

cluding atherosclerosis, which, notes Ewald, may be caused by *Chlamydia pneumoniae*, a very common infection. This issue is currently the subject of intense international research activity, but researchers have yet to prove a causal relation.

The bottom line is that the contentions here are carried too far. Ewald seems to believe that any deviation from the norm could be caused by an infection — even, he suggests, artistic creativity. I find this way of thinking frightening.

Ewald also stresses that the supposition that the infectious agent and its host will, over time, put selective pressures on each other toward the persistence of both and the least damage to either is not necessarily true. The evidence against this principle comes from situations in which the rapid transmission of an organism occurs, with selection for increased virulence — for instance, in locations where there is a high degree of crowding, mobility, and sexual contact as well as poor sanitary conditions. Ewald discusses such short-term success of highly virulent organisms as a revelation of “evolutionary biology.” However, Sir Macfarlane Burnet, one of the leading proponents of the theory that successful host–parasite relationships adapt in the direction of benignity, actually discussed it about 50 years ago: “If the microorganism causing an epidemic is susceptible to variations in the direction of higher virulence, then opportunities for rapid spread in susceptible persons will cause an apparent increase in virulence by selection of the fittest, i.e. most virulent, bacteria” (*The Natural History of Infectious Disease*. Cambridge, United Kingdom: Cambridge University Press, 1953).

This book has a somewhat self-congratulatory tone — the author likes to assert that he made predictions and then to congratulate himself for having been right, as in the case of an accurate prediction that the Spanish influenza strain would not reappear during the 1990s. That a population becomes susceptible to the epidemic spread of an infection because acquired immunity has waned does not necessarily mean that the pathogen is still sitting there, waiting to reinfect people.

There are also some errors and misinterpretations in this book. For example, Ewald asserts that the transmission of genital herpesvirus infections is caused by stress, which induces the replication of the virus, the formation of blisters, and the subsequent transmission of the virus from lesions. The current understanding, however, is that inapparent infection and the asymptomatic shedding of virus are probably responsible for transmission in most cases. In trachoma, blindness is not caused by “puffiness” of the conjunctiva but is, rather, secondary to the scarring of the conjunctiva. Genital chlamydial infections (with secretions, Ewald supposes, contaminating the garments of persons in areas where trachoma is endemic) are not considered to have a role in the transmission of trachoma. The model Ewald presents of the replication of human papillomavirus in his discussion of cervical cancer probably has little relevance for the actual transmission of that virus. Young, sexually active women have very high rates of inapparent infection, and long-lasting dysplasia reflects a persistence of infection. That women with multiple sexual partners have a higher prevalence of infection with the “high-risk” types of human papillomavirus need not reflect increased virulence (there is no evidence of changes in the virulence of specific types of the virus); it can, instead, be easily explained by increased exposure and the more persistent nature of the infection

caused by these types of the virus. The bottom line: an important subject, a disappointing book.

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CORRECTION

Efficacy of Mycophenolate Mofetil in Patients with Diffuse Proliferative Lupus Nephritis (February 1, 2001;344:382-3). On page 383, the authors should have been listed as Fotini B. Karassa, M.D., followed by David A. Isenberg, M.D.

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