative bacteria that ferments lactose, therefore it is not a fecal coliform. These seem to be a little more than indicators of fecal contamination since they are all on the the list of dangerous biological bioterrorism agents.

But that is only the tip of the iceberg. In 2001, a review of the scientific literature identified 1415 species of infectious organisms known to be pathogenic to humans, including 217 viruses and prions, 538 bacteria and rickettsiae, 307 fungi, 66 protozoa and 287 helminths. Of these, 61% were zoonotic and 12% were associated with diseases considered to be emerging (Taylor, Latham & Woolhouse, 2001). http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1088493/pdf/TB010983.pdf

As you can see there are many known types of exogenous (external) and endogenous (internal) disease causing organisms, that given the proper circumstances, can kill us. Fortunately, most of us have never encountered the dangerous ones and many that did never realized it due to their good immune system. Unfortunately, children, the elderly and those with compromised immune systems have not been so lucky.

Background on Biological Bioterrorism rule

Twenty-eleven has been a rather interesting year for the bioterrorism and food safety industries with the enactment of new or modified laws to protect our food supply as well as some of the most deadly foodborne outbreaks on record. The Agricultural Bioterrorism Protection Act of 2002 was very expensive and a major failure. This led to the Food and Drug Aministration (FDA) Food Safety Modernization Act of 2011 which is already very expensive. It has spawned a new industry of food safety experts who have no idea they are dealing with listed biological bioterrorism agents. This too is doomed to be a total failure since some biological agents currently used as fecal indicators not only infect humans, but also animals, marines species, plants and even trees.

The really bad news is that in the past 30 years researchers have manipulatied the genetic make-up of bacteria creating antibiotic resistant pathogens out of what were once thought to be benign organisms in the Enterbacteriaceae family. Federal agencies who have watched the benign bacteria turn into pathogens and scientists within the food, waste and water industry would prefer you never find out that these are the coliforms. This is no longer just a concern for children, the elderly and immune compromised people. It puts everyone's health at risk.

In 2011 there were two foodborne outbreak events: the Escherichia coli 104:H7 outbreak in Germany

(51 deaths, 851 kidney complications, over 4,000 infections) caused by contaminated bean sprouts and the Listeria outbreak in Colorado (29 deaths, 129 documented infections) caused by cantaloupes grown near a field where contaminated sewage sludge had been disposed of as a fertilizer. The blame for the contaminated bean sprouts was placed on Egyptian bean seeds that at some point had been exposed to E. coli 104:H4. The blame for the contaminated cantaloupes was placed on a piece of second-hand packing equipment that had been previously used to wash and dry potatoes. Yet, no one brought up the subject of potential bioterrorism bacterial agents in either case.

The two outbreaks appear to have little in common. However, both bacteria are on the Public Health Service, Department of Health and Human Services' 2002 bioterrorism list of dangerous biological agents that have the potential to pose a severe threat to public health and safety, to animal health, to plant health, or to animal and plant products.

The second factor in common is that while E. coli is referred to as a coliform as well as a fecal coliform, it is used as an indicator that Listeria and/or other deadly pathogens might be present in food or water, even though E. coli may be the more deadly bacteria. E. coli is the primary member of the Enterobacteriaceae group (i,e., coli-like-forms) of bacteria which the Public Health Service adopted in 1914 as an indicator of fecal contamination in food and water. The scientific opinion at that time was that they actually had no sanitary significance. Today, we know that the term fecal indicators has no sanitary significance as most are dangerous biological agents and have no relationship to other dangerous disease causing agents.

Since federal and state agencies use the generic coliform or fecal coliform test as fecal indicators for drinking water as well as sewage sludge (aka biosolids) and recycled sewage effluent used on food crops, there is no way we would know if terrorists were poisoning our food and water. Any outbreak could simply be business as usual for municipalities as they put our children at risk by dumping the contaminated sludge and recycled water on grazing land, food crops, parks and school grounds as well as home lawns or mix it with our drinking water. Our food and water safety is based on allowing exposure to a certain level of biological agents (coliform and/or fecal coliform colonies) – reported as individual bacteria.

Thirty years ago in 1981, the Environmental Protecting Agency (EPA), Food and Drug Administration (FDA), and U.S. Department of Agriculture (USDA) created a federal policy to dispose of biological active agents in contaminated sewage sludge on fruits and vegetables. EPA, FDA and USDA states that the safety of food grown on sludge is assured as long as the guidance is followed. There is a caveat, the "government can not offer any indemnity against product recall, seizure, or other enforcement actions, -- However, the risk of such enforcement actions would be no greater than the risks associated with normal farming and processing practice."
http://thewatchers.us/EPA/1981-policy-EPA-USDA-FDA.pdf

In 2002, the three agencies were given responsibility for protecting the public and agriculture from these same dangerous biological agents used as fecal indicators and spread throughout our environment as a component of beneficial products. Two of the coliform fecal indicators (E. coli and Salmonella) have been responsible for some of the most expensive and deadly foodborne outbreaks including those in spinach and peanuts. This has led to the new food safety laws.

According to FDA, “The FDA Food Safety Modernization Act (FSMA) was signed into law by

President Obama on January 4th, 2011. It aims to ensure the U.S. food supply is safe by shifting the focus of federal regulators from responding to contamination to preventing it."

The problem no one wants to discuss is that the agencies focus on easily stressed pathogenic coliform indicators to prevent food and waterborne infections and disease. In fact, some disease organisms can not be cultured yet. P. C. Y. Woo, et al., pointed out the problem in their 2008 study, "Then and now: use of 16S rDNA gene sequencing for bacterial identification and discovery of novel bacteria in clinical microbiology laboratories." They said:

> "Despite extensive investigations, a microbiological cause cannot be determined in approximately half of the patients with infectious disease. For some clinical syndromes, such as neutropenic fever, no microbiological cause can be found in up to 80% of patients who are believed to be suffering from infective causes. For some other syndromes, e.g. acute gastroenteritis and community-acquired pneumonia, the cause was undetermined in c. 40% of patients. Over the years, tremendous efforts have been made to determine the microorganisms associated with these 'unexplained infectious disease syndromes'. The discovery of novel aetiological agents responsible for 'unexplained infectious disease syndromes' relies quite heavily on the description of novel microbes. Although standard or unusual forms of known microbes are sometimes considered to be novel causes of 'unexplained infectious disease syndromes', most of the novel causes are indeed previously undescribed microbes."
> http://onlinelibrary.wiley.com/doi/10.1111/j.1469-0691.2008.02070.x/full

The FSMA is doomed to failure because the FDA does not actually test food at the optimum temperature of 98.6°F where bacteria infect humans and animals. Rather than testing for the bioterrorism biological agents in food and water, some of which it names, it raises the temperature of the test to inactivate most of the coliforms by heat stress and looks for thermotolerant strains of the same biological coliforms. In reviewing the "Bacteriological Analytical Manual," the truth is self-evident when it states: "E. coli is a member of the family Enterobacteriaceae, which includes many genera, including known pathogens such as Salmonella, Shigella, and Yersinia. 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. – Although the concept of using E. coli as an indirect indicator of health risk was sound, it was complicated in practice, due to the presence of other enteric bacteria like Citrobacter, Klebsiella and Enterobacter that can also ferment lactose and are similar to E. coli in phenotypic characteristics, so that they are not easily distinguished. As a result, the term "coliform" was coined to describe this group of enteric bacteria. 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 h at 35°C (95°F). – Fecal coliform analyses are done at 45.5°C (113.9°F) for food testing, except for water, shellfish and shellfish harvest water analyses, which use 44.5°C (112.1°F). The fecal coliform group consists mostly of E. coli but some other enterics such as Klebsiella can also ferment lactose at these temperatures and therefore, be considered as fecal coliforms."
http://www.fda.gov/food/scienceresearch/LaboratoryMethods/BacteriologicalAnalyticalManualBAM/ucm064948.htm

It would seem the German Federal Environment Agency (UBA) is one of the few in the world who

actually explains what the term coliform means. In the document "Coliform bacteria in drinking water Recommendations on risk assessment and measures in the case of systemic contamination," it states:

> "The term coliform bacteria covers a broad spectrum of bacteria families and species, which belong to the family of enterobacteriaceae. These species and families differ considerably in terms of their pathogenic properties and virulence. As already described, bacteria of the family of enterobacteriacae are not only found in the intestines of vertebrates and invertebrates, but can also be detected in large areas of the environment. In accordance with WHO Guidelines for Drinking Water Quality [22], in the assessment of health hazards not only ingestion, but also inhalation and contact (skin, mucous membranes) have to be considered as potential transmission paths of microorganisms in water for human consumption."

In 1990, the coliform test was for: "Escherichia, Klebsiella, Enterobacter, Citrobacter."
In 2001, the coliform test was for: "Escherichia, Klebsiella, Enterobacter, Citrobacter, Yersinia, Serratia, Hafnia, Pantoea, Kluyvera."
The 2001 alternate method was for: "Escherichia, Klebsiella, Enterobacter, Citrobacter, Yersinia, Serratia, Hafnia, Pantoea, Kluyvera, Cedecea, Ewingella, Moellerella, Leclercia, Rahnella, Yokenella."

They also state: "The coliform bacteria that are most frequently isolated in connection with nosocomial infections include:

- Klebsiella spp.
- Enterobacter spp.
- Citrobacter spp.
- Serratia spp."

http://www.umweltdaten.de/wasser-e/coliform_bacteria.pdf

However, according to Dr. Tarun Madappa on Medscape, "Escherichia coli is one of the most frequent causes of many common bacterial infections, including cholecystitis, bacteremia, cholangitis, urinary tract infection (UTI), and traveler's diarrhea, and other clinical infections such as neonatal meningitis and pneumonia." http://emedicine.medscape.com/article/217485-overview

These coliforms are all documented medical pathogens and some coliforms are so deadly they must be handled in a biosafety Hazard Level 3 laboratory. According to the 2010 Standards Unit, Evaluations and Standards Laboratory Centre for Infections at London, "IDENTIFICATION OF ENTEROBACTERIACEAE." It states, "All S[almonella]. Typhi, S. Paratyphi A, B and C, S. dysenteriae type 1, E. coli O157, Salmonella sendai and Salmonella cholera-suis, and Yersinia pestis are Hazard Group 3 organisms and suspected isolates must be handled in a containment level 3 room."

[Most laboratories handling disease causing organisms are certified Biosafety Hazard Level 2 containment. While it doesn't mention coliform, it does have a very detailed list of:]

> "Enterobacteriaceae reported to have caused human infections." These include:
> [species ------- serotypes]
>
> | | |
> |---|---|
> | Cedecea | davisae, lapagei, neteri, sp 3, sp 5 |
> | Citrobacter | amalonaticus, braakii, farmeri, freundii, koseri, rodentium, sedlakii, werkmanii, youngae |

Edwardsiella	hoshinae, ictaluri, tarda
Enterobacter	aerogenes, amnigenus, asburiae, cloacae, gergoviae, hormaechei, sakazakii, taylorae
Escherichia	coli, fergusonii, hermanii, vulneris
Ewingella	americana
Hafnia alvei	
Klebsiella	oxytoca, pneumoniae subspecies aerogenes, ozaenae, pneumoniae, and rhinoscleromatis
Kluyvera	ascorbata, cryocrescens, georgiana
Leclercia	adecarboxylata
Morganella	morganii
Pantoea	agglomerans, dispersa
Photorhabdus	luminescens
Proteus	mirabilis, penneri, vulgaris
Providencia	alcalifaciens, rettgeri, stuartii
Rahnella	aquatilis
Salmonella	enterica (>2000 serotypes)
Serratia	fonticola, grimesii, liquefaciens, marcescens, odorifera, plymuthica, proteamaculans, rubidaea
Shigella	boydii, dysenteriae, flexneri, sonnei
Tatumella	ptyseos
Yersinia	bercovieri, enterocolitica, intermedia, frederiksenii, kristensenii, mollaretti, pestis, pseudotuberculosis, rohdei
Yokenella	regensburgei

Other genera and species of the Enterobacteriaceae may rarely be associated with human disease."

http://www.hpa-standardmethods.org.uk/documents/bsopid/pdf/bsopid16.pdf

Enterobacteriaceae infectious organisms will meet the definition of a dangerous biological agent as defined in the "Public Health Security and Bioterrorism Preparedness and Response Act of 2002" (Public Law 107-188; June 12, 2002). This paper concerns seven of the named bacteria in the family Enterobacteriaceae on the list as well as another 23 in the same family that meet the definition in the Department of Health and Human services final bioterrorism rules that have the potential to pose a severe threat to public health and safety, to animal and plant health, or to animal and plant products.

Coliforms as Bioterrorism Agents

Congressman John Dingell of Michigan was a major force in writing many of the environmental protection laws, including the "Public Health Security and Bioterrorism Preparedness and Response Act of 2002" (Public Law 107-188; June 12, 2002), short title: 'Agricultural Bioterrorism Protection Act of 2002'. Dingell currently states on his website, "Thirty-five years ago, the United States had virtually no laws in place to protect the environment. Private individuals, industry and governments could burn into the air, pump into the water, or dump onto the ground virtually anything – with impunity." **http://www.dingellforcongress.com/the-issues/environment/**
http://www.selectagents.gov/resources/PL107-188.pdf

Thirty-five years later, by regulation, some private individuals, industry and government agencies can still dump onto the ground virtually anything – with impunity – as long as it is claimed to be for beneficial agricultural use. An example is the biological agents and toxins in sewage sludge (a Congressional mandated solid waste) and recycled sewage effluent water dumped on grazing land, crop land, parks, school grounds as well as home lawns and gardens. The dumping is allowed because the agencies only test for coliforms, fecal coliforms or E. coli. Think about that. Coliforms are dangerous biological agents at normal body temperature. Fecal coliforms are the same thermotolerant biological agents at 13.5 degrees above normal body temperature. E. coli is the primary coliform and the fecal coliforms. It is a case where regulation definitions don't always match definitions in the laws, therefore, the law no longer applies. To get a true picture of biological agents, we have to look at the Congressional definition of biological agents in the United States Codes.

Under the 01/07/2011 edition, "7 U.S.Code CHAPTER 110 - ENHANCING CONTROLS ON DANGEROUS BIOLOGICAL AGENTS AND TOXINS," Congress mandated that:

The Secretary of Agriculture shall by regulation establish and maintain a list of each biological agent and each toxin that the Secretary determines has the potential to pose a severe threat to animal or plant health, or to animal or plant products.

(1) The terms "biological agent" and "toxin" have the meanings given such terms in section 178 of title 18. http://uscode.house.gov/download/pls/07C110.txt

18 U.S.C. § 178 : US Code - Section 178: Definitions

(1) the term "biological agent" means any microorganism (including, but not limited to, bacteria, viruses, fungi, rickettsiae or protozoa), or infectious substance, or any naturally occurring, bioengineered or synthesized component of any such microorganism or infectious substance, capable of causing -

[(2002) Amended to: "means any micro-organism, virus, infectious substance, or biological product that may be engineered as a result of biotechnology, or any naturally occurring or bioengineered component of any such microorganism, virus, infectious substance, or biological product, capable of"] causing --

(A) death, disease, or other biological malfunction in a human, an animal, a plant, or another living organism;
(B) deterioration of food, water, equipment, supplies, or material of any kind; or
(C) deleterious alteration of the environment;

(2) the term "toxin" means the toxic material or product of plants, animals, microorganisms (including, but not limited to, bacteria, viruses, fungi, rickettsiae or protozoa), or infectious substances, or a recombinant or synthesized molecule, whatever their origin and method of production, and includes -

[Amended to: "means the toxic material of plants, animals, microorganisms, viruses, fungi, or infectious substances, or a recombinant molecule, whatever its origin or method of production, including -

(A) any poisonous substance or biological product that may be engineered as a result of biotechnology produced by a living organism; or
(B) any poisonous isomer or biological product, homolog, or derivative of such a

substance;

(3) the term "delivery system" means -
(A) any apparatus, equipment, device, or means of delivery specifically designed to deliver or disseminate a biological agent, toxin, or vector; or
(B) any vector;
(4) the term "vector" means a living organism, or molecule, including a recombinant or synthesized molecule, capable of carrying a biological agent or toxin to a host; and
(5) the term "national of the United States" has the meaning prescribed in section 101(a)(22) of the Immigration and Nationality Act (8 U.S.C. 1101(a)(22)).

http://codes.lp.findlaw.com/uscode/18/I/10/175

USDA (Title II), FDA (Title III) and EPA (Title IV) all refer to seven of the named biological agents on the Department of Health and Human Services list in their regulations as generic biological pollutants, pollutants, toxic pollutants, coliforms, contaminants, etiologic agents, fecal coliforms or pathogenic agents.

According to the Federal Food and Drug Administration, "The events of Sept. 11, 2001, reinforced the need to enhance the security of the United States. Congress responded by passing, the Public Health Security and Bioterrorism Preparedness and Response Act of 2002 (the Bioterrorism Act), which President Bush signed into law June 12, 2002. The Bioterrorism Act is divided into five titles:
Title I -- National Preparedness for Bioterrorism and Other Public Health Emergencies
http://energycommerce.house.gov/107/drafts/3448section.htm
Title II -- Enhancing Controls on Dangerous Biological Agents and Toxins
http://uscode.house.gov/download/pls/07C110.txt
Title III -- Protecting Safety and Security of Food and Drug Supply
http://www.fda.gov/RegulatoryInformation/Legislation/ucm155769.htm
Title IV -- Drinking Water Security and Safety
http://thomas.loc.gov/cgi-bin/cpquery/?&dbname=cp107&sid=cp107Q56vs&refer=&r_n=hr481.107&item=&sel=TOC_295697&
Title V -- Additional Provisions"
http://www.fda.gov/RegulatoryInformation/Legislation/ucm155786.htm
http://www.fda.gov/food/fooddefense/bioterrorism/ucm111086.htm

The Department of Agriculture (USDA) Animal and Plant Health Inspection Service rules are 7 CFR Part 331 for plants and 9 CFR Part 121 for animals. USDA states:

> The Public Health Security and Bioterrorism Preparedness and Response Act of 2002 (Pub. L. 107–188), provides for the regulation of certain biological agents 1 and toxins 2 that have the potential to pose a severe threat to public health and safety, to animal health, to plant health, or to animal and plant products. The Act also requires that the Secretary of Agriculture establish and enforce standards and procedures governing the possession and use of the listed biological agents and toxins, including the establishment and enforcement of safety requirements for the transfer of listed agents and toxins; the establishment and enforcement of safeguard and security measures to prevent access to listed agents and toxins for use in domestic or international terrorism or other criminal purpose; and the establishment of procedures to protect animal and plant health, and animal and plant products, in the event of a transfer in violation of the established safety

and security measures.

1. Any microorganism (including, but not limited to, bacteria, viruses, fungi, rickettsiae, or protozoa), or infectious substance, or any naturally occurring, bioengineered, or synthesized component of any such microorganism or infectious substance, capable of causing:

(1) Death, disease or other biological malfunction in a human, an animal, a plant, or another living organism;

(2) deterioration of food, water, equipment, supplies, or material of any kind; or

(3) deleterious alteration of the environment.

2. The toxic material or product of plants, animals, microorganisms (including, but not limited to, bacteria, viruses, fungi, rickettsiae, or protozoa), or infectious substances, or a recombinant or synthesized molecule, whatever their origin and method of production, and includes:

(1) Any poisonous substance or biological product that may be engineered as a result of biotechnology produced by a living organism; or

(2) any poisonous isomer or biological product, homolog, or derivative of such a substance.

http://www.selectagents.gov/resources/APHISFinalRule.pdf

The Public Health Security and Bioterrorism Preparedness and Response Act of 2002 requires that the United States improve its ability to prevent, prepare for, and respond to acts of bioterrorism and other public health emergencies that could threaten either public health and safety or American Agriculture. The Department of Health and Human Services regulation 42 C.F.R. § 72.3, "Transportation of materials containing certain etiologic agents; minimum packaging requirements" states:

"Notwithstanding the provisions of §72.2, no person may knowingly transport or cause to be transported in interstate traffic, directly or indirectly, any material [**other than biological products**] known to contain, or reasonably believed by such person to contain, one or more of the following etiologic agents unless such material is packaged, labeled, and shipped in accordance with the requirements specified in paragraphs [a]-[f] of this section: – The select agents and toxins identified in the final rules have the potential to pose a severe threat to public health and safety, to animal and plant health, or to animal and plant products." They are: "Acinetobacter calcoaceticus; Actinobacillus - all species; Actinomycetaceae - all members; Aeromonas hydrophilia; Arachnia propionica; Arizona hinshawii - all serotypes; Bacillus anthracis; Bacteroides spp.; Bartonella - all species; Bordetella - all species; Borrelia recurrentis, B. vincenti; Brucella - all species; Campylobacter (Vibrio) foetus, C. (Vibrio) jejuni; Chlamydia psittaci, C. trachomatis; Clostridium botulinum, Cl. chauvoei,Cl. haemolyticum, Cl. histolyticum,Cl. novyi, Cl. septicum, Cl. Tetani; Corynebacterium diphtheriae, C. equi,C. haemolyticum, C. pseudotuberculosis,C. pyogenes, C. renale; **Edwardsiella tarda**; Erysipelothrix insidiosa; **Escherichia coli, all enteropathogenic serotypes;** Francisella [Pasteurella] Tularensis; Haemophilus ducreyi, H. influenzae; **Klebsiella - all species and all serotypes**; Legionella - all species and all Legionella-like organisms; Leptospira interrogans - all serovars; Listeria - all species;Moraxella - all species; Mimae polymorhpa; Mycobacterium - all species; Neisseria gonorrhoeae, N. meningitidis; Nocardia asteroides; Pasteurella - all species; Plesiomonas shigelloides; **Proteus – all species**; Pseudomonas mallei; Pseudomonas pseudomallei; **Salmonella -**

all species and all serotypes; Shigella - all species and all serotypes; Sphaerophorus necrophorus; Staphylococcus aureus; Streptobacillus moniliformis; Streptococcus pneumoniae; Streptococcus pyogenes; Treponema careteum, T. pallidum, and T. pertenue; Vibrio cholerae, V. parahaemolyticus; and **Yersinia (Pasteurella) pestis,Y. Enterocolitica."**

`http://ors.uchc.edu/bio/resources/pdf/5.2.1.B.1.a_42cfr72.1-6.pdf

Since sludge (aka Biosolids) is considered to be a biological product, the rule are ignored. Seven out of the forty-eight bacterial families of biological agents on the complete list are normal and thermotolerant coliforms used to assure farmers sludge is a safe fertilizer. Another twenty-three coliforms meet the definition of a dangerous biological agent. The seven coliforms and fecal coliforms families on the biological agent list are: Edwardsiella tarda; Escherichia coli, all enteropathogenic serotypes; Klebsiella - all species and all serotypes; Proteus – all species; Salmonella - all species and all serotypes; Shigella - all species and all serotypes; and Yersinia (Pasteurella) pestis,Y. Enterocolitica.

Many of the coliforms not only infect humans, they also infect animals, birds, insects, plants and marine life. E. coli is the primary coliform and the fecal coliform, and a drinking water contaminant, therefore it will be addressed first.

Escherichia coli, all enteropathogenic serotypes

Escherichia coli (a coliform & fecal coliform) is the primary member of the Enterobacteriaceae. It was formerly known as Bacillus coli or Colibacillus. Some clones have become Coliform Superbugs immune to antibiotics. Infections include, appendicitis, aneurysms, septic arthritis, bacteremia, bone abcesses, cerebrovascular disease, cholecystitis, simple diarrhea, inflammatory diarrhea , destruction of red blood cells, developmental abnormalities, endophthalmitis, heart disease – endocarditis, Intra-abdominal abscesses, kidney failure (hemolytic-uremic syndrome), liver abscess, abscesses in the lining of the lungs (empyema), mastitis (breast infection), meningitis, neonatal meningitis, neonatal sepsis, neurologic complications, severe, lung infection , osteomyelitis, peritonitis, pneumonia, respiratory disease, skin and soft-tissue infections. sinusitis, suppurative thyroiditis, necrotizing "flesh eating" infections in the urinary tract such as urethritis/cystitis, symptomatic cystitis, pyelonephritis, acute prostatitis, prostatic abscess, urosepsis, reactions to endotoxin (cytokines) or lipopolysaccharides can lead to disseminated intravascular coagulation (blood clotting) and death.

EPA in public relations statements concerning coliform (including fecal coliform and E. coli) usually include some form of the statement "**Although they are generally not harmful themselves**, …." The qualifier is left off. The qualifier is that coliform infections (or any organism) that is promptly diagnosed and responds well to antibiotics are generally not harmful. However, if not promptly diagnosed, treated promptly and effectively, can quickly become life threatening by entering the bloodstream and spreading throughout the body. Acute and/or chronic effects may last a lifetime.

The same claim is iterated in the EPA Office of Water regulation for drinking water. The contaminate list states that "Total coliform (including fecal coliform and E. coli) [are] Not a health threat in itself; it is used to indicate whether other potentially harmful bacteria may be present [not only that but] No more than 5.0% [of the required monthly] samples [may be] total coliform-positive [fail the test] in a month. – [it is required that] Every sample that has total coliform must be analyzed for either

[thermotolerant] fecal coliforms or E. coli. – [it claims] Coliforms are naturally present in the environment; as well as feces; fecal coliforms and E. coli only come from human and animal fecal waste." http://water.epa.gov/drink/contaminants/index.cfm#Microorganisms

EPA appears to recognize coliforms are biological agents for potential bioterrorism. In fact, EPA states, "Bacteria can be difficult to sample and analyze, – many analytical methods have a low level of precision yet can be quite complex;" EPA's Office of Water has a tendency to mislead the public by requiring a coliform test, a fecal coliform test and an E. coli test in water when all three tests are just for E. coli. As an example, EPA states, "E. coli is a species of fecal coliform bacteria that is specific to fecal material from humans and other warm-blooded animals. – The most commonly tested fecal bacteria indicators are total coliforms, fecal coliforms, Escherichia coli, fecal streptococci, and enterococci. All but E. coli are composed of a number of species of bacteria that share common characteristics such as shape, habitat, or behavior; E. coli is a single species in the fecal coliform group. Members of two bacteria groups, coliforms and fecal streptococci, are used as indicators of possible sewage contamination because they are commonly found in human and animal feces. Although they are generally not harmful themselves, they indicate the possible presence of pathogenic (disease-causing) bacteria, viruses, and protozoans that also live in human and animal digestive systems." http://water.epa.gov/type/rsl/monitoring/vms511.cfm

Streptococci and Enterococci are not just indicators. You should know that Streptococci is responsible for millions of skin and throat infections each year as well as life-threatening invasive diseases such as septicemia, streptococcal toxic shock syndrome, necrotizing fasciitis, etc. Enterococci (Vancomycin-Resistant Enterococcus, or VRE) has emerged in the last 20 years in the U.S. to cause urinary tract infections, bacteremia, bacterial endocarditis, diverticulitis, meningitis, etc.

EPA's scientists seems to have missed the fact that even the least virulent E. coli can be a killer. Outside the stomach and colon, the endogenous coliform E. coli, with no virulence factors, have been implicated in respiratory tract infections such as pneumonia, blood poisoning (septicaemia), endotoxic shock, high blood pressure, scarring and eventually kidney failure, mental changes or confusion, neonatal meningitis, and urinary tract infections leading to serious infections of the prostate (prostatitis), bladder, and kidneys (pyelonephiritis) and death. According to Palomar College Microbiology Instructor, Ana Dowey, "50 to 80% of urinary tract infections in healthy people are produced by self-contamination from endogenous strains of E. coli." E. coli, with no virulence factors. This is called an opportunistic pathogen. That also means 20 to 50% of the urinary tract infections could be from taking a bath in E. coli contaminated tap water.

In 1900, Glasgow physician Edward McCharg reported fifty-seven genital infections during childbirth. Thirty one infections caused by streptococcus and bacillus coli were fatal. http://www.jstor.org/pss/20263452

Before E. coli took on Theodor Escherich's name, it was one of the first documented bacterial killers. By 1903, Scientists like William Savage MD were well aware that there were varying virulence levels between members of Bacillus coli (B. coli). However, he was of the opinion that only exposure to B. coli from humans was of concern in drinking water. His wish was to have a test that separated human B. coli from environmental strains of similar coli forms of bacteria. http://www.jstor.org/pss/3858994

In 1904, Christiaan Eijkman discovered some strains of E. coli could grow above 112.1°F. He claimed

the higher temperature differentiated them from environmental strains which scientists agreed had no sanitary significance. This strain of thermotolerant E. coli assumed the name fecal coliform. E. coli strains that grew at optimum growth temperature of 98.6°F were named coliform by the Public Health Service in 1914 and also considered to have no sanitary significance. It is amazing that after one hundred and seven years some scientists still believe the myth. Yet, the medical profession has always had a different view of what was then called Coli bacillus or Bacillus coli and tested it at normal body temperature.

In the 1910 article, "Discussion On Infections Of The Urinary Tract By Bacillus Coli In Infancy And Childhood," Charles R. Box, et al., reported the progression of the infections in infants and young children, including death. http://www.jstor.org/pss/25292509

In the 1919 edition of "Modern Surgery" John Chalmers Da Costa, stated, "This bacillus may be responsible for appendicitis, peritonitis [thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs], inflammation of the genito-urinary tract, pneumonia, inflammation of the intestine, leptomeningitis [form of meningitis which complicates the course of all of the acute infections], perineal abscess [between vaginal opening and rectum], cholangitis [infection of the common bile duct], cholecystitis [bacterial infection superimposed on an obstruction of the biliary tree], myelitis [inflammation of the spinal cord], puerperal fever [bacterial infection contracted by women during childbirth or miscarriage], wound infections and septicemia [bacteria contaminating the blood]. It is the cause of many abscesses about the intestine and responsible for many ischiorectal abscesses [region between the rectum and ishial tuberosity – sitz bone]."

In the 1921 study "ON BACILLUS COLI INFECTIONS OF THE URINARY TRACT, ESPECIALLY IN RELATION TO HAEMOLYTIC ORGANISMS," according to Leonard S. Dudgeon,
"A great point has been made by those who believe that all urinary infections are primarily via the blood stream in that the B. coli can be obtained in pure culture from the blood, but the fact that such bacilli may be so recovered does not point to the origin of the infection, since in view of Thiele and Embleton's experiments, even if the colon bacillus did start from the urethral mucous membrane it would still be found in the blood. A careful examination of the blood in B. coli infections of the urinary tract has shown that such bacilli may be isolated very frequently, especially if the blood is taken at the height of the rigor. Cabot and Crabtree (1916) obtained positive blood cultures in 40 percent of cases out of 32 examined. Conclusion:
(1) Bactllus coli in infected urine can be divided into two groups: (i) haemolytic; (ii) non-haemolytic.
(2) The haemolytic group is the common type in the infection in men and the non-haemolytic in women." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2207040/pdf/jhyg00382-0041.pdf

In the 1984 New England Journal of Medicine article "Mortality Associated with Nosocomial Urinary-Tract Infection," R. Platt and associates reported that hospital acquired UTIs were deadly. They stated, "Seventy-six patients (25 infected and 51 noninfected) died during hospitalization; death rates were 19 per cent in infected patients and 4 per cent in noninfected patients"
http://www.nejm.org/doi/full/10.1056/NEJM198209093071101

Urinary tract infections often occur from fecal material getting into the urethra. In 1934 study, Edith E. Nicholls reported on "THE INCIDENCE AND BIOLOGICAL CHARACTERISTICS OF THE HEMOLYTIC BACILLUS COLI IN THE STOOLS OF 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 hemolyic 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."
http://www.jci.org/articles/view/100599

According to EPA, "2% to 7% of children and elderly infected with E. coli [0157;H7], may develop hemolytic uremic syndrome, in which the red blood cells are destroyed and the kidneys fail; 33% of persons with hemolytic uremic syndrome have abnormal kidney function many years later, and a few require long-term dialysis, a smaller percentage of persons with hemolytic uremic syndrome develop high blood pressure, seizures, blindness, paralysis, and the effects of having part of their bowel removed." http://www.epa.gov/reg3wapd/cso/pdf/CSO_symptoms.pdf

In a 1935 study, "A Study of B. coli mutabile from an Outbreak of Diarrhea in the New-born," Anna Dean Dulaney, Ph.D., and I. D. Michelson, M.D. of the Medical School at the University of Tennessee, Memphis, reported the first outbreak of B. coli diarrhea among new-born infants in the Memphis General Hospital during the winter of 1933-34. The mortality rate was 47%.
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1559361/

Todar's Online Textbook of Bacteriology states, "It was not until 1935 that a strain of E. coli was shown to be the cause of an outbreak of diarrhea among infants. The GI tract of most warm-blooded animals is colonized by E. coli within hours or a few days after birth. The bacterium is ingested in foods or water or obtained directly from other individuals handling the infant. The human bowel is usually colonized within 40 hours of birth. E. coli can adhere to the mucus overlying the large intestine. Once established, an E. coli strain may persist for months or years. Resident strains shift over a long period (weeks to months), and more rapidly after enteric infection or antimicrobial chemotherapy that perturbs the normal flora. The basis for these shifts and the ecology of Escherichia coli in the intestine of humans are poorly understood despite the vast amount of information on almost every other aspect of the organism's existence." http://textbookofbacteriology.net/e.coli.html

Today, there are more than 200 hydrogen sulfide producing variants of E. coli and an unknown number of pathogenic chimeric clones including 3,520 unique strains of E. coli 0157:H7 reported to CDC PulseNet between 1996 and 2006. http://www.cdc.gov/mmwr/preview/mmwrhtml/mm55d926a1.htm

The "E. coli Reference Center (formerly Gastroenteric Disease Center) is a part of Animal Diagnostic Laboratory in the Department of Veterinary and Biomedical Sciences" at PennState houses "the largest repository for E. coli strains in America. It holds more than 70,000 strains collected over the last 50 years from animals, humans, birds and environment." http://ecoli.cas.psu.edu/

Since the mid-1970s E.coli has been the work-horse of the genetic engineering researchers. It has accepted genes inserted in the laboratory from other bacteria, viruses, yeast, plants and humans, including antibiotic resistant marker genes. Since that time it has become infamous for containing the Shiga-toxin genes such as stx1, stx2, rfb and EHEC hlyA (shiga-toxigenic Escherichia coli (STEC)) which causes bloody diarrhoea, haemorrhagic colitis (HC) and haemolytic uraemic syndrome (HUS). The use of antibiotics on STEC may cause an immediate release of deadly toxins, and shortly

thereafter, death.

In 1989, EPA claimed pathogenic strains of E. coli in "treated" sludge (biosolids) only caused gastroenteritis (diarrhea associated with nausea and vomiting). Even that small warning was removed from the final regulation. According to the International Escherichia and Klebsiella Centre (WHO), it has a very large strain collection of approximately 60,000 E. coli strains, most of which are clinical isolates. This collection includes test and reference strains for O, K, H and F antigens, various toxins and other E. coli virulence factors. The collection contains strains representing almost any possible sero- and virulence type.

2010, Morbidity and Mortality Weekly Report (MMWR), "Detection of Enterobacteriaceae Isolates Carrying Metallo-Beta-Lactamase --- United States, 2010," CDC reported, "During January--June 2010, three Enterobacteriaceae isolates carrying a newly described resistance mechanism, the New Delhi metallo-beta-lactamase (NDM-1) (1), were identified from three U.S. states at the CDC antimicrobial susceptibility laboratory. This is the first report of NDM-1 in the United States, and the first report of metallo-beta-lactamase carriage among Enterobacteriaceae in the United States. These isolates, which include an Escherichia coli, Klebsiella pneumoniae, and Enterobacter cloacae, carry blaNDM-1, which confers resistance to all beta-lactam agents except aztreonam (a monobactam antimicrobial) (1); all three isolates were aztreonam resistant, presumably by a different mechanism. In the United Kingdom, where these organisms are increasingly common, carriage of Enterobacteriaceae containing blaNDM-1 has been closely linked to receipt of medical care in India and Pakistan (2). All three U.S. isolates were from patients who received recent medical care in India."
http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5924a5.htm

E. coli in Animals

E. coli causes colibacillosis in animals. According to the Merk vetmanual, "Colibacillosis occurs as an acute fatal septicemia or subacute pericarditis and airsacculitis. It is a common systemic disease of economic importance in poultry and is seen worldwide."
http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/202000.htm

Baby Calf Health: Common Diarrheal Diseases
Colibacillosis usually occurs in calves 1-10 days old. "E. coli organisms are part of the normal flora of the intestinal tract. Many strains are harmless to the calf, but certain strains can cause moderate to severe scours and even death. E. coli typically produces a secretory diarrhea resulting from the intestinal epithelial cells being switched from an absorption mode to a secretion mode. E. coli is often referred to as "white" scours and is the most common cause of calf scours.
3 Types
Enteric. This is the most common type. The main clinical sign is severe diarrhea. The calf rapidly becomes weak and dehydrated with an initial fever that rapidly returns to normal (or subnormal). Dehydration can lead to death.
Enterotoxigenic. (K-99 strain) This infection runs a rapid, fatal course. Toxins cause so much fluid to be pumped into the intestine that the calf usually dies before external signs of diarrhea are present. This type of scours is one of the few that occur within the first 3 days of life.
Septicemic. This type acts like Salmonella by invading the blood stream and penetrating body tissues causing a general infection. Gross lesions are usually minimal. This is a rapid form of E. coli, often with no evidence of diarrhea. Colostrum deprived calves usually die of this form of E. coli.

http://www.merricks.com/tech_calfscours.html

As far back as 1891, bacteria was found to cause illness in cows. In the 1891 article, "OBSERVATIONS UPON A MASTITIS BACILLUS." By ALLAN MACFADYEN, M.D. (Ed.) He said, "In the Agricultural Year-Book of Switzerland for 1888, Professor Hess, of Berne, published the results of an inquiry into the causes of mastitis in cows. He proved that the infectious forms of mastitis are due to several and distinct kinds of bacteria, which penetrate through the milk canals into the milk glands. Within the latter they find a soil suitable for their growth, and for the development of their specific pathogenic properties. The resulting inflammation of the tissues is at times so mild, and the alterations in the secretion so slight, that the affected cows can still be milked, and the milk used for domestic purposes or for the manufacture of cheese. The milk from one affected cow, by being mixed with milk from healthy cows, can infect large quantities with the active bacteria. The determination of the nature and the action of these bacteria is, therefore, of hygienic and economic importance. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1328128/

In the 2003 study, "Severity of *E. coli* mastitis is mainly determined by cow factors," Christian Burvenich, et al., Ghent University at Merelbeke, Belgium, reported, "*Escherichia coli* causes inflammation of the mammary gland in dairy cows around parturition and during early lactation with striking local and sometimes severe systemic clinical symptoms. This disease affects many high producing cows in dairy herds and may cause several cases of death per year in the most severe cases. It is well known that bacterial, cow and environmental factors are interdependent and influence mastitis susceptibility. Many studies, executed during the last decade, indicate that the severity of *E. coli* mastitis is mainly determined by cow factors rather than by *E. coli* pathogenicity. During *E. coli* mastitis, the host defense status is a cardinal factor determining the outcome of the disease. Today, we know that the neutrophil is a key factor in the cows' defense against intramammary infection with *E. coli*. Effective elimination of the pathogen by neutrophils is important for the resolution of infection and the outcome of *E. coli* mastitis." https://biblio.ugent.be/input/download?func=downloadFile&fileOId=984758

In the 2009 study, "Human Health Hazards from Antimicrobial-Resistant Escherichia coli of Animal Origin," Anette M. Hammerum, et al., National Center for Antimicrobials and Infection Control, Statens Serum Institut at Copenhagen, reported, "E. coli is a commensal bacterium in the intestine of poultry, cattle, and pigs that are used for food production, and food of animal origin can be contaminated with E. coli during slaughter of the animals. E. coli from meat has mostly been associated with intestinal pathogenic E. coli (e.g., enteropathogenic, enterotoxigenic, and verotoxigenic E. coli), but recently, E. coli of animal origin has been shown to also be associated with extra-intestinal infections, such as urinary tract infections. In humans, the majority of infections caused by E. coli are not harmful (e.g., uncomplicated urinary tract infections), whereas other infections (e.g., blood stream infections) may be lethal. In many cases, the origin of E. coli that cause infection in humans remains unknown, and the significance of the animal reservoir of antimicrobial-resistant E. coli has not been quantified." http://cid.oxfordjournals.org/content/48/7/916.full

In the 2011 Virginia Cooperative Extension bulletin, "Escherichia coli: A Practical Summary for Controlling Mastitis," Christina S. Petersson-Wolfe, Assistant Professor, Dairy Science, Virginia Tech, and John Currin, Assistant Professor, Virginia-Maryland Regional College of Veterinary Medicine, reported, "... the control of environmental pathogens still remains a daunting task. Escherichia coli are Gram-negative bacteria, similar in structure to Klebsiella spp. E. coli mastitis is typically associated

with a quick onset and often severe clinical signs. -- These organisms are commonly found in organic matter including bedding and manure. -- E. coli will infect mammary glands through environmental contact. -- When E. coli bacteria die, a toxin is released; this toxin is the primary cause of the clinical signs observed in a local mastitis infection. Antibiotics act to kill bacteria and in the case of these infections, would then result in the toxin release. Therefore, intramammary antibiotic treatment is not a generally recommended practice for local infections." http://pubs.ext.vt.edu/404/404-224/404-224.html

In the 2011 bulletin, "Control of E. coli mastitis starts with vaccination," Pfizer Animal Health, said, "Research shows 60 percent to 70 percent of coliform mastitis infections become clinical.1 Coliform mastitis, when it occurs, can become severe and cause these negative impacts on your cows:
Fever
Abnormal milk
Excessive udder edema
Dramatic drop in milk production
Death"
http://www.dairyherd.com/dairy-resources/mastitis/Control-of-E-coli-mastitis-starts-with-vaccination-126774653.htm

E. coli in Plants

In a 2002 study, ""Transmission of Escherichia coli O157:H7 from Contaminated Manure and Irrigation Water to Lettuce Plant Tissue and Its Subsequent Internalization, Ethan B. Solomon, et al., Rutgers University at New Brunswick, reported, "The transmission of Escherichia coli O157:H7 from manure-contaminated soil and irrigation water to lettuce plants was demonstrated using laser scanning confocal microscopy, epifluorescence microscopy, and recovery of viable cells from the inner tissues of plants. E. coli O157:H7 migrated to internal locations in plant tissue and was thus protected from the action of sanitizing agents by virtue of its inaccessibility. Experiments demonstrate that E. coli O157:H7 can enter the lettuce plant through the root system and migrate throughout the edible portion of the plant." http://aem.asm.org/cgi/content/short/68/1/397

In the 2011 study, "Identification of the Cellular Location of Internalized Escherichia coli O157:H7 in Mung Bean, Vigna radiata, by Immunocytochemical Techniques," Amanda J Deering, et al., Purdue University, said, "Escherichia coli O157:H7 has been associated with numerous outbreaks involving fresh produce. Previous studies have shown that bacteria can be internalized within plant tissue and that this can be a source of protection from antimicrobial chemicals and environmental conditions. However, the types of tissue and cellular locations the bacteria occupy in the plant following internalization have not been addressed. In this study, immunocytochemical techniques were used to localize internalized E. coli O157:H7 expressing green fluorescent protein in germinated mung bean (Vigna radiata) hypocotyl tissue following contamination of intact seeds. An average of 13 bacteria per mm(3) were localized within the sampled tissue. The bacteria were found to be associated with every major tissue and corresponding cell type (cortex, phloem, xylem, epidermis, and pith). The cortical cells located on the outside of the vascular bundles contained the majority of the internalized bacteria (61%). In addition, the bacteria were localized primarily to the spaces between the cells (apoplast) and not within the cells. Growth experiments were also performed and demonstrated that mung bean plants could support the replication of bacteria to high levels (10(7) CFU per plant) following seed contamination and that these levels could be sustained over a 12-day period. Therefore, E. coli O157:H7 can be internalized in many different plant tissue types after a brief seed contamination event,

and the bacteria are able to grow and persist within the plant."
http://www.mendeley.com/research/identification-cellular-location-internalized-escherichia-coli-o157h7-mung-bean-vigna-radiata-immunocytochemical-techniques/

Edwardsiella tarda

In addition to E. coli, Edwardsiella (a coliform) is one of the Enterobacteriaceae that produces deadly hydrogen sulfide gas. Infections include: bacterial aneurysms, bacterial endocarditis, Gastroenteritis – inflammation of the stomach and intestines, Septicemia – bacteria in the blood (bacteremia), Wound infection, Cellullitis – skin infection, Cholecystitis – inflammation of the gallbladder, Meningitis – infection of the membranes covering the brain and spinal cord, Myonecrosis – necrosis (death) of muscle, Peritonitis – inflammation of the thin tissue that lines the inner wall of the abdomen and covers most of the abdominal organs, Salpingitis – infection of the uterus lining, fallopian tubes, or ovaries, Infected prostheses and soft tissue infection that can destroy the muscles, skin, and underlying tissue infections – necrotizing fasciitis.

In a 1980 study, "Extraintestinal human infection caused by Edwardsiella tarda," J. E. Clarridge, et al., reported, "Edwardsiella tarda is an uncommon enteric bacterium which has been found generally in animal hosts and occasionally in human feces. Three cases of extraintestinal infection caused by E. tarda which are described herein include a typhoid-like illness, peritonitis with sepsis, and cellulitis from a wound acquired while fishing. The microbiology of E. tarda and the previous reports of infection due to this organism are reviewed." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC273444/

In a 1989 study, "Serious infections with Edwardsiella tarda. A case report and review of the literature," J.P. Wilson, et al., University of Mississippi at Jackson, stated, "Edwardsiella tarda, a member of the family Enterobacteriaceae, has recently become recognized as pathogenic, especially in patients with an underlying illness. In the present report, a patient had sickle cell hemoglobinopathy and E tarda bacteremia. Other cases of serious infection with Edwardsiella are reported in the literature. Edwardsiella infection may present as bacteremia, enteric fever, gastroenteritis, localized infection, and an asymptomatic carrier state. On the basis of this review, bacteremia with E tarda often has been associated with septic shock and has a high mortality, but this may be related to the usual presence of a serious underlying illness in these patients." http://www.ncbi.nlm.nih.gov/pubmed/2643415

In the 1991 study, "Edwardsiella tarda: a causative agent in human infections," S. Jaruratanasirikul and S. Kalnauwakul, Prince of Songkhla University at Songkhla, reported, "In a retrospective study 45 specimens of E. tarda infection from 44 adult cases at Songklanagarind Hospital during February 1982 to March 1989 were reviewed. There were 24 males and 20 females, with a mean age of 48.20 years. Nearly all of E. tarda were isolated from extraintestinal sources, especially pus and urine and most of them were subsequently found to be nosocomial-acquired infections. About half were polomicrobial infections of E. tarda and gram negative bacilli. Forty one patients were cured of the infection. Three cases died from bacteremia and serious underlying diseases."
http://www.ncbi.nlm.nih.gov/pubmed/1948259

In a 1993 study, "Infections associated with the genus Edwardsiella: the role of Edwardsiella tarda in human disease," according to J.M. Janda and S.L. Abbott, "The role of the genus Edwardsiella in human illness is reviewed. Of the three recognized species, only Edwardsiella tarda has been

demonstrated to be pathogenic for humans. Chief infections associated with this species include bacterial gastroenteritis, wound infections such as cellulitis or gas gangrene associated with trauma to mucosal surfaces, and systemic disease such as septicemia, meningitis, cholecystitis, and osteomyelitis. Risk factors that are associated with E. tarda infections include exposure to aquatic environments or exotic animals (e.g., reptiles or amphibia), preexisting liver disease, conditions leading to iron overload, and dietary habits (e.g., raw fish ingestion). Although studies indicate that this bacterium is susceptible to most commonly prescribed antibiotics, fatal gastrointestinal and extraintestinal infections have been described." http://www.ncbi.nlm.nih.gov/pubmed/8268359

In a 2001 study, "Myonecrosis caused by Edwardsiella tarda: a case report and case series of extraintestinal E. tarda infections," E.M. Slaven, et al., Louisiana State University Health Sciences Center at New Orleans, reported, "Edwardsiella tarda is an unusual human pathogen. It is primarily associated with gastrointestinal disease, although recent reports of extraintestinal disease are broadening the current understanding of the clinical spectrum of E. tarda. A series of 11 cases of extraintestinal E. tarda infection is presented, including the first reported case of myonecrosis in an immunocompetent patient. Wound infections were the most common manifestation, and 3 of 5 patients with infected wounds had been exposed to a marine environment. One patient had bacteremia, and the remaining 5 patients developed abscesses that required surgical drainage. Four patients had E. tarda isolated in pure culture, including the patient with myonecrosis. Although it is often difficult to ascertain the contribution of E. tarda to infection when it is isolated as part of a mixed culture, this case series suggests that E. tarda is singularly capable of causing limb- and life-threatening infections." http://www.ncbi.nlm.nih.gov/pubmed/11317243

In a 2003 study, "Maternal Colonization and Neonatal Sepsis Caused by Edwardsiella tarda," Erin E. Mowbray, MD, et al., University of Louisville School of Medicine, stated, "A case of neonatal sepsis caused by Edwardsiella tarda, a bacterium usually associated with freshwater ecosystems, is described. The infant's mother was immersed in lake water during the sixth month of pregnancy and had vaginal and gastrointestinal colonization with the same strain of E tarda as the infant at the time of delivery. This case suggests that maternal exposures to contaminated bodies of water during pregnancy may represent a risk to newborns." http://pediatrics.aappublications.org/content/111/3/e296.full

In a 2005 study, "Extraintestinal manifestations of Edwardsiella tarda infection," I.K. Wang, et al., Chang Gung Memorial Hospital-Chiayi at Putz City, reported, "Edwardsiella tarda, a member of the family Enterobacteriaceae, is a rare human pathogen. Gastroenteritis is the most frequently reported manifestation of E. tarda infection. In contrast, extraintestinal infection with E. tarda has rarely been reported. This study made a retrospective case and microbiological data review of patients with extraintestinal E. tarda infections to further understand this disease. This study retrospectively reviewed the charts of all isolates of E. tarda cultures from clinical specimens other than faeces at Chang Gung Memorial Hospital, Taoyuan, Taiwan from October 1998 through December 2001. Edwardsiella tarda was isolated from 22 clinical specimens from 22 hospitalised patients (13 females and nine males). The extraintestinal manifestations of E. tarda infection included biliary tract infection, bacteraemia, skin and soft tissue infection, liver abscess, peritonitis, intra-abdominal abscess, and tubo-ovarian abscess. The major underlying diseases predisposing to E. tarda extraintestinal infection were hepatobiliary diseases, malignancy and diabetes mellitus. The overall mortality rate of E. tarda extraintestinal infection in the present series was 22.7% (5/22), and four (40%) of 10 patients with bacteraemia expired. Although rare, human E. tarda extraintestinal infections can have diverse clinical manifestations and moreover may cause severe and life-threatening infections. Consequently, E. tarda

should be considered a potentially important pathogen."
http://www.ncbi.nlm.nih.gov/pubmed/16033613

In a 2009 study, "Extraintestinal manifestations of Edwardsiella tarda infection: a 10-year retrospective review," J.J. Nelson, et al., University of South Alabama at Mobile, reported, "Edwardsiella tarda, a member of the family Enterobacteriaceae found in aquatic environments, is an unusual cause of human disease, presenting most frequently as gastroenteritis. Extraintestinal manifestations of E. tarda infection are rare but have included meningitis, cholecystitis, endocarditis, osteomyelitis, soft tissue infections, bacteremia, and septicemia. Over a 10-year period at our institution, 10 cases of extraintestinal infection related to E. tarda were identified. The infections ranged from soft tissue infections secondary to trauma to intra-abdominal infections with abscess formation. Several of the patients had documented factors predisposing them to infection including diabetes mellitus and C1 esterase deficiency. Interestingly, two of the patients had chronic idiopathic inflammatory bowel disease, and one patient developed a respiratory tract infection related to E. tarda, a previously unreported clinical manifestion. Although the mortality rate for extraintestinal E. tarda infections has been as high as 50% in some studies, antimicrobial treatment was eventually successful in each of the 10 cases at our institution." http://www.ncbi.nlm.nih.gov/pubmed/19489391

In a 2011 study, "Osteomyelitis due to trimethoprim/sulfamethoxazole-resistant Edwardsiella tarda infection in a patient with X-linked chronic granulomatous disease," T. Kawai, et al., National Center for Child Health and Development at Tokyo, reported, "Edwardsiella tarda, a catalase-positive bacillus widely distributed throughout nature, is generally susceptible to trimethoprim/sulfamethoxazole. We describe osteomyelitis [acute or chronic bone infection] due to trimethoprim/sulfamethoxazole-resistant E. tarda in a patient with chronic granulomatous disease (CGD). Once E. tarda acquires antibiotic resistance, infected CGD patients may develop severe infections with unforeseeable consequences." http://www.ncbi.nlm.nih.gov/pubmed/21246245

Edwardsiella in Animals, Birds and Fish

In a 1972 study, "Salmonellae and Edwardsiella tarda in Gull Feces: a Source of Contamination in Fish Processing Plants," R. W. Berg and A. W. Anderson, Oregon State University at Corvallis,said, "Edwarsiella tarda was isolated from four samples. Although the role of this organism in human intestinal infections is not precisely known, it seems worth recording the isolation of such organisms in a study of this type. This is apparently the first report of its isolation from gulls."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC376548/pdf/applmicro00123-0221.pdf

In a 1973 study, "Edwardsiella tarda, a New Pathogen of Channel Catfish (Ictalurus punctatus)," F. P. MEYER AND G. L. BULLOCK, Bureau of Sport Fisheries and Wildlife at Stuttgart, reported, "Edwardsiella tarda, an enteric, gram-negative bacterium, causes gas-filled, malodorous lesions in muscle tissue of channel catfish. Incidence and epizootiology of the disease are presented. – The majority of bacterial diseases among cultured catfishes are caused by aeromonads, pseudomonads. or myxobacteria (1). In July 1969, a septicemic disease among 38- to 50-cm channel catfish (Ictalurus punctatus) on a commercial farm in Arkansas was attributed to an enteric organism later identified as Edwardsiella tarda. The genus Edwardsiella was proposed in 1965 for a group of 37 organisms differing biochemically from other groups of Enterobacteriaceae (2). These 37 cultures were principally isolated from feces, blood, wounds, and urine of humans. All have been placed into a single species, E. tarda." http://aem.asm.org/cgi/reprint/25/1/155.pdf

In the 1991 study, "Pathogenic Properties of Edwardsiella Species", J. Michael Janda, et al., California Department of Health Services at Berkeley, said, "Although the genus originally consisted of only a single member (Edwardsiella tarda), at least three species are now known to exist. These species often inhabit freshwater sources and can also be recovered from cold-blooded vertebrates. Edwardsiellae additionally produce a wide range of infections in animals and are recognized as pathogens for eels, catfish, and high-order vertebrates. Edwardsiella ictaluri primarily causes enteric septicemia in channel catfish, while E. tarda has been implicated in the same animals as the causative agent of emphysematous putrefactive disease, a foul-smelling wound infection with abscess formation; other animal diseases caused by E. tarda include "red disease" in eels and enteritis in penguins. In humans, E. tarda is the only recognized pathogenic species primarily associated with sporadic cases of gastroenteritis; in rare instances, E. tarda has also been reported to cause extraintestinal disease, most commonly involving cases of septicemia or bacteremia. In the case of Edwardsiella hoshinae, although this species has been recovered from humans (in feces), it has been most often isolated from lizards and birds. A definite association between this species and its isolation between this species and its isolation as a bona fide pathogen has not been established to date."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC270248/pdf/jcm00045-0265.pdf

In another 1993 study, "An epizootic of Edwardsiella tarda in largemouth bass (Micropterus salmoides)," R. Francis-Floyd, et al., reported, "Edwardsiella tarda, an opportunistic bacterial pathogen, was isolated from dying largemouth bass (Micropterus salmoides) during an epizootic in a eutrophic lake system, Lochloosa Lake, Florida, USA. Approximately 1,500 adult fish died over a 6-wk period during the late summer and early fall of 1991. A mixed population of aerobic bacteria (E. tarda, Aeromonas hydrophila, and Pseudomonas sp.) was isolated from deep cutaneous ulcers and intestines of moribund bass. However, E. tarda in pure culture was the only bacterium isolated from several viscera of several fish; E. tarda may be the etiologic agent responsible for some episodes of seasonal mortality in largemouth bass." http://www.jwildlifedis.org/cgi/content/abstract/29/2/334

According to the 2009 "EAZWV Transmissible Disease Fact Sheet Sheet No. 80," Willem Schaftenaar, Head of the Veterinary Dept. of the Rotterdam Zoo, said, "E. tarda is a zoonotic problem and is a serious cause of gastroenteritis in humans. It has also been implicated in meningitis, biliary tract infections, peritonitis, liver and intra-abdominal abscesses, wound infections and septicaemia. It has been often isolated from catfish fillets in processing plants and can spread to man via the oral route or a penetrating wound. Contaminated water can also be a source of infection. – Susceptible animal groups: Fresh water and marine fish. Most diseases seem to occur at higher temperatures. Examples of affected species are channel catfish, Chinook salmon, Japanese eel, striped bass, striped mullet, Japanese flounder, yellowtail, tilapia, goldfish, carp, red sea bream. Reptiles and amphibians are common carriers." http://www.eaza.net/activities/tdfactsheets/080%20Edwardsiellosis.doc.pdf

Klebsiella - all species and all serotypes

Klebsiella (a coliform & fecal coliform) are part of the Enterobacteriaceae family. Some serotypes have become coliform superbugs immune to antibiotics. Infections include, but not limited to: aneurysms, endocarditis, urinary tract infection, pneumonia, lung destruction, surgical wound infections, blood infections (known as bacteremia), may progress to shock and death if not treated early in an aggressive fashion, especially with necrotizing fasciitis, i.e., "flesh eating" infections. Now

produces poisonous Hydrogen Sulfide (H2S) gas.

In the legal article, "Klebsiella Pneumoniae Disease Injury Lawsuits", Parker Waichman Alonso LLP, stated, "Klebsiella pneumoniae is a common hospital-acquired infectious agent, causing urinary tract and abdominal infections and hospital infected pneumonia. Klebsiella pneumoniae can be found in a person's mouth, skin, and intestines. Klebsiella is second to E. coli as the cause of urinary tract infections. The reported number of cases is up approximately 50% in the last five years and there is a 66% mortality rate in untreated patients." http://www.yourlawyer.com/

This information is not new. Joseph J. Curry, Assistant in Pathology, Harvard Medical School, was one of the first to report on the pathogenic aspect of Klebsiella pneumoniae in 1898. Curry reported on finding Klebsiella in Acute lobar pneumonia, Acute endocarditis, with gangrene of lung, Acute croupous pneumnonia complicated with acute otitis media (middle ear infection), Fracture of the skull, accompanied, by acute otitis media, throat infection, and Tonsillitis. He said, "Subcutaneous inoculation of guiniea pigs was fatal in from 5 to 7 days. Intra-peritoneal injection was fatal in 24 hours." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2121798/pdf/jbsms00030-0016.pdf

In a 1990 study, "Klebsiella pneumoniae gastroenteritis masked by Clostridium perfringens," R.P. Rennie, et al., University of Saskatchewan at Saskatoon, reported, "An unusual food-borne outbreak of gastroenteritis associated with contaminated turkey occurred at a catered company meal. The average incubation period was 10 h, and the predominant symptoms were watery diarrhea and cramps. Vomiting did not occur. Initial epidemiological features and cultures from turkey and feces of infected patients suggested that the causative agent was Clostridium perfringens, but Klebsiella pneumoniae of capsular type K15 was also isolated in large numbers from both the turkey and feces of the same patients. Plasmid analysis and enterotoxin results supported the role of K. pneumoniae as the causative agent in this outbreak. Organisms other than commonly identified pathogens should not be ignored if present in high concentrations in both food and feces of infected persons. http://www.ncbi.nlm.nih.gov/pubmed/2179254?dopt=Abstract

In the 1998 study, "A new variant of food poisoning: enteroinvasive Klebsiella pneumoniae and Escherichia coli sepsis from a contaminated hamburger." J.M. Sabota, et al., Northeastern Ohio Universities College of Medicine, Affiliated Hospitals at Canton, reported, "For the first time, we report Klebsiella pneumoniae as an enteroinvasive food-borne pathogen transmitted from a hamburger. A 28-year-old previously healthy African-American male ingested a portion of a hamburger from a fast food chain. Symptoms of gastroenteritis rapidly deteriorated to multiorgan failure. Blood and hamburger cultures grew Escherichia coli and Klebsiella pneumoniae. Since Klebsiella had not previously been reported as enteroinvasive, the isolates were compared. Full biochemical profiles, antimicrobial sensitivity, plasmid profile, and toxin assay by DNA hybridization probe were completely concordant. The patient survived the episode of food-borne sepsis. Deliberate or inadvertent employee contamination of food products with feces may be a potential source of life-threatening food-borne illness. " http://www.ncbi.nlm.nih.gov/pubmed/9448190

In a 2004 study, "Four cases of necrotizing fasciitis caused by Klebsiella species", C.H. Wong, et al., said, "Presented here are four cases of necrotizing fasciitis caused by Klebsiella spp. that were treated at one hospital over a 2-year period. Klebsiella necrotizing fasciitis can occur via direct inoculation, local trauma or, more commonly, hematogenous spread from other septic foci. Early, aggressive, surgical debridement and appropriate antimicrobial treatment are the cornerstones of treatment for this

condition. Necrotizing fasciitis due to Klebsiella spp. is unique in that it is commonly associated with multiple septic foci. While liver abscesses and endogenous endophthalmitis are better-known associations of disseminated Klebsiella infection, necrotizing fasciitis is increasingly recognized as one of the manifestations of this syndrome. When treating Klebsiella necrotizing fasciitis, awareness of the potential for multiorgan involvement should prompt a thorough search for associated foci of infection." http://www.springerlink.com/content/w3qmp64xc5dtpcr6/

In a 2004 study, "Outbreak of Klebsiella pneumoniae Producing a New Carbapenem-Hydrolyzing Class A ß-Lactamase, KPC-3, in a New York Medical Center," Neil Woodford, et al., reported, "Twenty-four patients in ICUs at the Tisch Hospital, NYU Medical Center, were colonized or infected with carbapenem-resistant K. pneumoniae between April 2000 and April 2001 (Table 2). Klebsiellae with this phenotype had not been detected in the hospital previously. All infections were nosocomially acquired, with the patients having been hospitalized from 9 to 374 days prior to isolation of the organism. Risk factors for acquisition included prolonged hospitalization, an ICU stay, and ventilator usage. Carbapenem-resistant organisms were isolated predominantly from respiratory secretions but also from urine and blood. Fourteen of the 24 patients were infected, and 8 of these died, with the Klebsiella infection considered causative or contributory."
http://aac.asm.org/cgi/content/full/48/12/4793

According to Jerome Groopman writing about the same outbreak in the August 11, 2008 edition of the New Yorker, "Superbugs: The new generation of resistant infections is almost impossible to treat," there were actually thirty-four patients with infections that year and nearly half died.
http://www.newyorker.com/reporting/2008/08/11/080811fa_fact_groopman#ixzz1ZSGRgeBH

In March 2007, a Klebsiella pneumoniae outbreak hit Israel health care facilities. According to Prof. Yehuda Carmeli, the head of the epidemiology unit at the Sourasky Medical Center in Tel Aviv, "Between 400 to 500 people have been infected by the bug, and 30 to 40 percent of them have already died." The average age was 74-75. http://www.ynetnews.com/articles/0,7340,L-3373478,00.html

In the 2011 Medscape article, "Klebsiella Infections" Obiamiwe Umeh, MBBS and Chief Editor: Burke A Cunha, MD, state, "Klebsiellae are ubiquitous in nature. In humans, they may colonize the skin, pharynx, or gastrointestinal tract. They may also colonize sterile wounds and urine. Carriage rates vary with different studies. Klebsiellae may be regarded as normal flora in many parts of the colon and intestinal tract and in the biliary tract. – Klebsiellae have also been incriminated in nosocomial infections. Common sites include the urinary tract, lower respiratory tract, biliary tract, and surgical wound sites. The spectrum of clinical syndromes includes pneumonia, bacteremia, thrombophlebitis, urinary tract infection (UTI), cholecystitis, diarrhea, upper respiratory tract infection, wound infection, osteomyelitis and meningitis" http://emedicine.medscape.com/article/219907-overview#a0104

The Bioinformatics Consortium of Taiwan, reports, "K. pneumoniae is a gram-negative enteric rod pathogen that can cause septicemia in immunocompromised patients. However, in the past fifteen years, it appears that there is a new clinical symptom of K. pneumoniae infection in Taiwan. Patients that have liver abscess, when infected with K. pneumoniae, can acquire complications such as metastatic meningitis or ophthalmitis. In addition, the new symptom brings about 10~30% mortality rate even though patients are treated with appropriate antibiotics therapy." http://cbs.ym.edu.tw/cbs-01/index.php?option=com_content&view=article&id=304&Itemid=330

Klebsiella in Animals

In the 1983 article "Isolation and Identification of Ropy Bacteria in Raw Milk", B. A. CHEUNG and D. C. WESTHOFF, University of Maryland, reported, "Approximately 4.2% of 4,000 Maryland-Virginia raw milk tanker samples developed ropiness when incubated at 10°C. Of the 56 bacterial isolates 30 were identified by species. Klebsiella oxytoca and Pseudomonas aeruginosa were isolated most frequently. Other ropy isolates were identified as Pseudomonas spp., Chromobacterium, Flavobacterium multivorum, presumptive gersinia pestis, Enterobacter agglomerans, Klebsiella pneumoniae, and Pasteurella-Actinobacter spp. Six of the Klebsiella oxytoca isolates were mesophilic (optimum temperatures of 32.0 to 37.8°C) with two isolates having psychrotrophic tendencies (optimum temperature of 26.8°C). All Pseudomonas aeruginosa isolates appeared to be psychrotrophic in their temperature requirements (optimum temperatures of 23.0 to 31.0°C)."
http://download.journals.elsevierhealth.com/pdfs/journals/0022-0302/PIIS0022030283820207.pdf

In the 2011 article, "Differentiation of Klebsiella pneumoniae and K. oxytoca by Multiplex Polymerase Chain Reaction", Yogesh Chander, et al., College of Veterinary Medicine, University of Minnesota at Saint Paul, reported, "In animals, Klebsiella are mostly associated with sepsis, infections of urinary and respiratory tracts, and mastitis. These disease syndromes cause serious economic consequences in some cattle herds. In fact, clinical mastitis due to Klebsiella infections result in higher milk losses than those due to Escherichia coli and may also result in the death of the affected cows. Both K. pneumoniae and K. oxytoca are frequently isolated from domestic animals."
http://www.jarvm.com/articles/Vol9Iss2/Vol9%20Iss2Goyal.pdf

According to John Hobkins Medicine, Noreen A. Hynes, M.D., M.P.H., D.T.M.&H., states, "Klebsiella granulomatis (formerly known as Calymmatobacterium granulomatis) is a member of the family Enterobacteriaceae; reclassification is based upon nucleotide relatedness to other Klebsiella spp. especially to K. rhinoscleromatis, another tropical infection (nasal). – Organism is difficult to demonstrate microbiologically because it does not grow on any standard microbiological laboratory media."
http://www.hopkinsguides.com/hopkins/ub/view/Johns_Hopkins_ABX_Guide/540301/all/Klebsiella_granulomatis__Granuloma_inguinale_Donovanosis

Klebsiella/Raoultella

In a 2009 letter to the editor, "Enteric Fever-Like Syndrome Caused by Raoultella ornithinolytica (Klebsiella ornithinolytica)," Victoria Pulian Morais, et al., Microbiology Service Complexo Hospitalario de Pontevedra, reported, "Raoultella ornithinolytica (formerly Klebsiella ornithinolytica) is a gram-negative aerobic bacillus in the family Enterobacteriaceae. This species has been related to histamine-producing bacteria causing subsequent fish poisoning (5). R. ornithinolytica has also been isolated from dentin of infected root canals (8). However, human infections caused by bacteria of the genus Raoultella are infrequent, and spontaneously occurring bacteremia cases have not been reported. Here, we present a case of enteric fever-like syndrome and bacteremia caused by R. ornithinolytica."
http://jcm.asm.org/cgi/content/long/47/3/868#FN1

In a 2009 study, "Isolation and characterization of Raoultella ornithinolytica from Clinical Specimens in Hilla city, Iraq," Samir M. Al-Hulu, et al., Babylon University, College of Medicine, Department of Microbiology, reported, "A total of 720 clinical samples were collected from three main hospitals in

Hilla city/ Babylon province, Iraq. Samples were screened for presence of Raoultella spp., as well as studying their expression of virulence factors. A total of 144 bacterial isolates were recovered and identified as Klebsiella-like organisms. Out of these, 11 isolates were identified as Raoultella ornithinolytica, which represent 7.6% of all Klebsiella-like organisms found. Many virulence factors expressed by R. ornithinolytica were studied in vitro. All isolates produced capsule and expressed CFA/I, and CFA/III.
Nine isolates (81.8%) were able to produce siderophores. Four isolates (36.6%) were able to produce bacteriocin. All R. ornithinolytica isolates were unable to produce extracellular protease, hemolysin, and histamine. All isolates of R. ornithinolytica were resistant to penicillin, ampicillin, gentamicin, chloramphenicol, rifampin, cephalothin, cephotaxime, streptomycin, amoxicillin.,but they showed high sensitivity to nitrofurantoin and ciprofloxacin, and all them were completely sensitive to meropenem. R. ornithinolytica expressed a high degree of sensitivity to the effect of human serum when they grew in human serum at 37 oC for 3 hrs. The present study represented the first record of occurrence of R. ornithinolytica in human clinical samples in Iraq."
http://www.uobabylon.edu.iq/uobcoleges/fileshare/articles/R.%20ornithinolytica-Univ%20website.pdf

In a 2007 case report, "A case of severe pancreatitis complicated by Raoultella planticola infection," M. S. Alves, et al., Universidade Federal de Juiz de Fora, Campus Universita´ rio, Bairro Martelos at Juiz de Fora, reported, "A 45-year-old-male presented with severe pancreatitis. Two bacterial isolates obtained from peritoneal fluid and abdominal purulent secretion were identified to the species level by 15 biochemical tests and four supplementary tests as Raoultella planticola. Identification was confirmed by rpoB gene sequencing. R. planticola is difficult to identify in the clinical laboratory, and the clinical significance of this isolation remains uncharacterized. This is the first report of pancreatitis with a primary infection by R. planticola."
http://jmm.sgmjournals.org/content/56/5/696.full.pdf

In a 2010 case report, "A Rare Case of Soft-Tissue Infection Caused by Raoultella planticola," Karina O' Connell, et al., Department of Medical Microbiology, University College Hospital at Galway, reported, "Raoultella species are Gram-negative, non-motile bacilli primarily considered to be environmental bacteria. Raoultella planticola is a rare cause of human infections. We report a case of serious soft-tissue infection in a young male tiler who presented with cellulitis of his left thumb. He had sustained a crush injury to his left thumb 10 days earlier in a soiled environment. He noted a minor break in the skin and he washed the wound out with running water. One week later, he experienced pain, erythema, and swelling of his thumb and attended his general practitioner who prescribed oral flucloxacillin and penicillin V. Despite this treatment, he noticed progressive erythema and swelling of his thumb requiring hospital admission 3 days later. He underwent washout and debridement of his thumb. Tissue obtained intraoperatively cultured Raoultella planticola. He was treated with broad-spectrum antibiotics including ciprofloxacin and made a full and rapid recovery."
http://downloads.hindawi.com/journals/crim/2010/134086.pdf

In a 2011 case report, "Sepsis caused by Raoultella terrigena," Muddassir Muhammad Shaikh and and Marina Morgan, Department of Microbiology, Royal Devon and Exeter NHS Foundation Trust, reported, "We describe a second reported case of human infection caused by Raoultella terrigena. – Raoultella terrigena (previously known as Klebsiella terrigena) is a rarely encountered gram-negative bacterium and mainly reported as aquatic and soil organism.1 However, in 2007 the first human infection caused by this organism was reported in a 45-year-old patient who developed endocarditis

due to R. terrigena post liver transplant.2 No other case reports of infections caused by this organism have been published, and the clinical spectrum of diseases caused by this organism is unknown. The correct identification of R. terrigena is not easily accomplished in most clinical microbiology laboratories, and isolates can be easily misidentified as Klebsiella pneumoniae or other Klebsiella species.3 We describe a patient with sepsis with a primary infection by R. terrigena." http://shortreports.rsmjournals.com/content/2/6/49.full

Klebsiella/Raoultella in Animals and Fish

Raoultella (formerly Klebsiella) is a gram-negative aerobic bacillus in the family Enterobacteriaceae. In a 2002 study, "Klebsiella pneumoniae Produces No Histamine: Raoultella planticola and Raoultella ornithinolytica Strains Are Histamine Producers,"Masashi Kanki, et al., Osaka Prefectural Institute of Public Health, reported, "Histamine fish poisoning is caused by histamine-producing bacteria (HPB). Klebsiella pneumoniae and Klebsiella oxytoca are the best-known HPB in fish. However, 22 strains of HPB from fish first identified as K. pneumoniae or K. oxytoca by commercialized systems were later correctly identified as Raoultella planticola (formerly Klebsiella planticola) by additional tests. Similarly, five strains of Raoultella ornithinolytica (formerly Klebsiella ornithinolytica) were isolated from fish as new HPB. R. planticola and R. ornithinolytica strains were equal in their histamine-producing capabilities and were determined to possess the hdc genes, encoding histidine decarboxylase. On the other hand, a collection of 61 strains of K. pneumoniae and 18 strains of K. oxytoca produced no histamine," http://www.ncbi.nlm.nih.gov/pmc/articles/PMC126807/

In a 2011 study, "Sources of Klebsiella and Raoultella species on dairy farms: Be careful where you walk", R.N. Zadoks, et al., Cornell University, reported, "Klebsiella spp. are a common cause of mastitis, milk loss, and culling on dairy farms. Control of Klebsiella mastitis is largely based on prevention of exposure of the udder to the pathogen. To identify critical control points for mastitis prevention, potential Klebsiella sources and transmission cycles in the farm environment were investigated, including oro-fecal transmission, transmission via the indoor environment, and transmission via the outdoor environment. A total of 305 samples was collected from 3 dairy farms in upstate New York in the summer of 2007, and included soil, feed crops, feed, water, rumen content, feces, bedding, and manure from alleyways and holding pens. Klebsiella spp. were detected in 100% of rumen samples, 89% of water samples, and approximately 64% of soil, feces, bedding, alleyway, and holding pen samples. Detection of Klebsiella spp. in feed crops and feed was less common. Genotypic identification of species using rpoB sequence data showed that Klebsiella pneumoniae was the most common species in rumen content, feces, and alleyways, whereas Klebsiella oxytoca, Klebsiella variicola, and Raoultella planticola were the most frequent species among isolates from soil and feed crops. Random amplified polymorphic DNA-based strain typing showed heterogeneity of Klebsiella spp. in rumen content and feces, with a median of 4 strains per 5 isolates. Observational and bacteriological data support the existence of an oro-fecal transmission cycle, which is primarily maintained through direct contact with fecal contamination or through ingestion of contaminated drinking water. Fecal shedding of Klebsiella spp. contributes to pathogen loads in the environment, including bedding, alleyways, and holding pens." http://www.journalofdairyscience.org/article/S0022-0302(11)00052-X/abstract

Proteus - all species

Proteus (a coliform) is one of the Enterobacteriaceae. Infections include, aneurysms, diarrhea of infants, endocarditis, wound and urinary tract infections, cystitis, secondary invader in various localized suppurative pathologic processes. It is antibiotic resistant and now produces poisonous Hydrogen Sulfide (H2S) gas.

In the 1957 article, “PROTEUS INFECTION OF URINARY TRACT, WITH SPECIAL REFERENCE TO TREATMENT WITH NITIROFURANTOIN,” J. E. MIDDLETON, B.M., M.R.C.P. Assistant Lecturer in Clinical Pathology, Louis Jenner Laboratory, St. Thomas's Hospital and Medical School, London, reported, “Infection produced by potentially pathogenic commensal organisms resistant to the chemotherapeutic agents in general use is becoming prevalent as their more virulent competitors are eliminated by these agents. Among such resistant organisms discussed by Bryer (1955) are members of the Proteus group. In the last few years there have been several reports in the American literature on the use of nitrofurantoin (" furadantin ") in infections of the urinary tract due 61 to proteus, notably by Richards et al. (1955). This relatively non-toxic drug, active in vitro against many Gram-positive and Gram negative bacteria, including the Proteus group, is excreted in high concentration in the urine following oral administration, although effective blood and tissue levels are not produced.” http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1962173/pdf/brmedj03118-0025.pdf

In a 1975 study, “INFECTION CAUSED BY PROTEUS MIRABILIS STRAINS WITH TRANSFERABLE GENTAMICIN-RESISTANCE FACTORS,” M.S. Shafi and Naomi Datta, Central Middlesex Hospital at London reported, “During a period of 10 weeks, four Summary patients in one hospital became infected with gentamicin-resistant Proteus mirabilis. In two of them, septicæmia associated with indwelling catheters developed, one had urinary tract and wound infections, and in the fourth patient the organism was isolated from a superficial wound. The P. mirabilisstrains showed multiple drug resistance. Strains from the first three patients were apparently identical and were sensitive to tobramycin. Their gentamicin resistance was not transferable to Escherichia coli K12, but could be transferred to another strain of P. mirabilis (PM13-3). The fourth strain was resistant to tobramycin; its gentamicin/tobramycin resistance was transferable to E. coli K12.” http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(75)92262-X/abstract

In a 1976 study, “NOSOCOMIAL INFECTION WITH HIGHLY RESISTANT PROTEUS RETTGERI,” RICHARD A. KASLOW, et al., Center for Disease Control at Atlanta, reported, “Over 22-1/2 months an epidemic of at least 127 cases of nosocomial infection developed from a strain of Proteus rettgeri resistant to all antibiotics commonly tested in hospital laboratories. Although there were at least four cases of septicemia and one related death, the majority of cases consisted of asymptomatic bacteriuria or clinically mild urinary tract infection. Indwelling urinary tract devices and antibiotic therapy were important predisposing factors. Data supported an association between increasing use of gentamicin and increasing rates of resistant infection. No common source was found, and contact spread appeared more likely. Control measures included efforts to reduce unnecessary exposure to the incriminated risk factors and to improve asepsis in the management of catheterized patients. An additional 36 cases and one related death were identified in the 7-1/2 months following the investigation and institution of control measures. Nosocomial infection with extremely resistant organisms may pose a serious hazard wherever indwelling urinary tract devices and antibiotics are used together intensively.” http://aje.oxfordjournals.org/content/104/3/278.abstract

In the 1982 study, "Factors influencing wound infection (a prospective study of 280 cases)," L.A. DeSa, et al., reported, "The bacteria isolated from nasal swabs, throat swabs and skin swabs after preparation are as shown in [Table - 3]. The importance of endogenous sources of contamination is further emphasised by [Table - 4] where it is seen that the concordance between organisms recovered from infected wounds and endogenous sources was better than that for exogenous sources. The single most common organism isolated from post-operative infected wounds was Staphylococcus aureus. However, mixed infections accounted for the maximum number of infected cases. Infections caused by mixed bacteria were of a 'severe' type as were infections caused by pure cultures of Proteus and Pseudomonas. Wound infections caused by the rest of the bacteria were of a 'moderate' type." http://www.jpgmonline.com/article.asp?issn=0022-3859;year=1984;volume=30;issue=4;spage=232;epage=6;aulast=deSa

In a 2000 report, "Sudden death caused by pulmonary thromboembolism in Proteus syndrome," A.M. Slavotinek, et al., Genetic Diseases Research Branch, National Human Genome Research Institute, National Institutes of Health, said, "We report 3 patients with Proteus syndrome (PS) who died suddenly from pulmonary embolism (PE). The first patient was a male diagnosed with PS at 12 years who had varicose veins, portal vein thrombosis, right iliac vein occlusion and recurrent PE. At age 25 years, he was admitted to the hospital with a severe headache. Despite therapeutic doses of warfarin, investigations for an acute episode of breathlessness showed PE and he was unable to be resuscitated. The second case was a 9-year-old male with PS who collapsed at home and could not be revived. Autopsy revealed that the cause of death was a PE associated with thrombosis of the deep veins (DVT). The third patient was a 17-year-old female undergoing inpatient treatment for sinusitis when she unexpectedly arrested. She could not be revived and a full autopsy revealed a large PE with no identified DVT. We conclude that PE is a serious complication of PS and recommend vigilance concerning the signs and symptoms of thrombosis and PE in individuals with PS, including children. Aggressive evaluation and treatment should be considered urgently in patients with PS and signs or symptoms of DVT." http://www.proteus-syndrome.org/wp-content/uploads/2011/06/Sudden-Death-Caused-by-PE.pdf

Health Canada, 2001, "Proteus spp. – MATERIAL SAFETY DATA SHEET - INFECTIOUS SUBSTANCES – Gram-negative, motile, aerobic rod shaped bacilli, urease positive, characteristic swarming; part of the normal flora of the GI tract . – Produces infections after leaving normal habitat in intestinal tract - Chronic urinary tract infections, bacteremia, pneumonia and focal lesions in debilitated patients or those receiving intravenous infusions, neonatal meningoencephalitis, empyema, osteomyelitis, cystitis, pyelonephritis, prostatitis. – RESERVOIR: Soil, water, sewage and part of normal flora of intestinal tract – Survives well out of host, especially in areas where animal protein is decomposing (sewage, soil, water) – Biosafety level 2 practices, containment equipment and facilities are recommended." http://www.msdsonline.com/resources/msds-resources/free-safety-data-sheet-index/proteus-spp.aspx

In a 2009 study, "Incidence of Proteus species in wound infections and their sensitivity pattern in the University of Benin Teaching Hospital," R. M. Mordi and M. I. Momoh, Department of Medical Microbiology, UBTH at Benin City, reported, "Proteus species are frequently recovered from infected wounds. They contaminate wounds and thus cause infections. This study was carried out at the University of Benin Teaching Hospital (UBTH) to determine the involvement of Proteus species as one of the major causative organisms in wound infections. The study also determined the sensitivity pattern

of the Proteus isolates. This was a prospective and cross-sectional study. Wound swabs and aspirates from various parts of the body and consisting of accidental, pathological and post-operative wounds were collected from patients who attended the clinics at the UBTH and examined by standard bacteriological methods. All isolates were tested for sensitivity against ciprofloxacin 5 #g, gentamycin 10 #g, streptomycin 10 #g, ofloxacin 5 mg/#g, chloramohenicol 10 #g, erythromycin 10 #g and tetracycline 10 #g. Of the 400 wound samples from various parts of the body 390 (97.5%) yielded growths and produced 560 isolates. Ten samples (2.5%) yielded no growths. Proteus species accounted for 150 (26.8%) of the isolates. Proteus mirabilis was the Proteus species most commonly isolated, 97 (17.3%), Proteus vulgaris 40 (7.1%), Proteus rettgeri 8 (1.40%), and Proteus morgagni 5 (0.9%). All the isolates were sensitive to ciprofoxacin, ofloxacin and gentamycin while all were resistant to tetracycline and erythromycin. Knowledge of the microbial flora of an environment and the sensitivity pattern are important tools in the management of wound infections especially those caused by Proteus species, and are also useful in formulating rational antibiotic policy."
http://www.ajol.info/index.php/ajb/article/viewFile/59935/48207

In a 2009 study, "Endophthalmitis caused by proteus species: antibiotic sensitivities and visual acuity outcomes", T. Leng, et al., University of Miami Miller School of Medicine at Miami, reported, "In the 13 patients identified, all cases followed intraocular surgery, and 1 was associated with a recurrent corneal ulcer. Of the 1,751 organisms isolated from intraocular culture during the study period, 244 were Gram negative. Proteus species represented 5.3% of gram-negative organisms and <1% of the total isolates identified. Endophthalmitis developed 2 days to 14 days postoperatively (median, 3.5 days), and patients were observed 1 month to 61 months after presentation (median, 17 months). Presenting vision ranged from light perception to 20/200. Ten patients had positive cultures for Proteus mirabilis, and three patients had a growth of Proteus morganii. Four patients (31%) were infected with >1 organism. All Proteus isolates were sensitive to the antibiotics clinically administered, including cefazolin, ceftazidime, gentamicin, and the fluoroquinolones. Five patients (38%) initially received intravitreal injections of antibiotics alone, 1 received an anterior chamber washout in combination with intravitreal injections, and 7 patients (54%) received pars plana vitrectomy in combination with intravitreal injections. Two of the patients (15%) who received vitrectomies had either an intraocular lens or retained nuclear fragments removed. Six patients (46%) received additional antibiotic injections during the clinical course, and 6 patients (46%) underwent additional surgical procedures. Final visual acuity was better than light perception in 5 patients (38%) and was light perception or no light perception in 8 patients (62%). Only 4 patients (31%) had a final vision acuity > or =5/200. CONCLUSION: Despite prompt treatment with appropriate antibiotics, the clinical outcome for Proteus species endophthalmitis is often poor." http://www.ncbi.nlm.nih.gov/pubmed/19584659

According to the Wales Public Health Service in 2010, "Most Proteus species are widely found in the environment. They are present in the normal human gut and also in soil, decomposing meat and sewage. The most common infections due to Proteus species are urinary tract infections. These can lead to infections of the blood (bacteraemia). As the organism is present in the gut, infections related to gut surgery can be caused by Proteus species and they can also cause medical device associated infections, such as urinary infections related to catheters and infections of intravenous lines."
http://www.wales.nhs.uk/sites3/page.cfm?orgid=379&pid=13070

In the 2010 study, "Clinical features of Proteus mirabilis pneumonia," Niro Okimoto, et al., reported, "In this study, we clinically reviewed 13 patients with Proteus mirabilis pneumonia who were admitted for treatment to Kawasaki Medical School Kawasaki Hospital, Okayama, Japan, between April 2006

and July 2009. Clinical features were retrospectively reviewed. Results showed that: (1) hospital-acquired pneumonia occurred in elderly patients with underlying diseases such as cerebrovascular disease; (2) some patients had complications of urinary tract infection due to P. mirabilis; (3) preadministration of antibacterial agents did not become a risk factor; (4) resistance for levofloxacin (LVFX) was observed; (5) prognosis was comparatively good (effective rate 84.7%)."
http://www.springerlink.com/content/423245k17w8k6568/

In a Medscape article "Proteus Infections" Kelley Struble; Chief Editor and Burke A Cunha, MD, said, "Proteus species are most commonly found in the human intestinal tract as part of normal human intestinal flora, – Proteus is also found in multiple environmental habitats, including long-term care facilities and hospitals. In hospital settings, it is not unusual for gram-negative bacilli to colonize both the skin and oral mucosa of both patients and hospital personnel. – Proteus mirabilis causes 90% of Proteus infections and can be considered a community-acquired infection. Proteus vulgaris and Proteus penneri are easily isolated from individuals in long-term care facilities and hospitals and from patients with underlying diseases or compromised immune systems. – Proteus can cause Urinary tract infections, gram-negative endotoxin-induced sepsis, resulting in systemic inflammatory response syndrome (SIRS), which carries a mortality rate of 20%-50%."
http://emedicine.medscape.com/article/226434-overview

In the article, "Treatment of endocarditis due to Proteus species: a literature review" International Journal of Infectious Diseases, 02/02/2011 Kalra A et al. – The authors report a case of endocarditis due to Proteus mirabilis that was successfully treated with ampicillin and gentamicin, and review the treatment regimens of previously published cases of Proteus endocarditis.
http://www.mdlinx.com/infectious-disease/news-article.cfm/3454627/endocarditis

According to the University of Michigan Medical School, "P. mirabilis is not a common cause of UTI in the normal host [12]. Surveys of uncomplicated cystitis or acute pyelonephritis show that P. mirabilis comprises only a few percent of cases. Even in patients with recurrent UTI, the incidence of infections by this organism is only a few percentage points higher. **Why have we conducted and now are proposing to continue intensive studies of the pathogenesis of P. mirabilis?** The answer lies in the fact that this organism infects much higher proportions (up to 44%) of patients with complicated urinary tracts; that is, those with functional or anatomic abnormalities or with chronic instrumentation such as long-term catheterization [5, 13-19], making it the most common nosocomial infection. While infecting the urinary tract, P. mirabilis has a predilection for the kidney [20]. Finally and importantly, not only does this bacterium cause cystitis and acute pyelonephritis [12, 21-23], but the production of urinary stones, a hallmark of infection with this organism [24], adds another dimension to these already complicated urinary tracts. Other urease-positive species such as Providencia and Morganella spp. that infect this patient population must also be taken into consideration, since these infections are often polymicrobial [5, 7]." http://www.umich.edu/~hltmlab/research/mirabillis/infection.htm

In a 2011 Medscape article, "Proteus Infections," Kelley Struble and Burke A Cunha, MD, reported:

> "Proteus species are part of the Enterobacteriaceae family of gram-negative bacilli. Proteus organisms are implicated as serious causes of infections in humans, along with Escherichia, Klebsiella, Enterobacter, and Serratia species.
> Proteus species are most commonly found in the human intestinal tract as part of normal human intestinal flora, along with Escherichia coli and Klebsiella species, of which E coli is the predominant resident. Proteus is also found in multiple environmental

habitats, including long-term care facilities and hospitals. In hospital settings, it is not unusual for gram-negative bacilli to colonize both the skin and oral mucosa of both patients and hospital personnel. Infection primarily occurs from these reservoirs. However, Proteus species are not the most common cause of nosocomial infections. Proteus mirabilis causes 90% of Proteus infections and can be considered a community acquired infection. Proteus vulgaris and Proteus penneri are easily isolated from individuals in long-term care facilities and hospitals and from patients with underlying diseases or compromised immune systems.

Patients with recurrent infections, those with structural abnormalities of the urinary tract, those who have had urethral instrumentation, and those whose infections were acquired in the hospital have an increased frequency of infection caused by Proteus and other organisms (eg, Klebsiella, Enterobacter, Pseudomonas,enterococci, staphylococci)."

http://emedicine.medscape.com/article/226434-overview

Proteus in Animals and Birds

In a 1983 study, "Septicemic Proteus Infection in Japanese Quail Chicks (Coturnix coturnix japonica)", R. L. Sah, et al., reported, "Proteus infection was incriminated as the cause of severe depression, coma, and high mortality in successive broods of quail chicks. The pathological lesions comprised congestion of lungs, liver, and kidneys and mucus exudation in the trachea. The organism, isolated from the heart blood and lungs of affected chicks, was identified on biochemical tests as Proteus mirabilis. Pathogenicity of the isolate was tested in young albino mice and week-old quail chicks, which succumbed to infection within 48 hours of inoculation. Association of P. mirabilis with septicemic disease in Japanese quails has apparently been demonstrated for the first time."
http://www.jstor.org/pss/1590396

From the NMC Newsletter "Udder Topics", December 2001, "Proteus spp. are not common mastitis pathogens in most herds. However, Proteus spp. can cause herd outbreaks. Infections tend to be chronic, and clinical cases are often severe. These infections respond poorly to antibiotic therapy."
http://www.nmconline.org/articles/protnotes.htm

According to the United States Geological Survey (USGS), "Proteus vulgaris – Occurs naturally in the intestines of humans and a wide variety of animals; also manure, soil and polluted waters. – Causes primary urinary tract infections and secondary infections – Associated Waterfowl Diseases – Bumblefoot (Pododermatitis, Chronic arthritis-synovitis) – Inflammation and bacterial infection of the connective tissues of the foot, linked to abrasive surfaces. – Salpingitis (Oviduct Infection, Oviduct Inflammation) – Inflammation of the oviduct, usually associated with infection, sometimes with associated peritonitis."
http://usgs.wildlifeinformation.org/S/0M_Gracilicutes/proteus/proteus_vulgaris.html#Summary

Salmonella - all species and all serotypes

Salmonella (a coliform & sometimes fecal coliform) is one of the Enterobacteriaceae family. Infections include, aneurysms, arterial infections or endocarditis, pneumonia or empyema, urinary tract infections, meningitis, septic arthritis and osteomyelitis, Enteric fever, rose spots) on the chest and abdomen,- intracranial, bone and joint, soft tissue, arterial, pancreatic, gallbladder and liver, kidneys

(glomerulitis), the genitourinary tract infections, Necrotizing fasciitis "flesh eating" infections, Typhoid (enteric fever), bacteremia, Pneumonia, heart valves (endocarditis), pericarditis, peritonitis, otitis media, cholecystitis, endophthalmitis, cutaneous abscesses, and infected cephalhematoma, It is antibiotic resistant and now produces poisonous Hydrogen Sulfide (H2S) gas.

In the 1975 study, "[Epidemiology of salmonellae and fertilizing of grassland with sewage sludge (author's transl)]," E. Hess and C. Breer, reported, "Our investigations prove that sludge contains Salmonellae in more than 90% of samples. The maximum number of organisms reached 10(7) per liter. One of our most important findings was the fact that neither aerobic stabilization nor anaerobic digestion significantly reduces the contamination with Salmonellae. Moreover we found that Salmonellae in sewage sludge spread on grass may survive up to 72 weeks. Fertilizing with unsanitized sludge may therefore lead to transmission from plant to animal. The increasing number of Salmonella carriers among our herds of cattle and their striking accumulation during the grazing period demonstrate that such transmission represents a growing danger. Sanitizing of sludge to be used as fertilizer is therefore urgent. We have investigated the sanitary effect of pasteurisation and of gamma irradiation on sewage sludge. After a proper pasteurisation in 5 plants (70 degrees C for 30 minutes) 98-100% of tested sludge samples contained less than 10 Enterobacteriaceae per gramm. The application of 300 krad resulted in a percentage of 97.2% of samples with less than 10 Enterobacteriaceae per gramm."
http://www.ncbi.nlm.nih.gov/pubmed/1189797

In the 1989 proposed Part 503 sewage sludge regulation, EPA acknowledged Salmonella to be a Primary pathogen in sludge and claimed that it only caused Gastroenteritis and enteric fever. However, Salmonella Typhi causes typhoid as does the different clones referred to as Salmonella paratyphi, while Salmonella Typhimurium causes a typhoid like disease in mice. Although Salmonella Typhi is host specific to humans, it and the other 2,500+ Salmonella Enteritidis clones that affect humans and animals may be spread through sewage products, food and water.

According to The Department of Biology at San Diego State University:

> "The three main serovars of Salmonella enterica are Typhimurium, Enteritidis, and Typhi [clones]. –
>
> Salmonella enterica serovar Typhimurium (Also called Salmonella Typhimurium or abbreviated to S. Typhimurium) Until recently the most common cause of food poisoning by Salmonella species was due to S. Typhimurium. As its name suggests, it causes a typhoid-like disease in mice. In humans S. Typhimurium does not cause as severe disease as S. Typhi, and is not normally fatal. The disease is characterized by diarrhea, abdominal cramps, vomiting and nausea, and generally lasts up to 7 days. Unfortunately, in immunocompromized people, that is the elderly, young, or people with depressed immune systems, Salmonella infections are often fatal if they are not treated with antibiotics. –
>
> Salmonella enterica serovar Enteritidis (Also called Salmonella Enteritidis or abbreviated to S. Enteritidis). In the last 20 years or so, S. Enteritidis has become the single most common cause of food poisoning in the United States. S. Enteritidis causes a disease almost identical to the very closely related S. Typhimurium. S. Enteritidis is particularly adept at infecting chicken flocks without causing visible disease, and

spreading from hen to hen rapidly. Many people have blamed the recent increase in the rise of S. Enteritidis infections on the use of mass production chicken farms. When tens or hundreds of thousands of chickens live together, die together, and are processed together a Salmonella infection can rapidly spread throughout the whole food chain. A compounding factor is that chickens from a single farm may be distributed over many cities, and even states, and hence Salmonella infections can be rapidly dispersed through millions of people. –

Salmonella enterica serovar Typhi. (Also called Salmonella Typhi or abbreviated to S. Typhi) This bacterium is the causative agent of typhoid fever. Although typhoid fever is not widespread in the United States, it is very common in under-developed countries, and causes a serious, often fatal disease. The symptoms of typhoid fever include nausea, vomiting, fever and death. Unlike the other Salmonella discussed below, S. Typhi can only infect humans, and no other host has been identified. The main source of S. Typhi infection is from swallowing infected water. Food may also be contaminated with S. Typhi, if it is washed or irrigated with contaminated water."
http://www.salmonella.org/info.html

In a 2001 study, "Soft Tissue and Cartilage Infection by Salmonella oranienburg in a Healthy Girl," Ariel R. PorcallA, MD, et al., National Institutes of Health, reported, "Compared with Salmonella typhi and S. paratyphi, whose transmission is solely person-to-person, nontyphoid salmonellal infections are acquired through contact with a number of sources such as poultry, livestock, pets, and animal products. Because of an increasing number of possible sources, the incidence of nontyphoid salmonellosis has increased dramatically since the early 1980s.[2] Children younger than 5 years of age, especially those younger than 1 year of age, appear to have the highest incidence of salmonellosis. – In the general population, focal suppurative extraintestinal complications have been recognized in 7% to 10% of cases of salmonellosis. These infections may occur almost anywhere in the body, but the most common sites are the bones and the meninges. Typically, salmonellal osteomyelitis and meningitis follow enteric fever or bacteremia. In infants, complications of salmonellosis include pneumonia, osteomyelitis, septic arthritis, pericarditis, peritonitis, otitis media, cholecystitis, endophthalmitis, cutaneous abscesses, and infected cephalhematoma."
http://www.medscape.com/viewarticle/410765

According to The European Bioinformatics Institute (EBI) "Salmonella enterica serovars often have a broad host range and some cause both gastrointestinal and systemic disease. The serovar Paratyphi A is restricted to humans and causes only systemic disease. The sequence and microarray analysis of the Paratyphi A genome indicates that it is similar to the Typhi genome but suggests that it has a more recent evolutionary origin. – Salmonella paratyphi is part of the Enterobacteriaceae family; it is a Gram-negative motile, aerobic rod which is facultatively anaerobic and there is serological identification of somatic and flagellar antigens. – Salmonella paratyphi causes bacterial enteric fever which is characterised by an abrupt onset, continued fever, malaise, headache, anorexia, enlargement of spleen, bradycardia, rose spots on trunk occur on approximately 25% of Caucasians, constipation is more common than diarrhea in adults; complications include perforation/hemorrhage/ulceration of the intestines, less frequently psychosis, hepatitis, cholecystitis, pneumonitis, and pericarditis. It is clinically similar to typhoid fever but milder with lower fatality rate. Common enterocolitis may result without enteric fever this is characterised by headache, abdominal pain, nausea, vomiting, diarrhea and dehydration. After entering the small intestine wall, the Salmonella invades through the lymphatic

system to the lymph nodes and after a period of multiplication invades the blood stream. From there the bacteria invades the liver, gall bladder, spleen, kidney and bone marrow where it multiplies and causes infection of these organs. From here they again invade the blood stream causing secondary bacteremia. The secondary bacteremia is responsible for causing fever and clinical illness. – It is transmitted by direct or indirect contact with faeces or rarely the urine of a patient or carrier, contaminated food, especially milk, milk products and shellfish, it may be contaminated by the hands of a carrier or flies may be a possible vector. A few outbreaks related to water supplies have been documented. The incubation period is 1 to 3 weeks."
http://www.ebi.ac.uk/2can/genomes/bacteria/Salmonella_paratyphi.html

In the 1990 study, "Evolutionary genetic relationships of clones of Salmonella serovars that cause human typhoid and other enteric fevers," R.K. Selander, et al., Institute of Molecular Evolutionary Genetics, Mueller Laboratory, Pennsylvania State University at University Park, stated, "Multilocus enzyme electrophoresis was employed to measure chromosomal genotypic diversity and evolutionary relationships among 761 isolates of the serovars Salmonella typhi, S. paratyphi A, S. paratyphi B, S. paratyphi C, and S. sendai, which are human-adapted agents of enteric fever, and S. miami and S. java, which are serotypically similar to S. sendai and S. paratyphi B, respectively, but cause gastroenteritis in both humans and animals. To determine the phylogenetic positions of the clones of these forms within the context of the salmonellae of subspecies I, comparative data for 22 other common serovars were utilized. Except for S. paratyphi A and S. sendai, the analysis revealed no close phylogenetic relationships among clones of different human-adapted serovars, which implies convergence in host adaptation and virulence factors. Clones of S. miami are not allied with those of S. sendai or S. paratyphi A, being, instead, closely related to strains of S. panama. Clones of S. paratyphi B and S. java belong to a large phylogenetic complex that includes clones of S. typhimurium, S. heidelberg, S. saintpaul, and S. muenchen. Most strains of S. paratyphi B belong to a globally distributed clone that is highly polymorphic in biotype, bacteriophage type, and several other characters, whereas strains of S. java represent seven diverse lineages. The flagellar monophasic forms of S. java are genotypically more similar to clones of S. typhimurium than to other clones of S. java or S. paratyphi B. Clones of S. paratyphi C are related to those of S. choleraesuis. DNA probing with a segment of the viaB region specific for the Vi capsular antigen genes indicated that the frequent failure of isolates of S. paratyphi C to express Vi antigen is almost entirely attributable to regulatory processes rather than to an absence of the structural determinant genes themselves. Two clones of S. typhisuis are related to those of S. choleraesuis and S. paratyphi C, but a third clone is not. Although the clones of S. decatur and S. choleraesuis are serologically and biochemically similar, they are genotypically very distinct. Two clones of S. typhi were distinguished, one globally distributed and another apparently confined to Africa; both clones are distantly related to those of all other serovars studied."
http://www.ncbi.nlm.nih.gov/pubmed/1973153

In the 1994 study, "Unusual manifestations of salmonellosis—a surgical problem," M.K. LALITHA and R. JOHN, reported, "From January 1981 to December 1992, of 6250 cases of salmonellosis treated at the Christian Medical college and Hospital, Vellore, India, 100 patients with focal pyogtenic infection caused by salmonellae required surgical intervention in addition to medical therapy. Thirty-one had involvement of the hepatobiliary system, and 10 more had other intra-abdominal infections. Involvement of bone and joint as well as soft tissue constituted 15% each. The site of infection in patients with soft tissue abscesses included skin (7), Parotid (2), thyroid (3), Breast (1), inguinal node (1), Branchial sinus (1), and injection site (1). Three patients had arterial infections. Noteworthy among the cases of genital infections was one case of salmonella infection in a pre-exiting hydrocele, and one

case of epididymo-orchitis with a loculated salmonella infection. Salmonella infection in a pre-existing ovarian cyst was seen in a patient with endometriosis. The salmonella serotypes most frequently encountered were S. typhi (36) and S. typhimurium(36) and S. paratyphi A (15). The importance of recognition of these protean manifestations of salmonellosis in an endemic setting is discussed. The microbilogical evaluation of properly obtained specimens is mandatory in such unusual Pyogenic infections." http://qjmed.oxfordjournals.org/content/87/5/301.abstract

According to the 2005 World Health Organization Fact Sheet, Fact sheet 9N°139, "Drug-resistant Salmonella), Salmonellosis, is one of the most common and widely distributed foodborne diseases. It constitutes a major public health burden and represents a significant cost in many countries. Millions of human cases are reported worldwide every year and the disease results in thousands of deaths. Salmonellosis is caused by the bacteria Salmonella. Today, there are over 2500 known types, or serotypes, of Salmonella. In recent years problems related to Salmonella have increased significantly, both in terms of incidence and severity of cases of human salmonellosis. – In the United States of America, an estimated 1.4 million non-typhoidal Salmonella infections, resulting in 168,000 visits to physicians, 15 000 hospitalizations and 580 deaths annually. Cost estimates per case of human salmonellosis range from approximately US$ 40 to US$ 4.6 million respectively for uncomplicated cases to cases ending with hospitalization and death. The total cost associated with Salmonella is estimated at US$ 3 billion annually in the United States. – S. Enteritidis caused the most recent epidemic, which peaked in humans in 1992 in many European countries. Its current slight decline sets the scene for re-emergence of S. Typhimurium as the most important serotype in human salmonellosis." http://www.who.int/mediacentre/factsheets/fs139/en/index.html

Salmonella in Animals

In the Virginia Cooperative Extension, Virginia Tech, and Virginia State University fact sheet 400-460 "Zoonotic Diseases of Cattle," Kevin D. Pelzer, Associate Professor, Large Animal Clinical Sciences, Virginia Tech; and Nancy Currin, D.V.M., Veterinary Extension Publication Specialist, Virginia Tech, report, "Salmonella are bacteria that are shed in the feces of infected animals. Many animals are susceptible to Salmonella, including cattle. Infection occurs as a result of the ingestion of contaminated feed, water, or grass. The bacterium can live for months to years in the environment, especially in wet and warm conditions. Young, stressed or pregnant animals are the most susceptible to Salmonella infection. Infection may result in fever, foul smelling diarrhea, and severe dehydration. People acquire Salmonella from undercooked contaminated meat, infected eggs, or unpasteurized milk products. If hands are not washed after direct contact with infected feces, then accidental ingestion of bacteria can occur. Humans may develop diarrhea, abdominal cramping, and fever, which can be very severe. Animals with diarrhea should be isolated and the area disinfected. Meat and eggs should be adequately cooked and proper food handling hygiene should be used. Always wash hands after touching or working with animals." http://pubs.ext.vt.edu/400/400-460/400-460.html

In the 2008 study, "Outbreak of Salmonella typhimurium in cats and humans associated with infection in wild birds," M. A. Tauni1and A. öSterlund, said, "An outbreak of Salmonella typhimurium infection in cats and humans in Sweden in 1999, associated with wild birds, is described. In the county of Värmland, 62 sick cats were examined. All were anorectic and lethargic, 57 per cent had vomiting and 31 per cent had diarrhoea. It was considered likely that salmonellosis was transmitted from cats to humans, but there were only a few such cases."
http://onlinelibrary.wiley.com/doi/10.1111/j.1748-5827.2000.tb03214.x/abstract

Salmonella in Plants

In 2011, Amanda J. Deering, et al., Purdue University at West Lafayette, reported on the "Examination of the Internalization of Salmonella serovar Typhimurium in Peanuts, Arachis hypogaea, Using Immunocytochemical Techniques." They said, "A variety of products of plant origin, such as tomatoes, melons, peppers and peanuts, have been implicated in Salmonella spp. associated outbreaks in recent years. Although these bacteria have been found to internalize within some plants associated with foodborne-related outbreaks, the internalization in peanut plants has not been examined to date. To investigate internalization and where the bacteria localize within the plant, intact peanut seeds were contaminated with Salmonella serovar Typhimurium expressing green fluorescent protein (GFP) for 30 min. and immunocytochemical techniques were used to localize the bacterium within the stem tissue of 16-day-old peanut plants. An average of 13.6 bacteria/mm3 were localized within the sampled tissue. The bacteria were found to be associated with every major tissue (cortical, vascular, epidermal and pith) and corresponding cell type. The cortical cells located to the outside of the vascular bundles contained the majority of the Salmonella cells (72.4%). Additional growth experiments demonstrated peanut seedlings could support the reproduction of Salmonella to high levels (109 CFU/plant) after 2 days following seed contamination. Together, these results show that Salmonella Typhimurium can internalize within many different plant tissue types after a brief seed contamination event and that the bacteria are able to grow and persist within the plant."
http://www.purdue.edu/newsroom/research/2011/story-print-deploy-layout_1_14364_14364.html

Shigella - all species and all serotypes

Shigella (a coliform) is one of the Enterobacteriaceae. Infections include, aneurysms, endocarditis, watery diarrhea, develop seizures, eye inflammation and reactive arthritis (Reiter's syndrome), intestinal perforation may occur, part of the rectum to be pushed out of the body, Permanent loss of bowel control can result, Necrotizing "flesh eating"enterocolitis: antimicrobial resistance. Now produces poisonous Hydrogen Sulfide (H2S) gas.

According to the FDA Bad Bug Book 2009, Shigella "Organisms are difficult to demonstrate in foods because methods are not developed or are insensitive. A genetic probe to the virulence plasmid has been developed by FDA and is currently under field test. However, the isolation procedures are still poor."

Shigella was one of the primary pathogen listed in the 1989 proposed 503 sludge regulation. EPA claimed Shigella is a Primary Pathogen in Sludge Biosolids that only causes Gastroenteritis. Even this small warning was removed from the final regulation.

According to the Merck Manual, Shigellosis is an acute infection of the intestine caused by Shigella sp. Symptoms include fever, nausea, vomiting, and diarrhea that is usually bloody. – Shigella causes disease by penetrating the lining of the intestine—primarily, the large intestine—resulting in swelling and sometimes shallow sores, abdominal pain and watery diarrhea. Intestinal perforation may occur as well as part of the rectum may be pushed out of the body. Permanent loss of bowel control can result, Patient may develop seizures, inflammation and reactive arthritis (Reiter's syndrome) as well as eye infections. Weight loss and dehydration may become severe and leads to shock and death. (Merck)

According to the Medscape article," Shigella Infection," Jaya Sureshbabu, et al., "These organisms are members of the coliform family, Enterobacteriaceae – The characteristic virulence trait is encoded on a large (220 kb) plasmid responsible for synthesis of polypeptides that cause cytotoxicity. Shigellae that lose the virulence plasmid are no longer pathogenic. Escherichia coli (E coli O157:H7) that harbor this plasmid clinically behave as Shigella bacteria. – Regarding chromosomally encoded enterotoxin, many pathogenic features of Shigella infection are due to the production of potent cytotoxins known as Stx, a potent protein synthesis–inhibiting exotoxin. – These toxins are lethal to animals; enterotoxic to ligated rabbit intestinal segments; and cytotoxic for vero, HeLa, and some selected endothelial cells (human renal vascular endothelial cells) manifesting as diarrhea, dysentery, and hemolytic-uremic syndrome (HUS). – Shigella chromosomes share most of their genes with E coli K12 strain MG1655, and the diversity of putative virulence genes acquired by means of bacteriophage-mediated lateral gene transfer is extensive. – According to recent CDC reports, Shigella infection accounted for 28% of all the enteric bacterial infections.[1] Children younger than 5 years had 7% of total reported cases, a rate indicating a disproportionate disease burden in this population." http://emedicine.medscape.com/article/968773-overview#a0199

Yersinia Enterocolitica.

Yersinia (a coliform & fecal coliform) is one of the Enterobacteriaceae. Infections include, aneurysms, endocarditis, severe abscess of the lung, diarrhea, hepatic and splenic, Focal (oral) infections, bacteremia, pharyngitis, meningitis, osteomyelitis, pyomyositis, conjunctivitis, pneumonia, acute proliferative glomerulonephritis, peritonitis, and primary cutaneous, necrotizlng "flesh eating" enterocolitis, pseudotuberculosis, acute gastroenteritis and mesenteric lymphadenitis, arthritis, septicemia, and erythema nodosum, Reiter's syndrome. It is antibiotic resistant and now produces poisonous Hydrogen Sulfide (H2S) gas.

Yersinia is a thermotolerant bacteria that has become very deadly since 1976. According to the 1976 study, "Lung abscess and osteomyelitis of rib due to Yersinia enterocolitica," by J. I. Sebes, et al., "Yersinia enterocolitica is a gram-negative organism that only recently has become known to infect man. Although the number of infections with Yersinia entercolitica has increased during the last few years, most reports came from Europe, Africa, Australia, and Japan. During the past year, Yersinia infections have been discovered in Canada and in the United States. – Most diseases caused by Yersinia entercolitica are relatively benign; however, we have recently observed one case that presented as a severe abscess of the lung. This case is unique in that it is not only the first report of pulmonary disease with this organism, but also demonstrates the potential aggressiveness of this organism in areas outside the gastrointestinal tract. Our purpose is to report this unusual occurrence and to acquaint the physician with this organism and some of its manifestations."
http://chestjournal.chestpubs.org/content/69/4/546.full.pdf

In the 1993 study, "Case report: Yersinia enterocolitica necrotizing pneumonia in an immunocompromised patient,**"** J.N. Greene, et al., said, "The authors report a rare case of Yersinia enterocolitica necrotizing pneumonia in an immunocompromised patient, who responded with resolution of the infection after 6 weeks of therapy with a third-generation cephalosporin but subsequently expired from the underlying lymphoma. In the few cases of Y. enterocolitica pulmonary infections that have been reported, the prognosis for cure of the infection is excellent with appropriate antibiotic therapy. Y. enterocolitica is likely to be recognized more frequently as a cause of serious

infection in the growing immunosuppressed population. Early recognition and appropriate therapy can improve survival significantly." http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&list_uids=8447337&dopt=Abstract

According to the 2006 study, "Molecular Biogrouping of Pathogenic Yersinia enterocolitica - Development of a Diagnostic PCR Assay With Histologic Correlation," Laura W. Lamps, MD, et al., reported, "Previous serologic, microbiologic, and molecular studies have implicated pathogenic strains of Y. enterocolitica as causative agents in numerous gastrointestinal inflammatory diseases, including appendicitis (granulomatous and suppurative), gastroenteritis, ileitis, colitis, and mesenteric lymphadenitis.[4,8,11,13-18] The spectrum of illness caused by Y. enterocolitica is extremely variable, ranging from acute self-limited gastroenteritis to fatal dissemination and sepsis. Food-borne outbreaks have been associated with virtually all pathogenic serovars. Serovar O:8 has characteristically been associated with more catastrophic human infection, whereas O:3 and O:9 have been linked to milder cases. – Six archival, formalin-fixed, paraffin-embedded patient specimens that had tested positive for pathogenic Y. enterocolitica chromosomal DNA using PCR analysis (see "Initial PCR Assay for the Y. enterocolitica ail Gene")[15,20,21] also were retrieved. Of the 6 cases, 3 were granulomatous appendicitis, 1 was malacoplakia of the appendix and right colon, 1 was a colon resection from a patient with acute Y. enterocolitica enterocolitis with perforation and sepsis, and 1 was a Y. enterocolitica soft tissue abscess."
http://www.medscape.com/viewarticle/530612_2 `

According to the 2009 American Academy of Pediatrics (AAP) Committee on Infectious Diseases Red Book Online, "Yersinia enterocolitica causes several age-specific syndromes and a variety of other less common presentations. Infection with Y. enterocolitica typically manifests as fever and diarrhea in young children; stool often contains leukocytes, blood, and mucus. Relapsing disease and, rarely, necrotizing enterocolitis also have been described. In older children and adults, a pseudoappendicitis syndrome (fever, abdominal pain, tenderness in the right lower quadrant of the abdomen, and leukocytosis) predominates. Bacteremia with Y. enterocolitica most often occurs in children younger than 1 year of age and in older children with predisposing conditions, such as excessive iron storage (e.g., desferrioxamine use, sickle cell disease, beta-thalassemia) and immunosuppressive states. Focal manifestations of Y. enterocolitica are uncommon and include pharyngitis, meningitis, osteomyelitis, pyomyositis, conjunctivitis, pneumonia, empyema, endocarditis, acute peritonitis, abscesses of the liver and spleen, and primary cutaneous infection. Post infectious sequelae with Y. enterocolitica infection include erythema nodosum, proliferative glomerulonephritis, and reactive arthritis; these sequelae occur most often in older children and adults, particularly people with HLA-B27 antigen."
http://aapredbook.aappublications.org/cgi/content/extract/2009/1/3.153

Yersinia enterocolitia in Animals

"Yersinia enterocolitica, is often carried by many animal species, especially pigs, and associated with sporadic diarrhea in humans and animals. Farmed deer are highly susceptible." Saunders Comprehensive Veterinary Dictionary, 3 ed. © 2007 Elsevier, Inc. All rights r eserved

Yersinia (Pasteurella) pestis,

Black Death -- Yersinia pestis causes the bubonic, pneumonic, and septicemic plagues . Human contraction of bubonic plague is usually through flea bites. Once inside the body, Y. pestis

releases a toxin which inhibits electron transport chain function. Swelling of the lymph nodes, skin blotches, and dilerium are sometimes observed within a few days of infection. Untreated infections usually result in death within a week of initial infection.
http://www.boisestate.edu/courses/westciv/plague/02.shtml

“Etymology: Alexandre E.J. Yersin; L, pestis, plague a species of small gram-negative bacteria that causes plague. The primary host is the rat, but other small rodents also harbor the organism. A person without symptoms may be a carrier, but this happens rarely. Yersinia pestis is hardy, living for long periods in infected carcasses, the soil of the host's habitat, or sputum. Also called Pasteurella pestis. See also plague.” Mosby's Medical Dictionary, 8th edition. © 2009, Elsevier.

In a 2007 study, “Multiple Antimicrobial Resistance in Plague: An Emerging Public Health Risk,” Timothy J. Welch, et al., United States Department of Agriculture (USDA), reported, “Antimicrobial resistance in Yersinia pestis is rare, yet constitutes a significant international public health and biodefense threat. In 1995, the first multidrug resistant (MDR) isolate of Y. pestis (strain IP275) was identified, and was shown to contain a self-transmissible plasmid (pIP1202) that conferred resistance to many of the antimicrobials recommended for plague treatment and prophylaxis. Comparative analysis of the DNA sequence of Y. pestis plasmid pIP1202 revealed a near identical IncA/C plasmid backbone that is shared by MDR plasmids isolated from Salmonella enterica serotype Newport SL254 and the fish pathogen Yersinia ruckeri YR71. The high degree of sequence identity and gene synteny between the plasmid backbones suggests recent acquisition of these plasmids from a common ancestor. In addition, the Y. pestis pIP1202-like plasmid backbone was detected in numerous MDR enterobacterial pathogens isolated from retail meat samples collected between 2002 and 2005 in the United States. Plasmid-positive strains were isolated from beef, chicken, turkey and pork, and were found in samples from the following states: California, Colorado, Connecticut, Georgia, Maryland, Minnesota, New Mexico, New York and Oregon. Our studies reveal that this common plasmid backbone is broadly disseminated among MDR zoonotic pathogens associated with agriculture. This reservoir of mobile resistance determinants has the potential to disseminate to Y. pestis and other human and zoonotic bacterial pathogens and therefore represents a significant public health concern.”
http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0000309

Plague is an endemic disease of rodents in the western United States, with occasional spread into human and non-rodent populations via enzootic or amplification hosts.

Contributor’s comment about the plague in mule deer and blacktailed deer states: “The mule deer had bilateral ocular infection due to Yersinia pestis. In addition to these changes there were acute necrotizing inflammatory lesions in lung, adrenals, lymph node, and liver with intralesional bacteria, and disseminated intravascular coagulation. – Plague is unusual in big game animals and ungulates are generally considered resistant to the disease. There is a published report of plague in a free-ranging mule deer in Wyoming,1 an unpublished, laboratory-confirmed case in a mule deer in Montana, 2 and bilateral plague- associated necrotizing panophthalmitis in a blacktailed deer in California.3 Ocular plague has been seen in Colorado (Dr. M. Miller, Colorado Division of Wildlife, unpublished observations).”
http://www.jwildlifedis.org/cgi/content/full/44/4/983
http://www.cwd.cc/Mule-deer_plagued_by_blindness.htm

Unlisted Primary Coliform Bioterrorism Biological Agents

The following unlisted primary coliforms are regulated Under The Public Health Security and Bioterrorism Preparedness and Response Act of 2002, as they are capable of causing: (1) Death, disease or other biological malfunction in a human, an animal, a plant, or another living organism; (2) deterioration of food, water, equipment, supplies, or material of any kind; or (3) deleterious alteration of the environment.

Citrobacter;

Citrobacter (a coliform & fecal colifom) is a genus of gram-negative bacteria in the family of the Enterobacteriaceae and a part of the normal intestinal flora of humans and animals and can be isolated from many environmental sources including food, soil, water, sewage, and sludge/biosolids. Infections include, aneurysms, endocarditis, urinary tract and infant meningitis, necrotizing meningo-encephalitis. It is multiple antibiotic resistant and now produces poisonous Hydrogen Sulfide (H2S) gas.

In the 1980 study, "Citrobacter Infections in Humans: Experience at the Seattle Veterans Administration Medical Center and a Review of the Literature," Benjamin A. Lipsky, et al., Veterans Administration Medical Center at Seattle, reported, "The genus Citrobacter is a distinct group of human pathogens comprising three species: Citrobacter freundii (biotypes a and b), Citrobacter amalonaticus, and Citrobacter diversus. In this review the clinical and microbiologic experience during 1972–1978 at the Seattle Veterans Administration Medical Center (Seattle, Wash.) with 298 isolates of Citrobacter is analyzed in relation to a survey of the literature. The most common sources of citrobacter isolates were urine, sputum, and soft tissue exudates. Members of this genus can cause neonatal meningitis and, perhaps, gastroenteritis in both children and adults. Although deep tissue infections due to Citrobacter have been reported only occasionally, in this study a large number of cultures of peritoneal fluid and bone contained Citrobacter. Most isolates of Citrobacter were from elderly, debilitated patients and either represented secondary infections or were of indeterminate clinical significance." http://cid.oxfordjournals.org/content/2/5/746.abstract

According to the 1996 "Medical Microbiology. 4th edition, Chapter 26," "The role of Citrobacter species in human disease is not as great as that of the other coliforms and Proteus. Citrobacter freundii and C diversus (C koseri) have been isolated predominantly as superinfecting agents from urinary and respiratory tract infections. Citrobacter septicemia may occur in patients with multiple predisposing factors; Citrobacter species also cause meningitis, septicemia, and pulmonary infections in neonates and young children. Neonatal meningitis produced by C diversus, while uncommon, is associated with a very high frequency of brain abscesses, death, and mental retardation in survivors." http://www.ncbi.nlm.nih.gov/books/NBK8035/

In the 1999 study, "Citrobacter freundii Invades and Replicates in Human Brain Microvascular Endothelial Cells ," Julie L. Badger, et al., Childrens Hospital Los Angeles, reported, "Neonatal bacterial meningitis remains a disease with unacceptable rates of morbidity and mortality despite the availability of effective antimicrobial therapy. Citrobacter spp. cause neonatal meningitis but are unique in their frequent association with brain abscess formation. – The fatality rate associated with neonatal meningitis is 25 to 50%; moreover, serious neurological sequelae result in 75% of survivors. Although the implication Citrobacter spp. in neonatal meningitis and brain abscess is clear, the mechanisms by

which these organisms cause disease have been poorly investigated."
http://iai.asm.org/cgi/content/full/67/8/4208

In a 2004 study, "Pneumocephalus in neonatal meningitis: diffuse, necrotizing meningo-encephalitis in Citrobacter meningitis presenting with pneumatosis oculi and pneumocephalus," S.K. Pooboni, et al., Glenfield Hospital at Leicester, said, "We report a case of a 19-day-old baby who presented with a rapid onset of septic shock complicated by progressively increasing gas accumulation within the brain and anterior chamber of the eye. – Despite effective antibiotic therapy and supportive management, the patient died with worsening accumulation of gas within the brain, resulting in brainstem death. – Citrobacter koseri was identified from the blood and cerebrospinal fluid cultures. – This case re-emphasises the importance of C. koseri as both a community-acquired and nosocomial neonatal pathogen." http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=PubMed&list_uids=15215013&dopt=AbstractPlus

In a 2008 study, "Resuscitation of eleven-year VBNC Citrobacter," Amel Dhiaf, et al, reported, "Citrobacter freundii strain WA1 was stressed by incubation in seawater microcosms for eleven years. After two years of starvation, no culturable strain was observed. Incubation of samples in nutrient-rich broth medium not supplemented with growth factors, however, allowed resuscitation of VBNC cells so that subsequent plating yielded observable colonies for significantly extended periods of time. Recovery of VBNC Citrobacter freundii was obtained by incubation in nutrient broth even after eleven years of starvation. To see whether the samples contained the same strain of Citrobacter freundii inoculated 11 years ago. The complete 16S rRNA gene was PCR amplified and sequenced from initial, stressed and revived strains of Citrobacter freundii strain WA1.The 16S rRNA gene sequences from eleven-year stressed strains were homologous with a high degree of similarity to the GenBank reference strain and were identical to each other."

In the 2009 study, "Endogenous endophthalmitis caused by Citrobacter koseri," C.H. Chiu, et al., Tri-Service General Hospital, National Defense Medical Center at Taipei, reported, "Endogenous endophthalmitis occurs when organisms are hematogenously disseminated in to the eye from a distant focus of infection. The most common isolated organisms that cause endogenous endophthalmitis are Klebsiella pneumoniae and Escherichia coli. Previous reports on endophthalmitis caused by Citrobacter species are limited. We present the first case of endogenous endophthalmitis caused by Citrobacter koseri bacteremia and renal abscesses." http://www.ncbi.nlm.nih.gov/pubmed/19834321

In a 2011 study, "Citrobacter freundii infection after acute necrotizing pancreatitis in a patient with a pancreatic pseudocyst: a case report," Antonio Lozano-Leon, et al., said, "We describe a Citrobacter freundii isolation by endoscopy ultrasound fine needle aspiration in a 80-year-old Caucasian man with pancreatic pseudocyst after acute necrotizing pancreatitis. – Conclusion:
Our case report confirms that this organism can be recovered in patients with a pancreatic pseudocyst. On-site cytology feedback was crucial to the successful outcome of this case as immediate interpretation of the fine needle aspiration sample directed the appropriate cultures and, ultimately, the curative therapy. To the best of our knowledge, this is the first reported case of isolated pancreatic C. freundii diagnosed by endoscopy ultrasound fine needle aspiration."
http://www.jmedicalcasereports.com/content/5/1/51

Enterobacter;

Enterobacter (a coliform & fecal coliform) is one of the Enterobacteriaceae. Infections include, aneurysms, endocarditis, hospital infections, urinary tract and respiratory tract infections, necrotizing enterocolitis "flesh eating". It is antibiotic resistant and now produces poisonous Hydrogen Sulfide (H2S) gas.

In a 1981 study, "Enteral feeds contaminated with Enterobacter cloacae as a cause of septicaemia," M. W. CASEWELL, et al., reported, "Artificial enteral feeds are increasingly used for patients with severe catabolic states associated with, for example, bowel pathology, burns, infection, and malignancy. One advantage claimed for using this route is the "virtual absence of the risk of infection."' Despite our previous study which showed that contaminated enteral feeds were a source of Klebsiella spp for intensive care patients,2 a recent Drugs and Therapeutics Bulletin on enteral feeding does not mention the hazard of infection.3 We report on a patient with septicaemia caused by Enterobacter cloacae derived from enteral feeds that had been contaminated by a detergent dispenser in a diet kitchen. – This case illustrates how contaminated enteral feeds provide a source of opportunistic Gram-negative bacilli that may colonise or seriously infect debilitated patients. Such organisms multiply readily at room temperature, and there are thus advantages of using commercially produced bacteriologically clean feeds, which do not require mixing with additives or diluents in the hospital environment, and disadvantages of continuous infusion of mixed feeds over several hours at room temperature. We suspect that other hospitals are similarly contaminating enteral feeds during their preparation and suggest that the unsuspected, but avoidable, infection hazards of this common form of treatment should be more widely recognised."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1504797/pdf/bmjcred00650-0047.pdf

In a 1987 study, "Outbreak of cephalosporin resistant Enterobacter cloacae infection in a neonatal intensive care unit," N. Modi, et al., reported, "Enterobacter cloacae resistant to third generation cephalosporins emerged rapidly during an outbreak of serious infections due to this organism in a neonatal intensive care unit where ampicillin and gentamicin were used as first line antibiotic treatment. Organisms resistant to cephalosporins were isolated from 12 infants, six of whom developed systemic infection. Two infants died. Isolates of E. cloacae from four of five infants treated with cefotaxime showed a loss of sensitivity to this antibiotic during treatment, but in the three infants who survived sensitive organisms were again isolated after treatment had stopped. Stopping treatment with the cephalosporins, closure of the unit to new admissions, and strict cohorting of colonised infants resulted in a prompt end to the outbreak. This outbreak suggests that the routine use of third generation cephalosporins for suspected sepsis may be inappropriate in the presence of a large reservoir of organisms with the potential for rapidly developing resistance. Routine bacteriological surveillance, however, might permit their use on a rotational basis." http://adc.bmj.com/content/62/2/148.abstract

In the 1998 study, "Occurrence of Virulence-Associated Properties in Enterobacter cloacae," Rogéria Keller, et al., Universidade Federal de São Paulo, reported, "Enterobacter cloacae is part of the normal flora of the gastrointestinal tract of 40 to 80% of people and is widely distributed in the environment (15, 19, 39). Like most members of the family Enterobacteriaceae, these organisms are capable of causing opportunistic infections in hospitalized or debilitated patients (18, 19). They were recognized as a minor cause of hospital infection in a survey published in 1981 (31). Since then, clinical awareness of the potential of E. cloacae strains to cause disease has been reflected in the increasing number of

epidemiologic studies of these microorganisms showing that they could be a serious cause of nosocomial gram-negative bacteremia (9, 17-19, 23)." http://iai.asm.org/cgi/content/full/66/2/645

In the 1999 study, "Outbreak of Enterobacter cloacae Related to Understaffing, Overcrowding, and Poor Hygiene Practices," Stephan Harbarth , MD, MS, et al., University Hospitals of Geneva, stated a "Retrospective cohort study in a neonatal intensive-care unit (NICU) from December 1996 to January 1997; environmental and laboratory investigations. – 60 infants hospitalized in the NICU during the outbreak period. – Of eight case-patients, two had bacteremia; one, pneumonia; one, soft-tissue infection; and four, respiratory colonization. – Several factors caused and aggravated this outbreak: (1) introduction of E cloacae into the NICU, likely by two previously colonized infants; (2) further transmission by HCWs' hands, facilitated by substantial overcrowding and understaffing in the unit; (3) possible contamination of multidose vials with E cloacae. Overcrowding and understaffing in periods of increased work load may result in outbreaks of nosocomial infections and should be avoided." http://www.jstor.org/stable/30142031

In a 2000 study, "Detection of Extended-Spectrum β-Lactamases in Clinical Isolates of Enterobacter cloacae and Enterobacter aerogenes," Eva Tzelepi, et al., Hellenic Pasteur Institute at Athens, said, "The aim of the present study was to investigate the frequency of extended-spectrum β-lactamases (ESBLs) in a consecutive collection of clinical isolates of Enterobacter spp. The abilities of various screening methods to detect ESBLs in enterobacters were simultaneously tested. Among the 68 consecutive isolates (56 Enterobacter cloacae and 12 Enterobacter aerogenes isolates) that were analyzed for β-lactamase content, 21 (25 and 58%, respectively) possessed transferable ESBLs with pIs of 8.2 and phenotypic characteristics of SHV-type enzymes, 8 (14.3%) of the E. cloacae isolates produced a previously nondescribed, clavulanate-susceptible ESBL that exhibited a pI of 6.9 and that conferred a ceftazidime resistance phenotype on Escherichia coli transconjugants, and 2 E. cloacae isolates produced both of these enzymes. Among the total of 31 isolates that were considered ESBL producers, the Vitek ESBL detection test was positive for 2 (6.5%) strains, and the conventional double-disk synergy test (DDST) with amoxicillin-clavulanate and with expanded-spectrum cephalosporins and aztreonam was positive for 5 (16%) strains. Modifications of the DDST consisting of closer application of the disks (at 20 instead of 30 mm), the use of cefepime, and the use of both modifications increased the sensitivity of this test to 71, 61, and 90%, respectively. Of the 37 isolates for which isoelectric focusing failed to determine ESBLs, the Vitek test was false positive for 1 isolate and the various forms of DDSTs were false-positive for 3 isolates." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC86144/

In a 2003 study, "Nosocomial Enterobacter Meningitis: Risk Factors, Management, and Treatment Outcomes," Stephen Parodi, et al., Veterans Affairs Greater Los Angeles Healthcare System, reported, "Enterobacter species are increasingly a cause of nosocomial meningitis among neurosurgery patients, but risk factors for these infections are not well defined. A review of all adult patients hospitalized at the University of California-Los Angeles (UCLA) Medical Center during an 8-year period identified 15 postneurosurgical cases of Enterobacter meningitis (EM). Cure was achieved in 14 cases (93%), and efficacy was similar for carbapenem- and cephalosporin-based treatment. – Although uncommon, the proportion of cases of nosocomial meningitis due to gram-negative organisms appears to be increasing [1–3]. Appropriate empirical antimicrobial therapy for the treatment of gram-negative bacillary meningitis is essential to prevent morbidity and mortality [3, 4], and treatment options are limited by emerging resistance to third-generation cephalosporins, especially among Enterobacter species [5–7]." http://cid.oxfordjournals.org/content/37/2/159.full

In a 2010 Medscape article, "Enterobacter Infections," Susan L Fraser, MD, stated, "Enterobacter species, particularly Enterobacter cloacae and Enterobacter aerogenes, are important nosocomial pathogens responsible for various infections, including bacteremia, lower respiratory tract infections, skin and soft-tissue infections, urinary tract infections (UTIs), endocarditis, intra-abdominal infections, septic arthritis, osteomyelitis, and ophthalmic infections. Enterobacter species can also cause various community-acquired infections, including UTIs, skin and soft-tissue infections, and wound infections, among others. – These "ICU bugs" cause significant morbidity and mortality, and infection management is complicated by resistance to multiple antibiotics."
http://emedicine.medscape.com/article/216845-overview

According to the Simon Fraser University, Brinkman Laboratory website at Vancouver, "Enterobacter sakazakii ATCC BAA-894, …. primarily causes, Meningitis, septicemia, necrotizing enterocolitis."
http://www.pathogenomics.sfu.ca/islandpath/update/IPindex.pl

The 2011 Austin Community College Microbology for the Health Sciences report on "Enterbacter cloacae, [states] Enterobacter bacteria are nosocomial opportunistic pathogens that cause infections that include
~5% of hospital-acquired septicemias
~5% of nosocomial pneumonias
~4% of nosocomial urinary tract infections
~10% of postsurgical peritonitis cases
Some usefulness to humans, such as E. cloacae used in biological control of plant diseases."
http://www.austincc.edu/rlewis3/docs/g-neg-info.html

Enterobacter in Plants

In 1982, John M. Gardner, et al., University of Florida at Lake Alfred, reported on "Bacteria in Rough Lemon Roots of Florida Citrus Trees." They said, "An aseptic vacuum extraction technique was used to obtain xylem fluid from the roots of rough lemon (Citrus jambhiri Lush.) rootstock of Florida citrus trees. Bacteria were consistently isolated from vascular fluid of both healthy and young tree decline-affected [dying] trees. Thirteen genera of bacteria were found, the most frequently occurring genera being Pseudomonas (40%), Enterobacter (18%), Bacillus, Corynebacterium, and other gram-positive bacteria (16%), and Serratia (6%). Xylem bacterial counts fluctuated seasonally. Bacterial populations ranged from 0.1 to 22 per mm3 of root tissue (about 102 to 2 x 104 bacteria per g of xylem) when bacterial counts were made on vascular fluid, but these numbers were 10- to 1,000-fold greater when aseptically homogenized xylem tissue was examined similarly. Some of the resident bacteria (4%) are potentially phytopathogenic. It is proposed that xylem bacteria have an important role in the physiology of citrus."
http://aem.asm.org/cgi/content/abstract/43/6/1335

In the 1993 article, "Enterobacter cloacae: internal yellowing of papaya (Plant Disease Pathogen)," K.A. Nishijima, University of Hawaii, said, "Enterobacter cloacae has been isolated from papaya flowers, homogenates of papaya seeds, and the crop and mid-gut of the oriental fruit fly (Dacus dorsalis Hendel), and recent studies claiming an apparent attractancy of D. dorsalis to E. cloacae, suggest that fruit flies may possibly be involved in the transmission of the bacterium to papaya. -- A report of E. cloacae isolated from homogenates of papaya seeds in 1972 suggests that this organism may have been

present in a non-pathogenic form for many years. Monthly samplings from five papaya packinghouses, that process fruit from different areas on the island of Hawaii, indicate that the incidence of internal yellowing is sporadic and may be affected by environmental factors."
http://www.extento.hawaii.edu/kbase/crop/type/e_cloac.htm

In the Fall 2005 UMass Extension Landscape, Nursery & Urban Forestry Program, fact sheet, "Wetwood and slime flux" we find that a human pathogen also kills trees. Daniel H. Gillman, Plant Pathologist said, "The bacteria Enterobacter cloacae along with several other bacteria commonly occur in elms in association with the water-soaked condition of wood called bacterial wetwood. – Wetwood and the bacteria consistently associated with it occur in nearly all elm (Ulmus) and poplar (Populus). In addition, fir (Abies), hemlock (Tsuga), maple (Acer), mulberry (Morus), oak (Quercus), and white pine (Pinus strobus) often have bacterial wetwood. – Wetwood occupies the trunk, branches, and roots of affected trees. Most bacteria associated with wetwood commonly inhabit soil and water."
http://www.umassgreeninfo.org/fact_sheets/diseases/wetwood_slime_flux.pdf

Unlisted Secondary and Emerging Coliform Biological Bioterrorism Agents

Averyella;

Averyella (a coliform) is one of the Enterobacteriaceae family.

In the 2005 study, "First Case of Septicemia Due to a Strain Belonging to Enteric Group 58 (Enterobacteriaceae) and Its Designation as Averyella dalhousiensis gen. nov., sp. nov., Based on Analysis of Strains from 20 Additional Cases," Andrew S. Johnson, et al., Queen Elizabeth II Health Sciences Centre, Dalhousie University at Halifax, reported, "When enteric group 58 was first described as a distinct new group of Enterobacteriaceae in 1985, there were only five known human isolates: four from wounds and one from feces. In 1996, we investigated the first blood isolate of enteric group 58, a case of sepsis in a 33-year-old woman receiving total parenteral nutrition. Fifteen additional clinical isolates have since been identified at CDC, including several recognized from a collection of "unidentified" strains dating back to 1973. – Enteric group 58 strains have been most frequently isolated from traumatic injuries, fractures, and wounds and rarely from feces. Defining its clinical significance and distinguishing infection from colonization requires further study, but our case report indicates that serious systemic infection can occur. – Very little is known about the epidemiology, pathogenesis, or clinical significance of this group of organisms, but existing information favors an environmental or exogenous source. We report the first blood culture isolate of enteric group 58 associated with infection of a central venous catheter and describe the phenotypic characteristics of 21 enteric group 58 strains isolated from clinical specimens."
http://jcm.asm.org/cgi/content/full/43/10/5195

Budvicia aquatica;

Budvicia (a coliform) is one of the Enterobacteriaceae family.

Zentralbl Bakteriol Mikrobiol Hyg {A}, 1983 Mar, 254(1), 95 - 108
"A hydrogen sulphide producing Gram-negative rod from water;" Aldova E et al., said, "The biochemical properties of a hydrogen sulphide-producing Gram-negative rod, provisionally designated HG group, were compared with those of the known H2S-producing and H2S-negative Enterobacteriaceae and related organisms . Sixty-four tests were used as a basis for numerical identification . All these tests demonstrated a distinctness of the HG group from other members of Enterobacteriaceae and related organisms . Results of numerical identification are discussed . According to the guanine-plus-cytosine molar content in DNA the bacterium could belong to the tribe Escherichiae of the family Enterobacteriaceae . Plasmids of different molecular size or linear fragments of DNA were found in 17 of 19 strains which indicates that the H2S production is not in correlation with the occurrence of a plasmid of definite size . So far, the only habitat of the HG group had been water, and it seems to be no rarity . Among 28 HG strains a single isolate HG16 was found which differs from HG group in biochemical properties . The distinctness of this single isolate has been confirmed also by numerical identification . Note: On the basis of DNA-DNA hybridization performed by Dr . P.A.D . Grimont and coworkers the HG group has been established as a new genus and a single species . The authors accordingly propose for the group the generic name Budvicia and the specific epithet aquatica."

http://www.bionewsonline.com/o/1/enterobacter_n.htm

In a 2007 study, "Budvicia aquatica sepsis in an immunocompromised patient following exposure to the aftermath of Hurricane Katrina," Angela Corbin, et al., Nicholls State University at Thibodaux, reported, "Budvicia aquatica has been found in surface water not associated with human faeces or sewage. The first known case is described of infection in an 85-year-old woman exposed to the aftermath of Hurricane Katrina, who tested positive for B. aquatica from both blood and urine samples." http://jmm.sgmjournals.org/content/56/8/1124.abstract

Budvicia in Animals

In the 2010 study, "Study of fecal bacterial diversity in Yunnan snub-nosed monkey (Rhinopithecus bieti) using phylogenetic analysis of cloned 16S rRNA gene sequences," Changfei Wu, et al., Yunnan normal University at Kunming, reported, "Of the 156 clones isolated, 40 (comprising 37 OTUs and represent 25.64% of the clones) had + 97% similarity of sequences with known species of bacteria, which included Pseudomonas sp., Pedobacter sp., Yersinia enterocolitica, Ruminococcus, Deefgea sp., **Budvicia aquatica,** Sphingobacterium sp., Pseudomonas syringae, Pelosinus sp., Mycetocola saprophilus, Comamonas sp. and some uncultured bacteria. – We also found bacteria such as Yersinia, Budvicia and Campylobacter, which were all pathogenic bacteria for humans and animals. Yersinia may lead to diarrhea or acute gastroenteritis (Gao et al., 1985). Campylobacter mainly caused chordapsus and food poisoning, as well as adjuvant arthritis, hepatitis and so on. It was unknown whether R. bieti suffers from these diseases."
http://www.academicjournals.org/AJB/PDF/pdf2010/20Sep/Wu%20et%20al.pdf

Buttiauxella noackiae

Buttiauxella (a coliform) is part of the Enterobacteriaceae family.

In the 2002 study, "Isolation of Buttiauxella gaviniae from a Spinal Cord Patient with Urinary Bladder Pathology," Thierry De Baere, et al., Ghent University Hospital, reported, "A gram-negative Buttiauxella gaviniae-like organism (LBV449) was isolated from a urine sample of a patient suffering from urine bladder pathology and neurological problems. The isolate was positive for adonitol fermentation and l-arginine dihydrolase and negative for melibiose and l-ornithine decarboxylase. The API 20E code was 3004113. Retrospectively, another isolate (ENT107), from a leg wound, was recovered from our collections and was shown to have similar biochemical characteristics. – To our knowledge, no clinical cases with Buttiauxella strains have been described thus far."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC130887/

In a 2006 study, "BUT-1: a new member in the chromosomal inducible class C β-lactamases family from a clinical isolate of Buttiauxella sp.," Vincent Fihman, et al., said, "The aim of this study was the characterization of the β-lactamase from a clinical isolate belonging to the genus Buttiauxella, an Enterobacteriacea usually isolated from snails and slugs[12], and conferring a phenotype typical of inducible cephalosporinase close to those described for E. cloacae and C. freundii. – Buttiauxella sp. BTN01 was isolated at Tenon Hospital (Paris, France) in December 2000 from the skull wound a patient received when ejected into side-road undergrowth from his crashed car. It was identified using the API-50CHE biochemical system (bioMérieux, Marcy-l'Etoile, France) as Buttiauxella agrestis. E.

coli HB101 [F−, leu B6, pro A2, rec A13, thi-1, ara-14, lac Y1, gal K2, xyl-5, mtl-1, rps L20, sup E44, hsd S20] (Bio-Rad Laboratories, Marnes-la-Coquette, France) was used as the host strain for cloning experiments. The pBK-CMV phagemid (Stratagene, La Jolla, CA, USA) which confers kanamycin resistance was the cloning vector."
http://onlinelibrary.wiley.com/doi/10.1111/j.1574-6968.2002.tb11293.x/full

Calymmatobacterium

Calymmatobacterium (a coliform) is part of the Enterobacteriaceae family.

According to the U.S. National Library of Medicine, National Institute of Health, "This microorganism causes granuloma inguinale, a venereal disease with local tissue destruction in the genital, inguinal, and perianal region. – It spreads to adjacent areas, and the regional lymph nodes also may be inflamed. Persistent granulomatous lesions tend to ulcerate, destroying skin and subcutaneous tissue. – Calymmatobacterium granulomatis is normally present in the gut flora and may be transmitted to the genital area by autoinoculation or sexual contact. – Superinfection of ulcers with other pathogenic organisms is possible. – In the United States, infection of blacks is seven times more frequent than infection of whites." http://www.ncbi.nlm.nih.gov/books/NBK7885/

Cedecea

Cedecea (a coliform) is part of the Enterobacteriaceae family.

In a 1983 study, "Cedecea davisae isolated from scrotal abscess," B. H. Bae and S. B. Sureka reported, "A strain of Cedecea davisae was isolated as the predominant organism from a patient with a scrotal abscess as well as chronic heart and liver diseases." www.ncbi.nlm.nih.gov

In a 1986 study, "Cedecea davisae Bacteremia," SANDRA R. PERKINS, et al., McLaren General Hospital at Flint, said, "A case of bacteremia caused by Cedecea davisae is presented. This is the first reported case of bacteremia caused by this organism. – The name Cedecea was proposed in 1980 for a new genus, in the family Enterobacteriaceae, formerly designated as CDC Enteric Group 15.... – Seventeen isolates of the species designated as Cedecea davisae have previously been reported (1). Sputum was the most common source. Other sources included a gall bladder, hand wounds, and an eye swab from a 4-day-old infant. None of the 17 strains was isolated from blood or spinal fluid. – This case represents the first known report of C. davisae bacteremia."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC269000/pdf/jcm00100-0195.pdf

In a 1998 study, "A Case of Cedecea davisae Peritonitis in a Liver Cirrhosis Patient," S.H. Shin, et al., Chosun University Medical School at Kwangju, reported, "Cedecea davisae is a motile, Gram-negative rod in the family Enterobacteriaceae which is positive for lipase, DNase and catalase, and negative for gelatinase and oxidase. This bacterium is rarely isolated in the clinical specimens. We isolated C. davisae from the ascitic fluid of a 49-year old male patient with liver cirrhosis who was diagnosed as acute bacterial peritonitis. Bacterial identification was performed by API 20E and VITEK. Antimicrobial susceptibility test showed that the isolate was susceptible to cefotaxime, piperacillin, and imipenem. Peritonitis of this patient was improved by imipenem therapy. This is the first reported case

of peritonitis caused by this organism."
http://www.koreamed.org/SearchBasic.php?RID=2086KJID/1998.30.1.97&DT=1

In a 2009 study, "Scrotal abscess with a rare cause," Khizer Mansoor, et al., reported, "A 4-year-old boy presented with a short history of right-sided acute scrotal pain and swelling. At exploration, pus was found in the hemiscrotum but no local cause could be found. Further exploration showed the pus coming through a patent processus vaginalis from a collection in the right iliac fossa secondary to acute appendicitis." http://www.mendeley.com/research/scrotal-abscess-rare-cause/

In a 2010 study, "Sirolimus oral ulcer with Cedecea davisae superinfection," H. Mawardi, et al., reported, "Cedecea davisae is a member of the Enterobacteriaceae family and is an uncommon pathogen. This organism has been isolated from the blood, sputum, and cutaneous ulcers of only a handful of patients, most of these being elderly or otherwise medically compromised. This is a report of a patient, status post renal transplantation, who developed an oral ulcer associated with sirolimus use and superinfected with C. davisae. According to the literature, this is the first case of C. davisae detected in the oral cavity. Antibiotic therapy led to prompt resolution of this very large ulcer."

In a 2011 study, "Cedecea davisae bacteremia in a neutropenic patient with acute myeloid leukemia," G. Abate, et al., Saint Louis University at Saint Louis, reported, "Cedecea are the new members of Enterobacteriacea. Because of their inherent resistance to some antibiotics, the clinical response could be unpredictable making management of Cedecea infection in immunocompromised patients challenging. We report a case of acute myeloid leukemia with central line-related Cedecea bacteremia." http://www.ncbi.nlm.nih.gov/pubmed/21571373

In another 2011 study, "Incidence and resistance profile of Cedecea sp. isolated from a hospital," Marcelo M. Antunes, et al., Brazil Hospital Barão de Lucena at Recife, stated, "The isolation of Cedecea in hospitals has been each more frequent, what it represents a significant importance in the determination of the etiology of these infections. A total of 52 Cedecea samples were collected from patients in Recife, Brazil, from March 2000 to June 2003. For the bacterial identification and determination of the sensitivity profile to the antimicrobials automated methodology from MicroScan was used, with panels neg. combo, according to the manufacturer's instructions. In a total of 52 isolated samples, 27 were identified as Cedecea lapagei (52%), 19 as Cedecea davisae (36%) and 6 as Cedecea sp. 5 (12%). The sensitivity profile of the isolates is presented, demonstrating a clear tendency to multi-resistance, with higher sensitivity for Fluoroquinolones and Carbapenems. It is possible to conclude that the incidence of Cedecea, what in our country is already real, corresponds to less than 1% of all registered infectious processes. The sensitivity profile presented few options to be used as treatment, being necessary not only to standardize the sensitivity tests, as well as to improve the studies in this area in order to achieve an efficient control of this pathogen in the hospital environment."
http://eproceedings.worldscinet.com/9789812837554/9789812837554_0106.html

Ewingella

Ewingella (a coliform) is part of the Enterobacteriaceae family.

In a 1983 study, "Ewingella americana gen.nov., sp.nov., a new Enterobacteriaceae isolated from clinical specimens," P.A. Grimont, et al., said, "We propose the name Ewingella gen.nov. for a new

group in the Enterobacteriaceae. Ewingella is phenotypically distinct from all other groups of Enterobacteriaceae. The members of this genus are lipase- and deoxyribonuclease-negative; Voges-Proskauer-positive; lysine-, ornithine- and arginine-decarboxylase-negative; anaerogenic; they produce acid from glucose in the presence (and absence) of iodoacetate, but fail to produce acid from L-arabinose, melibiose, raffinose, D-sorbitol or sucrose. DNA-relatedness studies (S1-nuclease method) showed that the 10 Ewingella strains studied form a single DNA-hybridization group which is less than 21% related to other members of the Enterobacteriaceae. This single DNA-hybridization group is named Ewingella americana sp. nov. The type strain of E. americana is CDC 1468-78 (= ATCC 33852 = CIP 8194). Although the 10 strains of E. americana were isolated from clinical sources in the United States, the clinical significance of these organisms is not known."
http://www.ncbi.nlm.nih.gov/pubmed/6847036

In another 1983 study, "Polymicrobial bacteremia caused by Ewingella americana (family Enterobacteriaceae) and an unusual Pseudomonas species," F.D. Pien, et al., stated, "Ewingella americana and a Pseudomonas species were isolated from three sets of blood cultures from a 41-year-old patient after coronary bypass surgery. This is the first well-described case of bacteremia due to E. americana. Based on data from 31 strains, a detailed description of E. americana is given."
http://www.ncbi.nlm.nih.gov/pubmed/6630449

In a 1991 study, "Isolation of Ewingella americana from a patient with conjunctivitis," W.R. Heizmann and R. Michel, reported, "Ewingella americana (family Enterobacteriaceae) was isolated separately from both eyes of a 30-year-old woman. Clinical signs and symptoms included adhesive eyelids, itching and edematous upper and lower lids. Therapy with amoxicillin-clavulanate resulted in the relief of symptoms. Ewingella americana can be isolated worldwide, but seems to be a rare pathogen."
http://www.ncbi.nlm.nih.gov/pubmed/1794367

In a 2003 study, "Fatal Waterhouse-Friderichsen syndrome due to Ewingella americana infection," M. Tsokos, Institute of Legal Medicine, Department of Forensic Pathology at Hamburg, reported, "A fatal case of Waterhouse-Friderichsen syndrome resulting from infection in a previously healthy 74-year-old woman is reported. The patient died suddenly within 14 hours after presentation. The diagnosis of Waterhouse-Friderichsen syndrome [adrenal gland failure] as the cause of death was established post mortem based on autopsy findings, microscopic examination, measurement of serum procalcitonin concentration (113 ng/ml), and outcome of postmortem bacteriologic cultures that grew in heart and spleen blood samples. Since the introduction of as a new group in the family in 1983, more recent case studies have established its clinical significance and pathogenic potential to cause severe, life-threatening bacteremia and sepsis. is a rare pathogen that should be added to the list of unusual bacteria causing Waterhouse-Friderichsen syndrome." http://www.ncbi.nlm.nih.gov/pubmed/12604997

In a 2005 study, "A Case of Pneumonia Caused by Ewingella americana in a Patient with Chronic Renal Failure," Nam-Hee Ryoo, et al., School of Medicine, Keimyung University at Daegu, stated, "Ewingella americana is the only species of the genus of Ewingella in the family Enterobacteriaceae, first described from clinical specimens in 1983 (1). The pathogenic significance and niches of the reservoir have not been clarified. This organism rarely causes human infections and has been identified from various clinical specimens including wound, sputum, urine, stool, blood (1, 2), conjunctiva (3) and peritoneal dialysate (4). We present a chronic renal failure patient with fever and haziness in right lung field on chest radiography. Since the blood and urine cultures showed no other bacterial growth, pneumonia caused by E. americana suspected to be the origin of fever. Ceftriaxone and isepamicin

(Yoohan, Seoul, Korea) were administered and the clinical and radiological findings were improved. As far as we know, this is the first report of a pneumonia caused by E. americana." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2808562/?tool=pmcentrez

In a 2011 case report, "Multidrug-Resistant Ewingella Americana: A Case Report and Review of the Literature," Melanie W Pound, PharmD BCPS, et al., Campbell University School of Pharmacy at Buies Creek, stated, ".... a case of multidrug-resistant Ewingella americana associated with exacerbation of chronic obstructive pulmonary disease (COPD). – To our knowledge, this is only the second case of an E. americana respiratory infection, and the only one in which multidrug resistance has been reported. – E. americana is a rare gram-negative bacillus that has infrequently been reported to cause infection. This organism has been reported in humans in the blood, sputum, conjunctiva, wounds, and peritoneal fluid. In several of these cases, as well as in our case, this organism appeared to occur more frequently in immunocompromised patients. Although generally susceptible to most antibiotics, our patient's organism was resistant to all antibiotics tested, with the exception of TMP/SMX, ticarcillin/clavulanate, and cefotetan." http://www.theannals.com/content/41/12/2066

Ewingella in Plants

In the 2007 study, "Isolation, identification and ecology of Ewingella americana (the causal agent of internal stipe necrosis) from cultivated mushrooms in New Zealand," P. Roy Chowdhury, et al., University of Canterbury at Christchurch, reported, "Internal stipe necrosis of cultivated mushrooms (Agaricus bisporus) is caused by the bacterium Ewingella americana, a member of the Enterobacteriaceae. Recently, E. americana was isolated from healthy cultivated button mushrooms grown in New Zealand and from mushrooms showing mild stipe browning. E. americana forms a part of the endogenous bacterial population present in mushroom sporocarp tissues. This is the first time that E. americana has been isolated from a non-human host in New Zealand. Previously, the bacterium has been found associated with human blood and sputum samples. Presented here are the details of the identification methods used in providing evidence that this strain of E. americana has the capacity to induce typical symptoms of internal stipe necrosis. Ecological studies give a possible explanation as to why E. americana has previously been unnoticed in New Zealand." http://www.publish.csiro.au/paper/AP07045

Hafnia alvei

Hafnia (a coliform) is part of the Enterobacteriacea family.

In a 1996 study, "Clinical Significance of Extraintestinal Hafnia alvei Isolates from 61 Patients and Review of the Literature," Huldrych Giinthard and Andreas Pennekamp, University of Zurich, reported, "Hafnia alvei is a gram-negative bacterium that is rarely isolated from human specimens and is rarely considered to be pathogenic. It has been associated with gastroenteritis, meningitis, bacteremia, pneumonia, nosocomial wound infections, endophthalmitis, and a buttock abscess. We studied 80 H. alvei isolates recovered from 61 patients within a period of 30 months. H. alvei was cultured from sites that included the respiratory tract (n = 38), the gastrointestinal tract (n = 16), and the urogenital tract (n = 12); the organism was found in blood cultures (n = 8), on central venous catheters (n = 3), and on the skin (n = 3). Only 25% of H. alvei isolates were recovered in pure cultures. Fifty-seven (93.4%) of the patients had an underlying illness. H. alvei proved to be the

etiologic agent in two episodes of septicemia and in one episode of peritonitis and was probably responsible for septicemia in two other patients and pneumonia in one. All six of these patients recovered after receiving antibiotic treatment and/or standard surgical treatment, when needed. Three of these infections were nosocomial, and three were community acquired. Of the strains of H. alvei tested in our study, 100% were susceptible to netilmicin, ciprofloxacin, and imipenem; 92% were susceptible to piperacillin; 90% were susceptible to co-trimoxazole; and 88% were susceptible to ceftriaxone and ceftazidime. In this study, we found H. alvei to be a rare but significant etiologic agent of nosocomial and community-acquired infections."
http://cid.oxfordjournals.org/content/22/6/1040.full.pdf

In a 2000 study, "Extraintestinal infection due to Hafnia alvei," A. Ramos and D. Dámaso, Hospital Universitario Clínica Puerta de Hierro, Universidad Autónoma de Madrid, said, "The aim of this study was to establish the clinical features of extraintestinal infections caused by Hafnia alvei. Over a 5-year period (1994-1998), data were collected regarding inpatients (n = 8) with nosocomial (n = 5) or community-acquired (n = 3) infections caused by Hafnia alvei. The mean age of the patients was 47 +/- 21 years. Three patients had hospital-acquired urinary tract infections. Hafnia alvei also caused community-acquired cholangitis, cholecystitis, appendicitis, psoas abscess and prosthetic endocarditis. Hafnia alvei was susceptible to amoxicillin/clavulanic acid and to first-generation cephalosporins in two cases. Susceptibility to aminoglycosides, imipenem, cotrimoxazole, ciprofloxacin, piperacillin and cefotaxime was very good (8/8). Four patients required invasive treatment."
http://www.hopkinsguides.com/hopkins/ub/citation/11057506/Extraintestinal_infection_due_to_Hafnia_alvei_

In a 2007 study, "Young-infant sepsis combined with urinary tract infection due to Hafnia alvei," C.H. Liu, et al., Tri-Service General Hospital, National Defense Medical Center at Taipei, reported, "Hafnia alvei infections are uncommon and occur mainly in adult patients featuring underlying illnesses. Its isolation in pediatric cases is even more unusual. We report a rare case of sepsis combined with urinary tract infection caused by H. alvei in a 39-day-old infant who did not appear to feature any underlying disease. The infant was successfully treated with ceftriaxone over a 14-day period. In this case, we want to remind clinicians that the possibility of an extraintestinal invasive infection such as bacteremia or urinary tract infection caused by H. alvei should be taken into account in young infants who feature no apparent underlying disease." http://www.ncbi.nlm.nih.gov/pubmed/17493908

In a 2008 study, "Graft versus host disease-related Hafnia alvei colonization and probable infection," Vincenzo Savini, et al., Spirito Santo Hospital at Pescara, said, "We describe the case of a graft versus host disease (GvHD) patient, in whom Hafnia alvei was cultured as a single organism, and at high bacterial counts from stool samples, from the onset of the disease until its resolution. This case is a further example of the contentious role of this species in causing human intestinal disease. Furthermore, it focuses on enteric damage by GvHD as a risk factor for acquiring H. alvei colonization, and probably infection. – H. alvei is a rare human pathogen. Some cases of gastroenteritis reported in the literature have been suspected to be due to this organism, but its role as an agent of enteric infection is still uncertain (Gunthard & Pennekamp, 1996; Janda & Abbott, 2006). Isolation from respiratory secretions is more common; although most of the isolates from airways do not seem to be clinically significant, sporadic reports of pneumonia, bronchopneumonia and pulmonary abscesses that were most likely due to H. alvei have been described (Gunthard & Pennekamp, 1996; Janda & Abbott, 2006). The organism is occasionally found in the urinary tract, usually as a commensal, although in a few cases it was considered to be clinically significant (Gunthard & Pennekamp, 1996; Janda &

Abbott, 2006). Rare bloodstream infections in which H. alvei was isolated from blood cultures have been reported. Bacteraemias were mostly community acquired, and the times of the first positive blood cultures usually ranged from 1 to 41 days after hospitalization. In some cases, the organism has been isolated from blood and hepatic abscesses, pancreatic pseudocyst fluid, pleural fluid, and central venous catheters, at the same time; although the source of bacteraemias mostly remained unknown, the main origin of the bloodstream infections was thought to be the respiratory or intestinal tract (Gunthard & Pennekamp, 1996; Janda & Abbott, 2006; Liu et al., 2007; Rodriguez-Guardado et al., 2006). Neonatal infections related to silent H. alvei vaginal carriage by mothers and a meningitis case in a 1-year-old patient have been reported too. Very few reports exist in the literature regarding wound colonization by this organism, and we found just two reports concerning H. alvei endophthalmitis (Gunthard & Pennekamp, 1996; Janda & Abbott, 2006). Some cases of isolation of this organism from abscesses, and one from a patient with septic arthritis, are known, but its role as a pathogen was uncertain since it was recovered as a part of a mixed bacterial flora (Gunthard & Pennekamp, 1996; Janda & Abbott, 2006). Finally, two cases of cholecystitis, a report of spontaneous bacterial peritonitis and a case of endocarditis have been described (Hazouard et al., 2006; Janda & Abbott, 2006; Loulergue et al., 2007). Interestingly, an outbreak of probable haemolytic uraemic syndrome has been cited by Janda and colleagues, in which a H. alvei strain producing a Vero cell active cytolytic toxin was isolated from faeces (Crandall et al., 2006)." http://jmm.sgmjournals.org/content/57/9/1167.full

In another 2008 study, "Invasion and intracellular survival of Hafnia alvei strains in human epithelial cells," D. Padilla, et al., reported, "The aim of this study was to investigate the invasion and intracellular survival of different Hafnia alvei strains in HeLa cells. – We performed different experiments on the bacterial invasion of different strains of H. alvei into the HeLa cell line using gentamicin protection assays and immunofluorescence. We also report the time course of cell internalization and the effects of inhibitors on the invasion of H. alvei. Levels of invasion varied depending on the conditions (strain, time and inoculum size) used. – This study revealed that H. alvei strains were able to enter and persist in a human epithelial cell line. – Our in vitro findings highlight the possibility that some H. alvei strains may exploit nonprofessional phagocytes or nonphagocytic cells to spread in vivo, which may be important for the persistence and establishment of an asymptomatic carrier state." http://www.mendeley.com/research/invasion-and-intracellular-survival-of-hafnia-alvei-strains-in-human-epithelial-cells/

In a 2011 study, "Hafnia alvei pyelonephritis in a renal transplant recipient: case report and review of an under-recognized nosocomial pathogen," A.P. Cardile, et al., Tripler Army Medical Center at Honolulu, said, "We describe the first case to our knowledge of Hafnia alvei pyelonephritis in a renal transplant recipient. Clinicians should consider this under-recognized pathogen when clinically evaluating immunosuppressed patients with a history of invasive procedures." http://www.ncbi.nlm.nih.gov/pubmed/21299775

Kluyvera

Kluyvera (a coliform) is part of the Enterobacteriaceae family.

In a 1981 study, "Kluyvera, a new (redefined) genus in the family Enterobacteriaceae: identification of Kluyvera ascorbata sp. nov. and Kluyvera cryocrescens sp. nov. in clinical specimens," J.J. Farmer, 3rd, et al., reported, "Kluyvera is proposed as a new genus for the group of organisms formerly known as

Enteric Group 8 (synonym = API group 1). Strains of Kluyvera share the properties of most members of the family Enterobacteriaceae: they are gram-negative rods, motile with peritrichous flagella, catalase positive, and oxidase negative; they grow on MacConkey agar, ferment D-glucose with the production of acid and gas, and are susceptible to many antibiotics. Strains are usually indole positive, methyl red positive, Voges-Proskauer negative, citrate positive, H2S (triple sugar iron) negative, urea negative, phenylalanine deaminase negative, lysine decarboxylase positive, arginine dihydrolase negative, and ornithine decarboxylase positive. Kluyvera strains ferment many of the sugars and polyhydroxyl alcohols used in identification. By deoxyribonucleic acid-deoxyribonucleic acid hybridization, strains of Kluyvera were divided into three groups. Kluyvera ascorbata is proposed as the type species for the genus. Most strains of K. ascorbata have been isolated from clinical specimens. K. cryocrescens is proposed as the second species. It was occasionally isolated from clinical specimens, but it was isolated more commonly from the environment. Kluyvera species group 3 was heterogeneous, but was distinct from the two named species by deoxyribonucleic acid hybridization. This group was rare, so no species name will be proposed at this time. K. ascorbata can be differentiated from K. cryocrescens by its positive ascorbate test, inability to grow at 5 degrees C in a refrigerator, and smaller zones of inhibition around carbenicillin and cephalothin disks. The test normally used for identification does not clearly differentiate these two species. Kluyvera species are probably infrequent opportunistic pathogens. The most common source is sputum, where they are probably not clinically significant. Five strains have been from blood cultures. More information is needed about the incidence and clinical significance of the genus Kluyvera."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC273917/

In 2001 study, "Infections Caused by Kluyvera Species in Humans," Juan C. Sarria, et al., said, "Kluyvera is a relatively newly described genus in the family Enterobacteriaceae that infrequently causes infections in humans. The organism has been isolated from various clinical specimens, but its significance has not been clearly established. In fact, it has been regarded alternatively as saprophytic, opportunistic, or pathogenic. Since the redefinition of this genus in 1981, case reports of diverse clinical infections occurring under various host conditions have been published. Here we present a critical review of all Kluyvera infections reported in the literature, along with our experience involving 5 additional cases. Most patients received prompt antimicrobial treatment on the basis of susceptibility testing, and overall the clinical outcomes were good. Antimicrobial agents active against most Kluyvera strains include third-generation cephalosporins, fluoroquinolones, and aminoglycosides. In contrast, the resistance to ampicillin, extended-spectrum penicillins, and first- and second-generation cephalosporins is significant. Kluyvera is a potentially virulent pathogen that deserves aggressive treatment designed with an awareness of the organism's antimicrobial resistance patterns."
http://cid.oxfordjournals.org/content/33/7/e69.full

In a 2005 case report, "Clinically Significant Kluyvera Infections," J. Elliot Carter, MD, and Tara N. Evans, MD, reported, "To determine the clinical significance of Kluyvera isolates at our institution, we retrospectively analyzed clinical microbiology data from January 1999 to September 2003. We identified 11 isolates classified as Kluyvera ascorbata, 7 of which were considered clinically significant pathogens: 3 cases represented urinary tract infections; 2, bacteremia; 1, a soft tissue infection of the finger; and 1, acute appendicitis with a subsequent intra-abdominal abscess. The age distribution of patients was wide, ranging from 2 months to 73 years. Antimicrobial susceptibility studies of the clinically significant and non–clinically significant Kluyvera isolates showed susceptibility patterns similar to those reported in the medical literature, namely trends of resistance to ampicillin and first- and second-generation cephalosporins. Of the 4 non–clinically significant isolates in our study, 1

was resistant to ciprofloxacin, a finding reported in only 1 other isolate of Kluyvera in the medical literature." http://ajcp.ascpjournals.org/content/123/3/334.full.pdf

In a 2010 case report, "Multidrug resistant Kluyvera ascorbata septicemia in an adult patient: a case report," Shannon Moonah, et al., Howard University Hospital at Washington DC, stated, "Kluyvera ascorbata is a gram negative microorganism belonging to the family Enterobacteriaceae. Although it causes infections infrequently, it is responsible for causing a wide range of infections including severe sepsis [1,2]. It is believed to be the source of genes encoding CTX-M-type extended spectrum B-lactamases (ESBLs) and it has the ability to transfer these genes to other Enterobacteriacae [3]. Only three cases of K. ascorbata isolated from the blood of adult patients have been reported [4-6]. We report what we believe to be the first case of a multidrug resistant K. ascorbata isolated from the blood of an adult patient with sepsis. – To the best of our knowledge, this is the first case report of a multidrug resistant Kluyvera ascorbata isolated from the blood in an adult patient with sepsis." http://www.jmedicalcasereports.com/content/4/1/197

In a 2011 study, "Kluyvera ascorbata Bacteremia and Meningitis: A Case Report and Review of the Literature," Thomas Walsh, MD, et al., stated, "Kluyvera is a small, motile, gram-negative bacillus within the Enterobacteriaceae family. It is part of the normal human flora of the respiratory tract and gastrointestinal tract. Although it was originally described as a benign colonizer and saprophyte, Kluyvera has also been described as causing a wide array of serious and potentially life-threatening infections and should not be discounted when isolated from clinical specimens. We report the case of a patient who developed Kluyvera ascorbata bacteremia and probable meningitis. It is the 17th reported case of Kluyvera bacteremia in the literature and would represent the first case of Kluyvera meningitis in a patient without a central nervous system shunt. We review the literature about reported cases of Kluyvera bacteremia and meningitis." http://journals.lww.com/infectdis/Abstract/2011/07000/Kluyvera_ascorbata_Bacteremia_and_Meningitis__A.6.aspx

Leclercia adecarboxylata

Leclercia (a coliform) is part of the Enterobacteriaceae family.

In a1997 study, "Leclercia adecarboxylata Infections: Case Report and Review," Zelalem Temesgen, et al., Mayo Clinic, Division of Infectious Diseases, reported, "Leclercia adecarboxylata has been rarely isolated from environmental and clinical specimens. On review of the world literature, we found two reports of L. adecarboxylata infection: one report described a patient with hepatic cirrhosis, and the other described a child dependent on total parenteral nutrition. L. adecarboxylata was isolated from five infected patients who were evaluated at our institution. Three patients had lower-extremity wound infections in which L. adecarboxylata was part of a mixed microbial growth. One patient had pneumonia due to multiple bacteria, including L. adecarboxylata, which were isolated from sputum. L. adecarboxylata was isolated from the blood of one patient with neutropenia and from the blood of the two patients reported in the literature. All patients except one had fever and leukocytosis. L. adecarboxylata isolates were susceptible to all the antimicrobials tested. L. adecarboxylata is most frequently isolated as part of a mixed microbial growth. Its role in these infections is not clear. However, the organism caused bacteremia in three patients." http://cid.oxfordjournals.org/content/25/1/79.abstract

In a 2000 report, "Leclercia adecarboxylata peritonitis in a child receiving chronic peritoneal dialysis," Oved Fattal and Jaime G. Deville, said, "A 5 year old boy with end-stage renal disease presented with clinical and laboratory findings of peritonitis. Peritoneal fluid revealed infection with Leclercia adecarboxylata. This is a motile, gram-negative bacillus, formerly designated enteric group 41 and Escherichia adecarboxylata. To our knowledge, this is the first reported case of peritonitis due to this organism." http://www.springerlink.com/content/t6455620xlx7hwdp/

In a 2001 letter reporting, "Isolation of Leclercia adecarboxylata from an Infant with Acute Lymphoblastic Leukemia," C. A. Longhurst and Daniel C. West, School of Medicine, University of California Davis, wrote, "Although Leclercia adecarboxylata was initially described in 1962 [1], reports of clinically significant infections involving this motile, gram-negative bacillus are uncommon. In the world's literature, 8 cases have been reported in which L. adecarboxylata was isolated from infected patients [2, 3]. In 4 of these cases, L. adecarboxylata was isolated from the blood of patients with an underlying medical condition (2 patients with hepatic cirrhosis, 1 child who was receiving long-term total parenteral nutrition, and 1 adult with neutropenia who had received a bone marrow transplant). In the other 4 cases, L. adecarboxylata was isolated from patients with mixed microbial infection (from lower extremity wound infections in 3 patients and from the sputum of 1 patient with adult Still's disease and pneumonia), which raises questions regarding the organism's role in these infections. We write to report another case involving an infant with acute lymphoblastic leukemia (ALL). In September 2000, we admitted to the hospital an 11-month-old girl with ALL and a chief complaint of chills and fever (temperature, 38.6°C). Her medical history was notable for a diagnosis of ALL at 4 months of age and multiple episodes of bacteremia during periods of neutropenia. The findings of a physical examination were significant for oropharyngeal mucositis, severe diaper dermatitis, and a small anal fissure." http://cid.oxfordjournals.org/content/32/11/1659.full

In another 2001 study, "Isolations of Leclercia adecarboxylata from a Patient with a Chronically Inflamed Gallbladder and from a Patient with Sepsis without Focus," Thierry de Baere, et al., Ghent University Hospital at Ghent, reported, "Leclercia adecarboxylata was isolated from a patient with a chronically inflamed gallbladder, together with Enterococcus sp. The organism was considered clinically significant and was susceptible to all antibiotics tested. Another strain of L. adecarboxylata was cultured from blood, together with Escherichia hermannii and E. faecalis, from a patient with sepsis."

In a 2008 study, "Leclercia adecarboxylata in an immunocompetent patient," Benjamin Hess, et al., Center for Family Medicine, at Sioux Falls, stated, "The infection reported in this case is of interest in that it is believed to be the first report in the literature of a pure culture – without other coinciding pathogens – of L. adecarboxylata from a wound infection of an immunocompetent patient. The success of this strain in infecting a healthy individual without the apparent aid of other organisms suggests that it may possess unique virulence factors that are absent from those strains responsible for the earlier reported cases. The day the culture results were reported, efforts were made to acquire a specimen for further biochemical and molecular analysis. Tragically, the culture had already been discarded by the clinical laboratory, per their routine practice for Enterobacteriaceae isolates." http://jmm.sgmjournals.org/content/57/7/896.full#aff-1

In a 2009 article, "Successful management of tunneled hemodialysis catheter-related bacteremia by Leclercia adecarboxylata without catheter removal: report of two cases," Mario Fernández-Ruiz, et al.,

University Hospital “12 de Octubre”, Madrid, said, “Leclercia adecarboxylata is a motile, aerobic, Gram-negative bacillus, previously known as enteric group 41 or Escherichia adecarboxylata.1 L. adecarboxylata strains are rarely isolated from clinical specimens of hospitalized patients and its actual relevance is not well established. Few cases of bacteremia in subjects with underlying medical conditions have been reported.1, 2, 3, 4 Catheter-related infection due to L. adecarboxylata represents an unusual complication of long-term, tunneled central venous catheters placed for total parenteral nutrition1 or chemotherapy.5 There are no previous reports of hemodialysis catheter-related bacteremia caused by L. adecarboxylata and successfully managed with catheter salvage. We report below the successful management of two such cases.” http://www.ijidonline.com/article/S1201-9712(09)00083-6/fulltext

In a 2011 study, “Leclercia Adecarboxylata Cellulitis in a Child with Acute Lymphoblastic Leukemia,” Avnee Shah, et al., Children’s Hospital of Philadelphia, reported, “Leclercia adecarboxylata is a rare, gram-negative rod that has been infrequently reported in the literature. The organism has been documented to cause solitary infections in immunocompromised hosts and polymicrobial wound infections in the immunocompetent. We present a case of an 8-year-old boy with significant past medical history of acute lymphobastic leukemia who developed cellulitis due to local infection by L. adecarboxylata. This case is presented to raise awareness of this rare organism’s ability to cause common cutaneous disease, especially in the immunocompromised.” http://onlinelibrary.wiley.com/doi/10.1111/j.1525-1470.2011.01348.x/abstract

Leminorella

Leminorella (a coliform) is part of the Enterobacteriaceae family.

In the 1985 study, “Leminorella, a New Genus of Enterobacteriaceae: Identification of Leminorella grimontii sp. nov. and Leminorella richardii sp. nov. Found in Clinical Specimens,” F. W. HICKMAN-BRENNER, et al., Centers for Disease Control at Atlanta, reported, “Leminorella is proposed as a new genus for the group of Enterobacteriaceae formerly known as Enteric Group 57. Strains of Leminorella gave positive tests for H2S production, acid production from L-arabinose and D-xylose, and tyrosine clearing; they were negative for indole production, Voges-Proskauer, urea hydrolysis, phenylalanine deaminase, motility, gelatin liquefaction, lysine and ornithine decarboxylases, arginine dihydrolase, growth in KCN, and acid production from adonitol, D-arabitol, cellobiose, erythritol, D-galactose, myo-inositol, lactose, maltose, D-mannitol, D-mannose, melibiose, a-CH3-glucoside, raffinose, L-rhamnose, salicin, D-sorbitol, sucrose, and trehalose. By DNA hybridization, strains of Leminorella were only 3 to 16% related to other Enterobacteriaceae and were divided into three groups. Leminorella grimontii is proposed as the type species for the genus and strain CDC 1944-81, ATCC 33999, is designated as the type strain. There were four strains of L. grimontii from stool specimens and two from urine specimens. L. richardii is proposed as the name for the second species (type strain, CDC 0978-82, ATCC 33998). All four L. richardii strains were from stool specimens. L. grimontii can be distinguished from L. richardii because it produces gas from glucose (100%) and acid from dulcitol (83%) and is methyl red positive (100%). One strain, CDC 3346-72, was more related to L. grimontii by DNA hybridization than to L. richardii, but the lower relatedness to both of these species indicated that it may be a third species. Biochemically it could not be distinguished from L. grimontii. All Leminorella strains were resistant (no zone of inhibition) to ampicillin, carbenicillin, and cephalothin. Some of the Leminorella strains were sent to us for

Salmonella serotyping, and two reacted weakly in Salmonella antisera. The clinical significance of Leminorella is unknown." http://jcm.asm.org/cgi/reprint/21/2/234.pdf

In a 2000 study, "Clinical Significance and Antibiotic Resistance Patterns of Leminorella spp., an Emerging Nosocomial Pathogen," LIDIA BLEKHER, et al., Tel Aviv Sourasky Medical Center, and Sackler Faculty of Medicine, Tel Aviv University, stated, "Although Leminorella spp., members of the family Enterobacteriaceae, were previously isolated from feces and urine specimens, clinical correlates have not been studied. We conducted a retrospective study to investigate the clinical significance and disease spectrum of these organisms, as well as their antibiotic susceptibility patterns. Identification and susceptibility testing were performed by an automated system. Eighteen cases were identified retrospectively during a 28-month period (1/97 to 4/99), representing an incidence of 11 cases per 100,000 patient admissions. The medical records of 14 patients were reviewed. The average patient age was 67 years, and 78% were males. Patients had multiple and diverse underlying conditions which might have predisposed them to infection. Leminorella spp. were classified as definite pathogens in 43% of the cases, probable pathogens in 29%, and possible pathogens in 21%. In one case of asymptomatic bacteriuria, the isolate had no clinical significance. All infections but one were nosocomial. Clinical syndromes included urinary tract infection in six patients, surgical site infection in three patients, and primary bacteremia, peritonitis, respiratory tract infection, and soft tissue infection in one patient each. Isolates were uniformly susceptible to imipenem. Other beta-lactam agents had poor activity against the isolates. We conclude that Leminorella spp. are significant nosocomial pathogens that are capable of causing a variety of clinical syndromes and are resistant to multiple antibiotic agents." http://jcm.asm.org/cgi/reprint/38/8/3036.pdf

In a 2005 study, "Spontaneous peritonitis caused by Leminorella grimontii," Maria Dalamaga, et al., said, "A case of spontaneous peritonitis caused by Leminorella grimontii in a 63-year-old man with cirrhosis is reported. To our knowledge, L. grimontii has never been reported as a cause of spontaneous bacterial peritonitis. The patient responded to antimicrobial therapy. Clinical and therapeutic implications are discussed." http://www.dmidjournal.com/article/S0732-8893(06)00119-2/abstract

Moellerella wisconsensis

Moellerella (a coliform) is a part of the Enterobacteriaceae family.

In a 1984 study, "Moellerella wisconsensis, a new genus and species of Enterobacteriaceae found in human stool specimens," F. W. Hickman-Brenner, et al., reported, "The name Moellerella wisconsensis is proposed for a group of the family Enterobacteriaceae previously called enteric group 46. The species name, wisconsensis, was coined because six of the nine strains were isolated in Wisconsin. M. wisconsensis strains were negative for indole production, Voges-Proskauer, H2S production, urea, phenylalanine deaminase, lysine and ornithine decarboxylases, arginine dihydrolase, gas production from D-glucose, acid production from trehalose, and motility; the strains were positive for methyl red, citrate (Simmons), and acid production from lactose and raffinose and resistant to colistin. DNAs from five strains of M. wisconsensis were highly related (80 to 93% in reactions assayed on hydroxyapatite at 60 degrees C and 78 to 97% at 75 degrees C) to 32P-labeled DNA of the proposed type strain (CDC 2896-78, ATCC 35017). Labeled DNA from this type strain was only 2 to 32% related (at 60 degrees C) to DNA from 49 strains of named and unnamed species of Enterobacteriaceae. Eight of nine M. wisconsensis strains were isolated from human stool samples. Clinical information on one strain was

available, and it was found to be associated with a case of diarrhea. On MacConkey agar, colonies of M. wisconsensis were bright red with precipitated bile around them and thus were indistinguishable from Escherichia coli colonies. Future studies should focus on the isolation of this new organism and its relationship to human disease." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC271095/

In a 1986 study, "The isolation of Moellerella wisconsensis from stool samples in the U.K.," A.R. Marshall, et al., said, "Three strains of Moellerella wisconsensis were isolated from a total of 400 stool specimens screened for this organism by means of a new selective medium developed in this laboratory. This is the first report of the isolation of this organism in the U.K. The exact role of M. wisconsensis in causing diarrhoea remains to be elucidated." http://www.ncbi.nlm.nih.gov/pubmed/3007629

In a 2009 case report, "First Case of Bacteremia Caused by Moellerella wisconsensis: Case Report and a Review of the Literature," A. Cardentey-Reyes, et al., stated, "Moellerella wisconsensis, a member of the Enterobacteriaceae family, is rarely isolated in clinical specimens. We report here a case of M. wisconsensis infection in a 46-year-old cirrhotic patient with acute cholecystitis. This is the first reported case of a M. wisconsensis infection in Belgium and the first reported case of human bacteremia caused by this bacterium. Our case report is followed by a review of the literature." http://www.springerlink.com/content/w74642t283425151/

In yet another 2009 report, "Isolation of Moellerella wisconsensis from blood culture from a patient with acute cholecystitis," A. I. Aller, et al., Hospital Universitario de Valme at Seville, said, "Moellerella wisconsensis is a Gram-negative bacillus of the family Enterobacteriaceae previously called Enteric Group 46 [1]. The name M. wisconsensis was proposed in 1984 by Hickman-Brenner et al. for strains from patients with diarrhoea, because the majority of the examined strains had been isolated from clinical specimens in the state of Wisconsin, USA. Isolates of M. wisconsensis have been recovered from animals, water and human food. Its pathogenicity in humans has not been established, although it has been isolated from clinical specimens other than stools, such as gall bladder tissue [2,3] or bile [4]. We report a case of isolation of this organism from blood culture." http://onlinelibrary.wiley.com/doi/10.1111/j.1469-0691.2009.03046.x/pdf

Moellerella in Animals

Moellerella (a coliform) is part of the Enterobacteria family.

In a 2002 report, "Moellerella wisconsensis Isolated from the Oral Cavity of a Wild Raccoon (Procyon lotor)," Rebecca F. Sandfort, et al., California Department of Health Services, Berkeley, said, "This report describes the isolation of Moellerella wisconsensis from the oral secretions of a wild raccoon in Northern California. Human enteric disease has previously been associated with this organism. This represents the first isolation of this rare enterobacterial species from a non-captive animal and only the third from a non-human source." http://www.liebertonline.com/doi/abs/10.1089/15303660260613765

In another 2009 report, "Isolation of Moellerella wisconsensis from the lung of a goat," F. CASALINUOVO and R. MUSARELLA, said, "This report describes the first isolation of Moellerella wisconsensis from the lung of a goat in Italy and represents the fourth isolation of this rare enterobacterial species from non-human sources reported in the literature. In fact, since its first isolation, M. wisconsensis has only been described in a few other occasions and often in clinical

samples associated with cases of human enteritis."
http://cat.inist.fr/?aModele=afficheN&cpsidt=21976698

Morganella

Morganella (a coliform) is part of the Enterobacteria family.

In a 1984 study, "Morganella morganii: Epidemiology of Bacteremic Disease," Carolyn McDermott and Joseph M. Mylotte, reported, "A retrospective review of microbiology records revealed 19 documented episodes of M. morganii bacteremia in 18 patients at a Veterans Administration hospital during a 5.5 year period. Thirteen of 19 bacteremias were related to nosocomial infections; 11 of the 13 nosocomial bacteremias occurred in surgical patients. Nine of the 13 patients with nosocomial bacteremia had received recent therapy with a beta-lactam antibiotic. The most common source of bacteremia was a postoperative wound infection (seven episodes). Only one episode was related to a urinary tract infection. Retrospective analysis showed that clusters of cases of M. morganii bacteremia had occurred almost yearly. This finding prompted a six-month period of prospective monitoring of all cultures for M. morganii to identify human reservoirs in our institution. Sixty percent of all cultures growing M. morganii came from urine cultures, 18% came from wound cultures, and the remaining 22% came from a variety of body fluids or tube drainage. Thirty-one percent of patients harboring M. morganii were on the Surgical Service. M. morganii bacteremia most commonly occurs in postoperative patients who receive beta-lactam antibiotics. From the data in this study, M. morganii is an infrequent cause of bacteremia, and its presence in blood cultures may be an indicator of an environment conducive for an outbreak of nosocomial infection." http://www.jstor.org/pss/30142630

In a 2003 letter to the Journal of Clinical Microbiology, "Morganella morganii-Associated Arthritis in a Diabetic Patient," Vikas Gautam, et al., Government Medical College and Hospital Sector 32 at Chandigarh, said, "Morganella morganii is a rare etiologic agent of septic arthritis (1-3; M. P. Jarrett and A. I. Grayzel, Letter, Arthritis Rheum. 23:128-129, 1980). In 1980, a 75-year-old man with a history of osteoarthritis had the first case of M. morganii septic arthritis of the knee. He responded to intravenous antibiotics, but his course was complicated by the development of synovitis secondary to gout and pseudogout (Jarrett and Grayzel, letter). The second case, in 1986, was that of a 53-year-old woman with severely deforming rheumatoid arthritis who responded well to antibiotics and closed drainage and subsequently underwent successful arthroplasty (1). The third case report described a 95-year-old man with a history of recurrent rectal adenocarcinoma who was hospitalized for evaluation of a persistent right shoulder effusion. The patient had slow resolution of the joint effusion when treated with antibiotics (3)." http://jcm.asm.org/cgi/content/full/41/7/3451

In a 2007 study, "Morganella Morganii Sepsis with Massive Hemolysis," Jong Hoon Kim, et al., Inje University Ilsan Paik Hospital at Goyang, reported, "Morganella morganii is a facultative gram-negative and anaerobic rod. It may be a cause of devastating infections in neonates and immunocompromised hosts. Some bacterial infections such as Clostridium and Vibrio are associated with hemolysis. However, massive hemolysis caused by M. morganii sepsis has not yet been reported. We observed a 59-yr-old man who had chemotherapy-induced neutropenia and was found to have massive hemolysis and metabolic acidosis due to sepsis. He died 6 hr after admission in spite of aggressive treatment. Two sets of blood cultures revealed the growth of M. morganii. We report here that M. morganii sepsis can cause fatal massive hemolysis leading to death."

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2694626/

In the 2009 Medscape article, "Morganella Infections," James R Miller, MD, stated:

> "Morganella morganii is a gram-negative rod commonly found in the environment and in the intestinal tracts of humans, mammals, and reptiles as normal flora. Despite its wide distribution, it is an uncommon cause of community-acquired infection and is most often encountered in postoperative and other nosocomial settings. M. morganii infections respond well to appropriate antibiotic therapy; however, its natural resistance to many beta-lactam antibiotics may lead to delays in proper treatment.
>
> The genus Morganella belongs to the tribe Proteeae of the family Enterobacteriaceae. The Proteeae, which also include the genera Proteus and Providencia, are important opportunistic pathogens capable of causing a wide variety of nosocomial infections. Currently, Morganella contains only a single species, M. morganii, with 2 subspecies, morganii and sibonii. M. morganii was previously classified under the genus Proteus as Proteus morganii.
>
> In the late 1930s, M. morganii was identified as a cause of urinary tract infections. Anecdotal reports of nosocomial infections began to appear in the literature in the 1950s and 1960s. Tucci and Isenberg reported a cluster epidemic of M. morganii infections occurring over a 3-month period at a general hospital in 1977.[1] Of these infections, 61% were wound infections and 39% were urinary tract infections.
>
> In 1984, McDermott reported 19 episodes of M. morganii bacteremia in 18 patients during a 5.5-year period at a Veterans Administration hospital.[2] Eleven of the episodes occurred in surgical patients. The most common source of bacteremia was postoperative wound infection, and most infections occurred in patients who had received recent therapy with a beta-lactam antibiotic. Other important epidemiological risk factors in these studies included the presence of diabetes mellitus or other serious underlying diseases and advanced age." http://emedicine.medscape.com/article/222443-overview

In a 2010 case report, "Morganella morganii septicemia and dermopathy," MELVER L. ANDERSON, MD, FACP and NASSRENE Y. ELMADHUN, MD, stated:

> "M. morganii is an increasingly common cause of nosocomial infection that presents as a life-threatening, community-acquired disease with severe dermatological manifestations. This case highlights the potential for M. morganii to affect nonimmunocompromised hosts through what would otherwise be viewed as a mild inoculum. M. morganii may be resistant to β-lactam antibiotics commonly selected for the empiric treatment of skin and soft tissue infections. It is a gram-negative rod found in soil and water and is present in normal fecal flora in humans and other mammals and in reptiles.
>
> Although a rare human pathogen, M. morganii has been reported as a cause of urinary tract infections, nosocomial surgical wound infections, peritonitis, CNS infection, endophthalmitis, pneumonia, chorioamnionitis, neonatal sepsis, pyomyositis, necrotizing fasciitis, and arthritis. Numerous cases of nosocomial infection have been described, usually as postsurgical wound infections or urinary tract infections. Patients in whom

bacteremia develops are typically immunocompromised, diabetic, or elderly or have at least 1 serious underlying disease.1 The overall mortality associated with M. morganii bacteremia is as high as 38%.2 Two dermatological observations reported with M. morganii bacteremia include hemorrhagic bullae and ecthyma gangrenosum–like eruptions.3,4." http://www.consultant360.com/content/morganella-morganii-septicemia-and-dermopathy

In a 2010 study, "[Meningoencephalitis due to Morganella morganii: a case report]," Ndiaye M, et al., said, "A central nervous system infection due to Morganella morganii is uncommon. We report a case diagnosed at the neurological department of Fann teaching hospital in Dakar, Senegal. A 12-year-old boy was hospitalized for acute meningoencephalitis. The CT scan was normal and the study of cerebrospinal fluid (CSF) revealed cytological and biochemical abnormalities and M. morganii. HIV and syphilitic serologies were negative and blood CD4 lymphocyte count showed 354 per mm(3). The treatment with cefotaxime associated with gentamicin for 6 weeks was successful. The outcome of infection depends on many factors such as the onset and quality of treatment, the virulence of the germ and the status of immune system." http://www.ncbi.nlm.nih.gov/pubmed/20431984

In a 2011 study, "Neonatal Morganella morganii sepsis: a case report and review of the literature," Hung-Yang Chang, et al., stated, "Neonatal sepsis remains a potentially lethal condition, especially in preterm neonates. Early-onset neonatal sepsis (EOS) occurs as a multisystemic illness in the first three days of life and is associated with the acquisition of microorganisms from the mother. The microorganisms most commonly associated with early-onset sepsis include group B Streptococcus, Escherichia coli, Haemophilus influenzae, and Listeria monocytogenes. Late-onset sepsis (LOS) syndrome occurs at 7–90 days of life and is often acquired from the environment. Organisms that have been implicated in causing LOS syndrome include coagulase-negative staphylococci, Staphylococcus aureus, E coli, Klebsiella, Pseudomonas, Enterobacter, Candida, and anaerobes. However, extensive use of intrapartum antibiotics has been associated with major changes in the spectrum of organisms involved and their susceptibility to antibiotics. Morganella morganii is rarely encountered but may cause serious invasive infection in the pregnant woman or neonate.1–10 This organism is characteristically resistant to many beta-lactam antibiotics which may lead to delays in proper treatment. We report an extremely premature infant with fatal early-onset M. morganii sepsis and provide a literature review of this disease." http://onlinelibrary.wiley.com/doi/10.1111/j.1442-200X.2010.03241.x/full

In a 2011 study, "Infected abdominal aortic aneurysm due to Morganella morganii: CT findings. [Case Reports, Journal Article]" O.Y. Kwon, et al., reported, "An infected aortic aneurysm, or mycotic aneurysm, is a rare arterial dilatation due to destruction of the infected vessel wall. Common pathogens resulting in an infected aortic aneurysm are Salmonella and Clostridium species, as well as Staphylococcus aureus; Morganella morganii, on the other hand, is very rare. An infected abdominal aortic aneurysm has tendencies to grow rapidly and to rupture. The mortality rate is high in patients undergoing emergent surgical intervention. We report the case of a 65-year-old man who presented with an infected abdominal aortic aneurysm caused by M. morganii. A high index of suspicion and imaging tests are necessary in order to diagnose an infected aortic aneurysm." http://www.unboundmedicine.com/medline/ebm/record/20352211/full_citation/Infected_abdominal_aortic_aneurysm_due_to_Morganella_morganii:_CT_findings_

Pantoea

Pantoea (a coliform) is a part of the Enterobacteriacea family.

In a 2000 case report, "Isolation of Pantoea agglomerans in Two Cases of Septic Monoarthritis after Plant Thorn and Wood Sliver Injuries," C. DE CHAMPS, et al., reported, "Arthritis after plant injury is often apparently aseptic. We report two cases due to Pantoea agglomerans. In one case, the bacterium was isolated only from the pediatric blood culture media, BACTEC Peds Plus, monitored in BACTEC 9240, and not from the other media inoculated with the joint fluid. This procedure could help improve the diagnosis of septic arthritis. – P. agglomerans, formerly named Enterobacter agglomerans (5), is a ubiquitous Enterobacteriaceae that is found in plants and in the feces of humans and animals. It is less often implicated in infection than Enterobacter aerogenes and Enterobacter cloacae and usually complicates debilitating illnesses (10). In the absence of preexisting disease, septic arthritis is rare, particularly in children or infants (6). Of the 294 patients admitted for septic arthritis to our hospital between 1979 and 1998, the two in this report were the only cases in which P. agglomerans was isolated." http://jcm.asm.org/cgi/reprint/38/1/460.pdf

In a 2007 study, "Pantoea agglomerans, a Plant Pathogen Causing Human Disease," Andrea T. Cruz, et al., Emergency Medicine, Baylor College of Medicine at Houston, said, "We present 53 pediatric cases of Pantoea agglomerans infections cultured from normally sterile sites in patients seen at a children's hospital over 6 years. Isolates included 23 from the bloodstream, 14 from abscesses, 10 from joints/bones, 4 from the urinary tract, and 1 each from the peritoneum and the thorax. P. agglomerans was most associated with penetrating trauma by vegetative material and catheter-related bacteremia." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1933083/

In another 2009 case report, "Six cases of sepsis caused by Pantoea agglomerans in a teaching hospital," Maria Carla Liberto, et al., Department of Medical Sciences, University "Magna Græcia" at Catanzaro, said, "Pantoea agglomerans is member of Enterobacteriaceae that inhabits plants, soil, water and such species includes bacteria reported as both commensal and pathogen of animals and humans (Gavini et al., 1989). The most common infection caused by P. agglomerans is septic arthritis or synovitis (Kratz et al., 2003), but Pantoea has been also involved in nationwide epidemic of septicaemia due to contaminated intravenous products (Mackel et al., 1975), an outbreak secondary to contaminated parental nutrition (Habsah H et al., 2005), osteitis (Laporte et al., 2002), colelithiasis (Flores et al., 2003), occupational respiratory infections and skin allergy (Milanowski et al., 2003), blood stream infection in an elderly person (De Baere et al., 2004) and peritonitis (Lim et al., 2006). Pantoea spp are clearly opportunistic pathogens and rarely cause disease in the otherwise healthy individuals (Sanders and Sanders, 1997). – Here we report on a 6 case outbreak in a teaching hospital. Within three months P. agglomerans was isolated from blood cultures of 5 patients from oncology and 1 patient from ICU departments. P. agglomerans was in pure culture in 5 cases, while in the last one Rahnella aquatilis and Candida famata were also isolated. Therefore, P. agglomerans is able to produce nosocomial infections in patients with primary pathology often associated with immune suppression." http://www.microbiologica.net/mb/pdf/2009/1/0148_Micro1_17_Liberto.pdf

In a 2011 case report, "Pantoea agglomerans pneumonia in a heart–lung transplant recipient: case report and a review of an emerging pathogen in immunocompromised hosts." A. Shubov, et al., reported, "Pantoea agglomerans is a gram-negative rod that is frequently found on the exterior of many

plants, fruits, vegetables, and in soil, and it is used as a biopesticide in the agriculture industry. Recent reports have implicated P. agglomerans in systemic infections of immunocompromised hosts and neonates, as well as more localized infections in healthy hosts. P. agglomerans as a cause of hospital-acquired pneumonia has not been well characterized. We report a case of P. agglomerans pneumonia in a heart–lung transplant recipient following transplantation. The organism was susceptible to multiple antimicrobial agents and treated successfully with ertapenem. We review the patient's course and the relevant literature, and discuss implications for the future."
http://onlinelibrary.wiley.com/doi/10.1111/j.1399-3062.2011.00630.x/abstract

Pantoea in Plants

In a 2009 study, "Pantoea ananatis: an unconventional plant pathogen," T.A. Coutinho and S.N. Venter, Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria at Pretoria, reported, "Pantoea ananatis causes disease symptoms in a wide range of economically important agricultural crops and forest tree species worldwide. It is regarded as an emerging pathogen based on the increasing number of reports of diseases occurring on previously unrecorded hosts in different parts of the world. Its unconventional nature lies in the fact that, unlike the majority of plant pathogenic microbes, P. ananatis is capable of infecting humans and occurs in diverse ecological niches, such as part of a bacterial community contaminating aviation jet fuel tanks and contributing to growth promotion in potato and pepper." http://www.ncbi.nlm.nih.gov/pubmed/19400836?ordinalpos=1&itool=PPMCLayout.PPMCAppController.PPMCArticlePage.PPMCPubmedRA&linkpos=5

In a 2009 study, "Recent evolution of bacterial pathogens: the gall-forming Pantoea agglomerans case," I. Barash and S. Manulis-Sasson, Department of Plant Sciences, Faculty of Life Sciences, Tel-Aviv University, said, "Pantoea agglomerans, a widespread epiphyte and commensal bacterium, has evolved into an Hrp-dependent and host-specific tumorigenic pathogen by acquiring a plasmid containing a pathogenicity island (PAI). The PAI was evolved on an iteron plasmid of the IncN family, which is distributed among genetically diverse populations of P. agglomerans. The structure of the PAI supports the premise of a recently evolved pathogen. This review offers insight into a unique model for emergence of new bacterial pathogens. It illustrates how horizontal gene transfer was the major driving force in the creation of the PAI, although a pathoadaptive mechanism might also be involved. It describes the crucial function of plant-produced indole-3-acetic acid (IAA) and cytokinines (CK) in gall initiation as opposed to the significant but secondary role of pathogen-secreted phytohormones. It also unveils the role of type III effectors in determination of host specificity and evolution of the pathogen into pathovars. Finally, it describes how interactions between the quorum sensing system, hrp regulatory genes, and bacterially secreted IAA or CKs affect gall formation and epiphytic fitness."
http://www.ncbi.nlm.nih.gov/pubmed/19400643

Photorhabdus

In a 1999 study, "Isolation, Identification, and Molecular Characterization of Strains of Photorhabdus luminescens from Infected Humans in Australia," Margaret M. Peel, et al., The University of Melbourne at Parkville, reported, " This is the first report of the isolation of P. luminescens from infected humans in Australia and the second report of the isolation of this species from infected humans worldwide. – We describe the isolation of Photorhabdus (Xenorhabdus) luminescens from four

Australian patients: two with multiple skin lesions, one with bacteremia only, and one with disseminated infection. One of the patients had multiple skin lesions following the bite of a spider, while the lesions in the other patient were possibly associated with a spider bite. The source of infection for the remaining two patients is unknown. As a member of the family Enterobacteriaceae, P. luminescens is unusual in that it fails to reduce nitrate and ferments only glucose and mannose." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC85716/

In the February 2003 edition of CDC's Emerging Infectious Diseases article, "Photorhabdus Species: Bioluminescent Bacteria as Human Pathogens?" John G. Gerrard, et al., Gold Coast Hospital at Southport, Queensland, reported, "We report two Australian patients with soft tissue infections due to Photorhabdus species. Recognized as important insect pathogens, Photorhabdus spp. are bioluminescent gram-negative bacilli. Bacteria belonging to the genus are emerging as a cause of both localized soft tissue and disseminated infections in humans in the United States and Australia. The source of infection in humans remains unknown." http://wwwnc.cdc.gov/eid/article/9/2/02-0222_article.htm

In a 2005 case report, "Photorhabdus asymbiotica, a Pathogen Emerging on Two Continents That Proves that There Is No Substitute for a Well-Trained Clinical Microbiologist," Alice S. Weissfeld, et al., Microbiology Specialists Incorporated, Houston, said, "A 54-year-old ranch hand presented to the emergency room with an alleged spider bite and multiple abscesses. Both wound and blood cultures grew Photorhabdus asymbiotica, an enteric gram-negative rod that was initially misidentified by the hospital's rapid identification system. Clinical laboratories should be aware of the limitations of their rapid identification systems and always use them as an adjunct to analysis of morphological and phenotypic traits." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1234010/

In another 2009 study, "The Emerging Human Pathogen Photorhabdus asymbiotica Is a Facultative Intracellular Bacterium and Induces Apoptosis of Macrophage-Like Cells," S. C. P. Costa, et al., Université Montpellier II, UMR1133 Laboratoire EMIP, at Montpellier, reported, "Photorhabdus species are gram-negative entomopathogenic bacteria of the family Enterobacteriaceae. Among the different members of the genus, one species, Photorhabdus asymbiotica, is a pathogen of both insects and humans. The pathogenicity mechanisms of this bacterium are unknown. Here we show that P. asymbiotica is a facultative intracellular pathogen that is able to replicate inside human macrophage-like cells. Furthermore, P. asymbiotica was shown for the first time in an intracellular location after insect infection. We also demonstrated that among Australian and American clinical isolates, only the Australian strains were able to invade nonphagocytic human cells. In cell culture infection experiments, Australian clinical isolates as well as cell-free bacterial culture supernatant induced strong apoptosis of a macrophage cell line at 6 h postinfection. American isolates also induced cellular death, but much later than that induced by Australian ones. Mammalian cultured cells analyzed for key features of apoptosis displayed apoptotic nuclear morphology, activation of the initiator caspases 8 and 9 and the executioner caspases 3 and 7, and poly(ADP-ribose) polymerase proteolysis, suggesting activation of both the intrinsic and extrinsic apoptotic pathways,"
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2643617/

Photorhabdus in Insects

In a 2009 study, "Photorhabdus asymbiotica with the insect pathogen Photorhabdus luminescens," Paul

Wilkinson, et al., University of Exeter in Cornwall at Penryn, reported, “The Gram-negative bacterium Photorhabdus asymbiotica (Pa) has been recovered from human infections in both North America and Australia. Recently, Pa has been shown to have a nematode vector that can also infect insects, like its sister species the insect pathogen P. luminescens (Pl). To understand the relationship between pathogenicity to insects and humans in Photorhabdus we have sequenced the complete genome of Pa strain ATCC43949 from North America. This strain (formerly referred to as Xenorhabdus luminescens strain 2) was isolated in 1977 from the blood of an 80 year old female patient with endocarditis, in Maryland, USA. Here we compare the complete genome of Pa ATCC43949 with that of the previously sequenced insect pathogen P. luminescens strain TT01 which was isolated from its entomopathogenic nematode vector collected from soil in Trinidad and Tobago.” http://www.biomedcentral.com/1471-2164/10/302

Providencia

Providencia (a coliform) is part of the Enterobacteriaceae family.

In a 1976 study, “PROVIDENCIA STUARTII, A HOSPITAL PATHOGEN: POTENTIAL FACTORS FOR ITS EMERGENCE AND TRANSMISSION,” RICHARD P. WENZEL, et al., reported, “The emergence of Providencia stuartii as a hospital pathogen in a burn unit was demonstrated by routine infection surveillance. The organism was initially recognized in a burn wound and subsequently in urine or sputum. Compared to controls, those patients harboring P. stuartii were similar in age and percentage of body surface burned and were more likely to have been in one of the two burn unit rooms, ($p < 0.02$). Infection with P. stuartii was independent of duration in the Intensive Care Unit or Burn Unit, and of number of visits to hydrotherapy or operating rooms (OR). Once patients were colonized with P. stuartii they had greater morbidity than non-colonized patients as evidenced by longer stays in the unit and increased visits to the OR for debridement. P. stuartii was isolated from air samples (5/14) more commonly than from the hands of personnel. In vitro tests suggested that extensive use of parenteral gentamicin and replacement of the antibacterial topical cream sulfamylon by silver sulfadiazine favored the emergence of P. stuartii over Pseudomonas aeruginosa as the predominant colonizing organism.” http://aje.oxfordjournals.org/content/104/2/170.abstract

In a 1980 study, “Nosocomial Multiply Resistant Providencia stuartii: a Long-Term Outbreak with Multiple Biotypes and Serotypes at One Hospital,” F. E. KOCKA, et al., The Chicago Medical School at North Chicago, said, “A long-term outbreak of urinary tract-associated multiply resistant Providencia stuartii occurred in a large medical facility that included a 513-bed chronic care unit. The unique characteristics of this outbreak were that from within a single medical facility, P. stuartii with multiple serotypes, biotypes, and antibiograms could be identified. The organisms isolated had five different biotypes, seven different antibiograms, and two major serotypes. All of the organisms were susceptible to amikacin, cefamandole, and cefoxitin. Application of standard infection control measures impeded the spread of this outbreak, and it slowly terminated 16 months later.” http://jcm.asm.org/cgi/reprint/11/2/167.pdf

In a 1986 study, “Providencia stuartii: A Common Cause of Antibiotic-Resistant Bacteriuria in Patients with Long-Term Indwelling Catheters,” John W. Warren, stated, “The long-term-catheterized urinary tract may offer a particular niche to Providencia stuartii, which is otherwise an uncommon clinical isolate. Published accounts of bacteriuria in patients catheterized for long periods indicate that P.

stuartii has often been found as frequently as familiar uropathogens such as Escherichia coli, Proteus mirabilis, enterococcus, and Pseudomonas aeruginosa. As in most nosocomial infections, the frequency of isolation of a given species has commonly differed among institutions. In the future P. stuartii may be more frequently encountered as a nosocomial pathogen in nursing homes and in acute care hospitals to which bacteriuric patients are transferred. This trend appears likely because of the increasingly large nursing-home population, the predilection of the bacterium for the long-term-catheterized urinary tract, the opportunity for nosocomial transmission from this reservoir, the resistance of the organism to multiple antibiotics, and the occasional systemic illness and bacteremia caused by P. stuartii." http://www.jstor.org/pss/4453809

In a 2004 study, "ESBL-producing multidrug-resistant Providencia stuartii infections in a university hospital," Mario Tumbarello1, et al., Università Cattolica at Roma, reported, "All consecutive episodes of P. stuartii infection that occurred during 1999–2002 were included in the study. For each patient, we recorded the area of hospitalization and drug susceptibility of the P. stuartii strains. Patients with ESBL-producing P. stuartii infection were considered cases and those with non-ESBL-producing P. stuartii infection were used as controls. – This 4 year surveillance of Providencia complaints clearly indicates that infections caused by ESBL-producing multidrug-resistant P. stuartii are an emerging problem." http://jac.oxfordjournals.org/content/53/2/277.full

In a 2009 Medscape article, "Providencia Infections," Evan G Brown, DO, said:

> "Kauffmann first proposed the genus name Providencia in 1951, referring to a group of organisms studied by Stuart and colleagues at Brown University in Providence, Rhode Island. By 1983, the 4 species in the Providencia genus at that time were fully differentiated with DNA hybridization and urea hydrolyzation. In 1986, Providencia heimbachae was the fifth species discovered.[1] The 5 species currently in the genus Providencia, in descending order of prevalence, include Providencia stuartii, Providencia rettgeri, Providencia alcalifaciens, Providencia rustigianii, and P heimbachae. – The genus Providencia includes urease-producing gram-negative bacilli that are responsible for a wide range of human infections. Although most Providencia infections involve the urinary tract, they are also associated with gastroenteritis and bacteremia. Providencia infections are uncommon and are usually nosocomial. They represent an emerging problem because of the increasing prevalence of antibiotic resistance secondary to extended-spectrum beta-lactamase (ESBL). – In humans, Providencia species have been isolated from urine (most common), stool, and blood, as well as from sputum, skin, and wound cultures. P stuartii septicemia is primarily of urinary origin. One case study has described P stuartii as the etiology of infective endocarditis.[4] Another case report found P rettgeri to be a cause of ocular infections, including keratitis, conjunctivitis, and endophthalmitis.[5] – Providencia species are found in multiple animal reservoirs, including flies, birds, cats, dogs, cattle, sheep, guinea pigs, and penguins, and are resident oral flora in reptiles such as pythons, vipers, and boas. Providencia species are also found commonly in soil, water, and sewage. Examples of Providencia infections in animals include neonatal diarrhea due to P stuartii infection in dairy cows and enteritis caused by P alcalifaciens infection in dogs. P rettgeri has been isolated in crocodiles with meningitis/septicemia and in chickens with enteritis.[2]P heimbachae has been isolated in penguin feces and an aborted bovine fetus.[3] "
> http://emedicine.medscape.com/article/226541-overview

In a 2010 study, "Bacterial Pericarditis due to Providencia stuartii An Atypical Case of Relapsing Pericarditis," Caterina Simon, MD, Department of Internal Medicine (M.D., A.B., S.F.C., M.S., F.S.), Radiology (P.B.), Pathology (S.P.), and Cardiovascular Surgery (C.S., P.F.), Ospedali Riuniti di Bergamo, reported, "A 58-year-old man was admitted for relapsing pericarditis. His past medical history included a transient ischemic attack when he was 47 years old, radical right nephrectomy for neoplasia 8 years earlier, and hypertension. – This is the first report of bacterial pericarditis due to P stuartii.2,3 The clinical course resembled that of recurrent "idiopathic" pericarditis, with the exception that fever and pain recurred soon after aspirin discontinuation."
http://circ.ahajournals.org/content/122/4/e401.full

In a 2011 study, "Nosocomial Providencia stuartii Meningitis: A Case Report," Ayhan Tekiner, et al., Neurosurgery Department, Ankara Training and Research Hospital at Ankara, said. "Providencia stuartii is an opportunistic pathogen and may cause health-care infections. They mostly cause urinary-catheter-related infections. Meningitis associated with this bacterium is extremely rare. Here we report a P. stuartii meningitis in a patient with external lumbar drainage in the neurosurgery unit. A fifty-seven-year old male patient was admitted to the neurosurgery department with headache and confusion. There was a subarachnoidal hemorrhage on computerized tomography (CT) scan and he was transferred to the intensive care unit. His neurological evaluation showed a grade 3a mental status according to the Yasargil classification and a Glasgow coma scale of 14. The CT-angiography and digital subtraction angiography revealed multiple arterial aneurisms. Coil embolization was made for three of the aneurisms. Since the patient had hydrocephalus on his follow-up on the 14th day, a lumbar drainage (LD) catheter was inserted. Daily cerebrospinal fluid (CSF) analyses were performed. On the 7th day of the LD the CSF findings revealed meningitis and P. stuartii was revealed from the three subsequent CSF cultures. The LD was removed and daily lumbar punctures and CSF cultures were performed. On the 7th day of his antibiotic therapy his laboratory findings returned to normal levels. Following CSF cultures were negative. The antibiotic therapy continued to 21 days. His meningitis was cured. To the authors knowledge there were only two patients with P. stuartii meningitis in the literature and this is the third one." http://www.neurores.org/index.php/neurores/article/viewArticle/14/9

Rahnella aquatilis

Rahnella (a coliform) is a part of the Enterobacteriaceae family.

In a 1979 study, "[Rahnella aquatilis, a new member of the Enterobacteriaceae (author's transl)]." D. Izard, et al., reported, "A DNA-DNA hybridization study was carried out to determine the taxonomic position of a new group of enterobacteria (group H2) previously studied by numerical taxonomy. All the strains of this group revealed relatively high reassociation binding ratios with the centrotype; 82% of the strains of the group showed more than 69% of reassociation with the centrotype. In spite of numerical taxonomy conclusions, there was no genetic relationship with the species Enterobacter cloacae (higher reassociation binding ratio: 37%). No significant genetic relationship with the other groups of enterobacteria was found. Rahnella aquatilis was defined from phenotypic and genetic data. The strain 133 (CIP 78-65) is proposed as type strain of the species."
http://www.ncbi.nlm.nih.gov/pubmed/484990

In a 1994 study, "Surgical wound infection caused by Rahnella aquatilis," S Maraki, et al., University Hospital of Heraklion at Crete, said, "Rahnella aquatilis is a water-residing gram-negative rod, a

member of the family Enterobacteriaceae, isolated rarely from clinical specimens of immunocompromised patients. A case of a surgical wound infection caused by R. aquatilis in a patient who underwent a prosthetic surgical intervention is reported. The presence of inducible beta-lactamase was suggested by the disk induction test and the conventional agar dilution assay. Literature on R. aquatilis infections in humans is reviewed."

In a 1996 study, "Infective endocarditis caused by an unusual gram-negative rod,Rahnella aquatilis," H. Matsukura, et al., reported on an "An 11-month-old girl with congenital heart disease developed infective endocarditis. Blood cultures revealed an unusual gram-negative rod,Rahnella aquatilis. The patient was successfully treated with a combination of netilmicin and ceftazidime. This is the first case report of infective endocarditis caused by this organism. R. aquatilis should be recognized as a clinical pathogen capable of causing life-threatening infection in children and adults."
http://www.springerlink.com/content/g4002g09n622k80l/

In a 2005 case report, "Rahnella aquatilis Bacteremia from a Suspected Urinary Source," Kaley Tash, Florida Infectious Disease Institute at Tampa, said, "A 76-year-old male with prostatic hyperplasia presented with acute pyelonephritis. Blood cultures yielded Rahnella aquatilis. Treatment with intravenous followed by oral levofloxacin resulted in cure. Important characteristics of this organism include its biochemical similarities to Enterobacter agglomerans, its apparent ability to cause bacteremia from a renal focus, and its response to quinolone therapy."
http://jcm.asm.org/cgi/reprint/43/5/2526.pdf

In a 2009 case report, "ISOLATION OF RAHNELLA AQUATILIS FROM BONE AND SOFT TISSUE OF A FOOT OF A PATIENT WITH DIABETES (CASE REPORT)," Elif AKTAŞ, et al., Zonguldak Karaelmas University, stated, "This is a case report on the first isolation of Rahnella aquatilis, a very rare enteric Gram negative rod, from bone and soft tissue of a foot of a patient with diabetes. Previous reports of isolation of R. aquatilis from patients are also summarized. It is concluded that clinical microbiologists must be aware of the diff erential characteristics of this rare microorganism, which is likely to be resistant to ampicillin and cephalothin, particularly in immunocompromised patients." http://tmc.dergisi.org/pdf/pdf_TMC_347.pdf

In a 2010 study, "Infection caused by Rahnella aquatilis," J. I. Gaitán, et al., University of Oklahoma Health Sciences Center at Oklahoma City, said, "The authors report a case of a 27-year-old African American woman with sickle cell disease who developed septic shock caused by a Rahnella aquatilis infection associated with a peripherally inserted central venous catheter. The infection was treated successfully with ciprofloxacin. R. aquatilis is a Gram-negative rod, first isolated from freshwater in 1976, which has been linked to human disease in rare instances, most commonly in immunosuppressed individuals. In addition to this case report, the authors also review the literature."
http://www.ncbi.nlm.nih.gov/pubmed/20545014

Serratia

Serratia (a coliform) is part of the Enterobacteriaceae family. It has a colorful history in religion, before it became pathogenic to humans, plants and coral.

Victor L. Yu, M.D., outlines some of the history in the article "**Serratia marcescens: Masquerader of**

Blood." Yu said, "Some strains of S. marcescens are capable of producing pigment, the intensity of which ranges from dark red to pale pink, depending on the age of the colonies. The pigment can be present after incubation at room temperature but usually disappears after subculturing. The pigment was extractred by 1902 and named "prodigiosin . – Serratia marcescens has a predilection for growth on foodstuffs, especially of the starchy variety, where the pigmented colonies were easily mistaken for drops of blood. As early as the sixth century B.C., Pythagoras had noted the appearance of a bloody concentration on foodstuffs, and in the 1800's, Ehrenberg uncovered almost 100 historical references to the miraculous appearance of blood on food."
http://www.antimicrobe.org/h04c.files/history/serratia.pdf

According to Attorneys Fred Pritzker's webpage, "Serratia Marcescens: Attorneys for Hospital Infection Lawsuit, – At least 9 people have died who had a Serratia marcescens infection due to contaminated IV bags at Alabama hospitals. – The following are some possible health effects of a Serratia marcescens infection:
•Bacteremia (blood infection, also called blood poisoning) and septicemia (severe bacteremia that can arise from from infections throughout the body, including infections in the lungs, abdomen, and urinary tract)
•Urinary tract infections
•Septic arthritis (bacteremia and septicemia can cause septic arthritis)
•Empyema (collection of pus)
•Lymphadenitis (inflammation of a lymph node)
•Endocarditis (inflammation of the heart—muscle, lining, valves)
•Meningitis (inflammation of the meninges, the protective membranes of the brain and spinal cord)
•Peritonitis (inflammation of the peritoneum, the membrane that lines the abdominal wall and covers most of the organs of the body)
•Fever
•Respiratory distress
•Shock
•Convulsions"
http://www.pritzkerlaw.com/section-unsafe-medical/medicines/heparin-serratia-marcescens-lawsuit.html

In a 1970 study, "Serratia marcescens: Biochemical, Serological, and Epidemiological Characteristics and Antibiotic Susceptibility of Strains Isolated at Boston City Hospital," James N. Wilfert, et al., Boston City Hospital, Harvard Medical School, National Communicable Disease Center, reported, "The biochemical, serological, and epidemiological characteristics of 95 strains of Serratia marcescens isolated at the Boston City Hospital were examined. Several strains were shown to be endemic, and the majority of isolates were cultured from urine or respiratory secretions. Serratia species were highly resistant to polymyxin B and the cephalosporins, and various proportions were also resistant to other antibiotics including kanamycin, but all of the isolates were sensitive to gentamicin. The appearance of resistance to kanamycin and nalidixic acid among endemic strains was demonstrated. The nosocomial nature of Serratia infections, particularly those involving the urinary tract, was confirmed. Many clinical bacteriology laboratories currently fail to identify the nonpigmented strains."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC376681/pdf/applmicro00100-0163.pdf

In a 1980 study, "ANTIBIOTIC-SENSITIVE SERRATIA MARCESCENS INFECTIONS

COMPLICATING CARDIOPULMONARY OPERATIONS: CONTAMINATED DISINFECTANT AS A RESERVOIR," N. Joel Ehrenkranz, et al., South Florida Hospital Consortium for Infection Control at Miami, said, "A cluster of Serratia marcescens infections complicating cardiopulmonary bypass operations was traced to contaminated quaternary ammonium disinfectant. Failure of hospital personnel to clean the disinfectant spray bottles before refilling them had enabled the organisms to survive and contaminate the environment, including the extracorporeal circulator. The organisms grew in two of four formulations of quaternary ammonium disinfectant. Serratia sensitivity to ampicillin and tetracycline was an epidemiological marker of a common- source outbreak." http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(80)92349-1/abstract

In a 1987 study, "Epidemic outbreak of Serratia marcescens infection in a cardiac surgery unit." I. Wilhelmi, et al., reported, "Between 2 February and 16 April 1985, an outbreak of Serratia marcescens infection involving 10 male patients occurred in a cardiac surgery unit. All the patients had surgical wound infection, five also had osteomyelitis (four sternal, one costal), and another had peritonitis secondary to peritoneal dialysis. Three patients had concomitant bacteremia. All Serratia strains isolated produced a cherry-red pigment, and all had the same biochemical and antibiotic susceptibility pattern. An intensive search for the origin of the outbreak was initially unsuccessful, and it proved impossible to isolate S. marcescens from cultures of numerous samples taken from hospital personnel and from the environment. The fact that all patients were male and had been shaved for surgery by the same team of barbers led us to investigate the shaving procedures. We finally isolated a strain of pigmented S. marcescens, corresponding to that involved in the outbreak, from samples taken from the hands and equipment of the barbers. After suitable action had been taken, the epidemic terminated." http://www.ncbi.nlm.nih.gov/pmc/articles/PMC269197/

In a 1993 Woodrow Wilson Biology Institute class outline, Micrococcus roseus or Serratia marcescens are used by students in "DEMONSTRATING AN EPIDEMIC," The course outline states, "This experiment allows students to experience a small scale "epidemic," demonstrating the ease with which disease organisms are spread, and enables the student to determine the originator of the "epidemic." Students will transfer live bacteria by hand contact, then transfer an inoculum to a nutrient agar plate for 24 hour incubation. After incubation, plates are observed for growth of the microbial agent. By arranging the plates in the order of hand contact, it can be determined what individual received the original contaminant and started the "epidemic," which individuals transferred the organism yet did not grow it out (carriers), and how dosage, or amount of contamination, affects getting a disease. It must be pointed out that in an actual epidemic a contaminated individual could feasibly contact many others and not just one as demonstrated in this procedure and that the organism will multiply in each host before being passed on. Other means of microbial transmission (air, water, body fluids, fomites) may also be discussed. (Fomites are inanimate objects such as combs, pencils, etc. which may carry microbes on the surface.)" http://www.woodrow.org/teachers/bi/1993/demonstrating.html

In a 1997 review article, "Serratia marcescens," A. HEJAZI and F. R. FALKINER, Trinity College and St James's Hospital at Dublin, reported, "Serratia marcescens, a gram-negative bacillus classified as a member of the Enterobacteriaceae, has been recognised as a cause of hospital-acquired infection for the last two decades. It is a widely distributed saprophytic bacterium, and has been found in food, particularly in starchy variants which provide , an excellent growth environment. While this organism was known formerly by a variety of names, including Chromobacterium prodigiosum [11, Gaughran et al. [2] used the name S. marcescens that had been assigned by Bizio in 1823. S. marcescens was considered originally to be an innocuous, non-pathogenic saprophytic water organism and was often

used as a biological marker because of its easily recognised red colonies. After a review in 1896 of a small number of incidents, Professor Scheurlen of the University of Strasbourg concluded that this organism contributed to more deaths than many pathogenic bacteria. The first description of nosocomial infection caused by S. marcescens was Wheat's report of 11 cases over a 6-month period in 1951 at Stanford University Hospital [3]. Infections caused by this organism have been reported with increasing frequency since 1960 [4]. In 1966, McCormack and Kunin [5] reported a nursery epidemic involving 27 babies, although only 15 cases of Serratia bacteraemia had been recorded by 1968 [6] . Its ability to cause infection was once thought to be limited to patients with chronic debilitating disorders, but S. marcescens has now been implicated as an aetiological agent in every conceivable kind of infection, including respiratory tract infection, urinary tract infection (UTI), septicaemia, meningitis and wound infections [7-9]. S. marcescens has been reported to cause infective endocarditis acquired in the community [l0] and in hospitals. In contrast to other gram-negative bacteria, it usually affects the left side of the heart [ll]. S. marcescens endocarditis acquired in the hospital is usually an exogenous infection associated with cardiac surgery [11]. Today, S. marcescens has attained the status of' a fully fledged pathogen that causes infections particularly in two disparate groups: heroin addicts and hospitalised patients. Environmental isolates of S. marcescens characteristically produce a red pigment, prodigiosin, and in early times such growth was often mistaken for fresh blood [2]. The pigmented bacterium is found in various ecological niches, including soil, water, air, plants and animals [12]. The ability to form prodigiosin is characteristic of S. marcescens [12], but the function of this red pigment remains unclear because clinical isolates are rarely pigmented."
http://jmm.sgmjournals.org/content/46/11/903.full.pdf

In a 2000 study, "An outbreak of multiply resistant Serratia marcescens: the importance of persistent carriage," S Knowles, et al., St James's Hospital at Dublin, stated, "An outbreak of multi-resistant Serratia marcescens involving 24 patients occurred in a bone marrow transplant and oncology unit, from September 1998 to June 1999, of whom 14 developed serious infection. This is the first such outbreak described in a BMT unit. All isolates demonstrated the same antimicrobial susceptibility pattern and were the same unusual serotype O21:K14. The antimicrobial susceptibility profile showed reduced susceptibility to ciprofloxacin, gentamicin and piperacillin-tazobactam. As the latter two antimicrobials are part of our empiric therapy for febrile neutropenia, they were substituted with meropenem and amikacin during the outbreak. Investigation revealed breaches in infection control practices. Subsequently, the outbreak was contained following implementation of strict infection control measures. A prominent feature of the outbreak was prolonged carriage in some patients. These patients may have acted as reservoirs for cross-infection. This report also indicates that patients who become colonised with Serratia marcescens may subsequently develop invasive infection during neutropenic periods." http://www.nature.com/bmt/journal/v25/n8/full/1702218a.html

According to a 2011 Medscape article, "Serratia," J. Ania Basilio, MD; said, "In 1819, Bartolomeo Bizio, a pharmacist from Padua, Italy, discovered and named S marcescens when he identified the bacterium as the cause of a miraculous bloody discoloration in a cornmeal mush called polenta. Bizio named Serratia in honor of an Italian physicist named Serrati, who invented the steamboat, and Bizio chose marcescens (from the Latin word for decaying) because the bloody pigment was found to deteriorate quickly.[6] Since 1906, physicians have used S marcescens as a biological marker for studying the transmission of microorganisms because, until the 1950s, this bacterium was generally considered a harmless saprophyte. Only since the 1960s has S marcescens been recognized as an opportunistic pathogen in humans.[7] – Serratia species are opportunistic gram-negative bacteria classified in the tribe Klebsielleae and the large family Enterobacteriaceae. Serratia are widespread in

the environment, but are not a common component of the human fecal flora.[1] – Serratia marcescens is the primary pathogenic species of Serratia. Rare reports have described disease resulting from infection with Serratia plymuthica, [2] Serratia liquefaciens, [3] Serratia rubidaea, [4] Serratia odorifera, and Serratia fonticola. [5] – In the hospital, Serratia species tend to colonize the respiratory and urinary tracts, rather than the gastrointestinal tract, in adults. Serratia infection is responsible for about 2% of nosocomial infections of the bloodstream, lower respiratory tract, urinary tract, surgical wounds, and skin and soft tissues in adult patients. Outbreaks of S marcescensmeningitis, wound infections, and arthritis have occurred in pediatric wards. Serratia infection has caused endocarditis and osteomyelitis in people addicted to heroin. Cases of Serratiaarthritis have been reported in outpatients receiving intra-articular injections." http://emedicine.medscape.com/article/228495-overview

In a 2011 study, "Rapidly controlled outbreak of Serratia marcescens infection/colonisations in a neonatal intensive care unit, Pescara General Hospital, Pescara, Italy, April 2011," E. Polilli, et al., Santo Spirito General Hospital at Pescara, reported, "In April 2011, an outbreak of Serratia marcescens infection/colonisations occurred in the neonatal intensive care unit of Pescara General Hospital. Rapid microbiological investigations lead to identification of five cases of likely cross-transmission from a neonate hospitalised for S. marcescens sepsis: four infections and one neonate colonised post-mortem. Two low birth weight neonates died. The environmental investigation detected S. marcescens from two soap dispensers. Strict hygiene measures lead to early interruption of the outbreak, without recurrences to date." http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19892

Serratia in Animals

In a 1997 study, "Pathology of Serratia marcescens mastitis in cattle," G. Di Guardo, et al., Istituto Zooprofilattico Sperimentale delle Regioni at Rome, reported, "Microbiological, cytological, histopathological, and immunohistochemical investigations were carried out on four dairy cows affected by Serratia marcescens mastitis. The animals under study were from a herd of 120 lactating cows bred in the province of Rome. In the above herd, S. marcescens mastitis showed a prevalence of 20.8%. S. marcescens was the only bacterial agent isolated, prior to and after slaughter, from the teat milk, the mammary gland and the supramammary lymph nodes of the four cows under study. Cytologically, the four subjects exhibited high cell counts in their milk, with an average of up to 5,570,000 cells/ml in S.marcescens-infected quarters. Macroscopically, nodular lesions were apparent scattered throughout the mammary parenchyma, with enlargement of the regional lymph nodes. Histologically, a chronic, non-purulent mastitis, characterized by a marked fibrous tissue proliferation and the coexistence of corpora amylacea within the glandular alveoli, was observed in association with chronic hyperplastic lymphadenitis involving the supramammary lymph nodes of the four cows. Immunohistochemically, S. marcescens was demonstrated, by means of monoclonal antibodies, both in the mammary gland and in the supramammary lymph nodes from these four animals" http://www.ncbi.nlm.nih.gov/pubmed/9451943

In a 2008 case report, "Necrotizing fasciitis caused by Serratia marcescens after tooth extraction in a Doberman Pinscher: a case report," T. Plavec, et al., Veterinary Faculty, University of Ljubljana, said, "A 3-year-old Doberman Pinscher was referred to the Clinic for Small Animal Medicine and Surgery, Veterinary Faculty of Ljubljana for cardiologic examination due to lethargy, inappetence and lateral abdominal wall oedema. The dog had been treated at the primary veterinary practice for tooth granuloma two days before the presentation. During the course of the disease a presumptive diagnosis

necrotizing fasciitis was ascertained and Serratia marcescens organism was isolated from the ventral body wall tissue, from the wound in the oral cavity and other organs in the body. Systemic signs developed concomitantly with the progression of the local disease. Due to grave prognosis the dog was euthanised. This is the first report of a necrotizing fasciitis in a dog caused by S. marcescens and also the first one suspected to occur after the dental procedure."
http://www.vri.cz/docs/vetmed/53-11-629.pdf

Serratia in Marine Coral

In a 2002 study, "The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, Acropora palmata," Kathryn L. Patterson, et al., Department of Marine Sciences and Institute of Ecology, University of Georgia at Athens, reported, "Populations of the shallow-water Caribbean elkhorn coral, Acropora palmata, are being decimated by white pox disease, with losses of living cover in the Florida Keys typically in excess of 70%. The rate of tissue loss is rapid, averaging 2.5 cm2·day−1, and is greatest during periods of seasonally elevated temperature. In Florida, the spread of white pox fits the contagion model, with nearest neighbors most susceptible to infection. In this report, we identify a common fecal enterobacterium, Serratia marcescens, as the causal agent of white pox. This is the first time, to our knowledge, that a bacterial species associated with the human gut has been shown to be a marine invertebrate pathogen." http://www.pnas.org/content/99/13/8725.full

In a follow up 2011 study, "Human Pathogen Shown to Cause Disease in the Threatened Eklhorn Coral Acropora palmata," Kathryn Patterson Sutherland, et al., Department of Biology, Rollins College at Winter Park, reported, "Coral reefs are in severe decline. Infections by the human pathogen Serratia marcescens have contributed to precipitous losses in the common Caribbean elkhorn coral, Acropora palmata, culminating in its listing under the United States Endangered Species Act. During a 2003 outbreak of this coral disease, called acroporid serratiosis (APS), a unique strain of the pathogen, Serratia marcescens strain PDR60, was identified from diseased A. palmata, human wastewater, the non-host coral Siderastrea siderea and the corallivorous snail Coralliophila abbreviata. In order to examine humans as a source and other marine invertebrates as vectors and/or reservoirs of the APS pathogen, challenge experiments were conducted with A. palmata maintained in closed aquaria to determine infectivity of strain PDR60 from reef and wastewater sources. Strain PDR60 from wastewater and diseased A. palmata caused disease signs in elkhorn coral in as little as four and five days, respectively, demonstrating that wastewater is a definitive source of APS and identifying human strain PDR60 as a coral pathogen through fulfillment of Koch's postulates. A. palmata inoculated with strain PDR60 from C. abbreviata showed limited virulence, with one of three inoculated fragments developing APS signs within 13 days. Strain PDR60 from non-host coral S. siderea showed a delayed pathogenic effect, with disease signs developing within an average of 20 days. These results suggest that C. abbreviata and non-host corals may function as reservoirs or vectors of the APS pathogen. Our results provide the first example of a marine "reverse zoonosis" involving the transmission of a human pathogen (S. marcescens) to a marine invertebrate (A. palmata). These findings underscore the interaction between public health practices and environmental health indices such as coral reef survival." http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0023468

Serratia in Plants

In a 2003 study, "Serratia marcescens, a phloem-colonizing, squash bug-transmitted bacterium: Causal agent of cucurbit yellow vine disease," BENNY D BRUTON, et al., ars.usd, reported, "In 1988,

Cucurbit yellow vine disease (CYVD) was first observed in squash and pumpkin in Central Texas and Oklahoma. In 1991, the disease was detected in watermelon and melon. Since then, the disease has also caused substantial damage to cucurbits in Arkansas, Tennessee, and Massachusetts. The diagnostic characteristic of CYVD affected plants is the honey-brown phloem discoloration in the crown and primary root. The objectives of the study were to isolate the phloem-inhabiting bacterium, develop an effective inoculation proceedure, prove Koch's Postulates, and determine the ability of squash bugs to transmit the bacterium to plants. Using PCR with CYVD-specific primers to confirm identity of the CYVD pathogen, the bacterium was isolated on nutrient agar. Koch's postulates were completed for cucurbit yellow vine disease, demonstrating that a strain of the cosmopolitan bacterium, Serratia marcescens, is the causal agent. Although S. marcescens is readily cultivated from various ecological niches, its cultivation from CYVD-affected cucurbits is significant because it represents the first successful growth of a phloem-colonizing bacterium on common laboratory media. The development of an effective mechanical inoculation method for S. marcescens on cucurbits was an essential step in this work. Infection rate was highly dependent on plant growth stage when inoculated. To achieve maximum disease development, plants should be inoculated when the seedlings first emerge and prior to cotyledons unfolding. Evidence presented from our field studies confirmed that the squash bug can transmit S. marcescens, the CYVD causal bacterium. The S. marcescens-Anasa tristis relationship described here is, to our knowledge, the first instance in which the squash bug has been identified as a vector of a plant pathogen." http://www.ars.usda.gov/research/publications/publications.htm?seq_no_115=132302

In a 2005 study, "Phytopathogenicity of Serratia marcescens strains in different plant host species," G. Luo, et al., OSU at Stillwater, said, "Strains of Seriatia marcescens (Sm), cause of cucurbit yellow vine disease (CYVD), colonize many niches (water, soil, humans, animals, insects, plants). To assess whether phytopathogenicity is strain-specific, tobacco leaves were needle-inoculated with various Sm strains. A HR-like response was observed within 24 hr with all Sm strains at 10**9 cells/ml, but at 10**6 cells/ml CYVD strains caused necrosis within 48 hr, while non-CYVD strains did so only after 60 hr. the response to SM, needle-inoculated to squash and carrot stems, and to onion bulbs (the latter two are non-hosts of CYVD), differed. Squash seedlings were stunted, onions were water-soaked and softened, and carrot seedlings wilted and died. Rates of infection also differed; 17% of squash plants inoculated with CYVD strains, but 0% of those receiving non-CYVD strains, showed symptoms. Surprisingly, only 0-33% of carrots and onions inoculated with CYVD strains showed necrosis, while 70-100% of those receiving non-CYVD strains did so. When Sm inoculum was dropped onto the surface of peeled onion bulbs to assess whether Sm could enter plants naturally, only non-CYVD strains caused symptoms. Our results demonstrate that Sm strains from non-plant niches can cause symptoms in plants, but their interactions with the plant host differ from those of CYVD strains." http://www.ars.usda.gov/research/publications/publications.htm?seq_no_115=199009

Tatumella

Tatumella (a coliform) is a part of the Enterobacteriaceae family.

In a 1981 study, "Tatumella ptyseos gen. nov., sp. nov., a member of the family Enterobacteriaceae found in clinical specimens," D. G. Hollis, et al., Centers for Disease Control at Atlanta, reported,"The name Tatumella ptyseos gen. nov., sp. nov., is proposed for a group of organisms (previously called group EF-9) isolated from clinical sources in the United States, Canada, and Puerto Rico. A total of

68% of these isolates were from sputum specimens. T. ptyseos strains are gram-negative, oxidase-negative, fermentative rods that grow on MacConkey agar. The distinctive biochemical characteristics of 44 T. ptyseos isolates were as follows: acid but no gas from D-glucose, sucrose, and, usually (71%), D-xylose (62% delayed); no acid from lactose, maltose, or D-mannitol; negative tests for indole, urea, methyl red, gelatin, L-lysine decarboxylase, and L-ornithine decarboxylase; L-arginine dihydrolase variable; phenylalanine deaminase positive; Voges-Proskauer positive by the Coblentz method but negative by the O'Meara method; nonmotile at 36 degrees C but 66% weakly motile (30% delayed) at 25 degrees C; Simmons citrate positive at 25 degrees C (89%) but Simmons citrate negative at 36 degrees C. Deoxyribonucleic acid-deoxyribonucleic acid relatedness studies on 26 T. ptyseos strains showed that they were 80 to 100% related at 60 degrees C, which indicated that they comprise a single species. The deoxyribonucleic acid relatedness to other species within the Enterobacteriaceae was 7 to 38%. This is evidence that this species belongs in this family, is distinct from all described species and is best placed in a new genus. The T. ptyseos isolates studied were susceptible to all of the antimicrobial agents tested by broth dilution; these antimicrobial agents were amikacin, ampicillin, cephalothin, chloramphenicol, gentamicin, kanamycin, tetracycline, and tobramycin. Three striking differences between T. ptyseos and other members of the Enterobacteriaceae were its large zone of inhibition around penicillin (mean diameter 24 mm), its tendency to die on some laboratory media (such as blood agar) within 7 days, and its small number (usually one) of flagella. Strain H36 (=ATCC 33301, =CDC D6168, =CDC 9591-78) is the type strain of this new species. T. ptyseos is the type species for the genus Tatumella."
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC271905/

In a 1989 study, "The first isolate of Tatumella ptyseos in Malaysia," S.C.Tan, et al., reported, "Tatumella ptyseos, the type species for the genus Tatumella, is a newly established member of the Family Enterobacteriaceae. It is a Gram-negative, oxidase negative, fermentative rod that grows on Mac Conkey agar. This first isolate was obtained from the blood culture of a neonate having neonatal jaundice with presumed sepsis. The organism was in vitro sensitive to Gentamicin, Chloramphenicol, Cotrimoxazole and Ampicillin. The patient was treated with Ampicillin and Gentamicin and recovered uneventfully." http://www.ncbi.nlm.nih.gov/pubmed/2632996

In a 2008 study, "Tatumella ptyseos causing severe human infection: report of the first two Brazilian cases," Paulo Sérgio Gonçalves Da Costa and Geyza Machado Ribeiro reported, "Tatumella ptyseos is the type species of the Tatumella genus (Enterobacteriaceae). This fermentative Gram-negative rod has only rarely been reported as a cause of human infections; there is very little information about it in the medical literature. We report here the first two Brazilian cases of T. ptyseos infections, both evolving to severe sepsis." http://www.mendeley.com/research/tatumella-ptyseos-causing-severe-human-infection-report-of-the-first-two-brazilian-cases/

In a 2011 pilot study, "GAMING CHIP SANITATION," Barbara Almanza PhD, RD, et al., Department of Hospitality and Tourism Management Professor, Purdue University, reported, "Those that work in and the customers who enjoy the casino environment may be at risk for exposure to infectious diseases, especially bacterial diseases. The purpose of this study to determine if and what type of bacterial microorganisms live on gaming chips. A total of 26 gaming chips (13 used actively in a casino and 13 never used) were utilized for the study. Swabs of the chips were performed and placed on blood agar Petri dishes where cultures were allowed to grow for 48 hours. The results of this growth showed a statistically significant number of bacteria and fungi development with a $p < 0.05$. Additional statistical analysis was performed on the level of contamination based on used versus unused chips and on the

location of the swab related to the obverse, reverse or rim of the chip, with overall results being statistically significant for the presence of pathogenic contaminants. – Saldmann, 2008, reported that illness causing virus and bacteria that can be spread through casual contact include: E. coli, ***Tatumella ptyseos,*** Serratia plymuthica, Citrobacter ferundii, Proteus penneri, Erwinia, and Helicobacter pylori. Each of these infectious diseases can spread through casual contact between people and can be spread from contact with objects that have been touched by individual carriers of these items. Further, if bacteria or viruses are deposited on an object, for example someone who is infected with human influenza and sneezes without covering their mouth, then the infectious organism can live from 1 to 48 hours, depending on the environmental conditions (Saldmann, 2008)."
http://scholarworks.umass.edu/cgi/viewcontent.cgi?article=1264&context=gradconf_hospitality&sei-redir=1&referer=http%3A%2F%2Fscholar.google.com%2Fscholar%3Fstart%3D10%26q%3Dtatumella%2Bptyseos%2Bdisease%26hl%3Den%26as_sdt%3D0%2C25%26as_vis%3D1#search=%22tatumella%20ptyseos%20disease%22

Tatumella in Plants

In a 2006 study, "Browning Discoloration by Reheating of Foods in Container-I: Isolation and Identification of Causal Bacteria of Browning Discoloration of Natudaidai," Report of Toyo Junior College of Food Technology and Toyo Institute of Food Technology, states, "Similarly to the well-known 'pink disease' of canned-pineapple, browning discoloration associated with bacteria has often occurred in canned Natsudaidai in syrup. Isolation of causal bacteria from a canning plant and raw materials was attempted. Contamination by causal bacteria was found at raw materials and carrying containers. Based on the sequences of 16S rDNA gene, these strains were identified to Tatumella ptyseos, Rahnella aquatilis and Enterobacter intermidius. AF1 agar, that is, modified PGG Agar added 5 ppm of Nystatin, constructed for the investigation of causal bacteria was effective for detection of causal bacteria. (author abst.)"
http://sciencelinks.jp/j-east/article/200704/000020070407A0095619.php

In a 2010 study, "Tatumella ptyseos, an Unrevealed Causative Agent of Pink Disease in Pineapple," Vianey Marín-Cevada, et al., reported, "Pink disease is a major problem in the pineapple canning industry. Affected fruit acquire a brownish pigment after pasteurization and can contaminate non-affected fruit before they are released to the consumer. In the last few years, Pantoea citrea has been described as the causative agent of pink disease. In this study, over 300 bacterial isolates from pineapple plants, growing in Mexican commercial fields, and from soil close to plant roots were recovered. Over 250 isolates showed a very high similarity in their phenotypic and genotypic traits with Tatumella ptyseos, a close relative of Pantoea. These isolates exhibited typical pathogenicity reactions in pineapple juice tests, pineapple slices and fruit. On this basis, molecular identification procedures for the Tatumella isolates associated with pink disease were implemented. In affected fruit populations around 106 CFU/g of fresh tissue were recovered. This is first time that T. ptyseos is demonstrated as a causal agent of pink disease."
http://onlinelibrary.wiley.com/doi/10.1111/j.1439-0434.2009.01575.x/abstract

Xenorhabdus

Xenorhabdus (a coliform) is a part of the Enterobacteriacea family.

In a 1989 study, "Xenorhabdus luminescens (DNA hybridization group 5) from human clinical specimens," J.J. Farmer, 3rd, et al., Centers for Disease Control at Atlanta, reported, "An unusual isolate from a human leg wound was identified as Xenorhabdus luminescens. This finding led to the discovery or isolation of four additional strains, two from blood and two from wounds. Three of the five strains were from patients in San Antonio, Tex. Three strains were studied by DNA-DNA hybridization (S1 nuclease-trichloroacetic acid method) and were 77 to 100% related to each other, 34% related to the type strain of X. luminescens, 35 to 40% related to three of Grimont's other DNA hybridization groups of X. luminescens, and 9% related to the type strain of Xenorhabdus nematophilus. The new group of five strains was designated X. luminescens DNA hybridization group 5. All five strains were very inactive biochemically and fermented only D-glucose and D-mannose. The key reactions for recognizing this new organism are yellow pigment production, negative test for nitrate reduction to nitrite, weak bioluminescence (10 to 15 min of dark adaptation is required to see the weak light produced), and a unique hemolytic reaction on sheep blood agar plates incubated at 25 degrees C. Two case histories of strains from wounds are given; these suggest that X. luminescens DNA hybridization group 5 may be a new bacterial agent that causes wound infections. The two cases of wound infection, along with the two blood isolates, suggest that the new organism is clinically significant." http://www.ncbi.nlm.nih.gov/pubmed/2768446

Xenorhabdus in Insect Vectors

In a 2004 study, "Stages of Infection during the Tripartite Interaction between Xenorhabdus nematophila, Its Nematode Vector, and Insect Hosts," Mathieu Sicard, et al., Laboratoire GPIA, Université Montpellier, reported, "Bacteria of the genus Xenorhabdus are mutually associated with entomopathogenic nematodes of the genus Steinernema and are pathogenic to a broad spectrum of insects. The nematodes act as vectors, transmitting the bacteria to insect larvae, which die within a few days of infection. We characterized the early stages of bacterial infection in the insects by constructing a constitutive green fluorescent protein (GFP)-labeled Xenorhabdus nematophila strain. We injected the GFP-labeled bacteria into insects and monitored infection. We found that the bacteria had an extracellular life cycle in the hemolymph and rapidly colonized the anterior midgut region in Spodoptera littoralis larvae. Electron microscopy showed that the bacteria occupied the extracellular matrix of connective tissues within the muscle layers of the Spodoptera midgut. We confirmed the existence of such a specific infection site in the natural route of infection by infesting Spodoptera littoralis larvae with nematodes harboring GFP-labeled Xenorhabdus. When the infective juvenile (IJ) nematodes reached the insect gut, the bacterial cells were rapidly released from the intestinal vesicle into the nematode intestine. Xenorhabdus began to escape from the anus of the nematodes when IJs were wedged in the insect intestinal wall toward the insect hemolymph. Following their release into the insect hemocoel, GFP-labeled bacteria were found only in the anterior midgut region and hemolymph of Spodoptera larvae. Comparative infection assays conducted with another insect, Locusta migratoria, also showed early bacterial colonization of connective tissues. This work shows that the extracellular matrix acts as a particular colonization site for X. nematophila within insects." http://aem.asm.org/cgi/content/full/70/11/6473

In a 2007 study, "Mutualism and pathogenesis in Xenorhabdus and Photorhabdus: two roads to the same destination," H. Goodrich-Blair and D.J. Clarke, University of Wisconsin at Madison, reported, "Photorhabdus and Xenorhabdus bacteria colonize the intestines of the infective soil-dwelling stage of entomophagous nematodes, Heterorhabditis and Steinernema, respectively. These nematodes infect susceptible insect larvae and release the bacteria into the insect blood. The bacteria kill the insect larvae

and convert the cadaver into a food source suitable for nematode growth and development. After several rounds of reproduction the nematodes are recolonized by the bacteria before emerging from the insect cadaver into the soil to search for a new host. Photorhabdus and Xenorhabdus bacteria therefore engage in both pathogenic and mutualistic interactions with different invertebrate hosts as obligate components of their life cycle. In this review we aim to describe current knowledge of the molecular mechanisms utilized by Photorhabdus and Xenorhabdus to control their host-dependent interactions. Recent work has established that there is a trade-off between pathogenicity and mutualism in both these species of bacteria suggesting that the transition between these interactions must be under regulatory control. Despite the superficial similarity between the life cycles of these bacteria, it is now apparent that the molecular components of the regulatory networks controlling pathogenicity and mutualism in Photorhabdus and Xenorhabdus are very different." http://www.ncbi.nlm.nih.gov/pubmed/17493120

Yokenella regensburgei

Yokenella (a coliform) is a part of the Enterobacteria family.

In a 1984 study, "Yokenella regensburgei gen. nov., sp. nov.: a new genus and species in the family Enterobacteriaceae," Y. Kosako, et al., reported, "The name Yokenella gen. nov. is proposed for a group of organisms in the family Enterobacteriaceae isolated from clinical sources and insects. Yokenella is a gram-negative, oxidase-negative, fermentative, motile rod possessing the characteristics of the family Enterobacteriaceae and the guanine plus cytosine contents of the DNA range from 58.0 to 59.3 mol%. Biochemical characteristics of this group and DNA hybridization studies indicate that the 11 strains studied here comprise a separate species which should be best placed in a new genus. This single DNA hybridization group is named Yokenella regensburgei sp. nov. The type strain of Y. regensburgei is NIH 725-83 (JCM 2403)." http://www.ncbi.nlm.nih.gov/pubmed/6503024

In a 1994 study, "Isolation of Yokenella regensburgei ("Koserella trabulsii") from a Patient with Transient Bacteremia and from a Patient with a Septic Knee," SHARON L. ABBOTT AND J. MICHAEL JANDA, Division of Communicable Disease Control, California Department of Health Services at Berkeley, said, "Yokenella regensburgei is one of a number of infrequently encountered members of the family Enterobacteriaceae that have only rarely been isolated from humans. Originally identified as NIH biogroup 9 by the National Institutes of Health in Japan (4) and as enteric group 45 by the Centers for Disease Control and Prevention (1), it most closely resembles Hafnia alvei biochemically. In 1984, Kosako et al. (4) proposed the name Y regensburgei for this new genus and species while the Centers for Disease Control and Prevention proposed the name "Koserella trabulsii" for enteric group 45 (1). Subsequently, it was recognized that Y regensburgei and "K. trabulsii" are objective synonyms (3) and Kosako and Sakazaki (2) proposed that the name Y. regensburgei had priority over "K. trabulsii" upon the basis of rule 24b of the Bacteriological Code. In 1991, the Centers for Disease Control and Prevention acknowledged that Y. regensburgei had priority upon the basis of prior publication and has since dropped the use of the name "K. trabulsii" (5). Y. regensburgei has been recovered from the intestinal tracts of insects, well water, and a number of anatomic sites in humans, including wounds of the limbs, the upper respiratory tract, urine, feces, and knee fluid (1, 4). Very little information is available on the clinical significance, epidemiology, and types of specimens associated with the recovery of Y, regensburgei. In this report, we describe two recent isolations of Y. regensburgei associated with extraintestinal sites in humans, including the first known recovery from blood."

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC264174/pdf/jcm00011-0236.pdf

In a 2010 study, "Abdominal Abscess and Septic Shock Secondary to Yokenella regensburgei," M. Fill & J. Stephens, Mercer University School of Medicine, reported, "Yokenella regensburgei is an uncommon clinical isolate of the family Enterobacteriaceae. We report a case of septic shock with bacteremia, abdominal abscess and pneumonia with Yokenella regensburgei. – Yokenella regensburgei, formerly known as Koserella trabulsii, is a relatively rare clinical isolate. Y. regensburgei has been isolated from rather obscure locations in nature, including water sources (including well water) and the intestinal tracts of insects; yet has also been isolated from the upper respiratory tract, urine, feces and synovial fluid of humans. However, no direct mechanism of Yokenella transmission from a specific source to humans has been elicited."
http://www.ispub.com/journal/the_internet_journal_of_infectious_diseases/volume_9_number_1_22/article/abdominal-abscess-and-septic-shock-secondary-to-yokenella-regensburgei.html

In a 2011 study, "Yokenella regensburgei in an immunocompromised host: a case report and review of the literature," Y.-C. Lo, et al., said, "Yokenella regensburgei belongs to the Enterobacteriaceae and shares some biochemical characteristics with Hafnia alvei. A few case reports have suggested that it is an opportunistic pathogen, but there is no strong evidence to support its clinical importance. Until recently, it was difficult to accurately differentiate between Y. regensburgei and H. alvei by use of routine identification techniques. Here, we present a case of soft tissue infection and bacteremia caused by Y. regensburgei, which was successfully treated by intravenous administration of ceftriaxone for three weeks, and review the previous literature."
http://www.springerlink.com/content/mt60m1m5213j0052/export-citation/

Conclusion

The Public Health Security and Bioterrorism Preparedness and Response Act of 2002 and some agency regulations that implement it make it very clear that the pathogenic coli-like-forms of gram negative bacteria referred to by scientists and regulators as Entrobacteriaceae, or colifroms, are in fact dangerous life threatening biological agents to humans, animals, birds, insects, plants and marine life.

The Act was designed to protect municipal water and sewage treatment plants as well as agricultural cropland. Therefore, scientists, municipal wastewater and health officials, state environmental and health departments as well as federal agencies are fully aware that sludge and recycled water is contaminated with dangerous life threatening biological agents they call coliform. The use of elevated levels of heat to stress the bacteria into inactivity is a deliberate effort to confuse and mislead farmers and the public about the danger of these biological agents in sludge and recycled water. The same is true for scientists who refer to thermotolerant stressed E. coli as Enterobacteriaceae or fecal coliform.

In examining the few studies reviewed in this paper on the thirty coliforms families, it is self-evident that Environmental Protection Specialist, Wesley Carr's, was misrepresenting the facts when he stated, **"there is no evidence that we have failed to protect the public health and environment"** The only justification for the statement is that municipal, state and federal officials have refused to investigate, or allow the investigation of, complaints from those exposed to the dangerous biological agents contaminating sewage sludge (aka biosolids) and recycled sewage water.

Since these dangerous biological agents are released to water under state and federal regulations, taken up in seeds, plants and vegetables when exposed to dangerous biological agents contaminating sewage sludge (aka biosolids) and recycled sewage water, FDA produce safety expert, Jim Gorny, was clearly wrong about the cantaloupe outbreak when he stated, "**It's only when a strange alliance of the stars occurs you get an extraordinary event like this**,"

Calling these dangerous coli-like-forms of gram negative bacteria coliforms or fecal coliforms and indicators of fecal contamination in food and water, confirmed by a test for E. coli is unethical as well as illegal, unless, the regulations override the intent and word of federal law.

It is time to demand a complete investigation and stop the lies.

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