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.

Victor S. Sloan
University of Medicine and Dentistry of New Jersey
New Brunswick, New Jersey, USA

References

1. Busch R, Mellins ED. Developing and shedding inhibitions: how MHC class II molecules reach maturity. Curr Opinion Immunol 1996;8:51-8.
2. Sloan VS, Zaller DM. Allelic specificity of the influence of HLA-DM on peptide repertoire. Arthritis Rheum 1996;39:S310.
3. Pinet V, Combe B, Avinens O, Caillat-Zucman S, Sany J, Clot J, Eliaou JF. Polymorphism of the HLA-DMA and HLA-DMB genes in rheumatoid arthritis. Arthritis Rheum 1997;40:854-8.
4. Chevrier D, Giral M, Bignon JD, Muller JY, Soulillou JP. Impact of the "new" MHC-encoded genes (HLA-DMA, -DMB and LMP2) on kidney graft outcome. Hum Immunol 1996;47:O717.
5. Giral M, Chevrier D, Muller JY, Bignon JD, Soulillou JP. TAP1*0201 and HLA-DMA*0103 markers and severe forms of membranous nephropathy. Hum Immunol 1996;47:0140.

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.

Neeloo Singh
Central Drug Research Institute
Lucknow, India

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

ceftriaxone and convenience of outpatient parenteral therapy, this antibiotic was continued to complete a 10-day course. On extensive questioning, the patient denied contact with any cat, dog, or other animal. He had recently traveled to Nigeria but denied even transient animal contact.

Since 1966, three cases of *P. multocida* epiglottitis have been reported (2-4). Although no direct culture of the epiglottis was performed in the present case, the clinical syndrome and the absence of any other focus accounting for *P. multocida* bacteremia strongly suggest that this organism caused the epiglottitis. Including the present case, three of the four reported cases have occurred since 1993, which suggests that either earlier cases were not recognized or the incidence of this condition may be increasing. In all three previous cases of *P. multocida* epiglottitis, the patients had cats as pets. As in the current case, the clinical features of *P. multocida* epiglottitis were indistinguishable from epiglottitis secondary to more common bacterial pathogens. However, the cases were all associated with positive blood cultures. In contrast, a 23% rate of bacteremia was reported in a series of epiglottitis cases in adults (including patients with blood cultures positive for *Haemophilus influenzae* type b or Group A streptococci)(5).

The vehicle of infection for this patient remains unknown, as human-to-human transmission has not been documented. This case demonstrates that epiglottitis due to *P. multocida*, a rare condition that may be increasing in frequency, need not be accompanied by recognized exposure to animals.

Michael Glickman and Robert S. Klein
Montefiore Medical Center and the Albert Einstein College of Medicine, New York, New York, USA

References

1. Weber DJ, Wolfson JS, Swartz MN, Hooper DC. *Pasteurella multocida* infections: report of 34 cases and review of the literature. Medicine 1984;63:133-54.
2. Johnson RH, Rumans LW. Unusual infections caused by *Pasteurella multocida*. JAMA 1977;237:146-7.
3. Leung R, Jassal J. Pasteurella epiglottitis. Aust N Z J Med 1994;24:218.
4. Rydberg J, White P. *Pasteurella multocida* as a cause of acute epiglottitis. Lancet 1993;341:381.
5. MayoSmith MF, Hirsch PJ, Wodzinski SF, Schiffman FJ. Acute epiglottitis in adults. An eight-year experience in the state of Rhode Island. N Engl J Med 1986;314:1133-9.

Hemolytic Uremic Syndrome Surveillance to Monitor Trends in Infection with *Escherichia coli* O157:H7 and Other Shiga Toxin-Producing *E. coli*

To the Editor: In the past 15 years, knowledge about the role of Shiga toxin-producing *Escherichia coli* (STEC) in human disease has expanded rapidly. The most distinctive complication of STEC infection is diarrhea-associated hemolytic uremic syndrome (HUS), a major cause of acute renal failure in U.S. children. Other manifestations of STEC infection can range from mild diarrhea to severe hemorrhagic colitis, thrombotic thrombocytopenic purpura, and death (1). In the United States, O157 is the most common STEC and causes an estimated 20,000 infections and 250 deaths annually. *E. Coli* O157 outbreaks associated with beef have caused concern among public health workers, clinicians, and the public, prompting major changes in clinical and laboratory practice, meat production, and food preparation. However, critical questions remain unanswered. Have prevention measures decreased risk? Are new sources of STEC infections emerging? Is the incidence of O157 infection changing? How much illness is due to STEC of serotypes other than O157?

Diarrhea-associated HUS is associated with Shiga toxin, which is produced in quantity only by STEC and by *Shigella dysenteriae* type 1; approximately 90% of HUS cases are diarrhea-associated (2,3). In the United States, where *S. dysenteriae* type 1 infections are very rare, STEC infections are the cause of virtually all diarrhea-associated HUS. The incidence of HUS in North America is about three cases per 100,000 children under 5 years of age per year; the rate among older children is somewhat lower, and the rate among adults is not known (2-6). HUS complicates approximately 5% to 10% of O157 infections and an unknown percentage of non-O157 STEC infections (1). Except for supportive care and hemodialysis, no treatment has been shown to decrease the severity of illness or to prevent complications. The sequelae of HUS—death in 3% to 5% of cases (2,3,5) and long-term renal dysfunction in 10% to 30% of survivors (6)—and the lack of specific therapy make prevention critical.