Streptococcus suis and Porcine Reproductive and Respiratory Syndrome, Vietnam

To the Editor: Streptococcus suis, an opportunistic pathogen of swine, is an emerging zoonotic pathogen among humans (1). In Vietnam, S. suis is the leading cause of human acute bacterial meningitis (2). Infection in humans is associated with direct exposure to infected pigs or infected raw or undercooked pork products (3). Of the 35 S. suis serotypes, only a limited number are pathogenic for pigs, and clinical cases in humans have most frequently been attributed to serotype 2 (SS2) (1). In Vietnam during September 2006–November 2007, the carrier rate of S. suis among slaughterhouse pigs was 41% (222/542); SS2 was the most frequently identified serotype in 14% (45/317) of S. suis isolations (4).

Porcine respiratory and reproductive syndrome (PRRS) is a major disease affecting the swine industry globally; the severity of PRRS in pigs can be increased by co-infection with S. suis (5). In 2010, PRRS outbreaks in swine were reported in 49 of 63 Vietnamese provinces (online Technical Appendix Figure). We investigated the presence of SS2 in blood and tissue samples from pigs on PRRS– and non-PRRS–affect ed farms by bacterial culture (online Technical Appendix Table). A total of 534 specimens from sick pigs yielded 9 (1.7%) SS2 isolates. One (2%) of 52 specimens from the healthy control pigs yielded a non-SS2 S. suis isolate. S. suis has been proposed to contribute to the spread of antimicrobial resistance genes to other human pathogenic streptococci (6). The antimicrobial susceptibility results of 9 SS2 isolates by disk diffusion (7) revealed a high prevalence (6/9, 66%) of resistance to tetracycline, tobramycin, enrofloxacin, and either marbofloxacin or chloramphenicol.

PCR amplification of the 16SrDNA gene (8) and the cps2J gene (2)
was performed on all blood samples to detect S. suis and SS2, respectively. Ninety-two (18%) of 521 sick pigs from PRRSV outbreak farms were systemically infected with S. suis. In contrast, no healthy pigs from control farms were positive for S. suis by PCR (online Technical Appendix Table). The SS2-cps2J-specific PCR was positive for 58 (11%) of 521 samples, and the S. suis-16SrDNA PCR was positive for 55 (11%). Twenty-one of the 16SrDNA-positive samples also were positive for cps2J-PCR, which indicated that 34 (7%) sick pigs were infected with non-SS2 strains. Therefore, SS2 accounted for most (58 [63%] of 92) S. suis–positive detections. The bacterial load of SS2 in blood ranged from 1 × 10^3 CFU/mL–1 to 8.3 × 10^6 CFU/mL–1 (median 9.2 × 10^3 CFU/mL–1). Overall, SS2 was found in 58 (11%) sick pigs and on 33 (32%) PRRS outbreak farms. The higher prevalence (92 [18%]) of systemic infections of S. suis and SS2 with high bacterial load in pigs from PRRS outbreak farms compared with prevalence on nonoutbreak farms (1 [2%] of 52) suggests increased systemic S. suis infections during swine PRRS outbreaks (p = 0.001, Fisher exact test).

We investigated the possible association between swine PRRS outbreaks and human S. suis infection. Case reports of confirmed human infections during 2007–2010 at the 2 tertiary referral hospitals in Hanoi and Ho Chi Minh City were reviewed. The number of human S. suis infection cases increased in August 2010 in southern Vietnam and doubled in northern Vietnam during May–August and October–November 2010 (Figure). Swine PRRS outbreaks were reported during June–September and March–December 2010 in southern and northern provinces, respectively (6) (online Technical Appendix Figure). Most patients with S. suis infection during these periods resided in provinces reporting swine PRRS outbreaks. Our data suggest a possible temporal association between swine PRRS outbreaks and human S. suis infections.

We demonstrated increased prevalence of systemic S. suis and SS2 infection in pigs co-infected with PRRSV during the 2010 swine outbreaks in Vietnam. The results indicate an increased risk for potential zoonotic transmission of S. suis to humans during outbreaks of PRRS in swine.

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Hepatitis E Virus Seroprevalence among Men Who Have Sex with Men, United Kingdom

To the Editor: Immunosuppression might be associated with chronic carriage of hepatitis E virus (HEV) (1,2). HIV-infected persons could be at increased risk for HEV acquisition (3). If HIV infection is a risk factor for HEV, the risk will probably be mediated by associated behavioral factors. Men who have sex with men (MSM) are known to be at risk for transmission of enteric infection (4). Because of increasing prevalence of chronic liver disease induced by various causes among HIV-infected persons, it is necessary to determine whether these patients are at risk for HEV acquisition and possible hepatic decompensation (5).

We aimed to establish the contribution of HIV infection and MSM to seroprevalence of HEV among banked serum specimens. We used an unlinked, anonymous HIV seroprevalence survey of sexual health clinic attendees in England, Wales, and Northern Ireland, compared results from testing of residual serum samples collected for routine syphilis testing from sentinel clinics, and analyzed basic epidemiologic data (6). We examined serum samples collected during a 3-year period (2006–2008) and stored at −80°C. All samples were from male patients, 20–44 years of age. IgG against HEV was measured by using ELISA (Wantai; Fortress Diagnostics, Antrim, UK). To further increase the specificity for a seroprevalence analysis, and in accordance with previous work (7), we defined only samples with an optical density/cutoff value ≥1.5 as reactive and those in the range 1.0–1.5 as weakly reactive.

We analyzed 422 serum samples collected during 2008, comprising 146 samples from MSM with positive HIV test results, 135 from MSM with negative HIV test results, and 141 from heterosexual men with negative HIV test results. Thirty (7.1%) serum samples showed IgG reactivity against HEV and 3 (0.7%) additional samples showed weak reactivity. We examined the effect of HIV infection on prevalence of IgG against HEV by comparing samples from HIV-infected MSM with those from HIV-negative MSM. Seroprevalence rates did not differ significantly (HIV-positive MSM 7.5%; HIV-negative MSM 10.4%; p = 0.4).

We then examined the effect of being MSM as a risk factor for HEV infection. Prevalence of IgG against HEV among HIV-negative heterosexual men was 3.5%, significantly lower than that among MSM (odds ratio 3.1, p = 0.025, for comparison with non-HIV–infected MSM). We examined the relationship of status of IgG against HEV among MSM to the presence of an acute non-HIV sexually transmitted infection (STI) at the time of serum sampling. No association was found (acute STI, 14 [9.1%] of 154 vs. no acute STI, 11 [8.7%] of 127; p = 0.9). Similarly, no statistical association was found between HEV antibody status and the location of the clinic that provided the sample serum (London, 21 [10.0%] of 211; United Kingdom excluding London, 4 [5.7%] of 70; p = 0.3). As has been observed for the general UK population (7), we

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