

when they arrived in Brazil is in agreement with previous findings of the differential distribution of TB and with a tuberculin survey on the African continent, supporting the hypothesis of native African TB (7,8). Therefore, the hypothesis of Africa as virgin soil for TB (1,9) cannot be easily supported. The incidence of TB among the slaves/Blacks in Rio de Janeiro was less than expected given their social and sanitary conditions (10), especially in a TB-endemic situation (4). Previous exposure to MTC might explain their apparent relative resistance.

Other evidence showing African contact with Europeans before the sixteenth century, supports the existence of TB in Africa (8), and TB was prevalent in urbanized centers along coastal areas of western Africa (7,8). Although some of those cases were probably the result of European contact, it is not possible to exclude that some were caused by TB native to Africa. We can affirm that persons buried in PNC, who were transported to Brazil as slaves from Africa, brought TB infection with them; whether the infection was caused by European TB endemic to Africa or by TB native to Africa is not known.

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### References

- Cummins SL. "Virgin soil"—and after: a working conception of tuberculosis in children, adolescents, and aborigines. *BMJ*. 1929;2:39–41. <http://dx.doi.org/10.1136/bmj.2.3575.39>
- Machado LC. Sítio cemitério dos Pretos Novos: análise biocultural. In: Dias O, Carvalho E, Zimmermann M, editors. *Estudos contemporâneos de arqueologia*. Palmas (Brazil): Fundação Universidade do Tocantins Neurosci, Instituto de Arqueologia Brasileira; 2006.
- Pereira JCMS. À flor da terra: o cemitério dos Pretos Novos no Rio de Janeiro. Rio de Janeiro: Garamond; 2007.
- Jaeger LH, Leles D, Lima VD, Silva LD, Neto OD, Iñiguez AM. *Mycobacterium tuberculosis* complex detection in human remains: tuberculosis spread since the 17th century in Rio de Janeiro, Brazil. *Infect Genet Evol*. 2012;12:642–8. <http://dx.doi.org/10.1016/j.meegid.2011.08.021>
- Harich N, Costa MD, Fernandes V, Kandil M, Pereira JB, Silva NM, et al. The trans-Saharan slave trade—clues from interpolation analyses and high-resolution characterization of mitochondrial DNA lineages. *BMC Evol Biol*. 2010;10:138. <http://dx.doi.org/10.1186/1471-2148-10-138>
- Salas A, Richards M, De la Fe T, Lareu MV, Sobrino B, Sanchez-Diz P, et al. The making of the African mtDNA landscape. *Am J Hum Genet*. 2002;71:1082–111. <http://dx.doi.org/10.1086/344348>
- Calmette A. Enquete sur l'épidéiologie de la tuberculose dans les colonies Françaises. *Ann Inst Pasteur (Paris)*. 1912;7:497–514.
- Daniel TM. The early history of tuberculosis in central East Africa: insights from the clinical records of the first twenty years of Mengo Hospital and review of relevant literature. *Int J Tuberc Lung Dis*. 1998;2:784–90.
- Stead WW. Tuberculosis in Africa. *Int J Tuberc Lung Dis*. 1998;2:791–2.
- Sigaud JFX. Do clima e das doenças do Brasil, estatística médica deste império. Rio de Janeiro: Editora Fiocruz. 2009.

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## Treatment of Listeriosis in First Trimester of Pregnancy

**To the Editor:** Foodborne infections with *Listeria monocytogenes* continue to be dangerous and disruptive. A 2011 outbreak in the United States, linked to cantaloupes, affected 147 persons; 33 persons died, and 1 pregnant woman experienced a miscarriage (1). Moreover, the incidence of listeriosis has been rising in several European countries (2). Compared with the general population, pregnant women are at markedly increased risk of acquiring listeriosis (3). Women who are infected with *L. monocytogenes* in the third trimester of pregnancy are typically treated with antimicrobial drugs until the child's delivery (3). However, the optimal treatment regimen for listeriosis early in pregnancy is unknown.

We cared for a 28-year-old, previously healthy woman who sought treatment at 12 weeks' gestational age with fever, headache, and neck stiffness; blood cultures were positive for *L. monocytogenes*. Lumbar puncture on admission to our hospital in Boston, Massachusetts, in December 2011, revealed clear fluid and an opening pressure of 15 mm Hg; 1 leukocyte was observed per high-powered field, and cultures of the cerebrospinal fluid were sterile. Pelvic ultrasound showed no abnormalities of the fetus, gestational sac, or uterus.

We treated the patient's condition with intravenous ampicillin

for 2 weeks, 2 g every 4 hours, and gentamicin, 100 mg every 8 hours, followed by ampicillin alone for 2 weeks. Shortly after the antimicrobial drugs were initiated, the patient defervesced and her blood cultures cleared. Her hospital course was complicated by spinal headache and transient acetaminophen-induced liver injury, but she was eventually discharged to her home in good condition. Blood cultures taken after discontinuation of antimicrobial agents were sterile, and the remainder of her pregnancy was unremarkable.

She ultimately gave birth to a healthy 2,405-g boy with Apgar scores of 4 and 7 (at 1 and 5 min, respectively) at 35.1 weeks' gestation by spontaneous vaginal delivery. Pathologic examination of the placenta showed no evidence of chorioamnionitis, villitis, or parenchymal abscesses, and placental cultures were sterile. The patient and her child are currently doing well without obvious sequelae of infection.

Listeriosis in early pregnancy presents a unique challenge for the infectious diseases clinician. Up to 30% of *L. monocytogenes* infections in pregnancy result in stillbirth, miscarriage, or preterm labor, and approximately two thirds of surviving neonates are infected (4). *L. monocytogenes* uses 2 surface proteins, InlA and InlB, to invade host cells, including the placenta (5). Once established within the placenta, *L. monocytogenes* forms microabscesses, which can lead to recurrence of infection (6). A recent study in which researchers used a guinea pig model suggests that eradication of microabscesses from the placenta may be critical to achieving the cure of the mother and the prevention of fetal illness and death (7).

What, then, is the optimal treatment strategy to cure the mother and sterilize the placenta? In a large case series of pregnant women with listeriosis, most patients were given a

b-lactam antimicrobial drug, with or without gentamicin (6). However, most women in this case series were in their third trimester of pregnancy and received treatment until delivery. In women who are infected in the first or second trimester, continuing intravenous antimicrobial drugs until delivery is impractical, and the efficacy of oral antimicrobial agents in preventing recurrence of infection is unknown.

Our case demonstrates that 4 weeks of intravenous therapy can sterilize the placenta and enable good maternal and fetal outcomes in a woman infected with listeriosis in the first trimester. We also identified 13 case reports of women in whom listeriosis developed in the first or second trimester of pregnancy (online Technical Appendix, [wwwnc.cdc.gov/EID/article/19/5/12-1397-Techapp1.pdf](http://wwwnc.cdc.gov/EID/article/19/5/12-1397-Techapp1.pdf)). Among these 13 case-patients, 8 instances occurred in which both mother and neonate survived without sequelae; all 8 patients had received ampicillin/penicillin with or without gentamicin.

The role of gentamicin in treatment of listeriosis in pregnancy is controversial. The combination of ampicillin and gentamicin has been thought to be synergistic, although in vivo evidence of clinical benefit, compared to that of treatment with ampicillin alone, is lacking (3,6). A particular concern in pregnancy is gentamicin's poor penetration into the intracellular space, where *L. monocytogenes* is likely to reside, in the placenta (8). Furthermore, some concern exists that gentamicin use in pregnancy could cause fetal ototoxicity, although few such cases have been reported, and several small cohort studies have not shown this association (9,10). Our patient's child had a normal result when standard audiology testing was performed several days after delivery.

Infectious diseases clinicians will likely see patients with listeriosis in early pregnancy, given the increasing incidence of this infection in many countries and the ongoing threat of

food-borne outbreaks. The collected experience from the cases reported here may be useful, particularly given the absence of high quality clinical data that support treatment recommendations for this population. Intravenous ampicillin, with or without gentamicin, effectively sterilizes the placenta and prevents maternal and fetal illness and death in cases of listeriosis in early pregnancy.

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## References

- Centers for Disease Control and Prevention. Multistate outbreak of listeriosis linked to whole cantaloupes from Jensen Farms, Colorado—listeriosis [cited 2012 Sep 13]. <http://www.cdc.gov/listeria/outbreaks/cantaloupes-jensen-farms/index.html>
- Allerberger F, Wagner M. Listeriosis: a resurgent foodborne infection. *Clin Microbiol Infect*. 2010;16:16–23. <http://dx.doi.org/10.1111/j.1469-0691.2009.03109.x>
- Janakiraman V. Listeriosis in pregnancy: diagnosis, treatment, and prevention. *Rev Obstet Gynecol*. 2008;1:179–85.
- Smith B, Kemp M, Ethelberg S, Schiellerup P, Bruun BG, Gerner-Smidt P, et al. *Listeria monocytogenes*: maternal-foetal infections in Denmark 1994–2005. *Scand J Infect Dis*. 2009;41:21–5. <http://dx.doi.org/10.1080/00365540802468094>
- Bonazzi M, Lecuit M, Cossart P. *Listeria monocytogenes* internalin and E-cadherin: from bench to bedside. *Cold Spring Harb Perspect Biol*. 2009;1:a003087. <http://dx.doi.org/10.1101/cshperspect.a003087>

6. Mylonakis E, Paliou M, Hohmann EL, Calderwood SB, Wing EJ. Listeriosis during pregnancy: a case series and review of 222 cases. *Medicine* (Baltimore). 2002;81:260–9. <http://dx.doi.org/10.1097/00005792-200207000-00002>
7. Bakardjiev AI, Theriot JA, Portnoy DA. *Listeria monocytogenes* traffics from maternal organs to the placenta and back. *PLoS Pathog*. 2006;2:e66. <http://dx.doi.org/10.1371/journal.ppat.0020066>
8. Hof H, Nichterlein T, Kretschmar M. Management of listeriosis. *Clin Microbiol Rev*. 1997;10:345–57.
9. Repchinsky CE, editor. *Compendium of pharmaceuticals and specialties: the Canadian drug reference for health professionals*. Ottawa (Ontario, Canada): Pharmacists Association/Association des Pharmaciens du Canada; 2003.
10. Kirkwood A, Harris C, Timar N, Koren G. Is gentamicin ototoxic to the fetus? *J Obstet Gynaecol Can*. 2007;29:140–5.

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## Correction: Vol. 16, No. 12

The name of author Sri Irianti was misspelled in the article Environmental Sampling for Avian Influenza A (H5N1) in Live-Bird Markets, Indonesia. The article has been corrected online ([www.wnc.cdc.gov/eid/article/16/12/10-0402](http://www.wnc.cdc.gov/eid/article/16/12/10-0402)).

# etymologia

## *Acinetobacter* [ascī-net'ō-bak'tər]

From the Greek *akineto* (immobile), a genus of gram-negative paired coccobacilli that are widely distributed in nature and can cause severe primary infections in compromised hosts. *Acinetobacter* was most likely first described as *Diplococcus mucosus* in 1908. In 1954, Brisou and Prévot proposed the genus *Acinetobacter* to indicate that the bacteria were nonmotile because they lacked flagella. *Acinetobacter* are still generally described as nonmotile, but most isolates exhibit “twitching” motility.

*Acinetobacter baumannii*—named in honor of American bacteriologists Paul and Linda Baumann—is a nosocomial pathogen with acquired multidrug resistance that is emerging as a major concern worldwide. Motility is linked to increased virulence in bacteria such as *Pseudomonas aeruginosa* and *Dichelobacter nodosus*; however, whether motility plays a role in the virulence of *A. baumannii* remains unclear.

### Sources

1. Allen DM, Hartman BJ. *Acinetobacter* species. In: Mandell GL, Bennett JE, Dolin R, editors. *Principles and practices of infectious diseases*. 7th ed. Philadelphia: Churchill Livingstone; 2010. p. 2881–5.
2. Bouvet PJ, Grimont PA. Taxonomy of the genus *Acinetobacter* with the recognition of *Acinetobacter baumannii* sp. nov., *Acinetobacter haemolyticus* sp. nov., *Acinetobacter johnsonii* sp. nov., and *Acinetobacter junii* sp. nov. and emended descriptions of *Acinetobacter calcoaceticus* and *Acinetobacter lwoffii*. *Int J Syst Bacteriol*. 1986;36:228–40. <http://dx.doi.org/10.1099/00207713-36-2-228>
3. Clemmer KM, Bonomo RA, Rather PN. Genetic analysis of surface motility in *Acinetobacter baumannii*. *Microbiology*. 2011;157:2534–44. <http://dx.doi.org/10.1099/mic.0.049791-0>
4. *Dorland's illustrated medical dictionary*. 32nd ed. Philadelphia: Elsevier Saunders; 2012.
5. Eijkelkamp BA, Stroehrer UH, Hassan KA, Papadimitriou MS, Paulsen IT, Brown MH. Adherence and motility characteristics of clinical *Acinetobacter baumannii* isolates. *FEMS Microbiol Lett*. 2011;323:44–51. <http://dx.doi.org/10.1111/j.1574-6968.2011.02362.x>
6. McConnell MJ, Actis L, Pachón J. *Acinetobacter baumannii*: human infections, factors contributing to pathogenesis, and animal models. *FEMS Microbiol Rev*. 2013;37:130–55.

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