Letters to the Editor

RE: "ASSOCIATION OF CATS AND TOXOPLASMOSIS"

Reference is made to the interesting and provocative article by Ganley and Comstock (1). In my opinion, the role of the cat in human toxoplasmosis is not as enigmatic as the authors imply. The questions they raised resulted from their evaluation of a number of retrospective seroepidemiologic studies, including their own. It seems to me that retrospective seroepidemiologic data obtained from a variety of sources, correlating toxoplasma antibody prevalence with exposure to cats, or to occupations and living conditions associated with presumed exposure to oocysts, can not provide uniform data unless it can be definitely established that the subjects never have been in contact with felids or felid feces. Correlation of positive serology and housecats is indeed too crude a discriminator to show an association. Since antibody to toxoplasma may persist for life, retrospective studies tend to evaluate events that have occurred so many years previously, they are beyond memory. The hypothesis stated by the authors that oocysts may be so ubiquitously distributed in nature that risk of acquiring infection is primarily a function of increasing age and nonspecific contact opportunities, is certainly supported by many epidemiologic observations (data indicating common source exposure). However, environmental conditions in certain geographic locations favor the persistence of oocysts in the environment and man's contact with them, more so than in other areas. Furthermore, the prevalence of acute toxoplasmosis (oocyst shedding) in cat populations may vary considerably depending on age and other factors (2). As mentioned by the authors, alley cats (stray cats) dependent on garbage (meat scraps) or wild rodents and birds for their food obviously would have a higher prevalence of infection than house cats primarily fed on cooked, commercially prepared pet food. The possibility that a stray or a neighbor's cat may defecate on someone's property even though a cat does not live there has to be considered also.

Therefore, simply asking the question of an adult 30 years or older, if there is a cat on the property where he or she is currently living, or had been living for 12 years, is not a valid method of evaluating the role of the cat in the transmission of the parasite to man. As the authors pointed out, raw or undercooked meat is a well-known source of toxoplasmosis for humans, yet this source of exposure in the population they observed was not mentioned. Also, a group of patients in the Ganley and Comstock study presumably had toxoplasma choreoretinitis. If so, they probably would have been infected congenitally and their exposure after birth to cats would be of no relevance and should not be included in the data. Furthermore, studies have demonstrated that the prevalence of antibody can vary considerably among different ethnic groups (with different lifestyles) inhabiting the same geographic area (3). This probably reflects differences in exposure to soil and oocyst-contaminated food, as well as to the consumption of raw meat. Although the specificity of antibody detected by indirect immunofluorescent tests cannot be proven in any one person not known to have been infected with toxoplasma, there is no evidence that I am aware of disputing the specificity of the test, which correlates well with the Sabin-Feldman dye test.
LETTERS TO THE EDITOR

Although the authors suggest the possibility of definitive hosts of toxoplasma other than felids, they did refer to data that do not support that hypothesis. In fact, there should be no doubt that the felid is the definitive host of *Toxoplasma gondii*. This is not only substantiated by many experimental studies, some of which were referred to by the authors, it is also supported by epidemiologic studies, one of which they did not mention. Data from this study (4) conducted in a group of remote South Pacific atolls, demonstrated that a human population (including adults) definitely known to have never been in contact with cats or cat feces had not been infected with *Toxoplasma*, whereas the prevalence of infection was high in ethnically similar populations that were in contact with cats and cat feces. This study, where infection by raw meat as well as exposure to cats throughout a lifetime could be evaluated, as well as observations on other Pacific Islands where felids had not been introduced (2), leaves little doubt that *Toxoplasma gondii* cannot be perpetuated in nature in the absence of cats.

What remains an enigma is the specific method by which toxoplasma oocysts infect man in specific cases. It seems obvious that small children, gardeners, farmers and others whose habits or work expose them to soil contaminated with oocysts, are at high risk of infection through the oral or perhaps respiratory route. In addition, as pointed out by Ganley and Comstock, transport hosts such as flies and cockroaches feeding on cat feces might be involved in contaminating human food with oocysts. In any event, pregnant women who are serologically negative are well-advised to avoid direct contact with cats (except housecats on a strict diet of cooked food), cat litter and other soil potentially contaminated with cat feces.

REFERENCES

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THE AUTHORS REPLY

We would like to thank Dr. Wallace for his remarks on our paper "Association of Cats and Toxoplasmosis" (1). We apologize for inadvertently overlooking his study in the South Pacific showing a low frequency of positive antibody titers on atolls where cats were not routinely found and a high frequency of positive titers on islands where cats were abundant (2). While this paper is further evidence for the association of cats and human infection with *Toxoplasma gondii*, the epidemiologic evidence for this association is still far from conclusive.

Dr. Wallace has stated, perhaps more eloquently than in our paper, that factors other than mere association with cats alone may account for the transmission of toxoplasmosis. We quite agree that cleanliness, hobbies, work environment, eating habits, etc. may be far more important factors than exposure to cats alone. In fact, these other factors may so dominate the transmission that mere exposure to cats or possession of cats are unimportant in the transmission of this disease.

We do not mean to deny that cats may be one source of dissemination for *T. gon-