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Listeriosis in Human Pregnancy: a systematic review

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Abstract

Listeria is commonly found in processed and prepared foods and listeriosis is associated with high morbidity and mortality. Preventative measures are well prescribed and monitoring and voluntary recall of contaminated products has resulted in a 44% reduction in the prevalence of perinatal listeriosis in the USA. Pregnant women are at high risk for listeriosis, but symptoms are non-specific and diagnosis is difficult. The intracellular life-cycle of *Listeria* protects the bacterium from host innate and adaptive immune responses. Antibiotic treatment requires agents able to penetrate, distribute, and remain stable within host cells. Prolonged use of high-dose ampicillin can significantly improve neonatal outcome.

Keywords

Listeriosis; Listeria monocytogenes; infection; foodborne; pregnancy; newborn; neonate; Epidemics

INTRODUCTION

Since the 1960s, and following the introduction and widespread use of refrigerators, processed foods, and the extended shelf-life of foods, listeriosis due to *Listeria monocytogenes* has become more widespread. Although rare in pregnancy, because of the potential for serious feto-maternal consequences, it is important that practicing obstetricians are familiar with the diagnosis, pathogenesis, treatment and prevention of *Listeria* infection. Accordingly, to review this problem, we conducted a computerized search using PubMed, Embase, Cinahl, Lilacs, ISI Web of Science, the Cochrane Central Register of Controlled Trials (all from inception or 1960 to September 2010), and Research Registries of ongoing trials using a combination of key and text words and Medical Subject Headings related to *Listeria monocytogenes,* listeriosis, Pregnancy, Newborn, Neonate, Infectious Disease Transmission, Vertical, Epidemics, and Food-borne infection.

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EPIDEMIOLOGY

In 1997, listeriosis was estimated to be responsible for 500 deaths annually in the USA [99], and in 2000 the prevalence was estimated to be 4 per million population [20]. In Europe, rates vary between 0.1 and 11.3 per million population, and approximately 20% involve neonatal infection [117]. Though no clear seasonal pattern exists, illness in humans tends to present in late summer and early autumn [81,98]. When the disease occurs in children, up to 54% have no apparent immunocompromising condition [22,151].

Listeriosis is mainly a food-borne infection, and sporadic cases as well as epidemics have been linked to contaminated food [18,43,85,87,130,144]. Human listeriosis typically occurs sporadically, but occasional nosocomial clusters have been reported [41,83,139]. In the USA, routine screening and surveillance often leads to a recall of food products due to concerns with respect to listerial contamination [72]. Since the same serotypes cause disease in humans and animals [123], it was originally believed that the disease was caused by direct transmission from animals; however, this has only rarely been documented [14]. In sporadic outbreaks, 11% of all food samples retrieved from refrigerators were contaminated and 64% of the refrigerators of infected cases contained at least one contaminated food item [112,134]. Table 1 demonstrates steps that can be taken by those at risk for listeriosis that help prevent the disease, based on foods known to be associated with infection or past outbreaks.

MICROBIOLOGY

Although, Hayem (France, 1891) and Henle (Germany, 1893) observed gram positive rods in the tissue sections from patients who died of a disease that, in retrospect, was almost certainly listeriosis, Hülphers (Sweden, 1911) appears to be the first to have recorded an encounter with the organism now known as *Listeria monocytogenes* [49]. Reflecting the association with liver abscesses and monocytosis in laboratory animals [26,103] (which is not a feature of disease in humans), the organism has been known as *Bacillus hepatitis* (1911) [66], *Bacterium monocytogenes* (1926) [33], *Listerella hepatolytica* (1927) [113], and following the first report in humans, *Bacterium monocytogenes hominis* (1929) [107]. The current name, *Listeria monocytogenes* [57], was finally agreed upon in 1940 in honour of Lord Joseph Lister, the “father of antisepsis” [45]. Of the seven species of *Listeria,* only four infect humans. Infection with *Listeria ivanii* is rare, and most cases, are due to three serotypes of *Listeria monocytogenes* (1/2a, 1/2b, and 4b), the last of which is responsible for the vast majority of listeriosis outbreaks [14,96,110,112].

L. monocytogenes is a small, facultatively anaerobic, Gram-positive, flagellated, linear motile rod, which is non-spore forming and microscopically difficult to distinguish from commensal *Diphtheroids* [17,106]. Therefore, it was initially thought to be of the genus *Corynebacterium* [80]. For an historical perspective on the microbiology of *Listeria,* the interested reader is referred to a comprehensive review by Farber and Peterkin [39].

Listeria grows well in broth, blood agar and most routine culture media [63]. Nevertheless, selective media, enrichment, and subculture may be necessary to avoid false-negatives from overgrowth of Gram-negative organisms from contaminated sites like the vagina and rectum [30,75,82]. Human pathogenic species produce a narrow zone of β-hemolysis on sheep blood agar [38,47,147], but while suspected to be a virulence factor, the presence of listeriolysin does not correlate with the severity of disease [44,54,78,79,109,140].

L. monocytogenes tolerates high and low temperatures as well as high salt concentrations. As a result, it replicates well in soil, dust, water, sewage, manure, silage and animal feeds [1,40,77,156,157]. *Listeria* grow at refrigerator temperatures (therefore contaminate

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refrigerated foods and raw meat), and can be detected in the feces of animals and humans [144]. Although it can survive for many months in soil [156], pasteurization and most disinfecting agents eliminate the organism [115]. While *Listeria* can be isolated from stool cultures of 5% of healthy individuals [12,52,53,76,85,129,134] and fecal carriage of *Listeria* occurs in 1-15% of the population, vaginal carriage is lower [85]. Pathogenic strains have been detected in the gastrointestinal (GI) tract of asymptomatic individuals, including those at risk (such as pregnant women or immunocompromised patients) [76,82,137].

COLONIZATION WITH LISTERIA

Exposure to and transient colonization of the GI tract by *L. monocytogenes* appears to be common, but invasive disease is rare. Using repeated sampling, *Listeria* can be detected in the feces of nearly 70% of healthy non-pregnant individuals and 44% of pregnant women [75]. Single samples of feces during pregnancy (with careful attention to culture techniques) have a 12% culture positive rate [75], yet others have reported lower recovery rates of 1.1-3.9% [48,82]. Pregnancy does not appear to affect the fecal, cervico-vaginal or oropharyngeal carriage rate of *Listeria,* but such carriage has been reported as a possible predisposing factor for perinatal listeriosis [82]. A study of 750 healthy pregnant women reported that 3.9% had a positive rectal culture while only one woman (0.13%) had a positive vaginal culture [85]. Four studies comprising more than 1,000 healthy pregnant women overall found positive cervicovaginal cultures of *L. monocytogenes* to be very rare [65,82,85,116].

PATHOGENESIS OF INFECTION

L. monocytogenes is an intracellular pathogen which, following ingestion of contaminated food, is phagocytosed and internalised in epithelial cells by the interaction between the bacterial surface protein, internalin, and its receptor on the epithelial surface, E-cadherin. The organism is vacuolated by macrophages, polymorphonuclear leucocytes and other plasma cells, and escapes the vacuole through the action of listeriolysin, finally entering the cytoplasm where proliferation occurs [88]. In the cytoplasm, Act A, another virulence factor, induces actin polymerization to form filaments which become a propulsion system for the organism [114]. The organism then migrates to the cell membrane and produces pseudopodia-like structures which extrude from the host cell, are recognized by adjacent cells and phagocytosed, so that the intracellular cycle continues [144]. This cell-to-cell spread accounts for the fact that antibodies, complement and neutrophils have little or no effect in host protection [63,88,114,144]. Since *L. monocytogenes* can enter the host without disrupting the integrity of the GI tract [144], it has been suggested that GI symptoms may reflect a non-specific response to a systematic infection. Alternatively, GI symptoms may represent a concurrent illness through other organisms (such as *Shigella)* in the GI tract, which disrupt mucosal integrity and facilitate invasion of *L. monocytogenes* [12,88,132,135]. Otherwise, GI symptoms may simply reflect the spectrum of illness of listeriosis itself.

Listeria activates T-cell mediated immunity which, under the influence of cytokines, attracts macrophages that produce inflammatory granulomata where bacteria are destroyed [91]. Memory T-cells provide an acquired resistance to *Listeria* infection, and this might explain why listeriosis is linked with malignancy, immunosuppressive therapy, AIDS, pregnancy and the neonate. This can also account for the observation that neutropenia and disorders of complement or immunoglobulin synthesis are not associated with excessive prevalence of the disease [8,13,32,69,91]. Apart from the immune status of the host, other factors which influence whether or not invasive disease occurs include the virulence of the infecting strain and the size of the inoculum [68,118,131]. The infective dose is unknown, but is estimated

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to be between 10^4-10^6 organisms/g of ingested product, although this estimate might be lower in groups at risk [61,101,128]. The duration of the incubation period is also unknown, but is likely to be about three weeks [112,134].

LISTERIOSIS IN PREGNANCY

Epidemiology

Listeriosis is 18 times more common in pregnancy (12/100,000) than in the non-pregnant population (0.7/100,000) [144], and 16-27% of all infections with *Listeria* occur in pregnant women [70,72,154]. Sporadic cases of perinatal listeriosis, as well as epidemic cases, have been reported with a prevalence that varies between 8.6 and 17.4/100,000 of live births [144,148]. The largest reported epidemic occurred in 1985 in Los Angeles, California, USA. Of 142 reported cases of listeriosis, 65.5% occurred in pregnant women [87]. The source was contaminated Mexican-style cheese made with unpasteurized milk, which is a well-known risk factor for the acquisition of listeriosis. Indeed, Hispanic (compared to non-Hispanic) women are at a significantly greater risk of pregnancy-associated listeriosis due to their consumption of food known to be at higher risk of contamination with *L. monocytogenes* [154].

Underlying maternal risk factors are uncommon (4.5%), and serious maternal disease is rare when other risk factors are absent [22,45]. However, one case of maternal meningo-encephalitis in a series of 248 cases of *Listeria* in pregnancy has been reported [95], as well as a case of maternal listerial endocarditis [64]. In addition, an analysis of mother-infant pairs with *Listeria* showed that one-third of mothers had *Listeria* isolated from blood cultures, and nearly half were hospitalized [70]. Listeriosis occurs mainly in the third trimester, perhaps due to deficient cell mediated immunity [14]; yet, cases have been observed at earlier gestational ages [48,111]. The incidence at lower gestational ages may be underestimated due to reluctance to culture aborted fetal tissue or products of conception.

Clinical Features

The presentation of listeriosis during pregnancy includes mild flu-like symptoms [10]. In a series of 191 cases of listeriosis in pregnancy, 32% of women had symptoms of a flu-like illness, 65% had a fever, and other symptoms included backache (21.5%) (which may be mistaken for a urinary tract infection), headache (10.5%), vomiting/diarrhea (7%), muscle pains (4%) and sore throat (4%). Approximately 29% of the women were asymptomatic [105].

Fetal transmission from maternal listeriosis is not invariable [67], and discordant infection *in utero* has been observed in a set of twins [142]. Once the placenta is infected, it becomes a reservoir for re-infection [6], and placental micro-abscesses may be histologically evident [31,152].

Listeriosis during pregnancy carries a poorer prognosis for fetuses who are affected at early gestations [105]as opposed to later gestations, and commonly results in spontaneous abortion or stillbirth [73,121]. The association with recurrent abortion has been reported in a series of cases in which *Listeria* was cultured from the cervix [119], though this has been questioned [4,92,93] mainly because of the inability to substantiate the presence of the organism microbiologically [2,50,86,125,126,150].

Preterm birth is common [9], and the highest mortality rate is predictably among infants born at the earliest gestations [95]. Two studies have been reported [95,105], one with 222 pregnant women comprising 11 culture-confirmed cases and 211 cases reported in the literature [105], and the other with 722 pregnant women who were either culture-positive or

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had clinical suspicion of listeriosis [95]. Spontaneous abortion occurred in 10-20% of cases, approximately 50% delivered preterm, intrauterine fetal death occurred in approximately 11% [95], 34% demonstrated fetal distress, and 75% developed meconium staining of the amniotic fluid particularly at early gestation [95,105].

In another study, tissue from preterm births or spontaneous abortions (which occurred around 3-7 days post infection) tested positive for *Listeria* in 1.6% of cases [46]. Of 34 women with a history of recurrent abortions and 87 controls, positive cultures were found in 73.5% and 0% respectively [119]. Other studies [4,84,92,93] failed to confirm these findings; therefore, routine genital tract culture and treatment are not recommended [138]. Nonetheless, although rare and because of the potentially serious consequences, it is important that practicing obstetricians are familiar with the diagnosis, treatment, and prevention of *Listeria* infection [124].

NEONATAL LISTERIOSIS

While maternal illness “due to listeriosis” may be mild, neonatal illness is often severe and may be fatal. Neonatal listeriosis may occur by vertical transmission of *L. monocytogenes* from mother to fetus, either by inhalation of infected amniotic fluid, transplacentally from the maternal circulation [9] or by ascending colonization from the vagina. Though vaginal colonization is rare, nearly half of asymptomatic mothers of infants born with neonatal listeriosis will have a positive vaginal culture for *L. monocytogenes* [85]. This observation is consistent with other perinatal infections like group B *Streptococcus* (GBS)., Similar to GBS, Listeria is likely to result from spread to the vagina from the lower GI tract [5,15,28]. Furthermore, ascending vaginal colonization is supported by the finding of listerial colonization solely in the firstborn of a set of twins [56] However, haematogenous spread may be more likely, and documented maternal bacteriuria and *in utero* fetal infection with positive amniotic fluid cultures prior to labor have been recorded [94]. In addition, nosocomial cross-infection has been reported [37,115,133] 55,115,116].

Neonatal listeriosis occurs in approximately 8.6/100,000 of live births [20], and is one of the most common causes of neonatal meningitis. The clinical manifestations of neonatal listeriosis are similar to GBS, and there is a high mortality rate (20-60%) [2,87,95,105,136]. One study reported that 68% of newborns whose mothers were diagnosed with listeriosis as a result of positive cultures from placenta, maternal blood or cervix developed neonatal infection; of those 68.2% made a complete recovery, 12.7% developed long-term neurological sequelae, and the infant mortality rate was 24.5% [105]. Like GBS, there are two forms of the disease in the neonate, early- and late-onset, suggesting different modes of transmission (vertical and nosocomial) [150].

Early Onset

Symptoms of early-onset listeriosis occur at a mean of 36 hours after birth [123]. In 50-74% of cases, the mother is likely to have had a flu-like illness with symptoms of fever, headache and myalgia [56,87,95,123], and isolation of *L. monocytogenes* from maternal blood or genital tract is common (44-89%) [87,95]. Neonates with early-onset infection are more frequently born preterm and associated with chorioamnionitis. The fetus presents with clinical features like septicaemia (81-88%), respiratory distress or pneumonia (38%), meningitis (24%) [87,95] and occasionally disseminated inflammatory granulomata (Granulomatosis infantisepticum) [7,17,65,73,119,120], from which pure culture of *L. monocytogenes* can be obtained [121,122]. Granulomatosis Infantisepticum was reported for the first time in 1893 (at which time, the disease was named pseudotuberculosis) [60], and is a pathognomonic feature of listeriosis [80].

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Late Onset

In contrast, late-onset neonatal listeriosis (commonly due to serotype 4b) [3], tends to occur between five days and two or more weeks postpartum, typically in term neonates [2,95]. Also typical of these cases is that the neonate is born to asymptomatic mothers with failure to isolate *L. monocytogenes* from maternal cultures [87,95]. The clinical features may be non-specific, but septicaemia (17-95%) and meningitis (67-93%) are common [115,141]. Neonatal listeriosis is one of the few congenital infections in which antibiotic therapy can improve outcome [23,42,105,159].

DIAGNOSIS

Diagnosis of maternal listerial infection may be difficult due to the lack of GI symptoms normally associated with food-borne pathogens and non-specific symptoms of malaise and influenza. Hematologically, a leucocytosis occurs rather than a monocytosis as the species name suggests. On wet mount microscopy, the organisms may be seen in characteristic “tumbling” motion, or may be evident on Gram stain, particularly if done on neonatal meconium before polymicrobial colonisation of the gut occurs. Gram stain is only useful in a minority of cases because, as an intracellular organism, it is easily missed and microscopically resembles many other bacteria commonly found in the vagina. This being the case, warning the microbiological microscopist of the possibility of listerial infection may improve the diagnosis using Gram stain [72]. Isolation is usually by culture from blood (maternal or neonatal), neonatal cerebrospinal fluid (CSF), amniotic fluid, the uterine cavity or the placenta [70,105]. The organism grows well from blood or CSF without the need for enrichment or selective media, and post-partum, histological evidence of micro-abscesses in the placenta can be found [31].

Using the ability of *Listeria* to grow at extreme temperatures, “cold enrichment” involving culture at 4°C (which discourages the growth of other organisms) may help identification [51]. However, this is time-consuming and not as accurate as selective media to isolate the organism from contaminated sites such as food or stools, which contain multiple species of *Listeria* [58]. Rapid detection, at least to the genus level, is possible using monoclonal antibody tests or DNA hybridization [25,29,45,143,155]. Serological testing for anti-listeriolysin may assist in diagnosing both invasive and non-invasive listeriosis [11,24,36], and identification to the species level is possible using specific primers for *L. monocytogenes*. Recently, a 16S rDNA assay with specific primers for the four major food-borne pathogens (*E. coli, Salmonella, Campylobacter* and *Listeria)* has been successfully tested [146].

PREVENTION OF LISTERIOSIS

In the USA, *Listeria* became nationally notifiable in 1999, and every occurrence should be reported to local health departments. Currently no vaccine for *Listeria* exists. Most cases of listeriosis are sporadic and not associated with an outbreak [105]. Most sporadic cases, as well as all large outbreaks, are due to manufactured foods [16], particularly ready-to-eat meats (especially hot dogs [97]), [71,98,105,138,144], and dairy products such as soft cheeses [21,87,141,148]. Most dairy-associated outbreaks are due to inadequate pasteurization, unpasteurized dairy products [87], or contamination post pasteurization [21].

The Centers for Disease Control and Prevention (CDC) have issued general recommendations for the prevention of listeriosis (Table 1). CDC recommendations state that all deli meats can be consumed safely if reheated until steaming hot. Prevention or avoidance of cross-contamination is an important preventative measure, and all utensils and surfaces should be cleaned after preparing meat dishes or cutting unprepared foods.

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Following the 1985 listeriosis outbreak [87], the CDC began monitoring dairy products and sporadic cases of listeriosis, including the voluntary recall of products and has maintained a zero tolerance policy for contaminated ready to eat products. This has resulted in a 44% reduction in the incidence of perinatal listeriosis in the USA (from 17.4/100,000 to 8.6/100,000 live births) [148].

Patient awareness

Despite the CDC recommendations, in a US nationalsurvey of 403 pregnant women, only 18% were familiar with listeriosis and less than 30% knew that it could be prevented by avoiding delicatessen meats, soft cheeses and unpasteurized dairy products. In focus groups, many had heard of *E. coli* and *Salmonella*, but few knew that pregnancy increased the risk for development of listeriosis, none had made any change to their food-handling habits, and all had continued to eat hot dogs and delicatessen meats during pregnancy [108]. Both commercially and in the home, individuals at low risk for listeriosis (who may therefore not be aware of the precautions needed to reduce the risk of listeriosis) often prepare food for pregnant women who are at higher risk. For these individuals, multimedia education has been recommended [19].

TREATMENT OF LISTERIOSIS

Treatment of listeriosis in pregnancy requires collaboration with an infectious diseases specialist, and should also involve pediatricians. Irrespective of the gestational age at which infection occurs in pregnancy, treatment is directed toward improving neonatal outcome. Early recognition and intervention are associated with improved outcome, and some physicians have advocated the use of antibiotics for the suspicion of *Listeria* infection for high risk cases after samples for culture have been obtained [138].

In contrast to other causes of chorioamnionitis in which induction of labor is the standard of care [138], treatment of maternal listeriosis in pregnancy and delay of delivery can result in the birth of a healthy infant at term [23,42,67,74,105,159]. Amniotic fluid cultures have been observed to change from positive to negative with appropriate antibiotic therapy [27]. Ideally, successful reports of *in utero* therapy should be accompanied by documentation of resolution by demonstrating negative amniotic fluid cultures.

Treatment challenges

No randomized controlled trials to assess type of antibiotic or duration of treatment have been performed. Therapeutic considerations include the fact that: 1) host susceptibility is linked to the atypical onset of the disease. The non-specific nature of the normal symptomatology may also make diagnosis and treatment difficult; and 2) the intracellular nature of the organism presents therapeutic challenges. Both of these factors may delay diagnosis and treatment. Bacteriostatic concentrations *in vivo* may be insufficient to be effective intracellularly. As a result, treatment recommendations are based mainly upon *in vitro* susceptibility testing or animal-model experimentation or are experience-based, following trials with small numbers of historical controls rather than being based on a foundation of robust medical evidence [149].

Antibiotics

The antibiotic of choice should penetrate host cells, distribute within the cells and remain stable in the intracellular environment [115,138,144]. As a result of these unusual antibiotic requirements, antibiotics may be ineffective in up to 70% of listeriosis cases [62].Most of the experience of treating *L. monoctogenes* is with the use of ampicillin, penicillin and amoxicillin [149], and to date, no bacterial resistance to penicillin has been detected [63]. *In*

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vitro, L. monocytogenes has a wide range of antibiotic sensitivities but is resistant to cephalosporins, clindamycin and chloramphenicol [34,105]. Chloramphenicol should not be used because of unacceptable rates of failure and relapse [21,69,88], and quinolones do not have adequate *in vitro* activity [35]. Cephalosporins are ineffective against *Listeria* because they do not bind to bacterial penicillin-bound protein-3 (PBP3) [144,153].

Though resistance to ampicillin has been reported [90], it is preferred over penicillin [10,105]. *L. monocytogenes* responds poorly to bacteriostatic antibiotics due to the intracellular nature of the pathogen, but ampicillin provides good host cell penetration without any significant change in pH or concentration which might compromise efficiency. Ampicillin also crosses the placenta in adequate amounts and binds to PBP3, causing bacterial death [63]. Gentamicin and tobramycin have been reported to have greater *in vitro* activity than streptomycin, kanamycin and amikacin [3,102,127]. Listerial resistance to vancomycin is rare [63], and its use (though limited) has been employed in endocarditis, as well as in listerial meningitis (in which cerebral intraventricular injections were administered) [104].

Synergy of ampicillin with gentamicin is claimed, but this has only been demonstrated *in vitro* [21,34,102,127,144]. Nevertheless, these two antibiotics are often used in combination in cases of meningitis despite concerns with respect to toxicity [149]. In patients with an allergy to ampicillin and/or gentamicin, erythromycin may be used [149], but transplacental passage of erythromycin provides sub-therapeutic concentrations in both amniotic fluid and fetal serum [59]. Alternatively, the combination of trimethoprim and sulfamethoxazole (which is bactericidal and achieves good serum and CSF concentrations) may be used [18,34,55,72,89,100,106,144,145,158]. However, there are concerns regarding the use of sulphonamides due to sulphur allergy and the risk of kernicterus (possible displacement of bilirubin from albumin) from use immediately prior to delivery. There are also concerns about possible teratogenic effects, such as neural tube defects due to the folate antagonism of trimethoprim during pregnancy, as well as hemolytic anemia in male infants due to glucose-6-phosphate deficiency. The preference for ampicillin over other options is such that it has been proposed to test sensitivity for suspected allergy before recourse to alternative therapy [72].

During pregnancy, the current recommendation for first line therapy is IV ampicillin ≥ 6g/day [149] and erythromycin 4g/day as second line IV. The duration for both regimes is 14 days, though the length of course may be longer in different clinical syndromes [45] and if the pregnant woman is immunosuppressed [72,149].

CONCLUSION

L. monocytogenes is an unusual pathogen due to the intracellular nature of its life cycle, which may explain the ready ease of transplacental passage. With an incidence of 12/100,000 pregnancies, listeriosis in pregnancy is still 18 times more common in pregnant women than in the general population. Pregnant women with co-morbidities are at increased risk due to compromised cell-mediated immunity. Maternal illness is usually mild, but neonatal disease carries a 20-30% mortality rate (although fetal and neonatal mortality are potentially preventable with early diagnosis and intervention). Ready-to-eat meats and dairy products made with unpasteurized milk should be avoided, and care should be taken to prevent cross contamination of foods by ensuring that preparation and cooking utensils and food preparation surfaces are clean. High dose ampicillin (≥ 6g/day) for a prolonged period of 14 days is recommended for treatment in pregnancy.

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Table 1

CDC Recommendations for the Prevention of *Listeriosis*

For the general public:

- Thoroughly cook raw food from animal sources, such as beef, pork, or poultry
- Wash raw vegetables thoroughly before eating
- Keep uncooked meats separate from vegetables and from cooked foods and ready-to-eat foods
- Avoid unpasteurized (raw) milk or foods made from unpasteurized milk
- Wash hands, knives, and cutting boards after the handling of uncooked foods
- Consume perishable and ready-to-eat foods as soon as possible

For persons at high-risk, such as pregnant women and persons with weakened immune systems, in addition to the recommendations listed above:

- Do not eat hot dogs, luncheon meats, or deli meats, unless they are reheated until steaming hot
- Avoid getting fluid from hot dog packages on other foods, utensils, and food preparation surfaces, and wash hands after handling hot dogs, luncheon meats, and deli meats
- Do not eat soft cheeses, such as feta, brie, and camembert; blue-veined cheeses; or Mexican-style cheeses, such as *queso blanco*, *queso fresco*, and *Panela*, unless they have labels that clearly state they are made from pasteurized milk
- Do not eat refrigerated pâtés or meat spreads. Canned or shelf-stable pâtés and meat spreads may be eaten.
- Do not eat refrigerated smoked seafood, unless it is contained in a cooked dish, such as a casserole. Refrigerated smoked seafood, such as salmon, trout, whitefish, cod, tuna or mackerel, is most often labeled as “nova-style,” “lox,” “kippered,” “smoked,” or “jerky.” The fish is found in the refrigerator section or sold at deli counters of grocery stores and delicatessens. Canned or shelf-stable smoked seafood may be eaten.

Source: Centers for Disease Control and Prevention Centers for Disease Control and Prevention Web site, Listeriosis technical information and FAQs *www.cdc.gov/ncidod/dbmd/diseaseinfo/listeriosis_t.htm* [Accessed April 7th 2010]