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SHORT REPORT: TRANSIENT ANTIBODY RESPONSE IN *TAENIA SOLIUM* INFECTION IN FIELD CONDITIONS—A MAJOR CONTRIBUTOR TO HIGH SEROPREVALENCE

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Abstract. The discordance between extremely high seroprevalence of *Taenia solium* antibodies in disease-endemic populations, relatively few symptomatic cases of neurocysticercosis, and high background levels of putatively inactive brain lesions (mainly calcifications) in seronegative controls have confused researchers, clinicians, and epidemiologists in the last decade. We reviewed longitudinal serologic data from general population serosurveys in 3 different disease-endemic areas of Peru and Colombia and found that ~40% of seropositive people were seronegative when resampled after 1 year (3 surveys) or after 3 years (1 survey). Transient antibodies may have significant implications for the epidemiology of and immunity to this disease.

Taenia solium cysticercosis is the main cause of acquired epilepsy in developing countries.[1] During the past 10 years, testing with the highly specific (100%) and sensitive (98%) enzyme-linked immunoelectrotransfer blot assay (EITB, Western blot)[2] showed unexpectedly high seroprevalence levels of antibodies to *T. solium* in populations of disease-endemic areas.[3–6] As more population-based survey data became available, it is clear that human taeniasis-cysticercosis resembles an ''iceberg'' with 3 tiers. The most noticeable tip of this iceberg consists of people who harbor established, symptomatic central nervous system (CNS) disease—that is, neurocysticercosis. Below this tier is a larger population with established cysticercosis infection outside the CNS or in the CNS but without discernible symptoms,[7–10] and below this, there are many more people who were exposed to failed infections, but became seropositive. The presence of extremely high seroprevalence of *Taenia solium* antibodies in disease-endemic populations and relatively few symptomatic cases of neurocysticercosis was assumed to be a result of a largely asymptomatic infected human population, with minimal changes in prevalence over time.

However, recent findings by our group challenge this view. We found that ~40% of seropositive people become seronegative in a short time—that is, $\leq$ 1 year. The first survey that showed this phenomenon was conducted in 1993 in Monterredondo, a coastal village in Peru. A village-wide sampling covering 40% of all inhabitants showed a prevalence of 16% (76 of 482) for serum antibodies to *T. solium* by EITB. One year later, 373 people from the same village were sampled again, and this time the prevalence was 27% (99 of 373). These 373 participants included 209 newly sampled people, 57 (27%) of which were found to be positive by EITB. Among the 164 people who had been previously surveyed, 36 of 145 (25%) who were seronegative 1 year before were now seropositive, and 13 of 19 (68%) of those who were seropositive remained positive, whereas the other 6 (32%; 95% confidence interval [CI], 11–52) seroconverted to negative. These data showed that a large number of villagers had been newly infected and that a significant proportion of seropositive people became seronegative within 1 year.

A few years later, between 1997 and 1999, we conducted a longitudinal serosurvey covering $>$ 90% of the total population of 2 villages in northern Colombia (details to be published elsewhere). In this study 750 (97%) out 776 residents were sampled in 1997, 677 of 756 (90%) in 1998, and 822 of 844 (97%) in 1999. Ten of 23 (43%; 95% CI, 23–64) people who were seropositive in 1997 became seronegative in 1998, and 11 of 32 (34%; 95% CI, 18–51) became seronegative between 1998 and 1999. In a third study, we examined 398 villagers from Quilcas in the central Peruvian highlands in 1996 (140 seropositive, 258 seronegative). When resampled in 1999, almost half of those seropositive (69 of 140, 50%; 95% CI, 41–58) were now seronegative, and 20 of 258 (8%) of the previously seronegative villagers had become seropositive (Table 1). The proportion of transient seropositive cases was statistically different from 0 in all cases ($P < 0.05$, 1-tailed Fisher's exact test).

This previously unrecorded phenomenon shows that *T. solium* is a dynamic infection in that many newly infected (or exposed) people will develop only a transient serologic antibody reaction. Because the EITB assay with purified gly-

TABLE 1
Frequency of transient antibody reactions to *Taenia solium* infection in endemic villages*

Village	Seroconverted to negative	Time lapse
Monteredondo (Peru)	6/16 (32%)	1 year
Montería (Colombia) 1997–1998	10/23 (43%), San Nicolas 3/6 (50%), Bijaito 7/17 (41%)	1 year
Montería (Colombia) 1998–1999	11/32 (34%), San Nicolas 6/11 (55%), Bijaito 5/21 (24%)	1 year
Quilcas (Peru)	69/140 (50%)	3 years

* Arithmetic mean for all series, 41.7%; weighted mean, 44.9%.

coprotein antigens is very specific and no cross-reactions have been reported to date, after ~12 years of field testing, we consider that all positive results reported herein correspond to antibody reactions to *T. solium.* The group of people with only transient antibodies may have been exposed to, but did not develop, a viable infection of *T. solium,* or they may have had cysticercosis that was self-cured. It is therefore logical to assume that many people who are currently seronegative may have been seropositive before. This could also explain the discrepant finding of high background levels of putatively inactive, calcified brain lesions in seronegative controls.[8–10]

The incidence of exposure or infection must therefore be much higher than was previously suspected in order to balance the effect of transient antibody reactions and still maintain an equilibrium of high prevalence levels. The relationship between the transient antibody and exposure, current infection, or the development of immunity to future infection remains to be elucidated.

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