PREVALENCE OF ANTIBODIES TO UNIQUE TAENIA SOLIUM ONCOSPHERE ANTIGENS IN TAIENAN AND HUMAN AND PORCINE CYSTICERCOSIS

MANUELA VERASTEGUI, ROBERT H. GILMAN, HECTOR H. GARCIA, ARMANDO E. GONZALEZ, YANINA ARANA, CESAR JERI, ISKRA TUERO, CESAR M. GAVIDIA, MIN LEVINE, VICTOR C. W. TSANG, AND THE CYSTICERCOSIS WORKING GROUP IN PERU

Department of Microbiology, Universidad Peruana Cayetano Heredia, Lima, Peru; Department of International Health, Johns Hopkins University Bloomberg School of Public Health, Baltimore, Maryland; Cysticercosis Unit, Instituto de Ciencias Neurológicas, Lima, Peru; Asociacion Benefica PRISMA, Lima, Peru; Public Health Section, School of Veterinary Medicine, Universidad Nacional Mayor de San Marcos, Lima, Peru; Immunology Branch, Division of Parasitic Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia.

Abstract. The presence of two oncosphere antigens (OAs) of 22.5 and 31.3 kD in whole and excretory/secretory (ES) OA preparations of both Taenia solium and T. saginata or in antigen preparations from T. solium metacestodes or immature tapeworms was assessed. This included an evaluation of whether antibodies to other cestodes cross-reacted to these OAs. The OAs were present in whole oncosphere extract and E/S antigens of T. solium, but were not present in other stages (immature tapeworm or metacestode) or in OAs of T. saginata. The majority (95%) of T. solium tapeworm carriers had antibodies to these OAs, while only 20% of active neurocysticercosis cases were positive. No antibodies to the OAs were found in healthy controls, subjects infected with Hymenolepis nana, patients with hydatid disease, T. saginata tapeworm carriers, hamsters infected with immature T. solium tapeworms, or dogs infected with Echinococcus granulosus. The OAs are stage and species specific to T. solium and antibodies to OAs are usually present in tapeworm carriers.

INTRODUCTION

Cysticercosis is a frequent infection in pigs and humans in developing countries. The cyst is the larval tissue stage of the pork tapeworm Taenia solium. When humans ingest raw or undercooked T. solium cysticercosis pork, the scolex within the cysticercus is freed, attaches in the human small intestine, and matures into the adult tapeworm within 10 weeks. When the tapeworm matures, microscopic eggs containing infective oncospheres are passed in human feces and ingested by the pig, the intermediate host. The egg hatches in the intestinal tract and the oncosphere is freed, migrates through the intestinal wall, and gains access via the circulatory system to the muscles, brain and other tissues of the host, where it transforms into the metacestode form or cysticercus. Humans develop neurocysticercosis when they ingest T. solium eggs and metacestodes develop in the brain.1

Control of cysticercosis has been achieved in developed countries through improved hygiene, sanitation, and properly maintained commercial piggeries. However, in developing countries the disease is highly endemic in all areas where pig raising is practiced. In these endemic regions, such as Latin America, mass treatment of pigs with cysticidal and people with taeniacide drugs as a sole strategy has not had sustainable success.2

Vaccination of pigs, the sole intermediate host, may be a feasible approach for control of T. solium transmission.3 Impunity to larval cestode infections is based on the concept that the oncosphere stage can be eliminated by immune mechanisms.4,5 In cysticercosis, the oncosphere and its secretions are potent source of immunogens.6,7 Indeed, several studies have demonstrated that vaccination with oncosphere antigens (OAs) will provide a high degree of protection in pigs.6,8 Other studies have shown that complete resistance to a second cysticercosis infection occurs after treatment of the initial cysticercosis infection.6,9 This protection seems to be associated with two OAs of 22.5 and 31.3 kD.6 The purpose of this study was to determine whether these two OAs are stage and species specific, and to determine their prevalence in human T. solium tapeworm carriers and patients with neurocysticercosis.

MATERIALS AND METHODS

Experimental design. Stage specificity was tested by using antisera to the OAs to examine whether antigen preparations from different stages of T. solium (immature tapeworm, metacestode, oncosphere) contained the two previously defined OAs (22.5 and 31.3 kD). Antisera (vaccine-protected pigs [VPP] sera) were obtained from pigs that were previously immunized with oncosphere extracts or viable oncospheres. These pigs had demonstrated antibodies to OAs and were not infected when challenged with viable oncospheres.9 Species specificity was tested using the same antisera to examine whether T. saginata stages (oncospheres and excretory/secretory [ES] antigen preparations) had OAs present.

Serum samples were examined using an enzyme-linked immunoelectrotransfer blot (EITB) to determine the prevalence of antibodies to OAs in patients with taeniasis and neurocysticercosis, in pigs with cysticercosis that were heavy and lightly infected with T. solium, as well as in pigs with T. solium cysticercosis after treatment with oxfendazole, an effective larvalcidal agent in pigs. We also determined whether there were cross-reacting antibodies to these OAs in sera from patients infected with T. saginata, Echinococcus granulosus, and Hymenolepis nana, and from dogs infected with E. granulosus tapeworms.

Animal protocols were reviewed and approved by the Animal Committee at the Johns Hopkins University School of Public Health and the School of Veterinary Medicine at San Marcos University in Lima. Protocols involving humans were reviewed and approved by the Institutional Review Boards of both the Johns Hopkins University School of Public Health and the Universidad Peruana Cayetano Heredia. Patients provided voluntary consent and signed written consent forms.

Preparation of oncospheres. Adult tapeworms of T. solium and T. saginata were collected from newly diagnosed patients, who had received a standard taeniacide treatment using two
grams of niclosamide given orally. This was preceded and followed two hours before and after by a colonic purgative (Nulityl, polyethylene glycol molecular weight = 3,350 and electrolytes; Asofarma SA, Buenos Aires, Argentina). Pregnant woman and children less than six years of age were not given this purgative. The feces were collected for 48 hours after treatment. The proglottides were collected by sieving and washed thoroughly with distilled water and stored in 25% glycerol supplemented with penicillin (1,000 IU/ml) and gentamicin (100 µg/mL) at 4°C until used. Differentiation of species was made by histopathology and polymerase chain reaction (PCR)–restriction enzyme analysis as previously described.10

The eggs obtained from gravid proglottides were maintained in a 2.5% potassium dichromate solution (Sigma, St. Louis, MO) at 4°C until used. Eggs were washed three times in distilled water prior to hatching. In vitro hatching of oncospheres was performed using 0.75% sodium hypochlorite for 10 minutes at 4°C as previously described.11–14 Oncospheres were washed three times in RPMI 1640 media (Sigma), resuspended in this media, and counted using a Neubauer chamber (Fisher Scientific, Atlanta, GA).

Whole OA. The OAs of T. solium and T. saginata for polycrylamide gel electrophoresis and Western blot analysis was prepared as follows. Oncospheres were resuspended in RPMI 1640 media (40,000 oncospheres/mL) and sonicated at 4°C at 70 Hz (Sonic-Dismembrator model 300; Fisher Scientific, Pittsburgh, PA) using three one-minute periods separated by one-minute rest intervals. The oncosphere preparation was centrifuged at 28,000 × g for 30 minutes at 4°C in a Sorvall (Newtown, CT) centrifuge and the supernatant was then used for both electrophoresis and Western blot analysis. Protein concentration of both antigens was estimated using the Bio-Protein Assay (Bio-Rad, Hercules CA), based on the method described by Bradford.15 The OA, prepared as described above, was used at a concentration of 0.028 mg/mL of protein. This generally represented 40,000 oncospheres/mL.

Oncosphere ES antigen (artificial intestinal fluid [AIF]–activated and non-AIF-activated oncospheres from T. solium and T. saginata). Whether oncosphere activation yielded increased OAs was determined using AIF as the activating stimulus as follows. Taenia solium and T. saginata oncospheres were prepared by treating T. solium eggs with sodium hypochlorite13,14 and then were dividing the oncospheres into two aliquots. One aliquot was used without further treatment while the other was treated with AIF (0.8% cholesterol, 2% dried bovine bile, 1% NaHCO₃, and 0.2% Na₂CO₃ in RPMI 1640 medium) for two hours at 37°C.16 Both non-AIF-treated and AIF-treated aliquots were then washed three times with minimal essential medium (MEM; Sigma). After washing, 1 × 10⁸ non-AIF-treated or AIF-treated oncospheres were inoculated into a 1.5-mL conical screw top tube with MEM containing penicillin (1,000 IU/mL) and gentamicin (100 µg/mL) and incubated at 37°C in an atmosphere of 5% CO₂ with frequent shaking. The percent viability of between 100 and 150 oncospheres was determined using 0.4% trypan blue (Sigma). Oncospheres that excluded the dye were considered viable. Every 24 hours, the tube was centrifuged at 20,000 × g and the media was harvested and replaced with new media. Harvested media was centrifuged at 28,000 × g for five minutes and stored at −70°C. This process was repeated for three days. The condition of the oncospheres was monitored daily by microscopy to determine viability and appearance. The concentration of protein in the ES antigen was higher than that of the crude antigen (0.118 mg per 100,000 oncospheres versus 0.075 mg/100,000 oncospheres).

Metacestode antigen. Taenia solium metacestodes were dissected immediately post-mortem from naturally infected pigs obtained from an endemic area of Peru. The metacestodes were homogenized in cold phosphate-buffered saline (PBS, 0.15M NaCl, 0.01 M phosphate) using a glass homogenizer and sonicated on ice three times (one minute on, one minute off) at 70 Hz (Sonic-Dismembrator model 300; Fisher Scientific). This metacestode preparation was centrifuged at 28,000 × g for 30 minutes at 4°C and the supernatant was stored at −70°C until used for both electrophoresis and Western blot analysis.

Immune worm antigen. Immunosuppressed hamsters were inoculated orally with cysts of T. solium and killed two months post-infection. Immature T. solium worms were harvested from the intestine following previously published methods.17 The worms were homogenized in cold PBS using a glass homogenizer and sonicated on ice three times (one minute on, one minute off) at 70 Hz (Sonic-Dismembrator model 300; Fisher Scientific). The preparation was centrifuged at 28,000 × g for 30 minutes at 4°C and the supernatant was stored at −70°C until used for both electrophoresis and Western blot analysis.

Test sera. The following groups of human (1−4) and animal (5−9) sera were tested for the presence of antibodies to OA.

1) Patients with taeniasis. Sera from 44 T. solium tapeworm carriers were also tested: 15 were diagnosed at the neurologic resource center and also had cerebral cysticercosis, 2 patients with neurologic symptoms were referred from a general medicine hospital; and 27 came from asymptomatic individuals detected by stool examination in parasitological field studies. All subjects, including the 27 asymptomatic individuals, were treated and expelled T. solium tapeworms. Tapeworm species was confirmed by microscopy and by PCR.16 Sera from six individuals with T. saginata tapeworms were also tested.

2) Patients with other larval or intestinal cestode infections. Sera were tested from 11 patients with confirmed hydatidosis and from 32 patients (16 children less than 14 years old and 16 adults) with stools positive for H. nana.

3) Patients with viable T. solium cysticercosis. Sera from 10 neurocysticercosis patients admitted with seizures, who had viable cysts on computed tomography but negative microscopic and coproantigen stool examination results for intestinal taeniasis17 was tested for antibodies to OA.

4) Healthy control subjects. Archived sera from 18 individuals living in a Lima shantytown that is non-endemic for cysticercosis transmission and 11 sera from Peruvian laboratory technicians were tested.

5) Cysticercosis-infected pigs. Sera that was EITB positive and obtained from cysticercosis-infected pigs bought in an endemic area were tested in heavily and lightly infected pigs. Heavily infected pigs (n = 16) were defined as those that on necropsy had > 100 cysts and lightly infected pigs (n = 19) as those with ≤ 100 cysts.

6) Treated cysticercosis-infected pigs. These sera came from naturally infected pigs from an endemic area. They were tongue positive, EITB positive for cysticercosis,18 and had
been treated with a single dose of oxfendazole.\textsuperscript{9,19–21} These oxfendazole-treated pigs were maintained in a non-endemic area under controlled conditions. Oxfendazole given at a single dose is highly effective in removing all cysts from infected pigs.\textsuperscript{9,19–21} These treated pigs were then returned three months later to the endemic area. None became infected.\textsuperscript{7} We have defined these pigs as protected post-treatment (PPT) pigs. There were 18 PPT pigs. Sera from these PPT pigs before and after oxfendazole treatment were tested for the presence of antibodies to OA.

7) \textit{Hamsters infected with T. solium}. Sixteen immunosuppressed hamsters were experimentally infected orally with \textit{T. solium} cysts and studied two months later to determine if they had antibodies in their sera to OAs. All hamsters studied had developed immature \textit{T. solium} worms (without eggs).\textsuperscript{17}

8) \textit{Control dogs}. Sera from 20 normal dogs living in Lima, a non-endemic area for \textit{E. granulosus}, and 16 dogs that expelled \textit{E. granulosus} tapeworms after an aracoline purge\textsuperscript{22} were studied.

9) \textit{Control pigs}. Sera from 45 healthy pigs raised on a high-standard pig farm located in a \textit{T. solium} non-endemic area of the country were tested. All 45 tested negative on EITB for cysticercosis using lectin-purified glycoprotein antigens.\textsuperscript{18}

\textbf{Sodium dodecyl-sulfate–polyacrylamide gel electrophoresis (SDS-PAGE)}. Both \textit{T. solium} and \textit{T. saginata} OAs were analyzed by SDS-PAGE under non-reducing condition using the Protein II System (Bio-Rad) and gradient polyacrylamide gels (5–22.5%).\textsuperscript{23} The OA was incubated at 65°C for 20 minutes in sample buffer (0.1% SDS, 0.025% [w/v] bromophenol blue, 1% glycerol, and 0.0025M Tris-HCl, pH 8.0). The protein concentration used was 0.056 µg/2 µL/mm of gel width. Gels were run at 5 mA/gel for the stacking gel and at 25 mA/gel for the separating gel.

\textbf{Silver stain for protein}. The OA was extracted at 4°C using 8 M urea. The running gels were stained with silver (Silver Stain Plus, Bio-Rad) to elucidate protein patterns.\textsuperscript{24,25}

\textbf{Western blotting (EITB)}. Oncosphere antigen (whole protein and ES antigen), metacestode antigen, immature adult worm antigen from \textit{T. solium}, and \textit{T. saginata} OA (whole protein and ES antigen) were analyzed by Western blot to elucidate antigen patterns and to determine the presence of OAs (22.5 kD and 33.1 kD). Immune sera from Vaccinated Protected Pig sera VPP\textsuperscript{6} was used as a known positive control to demonstrate the presence of OAs. Control sera were obtained from known negative pigs. Confirmation that negative control pigs were free of \textit{T. solium} cysticercosis was done by demonstrating the absence of antibodies against cysticercosis by Western blot.\textsuperscript{18}

Human, pig, and dog sera were assayed for antibodies to OAs (22.5 kD and 33.1 kD) as described previously.\textsuperscript{6} Antigens were visualized using an anti-human, anti-porcine, and anti-dog immunoglobulin (heavy plus light chain) peroxidase-labeled antibody and 3,3-diaminobenzidine (Sigma) as the substrate. The molecular weight of the antigens was calculated using protein markers with low and high molecular weights (Bio-Rad). All serum samples were run individually.

\section*{RESULTS}

\textbf{Oncosphere antigens in antigen preparations}. Silver staining of whole and ES \textit{T. solium} extracts demonstrated the presence of multiple bands including the two OA (22.5 kD and 31.3 kD) bands (Figure 1). When incubated with either total oncosphere extract antigens (AIF treated and non-treated) or ES antigen of \textit{T. solium}, VPP sera demonstrated specific antibody reactions to both OAs. In contrast, VPP did not show reactions to these bands when compared with antigens from immature \textit{T. solium} tapeworms or \textit{T. solium} metacestodes (Figure 2 and Table 1), or \textit{T. saginata} antigens (whole oncosphere extract or ES fluid) (Figure 3).

In addition to the two OAs, an 88.2-kD antigen was identified by Western blot using oncosphere ES antigen and whole oncosphere extract. The antibody from VPP reacted more strongly with the 88.2-kD antigen in ES antigen then in whole OA. Only five (62%) of eight of sera from VPP reacted with the 88.2-kD antigen. All pre-immunization sera were negative for this band. In immature \textit{T. solium} tapeworm antigen, a 21-kD protein was present. The OAs were also absent from \textit{T. saginata} whole oncosphere extract and ES antigen fluid. These results strongly suggest that antibodies to the OAs (22.5 kD and 31.5 kD) are present in sera from protected animals and are unique to the \textit{T. solium} oncosphere since they are not present in either \textit{T. solium} immature tapeworms or metacestodes or in \textit{T. saginata} oncospheres. In contrast, the VPP sera does not consistently have antibodies to the 88.2-kD antigen, although it is unique to the oncosphere.

\textbf{Oncosphere antigens in sera from humans or animals with other cestode infections. Intestinal cestodes}. None of six patients infected with \textit{T. saginata} tapeworms had antibodies to the OAs. Two of these patients had a 32-kD band that based on gradient gel analysis, was separate from the 31.3-kD \textit{T. solium} OA. Sera from 32 patients infected with \textit{H. nana} and
16 dogs infected with *E. granulosus* tapeworms were all negative for antibodies to OAs (Table 2).

**Larval cestodes (hydatid disease).** None of 11 patients with hydatid disease had antibodies to either OA (Table 2).

**Normal sera.** Reactions to OAs were also absent in sera from 29 healthy volunteers living in a non-endemic area and from 20 normal dogs.

**Oncosphere antigens in T. solium taeniasis.** Sera from 42 (95.5%) of 44 *T. solium* carriers had antibodies to the OAs (26 with both bands, 11 with only the 22.5-kD antigen, and 5 with only the 31.3-kD antigen) (Table 2). In addition, sera from 16 immunosuppressed hamsters that harbored immature *T. solium* worms did not have antibodies to OAs when tested against *T. solium* whole OA. Immature worms in hamsters do not develop eggs; thus, oncospheres are not present.

**Oncosphere antigens in T. solium cysticercosis.** Of 10 patients with symptomatic neurocysticercosis who did not carry a tapeworm at the time of diagnosis, two (20%) had antibodies to the OAs (Table 2). Both patients reacted only to the 22.5-kD band. Antibodies against OAs were present in the sera of 14 of 15 heavily infected pigs with *T. solium* cysticercosis, but were less frequent in sera from lightly infected pigs (14 of 15 [93%] versus 12 of 19 [61%]; *P = 0.067*). All positive pigs had the 22.5-kD band, while nearly half did not have the 31.3-kD band. No pigs had only the 31.3-kD band. In addition, all 18 oxfendazole-treated PPT pigs had antibodies to OAs before and after treatment with this drug.

**Discussion**

Our data support conclusions reached by Bogh and others in studies of *T. taeniformis* that cestodes have unique stage-specific antigens. These OAs are thought to be unique to *T. solium* oncospheres for two reasons. First, the OAs did not react with antisera to other cestode infections such as *T. saginata*, *E. granulosus*, and *H. nana*. Second, sera from pigs with antibodies to OA did not cross-react with extracts from *T. solium* metacestodes, immature *T. solium* worms, and *T. saginata* whole oncosphere and ES antigens.

In a previous study, we demonstrated that antibodies to two unique antigens of the oncosphere of *T. solium* (OAs) with molecular masses of 22.5 kD and 31.3 kD were associated with protection against the development of cysts after infection of oncospheres intramuscularly. In this study, we demonstrate that these antigens are indeed unique to *T. solium* oncospheres. We also demonstrate that nearly 100% of all human *T. solium* tapeworm carriers have antibodies to one or both of these OAs.

Molinari and others have previously demonstrated the presence of a 22-kD antigen in sera of patients infected with *T. solium* cysticerci when tested against an OA. However, these investigators did not examine whether this antigen was stage specific. Garcia-Allan and others found only high molecular mass (≥ 45 kD) antigens to be specific for *T. solium* oncospheres. Our OAs are less than 45 kD, with the exception of the 88.2-kD antigen. None of these antigens were described by Garcia-Allan and others.

When human tapeworm carriers were tested, 95% of all human sera tested from *T. solium* tapeworm carriers had antibody to one or both of the OAs, while only 20% of humans with active neurocysticercosis infection (metacestodes in the brain) had antibody to OAs. This is quite surprising, since the oncosphere is not the infective stage leading to the adult tapeworm, but is the infective stage leading to cysticercosis. When pigs with cysticercosis were examined for antibody to OAs, those with heavy infection were nearly all positive, but only 61% of lightly infected pigs had a positive reaction. All PPT pigs had antibody to OAs both before and after treatment with oxfendazole. These pigs were all tongue positive and presumably were heavily infected. After oxfendazole treatment, these pigs were immune to a challenge with *T. solium* oncospheres. In addition to the two protective OAs (22.5 kD and 31.3 kD), we also found an 88.2-kD antigen that was oncosphere stage specific, but was less strongly associated with protection since only two thirds of the protected pigs had antibody to this antigen.

It appears that there are two types of immunity to *T. solium* infection. The first is an immune response to the *T. solium* oncosphere, which is stage-specific and does not cross with other cestodes. The second is an immune response to the metacestode. This response also appears to be stage and species specific as described in studies on *E. granulosus*, *T. ovis*, and *T. hydatigena*. The oncosphere immune response blocks the development of both the oncosphere and metacestode stages of *T. solium* infection. In contrast, metacestode antibody appears to be only partially protective. Animals vaccinated with metacestodes and challenged are able to
develop either live cysts or cysts that are killed after their development by metacestode immunity. This data suggests that porcine vaccines against cysticercosis will best be accomplished using oncosphere-derived antigens.

It is clear that both pigs and humans are initially infected with the oncosphere and that they have a reaction to the two OA’s. The persistence of this antibody in pigs and humans with active metacestode infection probably depends on the initial intensity of oncosphere infection and the duration of the disease. This hypothesis is supported by the fact that all heavily infected pigs had antibody to these OAs, while only about 60% of lightly infected pigs reacted to these antigens. In humans with active *T. solium* cysticercosis infection, only 20% of the individuals had antibodies to the OA. However, most human larval cysticercosis infections are light and exposure to oncospheres is most likely relatively limited. Also, initial infection may have occurred many years earlier, so that the antibody response to the oncosphere may have waned. In contrast, nearly all *T. solium* tapeworm carriers had antibodies to the OAs.

At the present time, the coproantigen test cannot determine the species of *Taenia* in the patient. However, the specific *T. solium* oncosphere antigens (22.5 kD and 31.3 kD) may well serve as useful diagnostic reagents in sera or stool since speciation of *Taenia* infection may be possible without having to recover the worm.

From a clinical standpoint, the most important finding was that 95% of the infected patients with *T. solium* tapeworms had antibodies to one or both OAs. Nearly two thirds of these patients were asymptomatic and had their tapeworms detected only because of their participation in a stool survey. This suggests that all patients with *T. solium* tapeworms are exposed to *T. solium* OAs at the mucosa level. Whether these antibodies occur with mucosal exposure without invasion or if invasion is necessary is not known. Our data suggest that autoinfection with *T. solium* eggs may occur much more commonly than previously thought. If autoinfection is occurring, whether this occurs directly by oncospheres hatching in the intestine or by external infection of eggs by the fecal–oral route is not clear. Also, since pigs clearly have antibodies against OAs that are specific to oncospheres, ingestion of eggs in the environment, at least in this species, is enough to stimulate antibodies to the OAs. Whether the same is true in humans is not clear.

It appears that antibodies to OAs are associated with protection and may be an excellent marker for demonstrating which patients may develop cysticercosis in the future. Longitudinal studies that focus on those patients who have such antigens compared with those that do not will need to be performed to understand more about the natural course of this disease. Why individuals with tapeworms are not pro-

### FIGURE 3

Western blot of *Taenia solium* and *T. saginata* antigens at different stages. Lane A, oncosphere antigen of *T. saginata* (0.56 μg); B, oncosphere antigen of *T. solium* (0.56 μg); C, excretory/secretory oncosphere antigen of *T. solium* (0.56 μg). Antisera were obtained from vaccine-protected pigs. The arrows show the two oncosphere antigens associated with immune protection to oncosphere challenge (31.3 and 22.5 kD).

### TABLE 1

<table>
<thead>
<tr>
<th>Antigen</th>
<th>22.4 kD</th>
<th>31.5 kD</th>
<th>88.2 kD</th>
<th>Antisera</th>
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<tr>
<td><em>T. solium</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Whole oncosphere</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>Vaccinated protected pig sera (VPP)†</td>
</tr>
<tr>
<td>ES oncosphere</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Metacestode</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td></td>
</tr>
<tr>
<td>Immature worm</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td></td>
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<tr>
<td><em>T. saginata</em></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Whole oncosphere</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td><em>T. saginata</em> infection</td>
</tr>
<tr>
<td>ES oncosphere</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td><em>T. saginata</em> infection</td>
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<tr>
<td><em>T. solium</em></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Whole oncosphere</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td><em>T. saginata</em> infection</td>
</tr>
<tr>
<td><em>T. saginata</em></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Whole oncosphere‡</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td><em>T. saginata</em> infection</td>
</tr>
</tbody>
</table>

*ES = excretory/secretory.
† Pool of pig sera post-vaccination with whole oncosphere antigen.
‡ Multiple protein bands were seen, none of which were similar to the two OAs of *T. solium*. 
Antibodies to *T. solium* oncosphere antigens in human or pigs with infected *T. solium* (tapeworm or cysticercosis) compared to sera from human or animals infected with other cestodes

<table>
<thead>
<tr>
<th>Infection</th>
<th>n</th>
<th>22.5 kD</th>
<th>31.3 kD</th>
<th>Both</th>
<th>Either</th>
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<tr>
<td><em>T. solium</em> taeniasis</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Taeniasis (human)</td>
<td>44</td>
<td>37 (84%)</td>
<td>31 (70%)</td>
<td>26 (59%)</td>
<td>42 (95%)</td>
</tr>
<tr>
<td>Taeniasis (hamsters)</td>
<td>16</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><em>T. solium</em> cysticercosis</td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>Neurocysticercosis (humans)</td>
<td>10</td>
<td>2 (20%)</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Heavy infections (pigs)</td>
<td>15</td>
<td>14 (93%)</td>
<td>8 (53%)</td>
<td>8 (55%)</td>
<td>14 (93%)</td>
</tr>
<tr>
<td>Light infections (pigs)</td>
<td>19</td>
<td>12 (63%)</td>
<td>5 (26%)</td>
<td>5 (26%)</td>
<td>12 (63%)</td>
</tr>
<tr>
<td>Intestinal cestode infections</td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>T. saginata</em> (humans)</td>
<td>6</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><em>Hymenolepis nana</em> (humans)</td>
<td>32</td>
<td>—</td>
<td>—</td>
<td>—</td>
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</tr>
<tr>
<td><em>Echinococcus granulosus</em> (dogs)</td>
<td>16</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Larval cestode</td>
<td></td>
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<tr>
<td>Hydatid disease (humans)</td>
<td>11</td>
<td>—</td>
<td>—</td>
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<tr>
<td>Noninfected sera</td>
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<td></td>
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</tr>
<tr>
<td>Humans</td>
<td>29</td>
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<td>—</td>
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<tr>
<td>Dogs</td>
<td>20</td>
<td>—</td>
<td>—</td>
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Authors’ addresses: Manuela Verastegui, Yanina Arana, Cesar Jeri, and Iskra Tuero, Department of Microbiology, Universidad Peruana Cayetano Heredia, PO Box 5045, Lima, Peru. Robert H. Gilman, Department of International Health, Johns Hopkins University School of Hygiene and Public Health, 615 North Wolfe Street, Room 3501, Baltimore, Maryland 21205, Telephone: 410-614-3959, Fax: 410-614-6000, E-mail: rgilman@jhsph.edu. Hector H. Garcia, Cysticercosis Unit, Instituto de Ciencias Neurologicas, Jr. Ancash 1271, Barrios Altos, Lima, Peru. Armando E. Gonzalez and Cesar Gavida, Public Health Section, School of Veterinary Medicine, Universidad Nacional Mayor de San Marcos, Apartado 03-5113, Lima 03, Peru. Min Levine and Victor C. W. Tsang, Immunology Branch, Division of Parasitic Diseases, Centers for Disease Control and Prevention, Atlanta GA 30341.

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