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International Travel and Sexually Transmitted Disease

To the Editor: Recent articles in the professional literature (1–3) have offered advice regarding the importance of taking a careful travel history, particularly in this time of unprecedented levels of international travel

(4). Such screening serves an important public health purpose as well, especially for sexually transmitted disease (STD) control.

Sexual behaviors associated with travel can change the level of risks for STD transmission (5–7), and the epidemiology of STDs is not uniform throughout the world (8,9). These geographic differences may increase the risk of a traveler's becoming infected, or, conversely, increase the risk of a traveler's introducing a sexually transmitted pathogen, possibly one that is resistant to treatment, into a low-incidence area (10). In addition, different strains of pathogens may be common in different parts of the world (11–14). For example, quinolone-resistant *Neisseria gonorrhoeae* (QRNG) is much more common in Asia (up to 40% of all isolates) (15). These strains of QRNG were first introduced in the United States by persons who engaged in sexual activity abroad, but now California and Hawaii have an increasing incidence of infection attributable to these strains (16). Indeed, QRNG has become endemic in those states, and incidence is no longer related to travel. During 1999–2001, only 3 QRNG isolates (0.28%) were identified among the 1,066 gonococcal isolates cultured in the STD Laboratory, State Laboratory Institute, Massachusetts Department of Public Health (Massachusetts Department of Public Health, unpub. data). However, in 2002, 9 (2.1%) of 425 isolates of *Neisseria gonorrhoeae* were quinolone resistant. None of the persons recently infected reported a history of travel outside of New England. Unfortunately, few had reliable information to identify their partner(s). Those partners who were identified were either not located or did not agree to speak with the disease intervention specialist.

This experience with antimicrobial resistance of *Neisseria gonorrhoeae* should serve as a model for STD prevention planning and programming. It highlights the importance of retaining the laboratory capacity to monitor antimicrobial susceptibilities of bacterial STD isolates. Treatment protocols should be adjusted in light of the prevalence of resistant strains of sexually transmitted pathogens. In cases in which symptoms associated with a bacterial STD persist after what is usually considered appropriate treatment, clinicians should obtain cultures and perform susceptibility tests on isolates. Nucleic acid amplification technologies do not provide critical antibiotic susceptibility information. In this situation, the public health STD program or laboratory should be contacted for guidance. Determining the sensitivity pattern of the pathogen in an expeditious fashion will ensure that appropriate and timely therapy can be initiated for the infected patient as well as enable more effective follow-up and treatment to sexual contacts. Asking patients who seek treatment for a possible STD about their own and their partner's travel histories is important to broaden the differential diagnosis (17). The increase in population mixing facilitated by travel and Internet-generated contacts may be diminishing the importance of the focality of traditional STD epidemiology. Finally, STD prevention messages should be a part of the health advice offered to travelers (7,18,19).

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Salmonella in Denmark

To the Editor: In the large study by Evans and Wegener recently published in Emerging Infectious Diseases (1), salmonellae in broiler chickens and pigs significantly decreased after routine in-feed antimicrobial drug use for growth promotion was terminated in Denmark. Avoparcin was a frequently used growth promoter in poultry until its ban in Denmark in 1995 because of its association with the development and spread of vancomycin-resistant enterococci. On examining Evans and Wegener's data, I noticed that a precipitous drop in salmonellae in broiler chickens appeared to have occurred in early 1996. Do the authors think this drop was due to the withdrawal of avoparcin? As the authors note, avoparcin has been associated with increased shedding of salmonellae (including a dose-response effect) in a number of studies (2,3). If the large drop (from approximately 25% positive samples in 1995 to approximately 10% in 1996) is not due to withdrawal of avoparcin, what do the authors suggest could have caused it?

Do the authors have sufficient numbers of samples to reanalyze their data in broiler chickens for three periods instead of just two (i.e., use the periods January 1995–December 1995, January 1996–December 1997, and January 1998–December 2000)? This change would take into account the potential effect of avoparcin withdrawal in 1995.

Also, the most important reason for decreasing food animals' carriage of salmonellae is to protect people from becoming ill with *Salmonella*. Do the authors have any figures on domestically acquired human infections with salmonellae in Denmark since early 1995? Is there any temporal association with the withdrawal of growth promoters?

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In Reply: The drop in *Salmonella* organisms in broiler chickens becomes evident in September 1995. The ban on avoparcin occurred in May 1995. These two facts suggest that the first flocks of broiler chickens produced without avoparcin were slaughtered in August 1995. Thus, the temporal relationship is evident. We have reanalyzed the data for the three strata January 1994–December 1995, January 1996–December 1997, and January 1998–December 2000. Each stratum is significantly different from the two others ($p < 0.0001$).

Arguing in favor of a causal relationship, apart from the temporal relationship, one would say that no changes in the Salmonella control program in this period could explain this reduction. Arguing against a causal relationship, one would say that the levels momentarily bounced back to nearly the pre-ban level in 1997, despite the avoparcin ban. The subsequent drop and consistent low level could be explained by changes in the control program (introduction of serologic *Salmonella* monitoring in 1997 to 1998). On the basis of our data, drawing a conclusion one or the other is not possible.

There is a clear temporal association between reduction in *Salmonella* in broiler chickens and reduced incidence of domestically acquired *Salmonella* infections that can be attributed to domestically produced broilers. This finding was recently reported in this journal (1).

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