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Reply

SIR—We thank Muñoz et al. [1] for their correspondence regarding our article on infectious complications among heart transplant patients at Stanford University Medical Center (Stanford, CA) [2]. Dr. Muñoz and colleagues acknowledge that there are few published data to support the use of trimethoprim-sulfamethoxazole (TMP-SMZ) alone for the prevention of toxoplasmosis in heart transplant recipients. However, their data, along with findings on the use of TMP-SMZ for prophylaxis and treatment of toxoplasmosis in HIV-positive patients, are compelling.

We agree that the use of TMP-SMZ alone may be sufficient to prevent toxoplasmosis in patients who are seronegative for Toxoplasma gondii IgG antibodies and who receive a heart transplant from donors seropositive for T. gondii IgG antibodies (i.e., D+/R− patients). The optimal schedule of administration of TMP-SMZ to this group of patients is less clear and requires further study. Until these studies are performed, physicians must decide whether a schedule of daily administration or administration 3 times a week is to be used. For HIV-infected patients, we routinely recommend daily use of TMP-SMZ whenever feasible [3].

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Putting Salmonella Contamination in Perspective

SIR—The complexity of the food chain and the emergence of the “farm-to-fork” model of the animal feed/food-animal production cycle (and the model’s implied concomitant preventive controls) will remain formidable challenges for all who are objectively committed to minimizing and preventing hazards that cause foodborne illnesses. In support of their own study, Crump et al. [1] reintroduced a hypothesis that first surfaced ~30 years ago [2]. Clark et al. [2] described an outbreak of 17 cases of Salmonella enterica serotype Agona infection among persons in Paragould, Arkansas. This outbreak was traced to the consumption of poultry that had been reared on a Mississippi poultry farm. Epidemiological analysis indicated that the farm used fish meal imported from Peru as a protein supplement in the feed ration and that the fish meal had been contaminated with S. enterica serotype Agona. The convoluted farm-to-fork model has since been used as a basis for the early determination that bacterial contamination of animal feed could have an important causal relationship to foodborne illness in humans.

The study of Clark et al. [2] was amplified during Crump et al.’s [1] examination of the potential for feed and, especially, the animal protein component of feed to contribute to the bacterial colonization and infection of food-producing animals. Crump and colleagues suggested that such colonization and contamination could subsequently “contaminate animal carcasses at slaughter or cross-contaminate other food items, leading to human illness” [1, p. 859]. However, the study of Clark et al. [1] lacked many of the elements pointing to a finite causal association. Therefore, the concepts of “sufficient cause” and “component cause” require elucidation.

A cause of a disease event is an occurrence, condition, or characteristic that preceded the disease event, and that, without which, the disease outbreak would not have occurred. Component causes can be based on either strong or weak evidence. However, the causal inference proposed by Clark et al. [2], in which animal feed was linked to foodborne illness in humans, was based on little more than the mere coincidence of events and was an example of the logical fallacy of post hoc ergo propter hoc (i.e., “after this, therefore, on account of this”). Unfortunately, the report by Clark et al. [2] has been used to exacerbate the concerns of regulatory authorities regarding the implication of rendered animal protein feed as a possible source of foodborne disease in humans. However, the evidence was never more than anecdotal, because, in the “stellar” case reported by Clark et al. [2],
samples obtained from the chickens at the Mississippi farm were not cultured for determination of the source of infection.

Instead of delving into aspects of microbial myths, Crump et al. [1] need to consider some of the serious environmental concerns and questions associated with the farm-to-fork model. For example, is it possible to establish Salmonella-free areas in the production environment in which poultry and livestock are reared? Can we protect livestock and poultry against the environmental contaminants associated with animal husbandry (e.g., dust and dirt, water, fecal matter, excreta, pests [e.g., rodents], wild animals, and slurry and sludge)? How do we epidemiologically evaluate the significance of asymptomatic Salmonella carriage and the resultant implications for the transmission and perpetuation of salmonellosis? How do we prevent the stress to livestock and poultry that naturally occurs during transport and gives rise to increased shedding of infection-causing organisms, and, likely, to cross-infection? How do we preclude post-process contamination of feed on the farm by feces and urine (from rats and wild birds) and other risk factors associated with poor environmental hygiene (e.g., flies, roaches, and contaminated feed troughs)? Is airborne transmission of Salmonella organisms a relevant environmental concern, and to what degree is such transmission a problem at the farm or in the food-processing environment? Are all of the approximately 2300 Salmonella serotypes potentially infectious, and what relevance does this question have to preventive controls?

We need to work together collectively, as a society, by bringing different resources together under one umbrella to objectively examine the challenges associated with rearing healthy animals that will produce safe and healthy foods for human consumption. The elimination of foodborne diseases remains a distinct challenge. The words of my former administrator, Donald L. Houston, in an address presented at one of the early international symposia on Salmonella species, remain true to this day: “Because it is a chronic public health problem, salmonella confronts government, industry, and the scientific community as both a challenge and a reproach. It is a challenge because it sometimes appears that with our science and technology we are better able to strive toward a certain well-defined objective, like the moon, than to overcome a chronic, food-poisoning hazard” [3, p. 2].

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References

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Reply
Sir—we agree with Dr. Franco [1] that efforts to reduce the human health burden due to foodborne diseases are needed throughout the food chain (i.e., from “farm to fork”). Such efforts should include preventing the introduction of human pathogens at all points of the food chain [2]. Lack of attention to events that occur early in the food chain (i.e., at the level of food animal production) has led to adverse consequences for human health. Such consequences include the emergence of variant Creutzfeldt-Jakob disease [3] and the accelerated development of antimicrobial resistance among Salmonella organisms and other human pathogens, for which food animals are major reservoirs [4]. Conversely, other important zoonoses, such as brucellosis and Mycobacterium bovis infection, have been controlled and, in some areas, eliminated by measures implemented on the farm and among food animals.

The ecologies of the non-Typhi serotypes of Salmonella enterica, an important group of foodborne zoonoses, are indeed complex. Although non-Typhi Salmonella serotypes may vary in degree of virulence, we presume that all are pathogenic. Human volunteer feeding studies (performed during the 1950s) of putative “nonvirulent” Salmonella serotypes established that all of the tested serotypes caused disease if given in sufficient doses [5]. Although non-Typhi Salmonella infection among humans continues to develop at a high rate in the United States, salmonellosis in humans has been successfully controlled in several European countries by use of a comprehensive farm-to-fork approach that includes prevention of Salmonella contamination of animal feed in concert with other improvements in animal husbandry practices [6]. Elsewhere, implementation of biosecurity practices that include controls on the importation of animal feed and of feed ingredients has prevented some pandemic non-Typhi Salmonella serotypes from emerging as human public health problems [7]. Therefore, it is not surprising that investigations of several outbreaks of foodborne illness in humans in the United States have implicated animal feed as an ultimate source [2]. In the United States, surveillance of animal feed and other sources for Salmonella organisms is currently limited and is not integrated with surveillance of food animals, food, and humans for Salmonella organisms. Consequently, it is likely that other outbreaks of Salmonella infection associated with animal feed have occurred that could not be traced.

The control of salmonellosis and other