Paratyphoid Fever Due to *Salmonella enterica* Serotype Paratyphi A

To the Editor: An outbreak of paratyphoid fever caused by *S.* Paratyphi A occurred during September and October 1996 in a residential area of New Delhi, India.

S. Paratyphi A has been responsible for 3% to 17% of cases of enteric fever in India (1). We suspected an outbreak because the *S.* Paratyphi A isolation rates exceeded the expected frequency based on the blood culture-positive rates from the cases of enteric fever reported by the Department of Microbiology at the All India Institute of Medical Sciences, New Delhi, the previous September and October (nine cases in 1995, 36 cases in 1996).

Thirty-six cases of culture-positive enteric fever due to *S.* Paratyphi A were reported on the basis of blood cultures received by the Department of Microbiology at the All India Institute of Medical Sciences Hospital, New Delhi, during September and October 1996. All the patients lived in the same residential area of 428 homes. The male to female ratio was 2:1, and most cases were in young adults (mean age = 20.1 yrs). All patients had a history of fever of 3 to 5 days' duration. The first culture-confirmed case was reported on September 12, 1996. After the initial case, 14 cases were reported in week 1, 10 cases in week 2, five cases in week 3, three cases in week 4, two cases in week 5, and two in week 6. Four households reported two cases each; the rest reported only one case per household. All the patients responded to ciprofloxacin treatment. All the isolates were sensitive to chloramphenicol, amoxycillin, cotrimoxazole, ciprofloxacin, gentamicin, and ceftriaxone. All the strains belonged to phage type 1.

The first suspected source of infection was contaminated food because two important Hindu festivals were celebrated on August 28 and September 5, 1996, respectively, just before the first culture-positive report on September 12. Investigators visited the affected households and distributed a questionnaire regarding demographic information, history of fever, food consumption from a common source, festival attendance, and type of water supply used. All the household contacts were also questioned. The information gathered did not indicate a foodborne outbreak. The second suspected source of infection was the water supply. The residential area receives water intermittently from a central reservoir. The water and sewage pipelines lie close to each other; the sewer line has many joints close to the water pipes, so the water may become contaminated with human excreta from the sewer line. New Delhi had a heavy rainfall toward the end of August and the beginning of September 1996, which led to waterlogging in the residential area. The contaminated soil might have entered the water pipes (because of negative pressure inside the pipes created by intermittent water supply) and contaminated the water supplied to these households. Water samples from these households during the last week of September did not contain fecal coliform. Soil samples from different sites did not contain salmonellae. Since *S. typhi* does not survive long in the environment, isolating the organism from the source is difficult by the time the outbreak is suspected (2). This may also be true for *S.* Paratyphi A.

An outbreak of enteric fever due to *S.* Paratyphi A has never been reported. Although we could not isolate the organism from the water or the soil by the time the outbreak was suspected, epidemiologic evidence suggests a waterborne outbreak.

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MHC and Infectious Diseases

To the Editor: The review on the importance of the major histocompatibility complex (MHC) in infectious diseases by Singh et al. (Emerg Infect Dis 1997;3:41-9) failed to mention the potential role of human leukocyte antigen (HLA)-DM in conferring susceptibility to infectious diseases. HLA-DM is an MHC class II-like molecule essential for normal antigen processing and presentation (1). HLA-DM has been shown to function as a peptide editor, in that it influences the repertoire of peptides bound to HLA-DR.

Furthermore, this influence occurs in an allele-specific fashion (2). In addition, HLA-DM polymorphisms have been reported to confer an increased relative risk for such varied entities as rheumatoid arthritis (3), kidney transplant rejection (4), and membranous nephropathy (5). Since HLA-DM is important in determining which peptides are immunogenic, it may be as important as MHC class II molecules in regulating the immune response and therefore in conferring susceptibility to infectious diseases.

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Reply to V.S. Sloan: Dr. Sloan has rightly pointed out the importance of HLA-DM in regulating the immune response in rheumatoid arthritis, kidney transplant rejection, and membranous nephropathy. We did not mention the role of HLA-DM because our review dealt solely with infectious diseases that have well-established HLA associations.

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Acute Epiglottitis due to *Pasteurella multocida* in an Adult without Animal Exposure

To the Editor: *Pasteurella multocida* infection in humans usually involves animal contact, most commonly with a domestic dog or cat (1). Epiglottitis due to human *P. multocida* infection associated with animal contact is very rare (2-4). We report a case of epiglottitis due to *P. multocida* not associated with animal contact.

A 44-year-old patient was admitted to the hospital with fever, throat fullness, and drooling. He had been healthy until 12 hours before admission when he noticed difficulty in swallowing liquids; anterior neck discomfort and fever followed, and soon he could not swallow his saliva.

When he arrived at the Emergency Department of Montefiore Medical Center on September 23, 1996, the patient was mildly toxic and had an oral temperature of 103.2°F. Pulse was 110 and blood pressure 110/70. He was drooling. He had mild anterior neck tenderness, no cervical adenopathy, no pharyngitis on inspection of the oropharynx, and no palate deviation. The heart, lungs, abdomen, and skin showed no abnormalities. A lateral neck radiograph showed an enlarged epiglottis ("thumb sign"). Indirect laryngoscopy confirmed inflamed and edematous epiglottis and supraglottic structures. A culture of the epiglottis was not performed.

On admission, the patient had a hemoglobin of 1.9 g/dL; hematocrit was 48%; white blood cell count was 14,100/mm^3; and platelet count was 170,000/mm^3. A machine differential count showed 86% granulocytes, 9% lymphocytes, and 5% monocytes.

The patient was treated with dexamethasone and ceftriaxone. The fever abated rapidly, and all symptoms resolved. Repeat laryngoscopy on day 3 confirmed resolving epiglottitis. Blood cultures taken on admission grew gram-negative, oxidase-positive bacilli that did not grow on MacConkey agar (BBL, Cockeysville, MD) in two sets, both aerobically and anaerobically. The isolate was identified as *P. multocida* by the Vitek GNI card (BioMérieux-Vitek, Inc., Hazelwood, MO). Kirby-Bauer susceptibility testing demonstrated susceptibility to penicillin. Because of the patient's marked improvement after treatment with