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Health & Medicine, Disease

Research Topic: Listeria monocytogenes, Haemophilus influenzae, and Mycobacterium ulcerans Part 1: Listeria monocytogenes Listeria monocytogenes is a bacterium that is originated infoodand can cause the serious illness of Listeriosis. Listeriosis is a serious disease for humans; the overt form of the disease has a mortality rate greater than 25 percent1. This is a Gram-positive bacterium, and is mobile by means of flagella. Listeria monocytogenes can be found in nature in soil, water and animal feces, meaning it can be also be tracked to animals.

In addition to humans, at least 42 species of wild and domestic mammals and 17 types of birds can harbor listeria monocytogenes as well as crustaceans, fish, oysters, ticks, and flies. It is also reportedly carried in the intestinal tract of 5-10% of the human population without any apparent symptoms of disease1. Listeria monocytogenes is able to resist the deleterious effects of freezing, drying, and heat remarkably well for a bacterium that does not form spores2. A human can consume this bacterium by eating a raw vegetable that grew in infected soil, or by eating improperly processed deli meats and unpasteurized milk products3.

Healthy people rarely become ill from listeria monocytogenes infection. Possible complications due to the bacteria are blood infections and inflammation of the membranes and fluid surrounding the brain (meningitis). Other side effects that may develop are typical cold or flu-like symptoms. The real risk of infection is in the elderly, new born children, and pregnant women. During pregnancy, a listeria monocytogenes infection is likely to

cause only mild signs and symptoms in the mother. The consequences for the baby, however, may be devastating.

The baby may die unexpectedly before birth or experience a life-threatening infection within the first few days after birth3. The dangerous factor about the listeria monocytogenes is that the bacterium is able to grow in fresh food. The organisms can grow at 4o C which means that organism replication continues in refrigerated foods4. Therefore it is impossible for someone to know if he/she is purchasing food that 100% does not contain this bacterium. The current method the FDA uses to analyze food for possible contamination is complex and time consuming. The method requires 24 and 48 hours of enrichment, followed by a variety of other tests.

Total time to identification is from 5 to 7 days, but the announcement of specific non-radiolabeled DNA probes should soon allow a simpler and faster confirmation of suspect isolates. With new DNAtechnologymay even permit 2-3 day positive analysis in the future. Currently, FDA is collaborating in adapting its methodology to quantitate very low numbers of the organisms in foods2. This should drastically help prevent diseases that are caused through food consumption. Cases of Listeriosis in humans were not reported till about 1960, as the infection was only previous seen in animals.

In 1981, there was an outbreak that involved over 100 people in Canada. Thirty-four of the infections occurred in pregnant women, among whom there were 9 stillbirths, 23 infants born infected, and only two live healthy births. Among 77 non-pregnant adults who developed overt disease, there was nearly 30% mortality. The source of the outbreak was coleslaw produced

by a local manufacturer1. Even with increasing awareness of the bacteria and ways to prevent it from getting into food through processes developed by the FDA, there have still been recent outbreaks.

As of October 26, 2012, there have been twenty people reportedly hospitalized due to the listeria monocytogenes bacterium in 13 different states and the District of Columbia. Nine of the illnesses were related to a pregnancy; three of these illnesses were diagnosed in newborns. The other 13 ill persons range in age from 30 years to 87 years, with a median age of 77 years. Four deaths have been reported, one each from Minnesota, New York, Nebraska, and California4. An investigation was conducted by officials in local, state, and federal publichealthagencies to see what may have caused this outbreak.

They came to the conclusion that an imported brand of Frescolina ricotta salata cheese distributed by Forever Cheese, Inc. is the likely source of this outbreak of Listeriosis. In interviews, ill persons answered questions about foods consumed and other exposures in the month before becoming ill. Twelve of fourteen sick people interviewed reported consuming a soft cheese4. The investigation focused on identifying intact cheeses shipped to multiple retail locations where ill persons purchased cut and repackaged cheese. There is no report of a direct link to one specific retailer or location of where the cheese was produced.

No one cheese was reported by the majority of ill persons, suggesting that cross-contamination of other cheeses through cutting boards and utensils may have played a role. The investigation focused on identifying intact

cheeses shipped to multiple retail locations where ill persons purchased cut and repackaged cheese4. There is still a chance of more ill people being reported because it can take up to 2 months after eating contaminated food for Listeriosis to develop. Works Cited: 1. Todar K. 2012. Listeria monocytogenes. Todar's Online Textbook of Bacteriology. 1-3 2. [FDA] Federal Drug Administration. 012. Foodborne Pathogenic Microorganisms and Natural Toxins Handbook: Listeria monocytogenes. Bad Bug Book. 3. http://www. mayoclinic. com/print/listeria-infection/DS00963/DSECTION= all&METHOD= print 4. [CDC} Center of Disease Control. 2012. Multistate Outbreak of Listeriosis Linked to Imported Frescolina Marte Brand Ricotta Salata Cheese. Listeria (Listeriosis). Part 2: Haemophilus influenzae Just by looking at the name of the bacterium Haemophilus influenza, one would guess this is the microbe that causes influenza. It was first found during the influenza pandemic of 1890.

It was mistakenly thought to be the cause of the disease influenza, and it was named accordingly. Probably, H. influenzae was an important secondary invader to the influenza virus in the 1890 pandemic, as it has been during many subsequent influenza epidemics1. There are similarities between human influenza virus and H. influenzae, as was observed in infant rats. H. influenzae is a small Gram negative bacillus which can be grown on chocolate agar (heated blood)2. H. influenzae is highly adapted to its human host. It is present in approximately 75 percent of healthy children and adults.

It is rarely encountered in the oral cavity, and it has not been detected in any other animal species1. A minority of healthy individuals have H.

influenzae type b encapsulated in their upper respiratory tract. Haemophilus influenzae type b, or Hib, is a bacterium estimated to be responsible for some three million serious illnesses and an estimated 386, 000 deaths per year, mainly through meningitis and pneumonia3. Most victims are children under the age of five. Sickness due to these bacteria is most common in underdeveloped or third-world countries.

This is where the vast majority of Hib deaths occur. Hib can cause infections such as pneumonia, sinusitis, tracheobronchitis, and meningitis. Pneumonia accounts for a larger number of deaths than meningitis. However, Hib meningitis is more of a serious problem in first world countries. It leaves 15 to 35% of survivors with permanent disabilities such as mental retardation or deafness3. If a H. influenzae infection goes untreated, it will almost certainly kill the individual. The body’s immune system is incapable of fighting off the effects of the bacteria.

The good news is virtually all patients treated early in the course of H. influenzae meningitis are cured. The mortality rate of other Hib infections is less than 10 percent, but nearly 30 percent of the children who recover have residual neurologic effects1. Ampicillin is typically the drug used to fight Hib, but there have been strands of Hib found to be immune to it, so other drugs developed and used successfully in treatment. Before 1985, Hib was the most common cause of bacterial meningitis in children under 5 years of age (approximately 12, 000 cases per year, most in children younger than 18 months).

Approximately 5% of affected children died, and different neurological problems developed in 15% to 30% of the surviving children. An additional estimated 7, 500 cases of other invasive Hib infections also occurred annually in young children. The cumulative risk for a Hib invasive disease before the age of 5 was one in 200 children. The first Hib vaccines were licensed for use in the United States in 1985. These vaccines contained purified polyribosylribitol phosphate (PRP). PRP vaccines were ineffective in children less than 18 months of age because of the T-cell-independent nature of the immune response to PRP polysaccharide1.

Research proved that this vaccine was effective in the body being immune to Hib. In 1989, the first Hib conjugate vaccines were licensed for use among children 15 months of age or older. In 1990, two new vaccines were approved for use among infants1. The incidence of Hib invasive disease among children aged 4 years or younger has declined by 98% since the introduction of Hib conjugate vaccines. However there are still hundreds of thousands of children die each year from a disease related to Hib. There are two major obstacles when trying to prevent Hib, the shortage of both information andmoney.

The information shortage is largely due to the difficulty of diagnosing Hib disease; it claims most of its victims without ever being recognized. In addition, Hib vaccine is more expensive to produce and thus more highly priced than classicchildhoodvaccines. It costs roughly seven times the total cost of vaccines against measles, polio, tuberculosis, diphtheria, tetanus, and pertussis4. The Hib vaccine should be distributed to all children during their

first few years of life just like other types of vaccines. More than 90% of infants obtain long term immunity with 2-3 doses of the vaccine1.

With a strong statistic like that most parents would not hesitate to have the vaccine given to their children. The issue is not all doctors are recommending the vaccine and they do not have it available to them in order to give to their patients. A few common misconceptions about the particular vaccine for Hib are that it can prevent ear infections and meningitis. It is not clear if the particular vaccine can reduce the likelihood of ear infections, but there is no evidence to say it prevents it. As for meningitis, there are different types of bacteria that can cause it and the vaccine only protects against the H. nfluenzae bacteria itself. Children must receive other vaccines to be fully immune to meningitis. Works Cited: 1. Todar K. 2012. Haemophilus influenzae and Hib Meningitis. Todar's Online Textbook of Bacteriology. 1-4 2. Ghaffar A, 2010, Bacteriology Chapter 18: Bordetella, Haemophilus and Legionella. Microbiology and Immunology On-Line. 3. http://www. who. int/mediacentre/factsheets/fs294/en/index. html 4. Musher DM. Haemophilus Species. In: Baron S, editor. Medical Microbiology. 4th edition. Galveston (TX): University of Texas Medical Branch at Galveston; 1996. Chapter 30. Part 3: Mycobacterium ulcerans

We commonly think of an ulcer as a small, yet painful sore that develops on lips, inside mouths, or other places of the body. These are common, and are generally not serious in nature as they heal on their own. However there are other types of ulcers that are more serious that are caused by bacterial infections. The Mycobacterium ulcerans, is a bacterium that causes large

ulcers to form on the skin on different parts of the body. The infection due to the type of bacterium is called the Buruli ulcer. This may range from a painless nodule to large, ulcerative lesions that heal spontaneously but slowly.

Along with scarring and deterioration of the skin, a severe enough case of it can lead to permanent bone damage. After tuberculosis and leprosy, the Buruli ulcer is the most common mycobacterial infection of humans1. The mode of transmission is not known, but recent evidence suggests that aquatic insects and fish, and even different types of animals such as koalas, possums, horses, cats and dogs may be able to carry the bacteria2. Open wounds on the skin seem to be the most logical way the organism enters the body. There is little proven evidence of bacteria being able to be spread through human to human touch2.

About 70% of those affected are children under the age of 15 years2. Mortality due to the disease is low; however the likelihood of permanent scarring and amputation is high. The disease is mostly found in only is Western Africa and Australia. Prevalence rates have been estimated at 16% in some communities in the Ivory Coast and at 22% in a community in Ghana2. A few cases have been reported in non-endemic areas in North America and Europe, most likely as a result of international travel. Lack of familiarity with the Buruli ulcer has frequently resulted in significant delays in the diagnosis and treatment of these cases.

The current economic and social burden imposed by the Buruli ulcer is enormous. Skilled surgery, expert post-operativenursingcare, and restorative

physiotherapy are often required to achieve good outcomes3. In Ghana, the average cost of treatment per patient is estimated to be $ 7802. This may not seem like a lot to us for the severity of disease, but in countries like Ghana not many people can afford this, or the cost or accessibility to the proper drugs is beyond the ability of the doctors in their health care system.

There are many graphic pictures that can easily be found onGooglethat show people who do not have the ability to get their infection treated. These are the types of people with the most severe side effects. Untreated Buruli ulcer will eventually subside with the gradual development of host immunity in most cases. However, by this time, tissue damage may be very extensive and healing by scar can lead to permanent functional and cosmetic deformity3. Successful treatment will shorten the course of the disease and minimize deformity.

In recent years, research has been conducted to see where the microbe may have originated, and what it genetically contains. Unlike Hib, which was discussed in part 2, there is currently no vaccine on the market to prevent people from getting the Buruli ulcer. Scientists have been working on three different vaccine candidates: mycolactone-directed vaccines, attenuated live vaccines, and subunit protein vaccines4. An M. ulcerans bacterium causes harm through its toxin mycolactone; therefore a vaccine directed against this toxin may provide protection. Several constructs are under development and confer some protection in mice4.

These recent findings have been a big step forward in the understanding of the mechanisms by which mycolactone mediates its biological effects in the

skin. However, it is not at the stage to be tested on people yet. There have also been developments of vaccines in Switzerland of a different kind. This kind of vaccine uses 'typical' parts of the mycobacterium as target for the immune systems, instead of complete mycobacteria4. There is a lot less chance of negative side effects, but it is not always as effective. In test results, it has shown to be effective in preventing the growth of the M. lcerans in the short term, but the effects wear off and the vaccine would have to be re-administered. Even with all the progress toward a new vaccine, the problem still exists of how it would be properly administered. The areas that are most affected, as stated earlier, would not be able to afford these vaccinations for their children. The only area with the epidemic of the Buruli ulcers that could extremely benefit from the possible development of vaccinations would be southern Australia. For this reasonI believeother projects will be prioritized higher because of more economic benefit to the developers and sellers.

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