Am. J. Trop. Med. Hyg., 76(2), 2007, pp. 376–383

SWINE CYSTICERCOSIS HOTSPOTS SURROUNDING *TAENIA SOLIUM* TAPEWORM CARRIERS

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Abstract. We estimated the *Taenia solium* swine cysticercosis risk gradient surrounding tapeworm carriers in seven rural communities in Peru. At baseline, the prevalences of taeniasis by microscopy and swine cysticercosis by serology were 1.2% (11 of 898) and 30.8% (280 of 908), respectively. The four-month cumulative seroincidence was 9.8% (30 of 307). The unadjusted swine seroprevalence and seroincidence rates increased exponentially by 12.0% (95% confidence [CI] = 9.7–14.3%) and 32.8% (95% CI = 25.0–41.0%), respectively when distance to carriers decreased by half. Swine seroprevalence was 18.4% at > 500 meters from a carrier, 36.5% between 51 and 500 meters, and 68.9% within 50 meters ($P < 0.001$). Swine seroincidence also displayed a strong gradient near tapeworm carriers (3.8%, 12.2%, and 44.0%; $P < 0.001$). Within 50 meters, swine seroprevalence appeared unaffected if the owners harbored tapeworms, although pigs owned by a tapeworm carrier had a four times higher seroincidence compared with other pigs ($P = 0.005$). In rural areas, swine cysticercosis occurs in high-risk hotspots around carriers where control interventions could be delivered.

INTRODUCTION

Neurocysticercosis is the main cause of adult onset epilepsy and a prevalent health hazard in Latin America,[1] where an estimated 400,000 people live with symptomatic disease.[2] Both human and swine disease is caused by the larval stage of the cestode *Taenia solium*, and ingestion of infected, improperly cooked pork maintains the transmission cycle. Humans host the adult tapeworm and disseminate infective *T. solium* eggs in their feces, constituting a major risk factor for neurocysticercosis.[3–8] Tapeworm carriers not only increase their risk for neurocysticercosis but also place other household members at substantially elevated risk.[9,10]

Existing evidence suggests that *T. solium* tapeworm carriers could contaminate the environment beyond their households. Higher frequency of human and animal cases in households neighboring *T. solium* carriers has been reported,[9,11] although never assessed quantitatively. Experimental studies on *T. hydatigena* demonstrated increased infection rates in sheep 25 and 80 meters around tapeworm carriers,[12] but it is unclear whether these risk patterns are valid for *T. solium* cysticercosis. Swine tend to be coprophagic[13–15] and sheep and cattle are mostly coprophobic.[13,16,17] Because of such differences in exposure mechanism and doses, risk-clustering patterns around tapeworm carriers may be species specific and require further study to interpret findings from different taeniid species. We estimated the swine cysticercosis risk gradient around *T. solium* tapeworm carriers in seven rural Peruvian communities to evaluate the presence and size of infection foci. The existence of *T. solium* cysticercosis hotspots surrounding tapeworm carriers could be used later to develop and evaluate the feasibility and effectiveness focused control interventions.

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MATERIALS AND METHODS

Study site. The study took place in seven rural villages in the district of Matapalo, Tumbes, along the northern coast of Peru near the border with Ecuador (S 3°40′, W 80°11′, elevation = 54 meters). The main economic activities in the area are agriculture and small animal farming. Pig farming is common and swine roam free throughout the area. Matapalo is the poorest district in Tumbes with only 5% of the families having all their basic needs covered.[18]

Study design. Mass treatment of human tapeworms and swine cysticercosis serosurveys were conducted in Matapalo as part of a longitudinal study evaluating control measures for *T. solium* cysticercosis. We studied the swine cysticercosis seropositivity distance gradient surrounding tapeworm carriers identified during the first two mass-treatment rounds conducted between 1999 and 2000. Seropositivity was determined by seroprevalence at baseline and cumulative seroincidence between swine surveys.

Research ethics. The study protocol was reviewed and approved by the institutional review boards of the Cayetano Heredia Peruvian University, the Centers for Disease Control and Prevention, and the Johns Hopkins Bloomberg School of Public Health. All study subjects provided informed consent for their participation. Approval from legal guardians and child assent was obtained for legal minors. A single consent form was used for all study procedures, and prospective participants were told that they could refuse to participate in specific procedures.

Taeniasis mass treatment. Two mass-treatment rounds for human taeniasis took place in November–December 1999 and May 2000, respectively. A census was conducted during the first round, recording the age and sex of all household members, number of pigs owned, and type of sanitary facilities available. Household coordinates were recorded using global positioning system (GPS) hand-held receivers (GeoExplorer II™; Trimble, Sunnyvale, CA), with sub-meter accuracy after differential correction. No formal borders delimit communities but inhabitants reported consistently which village they live in. Village-membership was used to present approximate visual boundaries in maps but such borders were not used in statistical analysis.

Residents excluding children less than five years of age and pregnant women received a single oral dose of niclosamide (Pharmamed, Malta) to treat *T. solium* tapeworms; 1 gram for children 5–6 years of age and 2 grams otherwise. Women of childbearing age were questioned to exclude the possibility of pregnancy. Niclosamide remains to be the treatment of choice for intestinal tapeworms, and is reported to achieve 95% cure rates with mild and infrequent side effects.[19] Laxatives were not provided with this mass treatment.

Tapeworm detection. Single stool samples were requested from all participants regardless of age, pregnancy, or treatment status before and after the first treatment round and after the second treatment round. Fieldworkers provided disposable 500-mL plastic containers for stool collection as well as toilet paper and soap, instructing residents in basic hygiene procedures to avoid self-contamination. Specimens were examined for *Taenia sp.* eggs by standard stool microscopy.[20] Speciation was performed by morphologic differentiation of segments when available, although species identification was not a study outcome because both cattle farming and bovine cysticercosis are uncommon in the area. After five years of stool surveys, *T. saginata* has not been reported in Matapalo and has been found in less than 10% of tapeworm carriers in Tumbes (Garcia HH, unpublished data).

Swine serosurveys. Two swine serosurveys were conducted in November 1999–January 2000 and April 2000, respectively. Pigs $\geq$ 2 months of age excluding pregnant sows were captured, ear-tagged, and bled. A 6–8-mL serum sample was obtained from vena cava puncture. Vaccination against hog cholera (Pestiffa®; Merial, Sheffield, United Kingdom) after bleeding was offered as an incentive for participation. All captured pigs in the second sampling were treated for swine cysticercosis with oxfendazole (Synanthic®; Fort Dodge Animal Health, Overland Park, KS) at a dose of 30 mg/kg live weight[21] as one of the control interventions being tested by the parent study. The age of piglets declared by the owners was corroborated using conventional teeth-eruption indicators.[22] The serum enzyme-linked immuno-electrotransfer blot (EITB) assay was used to determine the presence of cysticercosis-specific antibodies. This assay identifies seven bands commonly present in the serum of human and swine cases, and is 94–98% sensitive and 100% specific.[23,24] Positivity is defined by the presence of one or more positive bands. The hog cholera vaccine does not interfere with EITB results, and oxfendazole treatment in the second round was administered only after bleeding to eliminate all chances of affecting EITB results. Coverage rates were not assessed, but previous studies conducted by our group experienced only a few refusals.[25]

Statistical analyses. Two main outcomes were analyzed: 1) percent seroprevalence in the first swine serosurvey and 2) cumulative (percent) seroincidence between the first and second swine serosurveys. These two outcomes were calculated aggregated at the household (herd) level, and seroincidence was measured only among seronegative pigs in the first serosurvey recaptured in the second serosurvey. The main covariate was the distance to the location of the nearest confirmed tapeworm carrier, calculated by assigning each swine herd the GPS coordinates of its owner's household. Distances were calculated in meters using equator equivalences of 110.57 and 111.32 km per degree of latitude and longitude, respectively.[26] For seroprevalence analysis, the distance to the nearest carrier was calculated including only tapeworms found at the baseline stool survey, while for seroincidence analysis, carriers found in any of both stool surveys were included to determine the distance to the closest carrier.

We assessed the association between swine cysticercosis seropositivity and the distance to the nearest tapeworm carrier by separately estimating seroprevalence and seroincidence distance gradients using three different approaches. First, we described the shape of the distance gradient using piecewise cubic splines. Splines with 2–7 sections were defined with equal numbers of seropositive pigs in each section and the best-fitting spline was chosen. Second, we evaluated if seropositivity increased exponentially near carriers testing the linear association between seropositivity and the binary logarithm of the distance [$-\log_2$ (distance + 1)]. Finally, seropositivity rates were estimated for specific distance ranges, forming a step function defined to maximize the differences in seropositivity between ranges. Identical distance ranges were used for seroprevalence and seroincidence to compare the distance gradients of seroprevalence and seroincidence. An additional analysis by distance ranges was conducted to evaluate the probability of finding tapeworm carriers in the proximity (50 meters) of high seropositivity herds.

Swine cysticercosis seroprevalence and seroincidence distance gradients were statistically assessed with generalized linear models,[27] using a binomial family, logarithmic link to calculate prevalence ratios (PR) and cumulative incidence ratios (CIR). The rate of increase in seropositivity rates as the distance from pigs to tapeworm carriers decreased by half were estimated from the PRs and CIRs of the $-\log_2$ (distance + 1). Statistical significance was determined with likelihood ratio tests (LRTs) and model selection was evaluated with the Akaike's Information Criterion (AIC).

Two additional analyses of the seroincidence distance gradient were conducted to assess the consistency of our findings. First, incidence density rates were calculated instead of cumulative (percent) incidence, using the time between serosurveys as the offset for Poisson family generalized linear regression models. Second, potential serorevertors (seropositive pigs at baseline found seronegative in the second serosurvey) were included in the analysis as seronegative. Seroreversion has been reported to occur in 15–24% of seropositive pigs,[25] usually in piglets 2–3 months of age carrying maternal antibodies.[28]

The association between swine cysticercosis seropositivity and sociodemographic and swine farming covariates was also assessed. Neighborhood population density was measured as the number of households in a 100-meter radius and in-house crowding was measured with the ratio of household members per bedroom. Neighborhood density, crowding and number of pigs owned were categorized in tertiles. All these covariates were included in nested, sequential models to estimate the regression-adjusted exponential seropositivity gradients. Robust confidence intervals were calculated when analyzing pig-level covariates to account for the correlation in the seropositivity of pigs from the same herd.

Statistical analyses were performed using Stata version 8.0 (Stata Corporation, College Station, TX) and all confidence intervals (CI) were calculated at the 95% level. Maps were prepared with ArcMap version 9.0 (Environmental Sciences Research Institute, Redlands, CA).

RESULTS

Study population. At baseline, there were 898 permanent residents in 212 households (Table 1) after excluding 16 households that were temporary dwellings or had incomplete coordinates. Matapalo was the largest village, had the highest population density ($P < 0.001$, by analysis of variance), and was the only village with electricity and a sewage system (46% and 28% of households, respectively). Latrines were more frequently present in Matapalo households than in other villages. The greatest distance between any two households within one village ranged from 866 to 3,284 meters.

Tapeworm detection and treatment. Overall tapeworm mass treatment coverage rates were 95% and 94% in the two rounds, and 88% and 40% of all residents provided stool specimens in each round, respectively. Eleven tapeworm carriers in nine households were identified from the first round pre-/post-treatment stool samples (age range = 5–98 years, five male), giving a taeniasis prevalence of 1.2% (11 of 898). Tapeworms were found in areas with slightly higher population density ($P = 0.089$, by Wilcoxon test). All carriers were treated with niclosamide except for a pregnant woman in Isla Noblecilla. She was treated post-delivery, which was past the study period.

Three tapeworms carriers were found during the second round: a person from Matapalo found positive and treated in the first round, the untreated pregnant woman from Isla Noblecilla, and a new resident in Nuevo Progreso. These three carriers were considered active during seroincidence analyses and the other nine were considered cleared.

Treatment effectiveness. Nine of the 10 tapeworm carriers treated at baseline were re-tested for tapeworms at follow-up, and one carrier moved out of the area between surveys. Assuming that the one recurrent carrier found was a treatment failure, treatment effectiveness was 89%.

Pig farming practices. Nearly two of three households reared pigs (mean = 6.6 pigs per household, Table 1). Villages differed in the proportion of households that had pigs ($P < 0.001$, by χ^2 test) and number of pigs owned per household ($P < 0.001$, by analysis of variance). Pig farming was more intense in less-populated areas (Spearman's $\rho = -0.19$, $P = 0.007$).

The four-month pig recapture rate was 50%, which was slightly higher than in previous swine longitudinal surveys.[25] One-tenth of all recaptured pigs were found at a different household, and we refer to these pigs as relocated. Relocated pigs were often found in the same village (88%), mostly sold to new owners.

Distance gradients. The baseline seroprevalence of swine cysticercosis was 30.8% (280 of 909), and the four-month cumulative seroincidence rate between surveys was 9.8% (30 of 307). Splines showed increasing seroprevalence and seroincidence rates surrounding to tapeworm carriers (Figure 1). A sharp increase was observed in the immediate proximity of carriers, specifically within 200 meters for seroprevalence and within 50 meters for seroincidence. Both seroprevalence and seroincidence showed a clear distance gradient and sustained higher rates nearer carriers, increasing by 12.0% (95% CI = 9.7–14.3%, $P < 0.001$, by LRT) and 32.7% (95% CI = 25.0–

TABLE 1

Baseline sociodemographics of the human and swine population, prevalence of taeniasis and swine cysticercosis seropositivity, Tumbes, Peru, 1999–2000

	Isla Noblecilla	Leandro Campos	Matapalo	Nuevo Progreso	Quebrada Seca	Totora	Tutumo	Total
No. of households	11	19	61	37	28	20	36	212
No. of residents	49	92	269	164	106	87	131	898
Mean residents per household	4.5	4.8	4.4	4.4	3.8	4.4	3.6	4.2
Average no. of households within 100 meters	3.5	2.7	11.2	6.0	6.3	5.2	3.2	6.6
Households with latrines (%)	0.0	0.0	51.7	17.1	38.5	10.5	28.6	28.9
Taeniasis prevalence (%)	1/49 (2.0)	0/92 (0)	5/269 (1.9)	1/164 (0.6)	3/106 (2.8)	1/87 (1.1)	0/131 (0)	11/898 (1.2)
Niclosamide treatment coverage (%)	95.7	97.4	93.7	97.9	94.6	91.9	96.6	95.3
Families having pigs (%), baseline	63.6	84.2	60.7	89.2	42.9	35.0	69.4	64.6
Mean no. of pigs per household, baseline	3.1	7.3	4.9	7.0	3.9	5.3	11.0	6.6
Swine cysticercosis seropositivity (%)								
Seroprevalence serosurvey 1	9/22 (40.9)	28/116 (24.1)	87/181 (48.1)	82/230 (35.7)	18/47 (38.3)	11/37 (29.7)	45/275 (16.4)	280/908 (30.8)
Seroprevalence serosurvey 2	9/19 (47.4)	8/93 (8.6)	41/132 (31.1)	46/209 (22.0)	21/49 (42.3)	2/23 (8.7)	27/214 (12.6)	154/739 (20.8)
Four-month cumulative seroincidence*	6/11 (54.6)	2/42 (4.8)	3/44 (6.8)	13/65 (20.0)	2/15 (13.3)	1/15 (6.7)	3/115 (2.6)	30/307 (9.8)
Maximum distance between houses (meters)	1,177	1,619	1,615	1,447	2,324	866	3,284	–

* Cumulative seroincidence is measured among pigs seronegative at baseline and bled again in the second serosurvey.

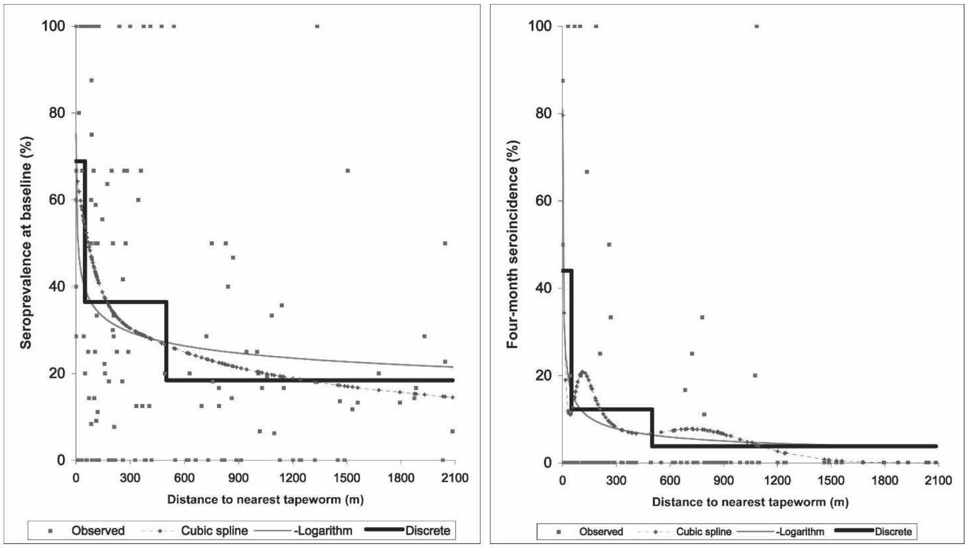

FIGURE 1. Gradients of swine cysticercosis seroprevalence and four-month cumulative seroincidence by distance to the nearest *Taenia solium* tapeworm carrier, Tumbes, Peru, 1999–2000.

41.0%, $P < 0.001$ by LRT), respectively, when the distance to the nearest carrier decreased by half.

Three discrete distance ranges were analyzed (> 500 meters, 51–500 meters, and < 50 meters), with separate analysis for swine owned by a tapeworm carrier. Swine seroprevalence was 18% (82 of 445) at > 500 meters, 36% (136 of 773) between 51 and 500 meters, and 69% (62 of 90) within 50 meters, which were significantly different at each of these three levels ($P < 0.001$, by Wald tests). Within 50 meters of a carrier, swine seroprevalence was 3.74 times higher than that at > 500 meters (95% CI = 2.94–4.75), and was similar between pigs owned by households with carriers (38 of 52 = 73%) compared with pigs owned by households without carriers (24 of 38 = 63%; $P = 0.3$, by χ^2 test). The swine cysticercosis seroincidence rate was 4% (7 of 184) at > 500 meters, 12% (12 of 98) at 51–500 meters, and 44% (11 of 25) within 50 meters, which were also significantly different at each of these three levels ($P < 0.05$, by Wald tests). Within 50 meters of a tapeworm, the seroincidence was 11.57 times higher than at > 500 meters (95% CI = 4.94–27.06), and higher in pigs owned by the carrier compared with all other pigs (8 of 10 = 80% versus 3 of 15 = 20%; $P = 0.005$, by Fisher's exact test).

All three analytical approaches used resulted in highly statistically significant distance gradients with different goodness of fit. According to the AIC, best fit for the seroprevalence distance gradient was obtained with the step function (3.47) followed by the splines (3.56) and exponential function (3.69). For the seroincidence distance gradient, however, the exponential function gave the best fit (1.30), followed by the splines (1.33) and step function (1.36).

Seroreversion was apparently common but did not alter the association between swine cysticercosis seroincidence and distance to the nearest carrier. Half of the recaptured seropositive pigs at baseline were seronegative during the second visit (73 of 146) and ~60% of these apparent serorevertors were < 4 months of age at baseline. Both piglets 2–3 months of age and pigs with a weak immunoassay result (only one positive band) had higher seroreversion rates ($P < 0.001$ and $P = 0.003$, respectively, by χ^2 test). Including serorevertor pigs as negative results reduced the seroincidence rates but did not alter substantially the rate of increase of seroincidence nearer carriers (27.4%, 95% CI = 18.2–37.3%, $P < 0.001$, by LRT). Similarly, the swine cysticercosis seroincidence distance gradient remained virtually unchanged when estimated using Poisson incidence-density rates. Seroincidence density rates increased by 32.9% as the distance to the nearest carrier was halved (95% CI = 21.3–45.5%, $P < 0.001$, by LRT).

Risk factors for seroprevalence. The seroprevalence of swine cysticercosis (Table 2) varied significantly across communities and was correlated with the prevalence of taeniasis at the village level (Spearman's $\rho = 0.81$, $P = 0.027$). The infection was present across the study area as approximately three-fourths of all pig farmers owned one or more seroprevalent pigs (Figure 2). Seroprevalence was higher in sows compared with male pigs and almost doubled after 9 months of age ($P < 0.001$, by χ^2 test). Age adjustment eliminated the seroprevalence differences between sexes because female pigs were older than male pigs (10.4 versus 5.1 months; $P < 0.001$, by Student's *t*-test). Swine seroprevalence increased steadily with higher human population density and less pigs owned. Crowding and presence of a latrine were weakly associated with swine seroprevalence.

The distance to the nearest tapeworm accounted for the seroprevalence gradient related to crowding and number of pigs owned, and also accounted for part of the variability in seroprevalence rates between villages, reducing the difference between the highest and lowest village-level seroprevalence rates from 32% to 19%. After multivariate adjustment, the rate of increase in seroprevalence nearer tapeworm carriers remained essentially unchanged (12.2%, 95% CI = 8.8–15.8%, $P < 0.001$). All other covariates significantly associated with seroprevalence are shown in Table 3. Despite the strong association between distance to the tapeworm and swine seroprevalence, the chances of finding a tapeworm 50 meters around the herds with highest seroprevalence never surpassed 37.5%.

TABLE 2

Baseline seroprevalence of swine cysticercosis by selected risk factors, Tumbes, Peru, 1999–2000

Variable	No. positive	No. tested	Prevalence	Prevalence ratio	*P*
Village					< 0.001
Tutumo	45	275	16.4	1.00	
Leandro Campos	28	116	24.1	1.48	
Totora	11	37	29.7	1.82	
Nuevo Progreso	82	230	35.7	2.18	
Quebrada Seca	18	47	38.3	2.34	
Isla Noblecilla	9	22	40.9	2.50	
Matapalo	87	181	48.1	2.94	
Swine age (months)					< 0.001
2–3	84	323	26.0	1.00	
4–5	30	152	19.7	0.76	
6–7	42	143	29.4	1.13	
8–9	14	66	21.2	0.82	
10–11	5	8	62.5	2.40	
12–13	23	63	36.5	1.40	
> 13	82	153	53.6	2.06	
Swine sex					0.018
Female	179	528	33.9	1.00	
Male	101	380	26.6	0.78	
No. of pigs in household					0.005
1–3 (1st tertile)	34	78	43.6	1.00	
4–7 (2nd tertile)	84	243	34.6	0.79	
> 7 (3rd tertile)	162	587	27.6	0.63	
Households within a 100-meter radius					< 0.001
1–3 (1st tertile)	101	484	20.9	1.00	
4–8 (2nd tertile)	88	248	35.5	1.70	
> 8 (3rd tertile)	91	176	51.7	2.48	
Crowding (people/room)*					0.143
0.3–1.2 (1st tertile)	64	190	33.7	1.00	
1.3–2.2 (2nd tertile)	104	348	29.9	0.89	
> 2.2 (3rd tertile)	83	323	25.7	0.76	
Latrine in household*					0.177
No	175	607	28.8	1.00	
Yes	92	276	33.3	1.16	
Total	280	908	30.8		

* Information about number of rooms and presence of latrines was not obtained from all households.

Risk factors for seroincidence. The four-month cumulative seroincidence differed between villages: 3% in two communities where no carriers were found, 10% in two villages with only cleared tapeworms, and 18% in three areas with at least one active tapeworm ($P < 0.001$, by χ^2 test, Table 4). Nearly 75% of all seroincident pigs lived in villages with active tapeworms (Figure 2). Swine cysticercosis seroincidence was significantly associated with population density and crowding, and only marginally associated with latrine presence and number of pigs owned. The age and sex of the pigs were not associated with differences in seroincidence. The distance to the nearest tapeworm remained significantly associated with swine seroincidence after adjustment for all the covariates listed above, although the small number of pigs 50 meters around carriers only allowed adjustment by one variable at a time. Similarly to seroprevalence, the chances of finding a tapeworm 50 meters around the herds with highest seroincidence never surpassed 33%.

DISCUSSION

Our findings demonstrate the existence of 50-meter *T. solium* cysticercosis hotspots around the homes of tapeworm carriers. Within hotspots, swine seroprevalence was 69% and the four-month cumulative seroincidence reached 44%, which were 2.9–4.8 and 4.9–27.1 times higher, respectively, compared with being 500 meters from carriers. The excess risk within hotspots was consistently present across different analytical approaches and is compatible with sustained increases in swine cysticercosis seropositivity for herds living closer to carriers. These apparently exponential gradients suggest that swine cysticercosis seropositivity remains increased beyond the immediate surroundings of tapeworm carriers. These results are consistent with previous field observations[8,10] and evidence from studies on other tapeworm species.[11,12] The steep distance gradient observed both for swine cysticercosis seroprevalence and seroincidence highlight the importance of evaluating and analyzing the proximity to tapeworm carriers, the actual exposure sources, as a critical risk factor.

Incidence rates displayed a steeper gradient around tapeworm carriers than prevalence rates, which was probably due to the multiple temporal biases that affect associations involving prevalence rates. Soon after a new tapeworm is introduced, environmental contamination builds up in the immediate surroundings[12,29] and forms a sharp incidence gradient. Pigs living farther are exposed later probably due to defecation and transit patterns, and potentially, dispersion mechanisms such as second-hand pig-pig transmission.[30] A diluted, extended prevalence distance gradient appears. Over time, tapeworms die and leave residual environmental contamination and *Taenia*-less seroprevalence hotspots. Additionally,

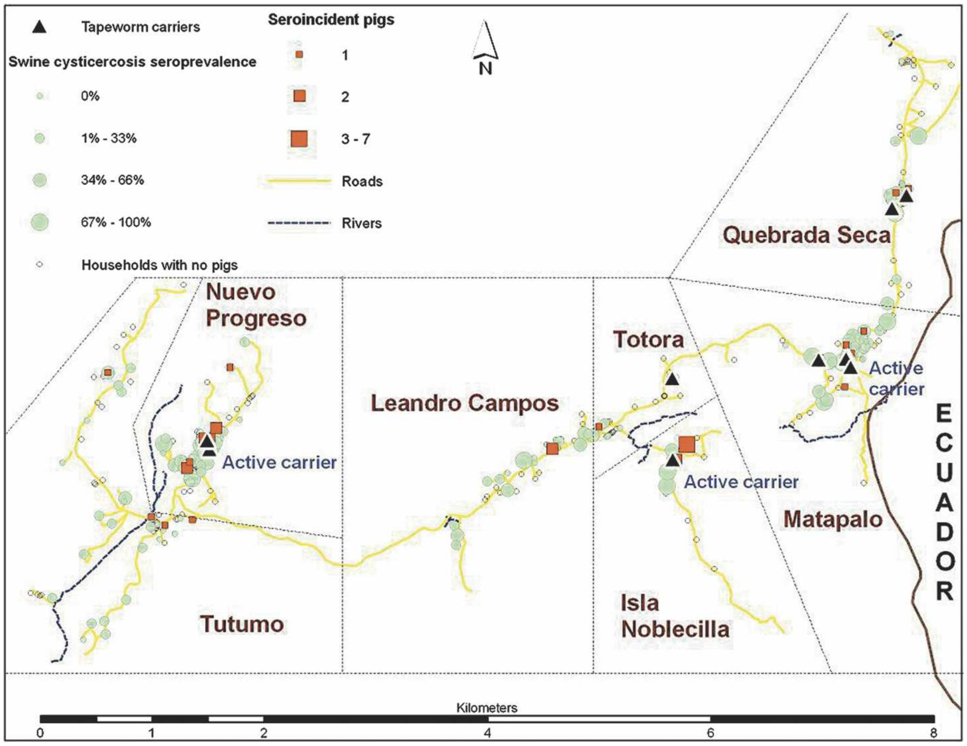

FIGURE 2. Seroprevalence and four-month seroincidence of swine cysticercosis and location of *Taenia solium* tapeworm carriers, Tumbes, Peru, 1999–2000. Two households with tapeworm carriers in Matapalo are virtually superimposed and cannot be differentiated visually. All three active tapeworm carriers are labeled as such. Villages were delimited considering households belonging to each community but using arbitrary divisions in the absence of actual community-defined boundaries.

the swine population has a quick turnover.[25] All these factors are constantly interacting over time, and probably account for the unexplained variability observed in multiple regression models.

The statistical power of this study was probably affected by the limited variability introduced by only 10 households with carriers, and inaccurate tapeworm detection due to the low stool collection coverage, the sensitivity of microscopy, and the variable excretion of *T. solium* eggs.[31] Additionally, our distance assessments cannot accurately reflect nor adjust for the free-range transit of pigs, introducing exposure misclassification and potentially weakening the associations estimated. Despite these limitations, we have observed across different analytical approaches a clear, monotonic increase in both swine cysticercosis seroprevalence and seroincidence closer to tapeworm carriers, and substantial excess risk in the immediate surrounding of carriers. Additionally, the effect sizes of estimates from different analysis of seroincidence were highly comparable. Therefore, although biases or confounding cannot be conclusively ruled out, the strength of the association, presence of an exposure-response relationship, and compatibility with findings in other cestodes[3,4,12,13] support the validity of our conclusions.

TABLE 3

Multiple regression analysis* of baseline seroprevalence of swine cysticercosis by selected risk factors, Tumbes, Peru, 1999–2000

Variable	Seroprevalence ratios	95% confidence intervals	*P* value Wald test
Swine age (months)			
2–9	1.00	–	–
> 9	2.07	1.72–2.48	< 0.001
Households within a 100-meter radius			
1–8 (1st/2nd tertiles)	1.00	–	–
> 8 (3rd tertile)	1.41	1.12–1.78	0.004
Logarithm of the distance to nearest tapeworm (meters)	1.12	1.09–1.16	< 0.001

* Adjusted by all three variables shown in the table.

We observed that most tapeworm carriers are located within high seropositivity hotspots but paradoxically only 37.5% of herds with high seroprevalence and 33% of herds with high seroincidence had tapeworm carriers within 50 meters. These high transmission spots without known, nearby carriers could be due to tapeworms that died recently, were briefly present in the area only, came from the outside but frequently spend time in the area, or were just simply undetected, and thus require additional search efforts to rule out the presence of potential carriers in the surroundings. Focalized control interventions may need to prioritize cysticercosis hotspots around a known tapeworm carrier, but cannot ignore the transmission potential of seropositivity-only hotspots. Targeting both the definitive human host and the intermediate porcine host in a single intervention may enhance control efforts.

In summary, we have demonstrated evidence of strong clustering of swine cysticercosis risk in clearly defined hotspots around *T. solium* tapeworm carriers, with additional spread of environmental contamination in broader areas. These hotspots combine both infected swine and active carriers, and offer control interventions the opportunity to target both branches of the transmission cycle in a single effort.

TABLE 4

Seroincidence rate of swine cysticercosis by selected risk factors, Tumbes, Peru, 1999–2000*

Variable	No. positive	No. tested	Cumulative incidence	Unadjusted CIR	*P* value χ^2	Adjusted by distance to carrier CIR	95% CI†	*P* value LRT
Village					< 0.001			0.562
Tutumo	3	115	2.6	1.00		1.00	–	
Leandro Campos	2	42	4.8	1.83		1.90	0.33–10.99	
Totora	1	15	6.7	2.56		2.11	0.23–19.04	
Matapalo	3	44	6.8	2.61		1.34	0.27–6.58	
Quebrada Seca	2	15	13.3	5.11		2.39	0.42–13.65	
Nuevo Progreso	13	65	20.0	7.67		3.43	0.88–13.30	
Isla Noblecilla	6	11	54.5	20.91		3.75	0.82–17.12	
Swine age (months)					0.697			0.275
2–3	15	146	10.3	1.00		1.00	–	
4–5	4	56	7.1	0.70		1.48	0.45–4.86	
6–7	7	46	15.2	1.48		2.79	1.13–6.89	
8–9	1	18	5.6	0.54		1.33	0.15–11.46	
> 9	3	41	7.3	0.71		1.29	0.19–8.85	
Swine sex					0.529			0.630
Female	14	160	8.8	1.00		1.00	–	
Male	16	147	10.9	1.24		1.13	0.65–1.96	
No. of pigs in household					0.117			0.542
1–3 (1st tertile)	5	35	14.3	1.00		1.00	–	
4–7 (2nd tertile)	11	76	14.5	1.01		0.65	0.27–1.56	
> 7 (3rd tertile)	13	185	7.0	0.49		0.59	0.25–1.40	
Households within a 100-meter radius					0.003			0.968
1–3 (1st tertile)	9	179	5.0	1.00		1.00	–	
4–8 (2nd tertile)	12	81	14.8	2.95		1.05	0.39–2.79	
> 8 (3rd tertile)	9	47	19.1	3.81		0.98	0.36–2.67	
Crowding (people/room)‡					0.043			0.155
0.3–1.2 (1st tertile)	8	59	13.6	1.00		1.00	–	
1.3–2.2 (2nd tertile)	5	114	4.4	0.32		0.48	0.17–.135	
> 2.2 (3rd tertile)	16	121	13.2	0.98		1.16	0.68–1.98	
Latrine in household‡					0.122			0.408
No	22	188	11.7	1.00		1.00	–	
Yes	7	112	6.3	0.53		0.71	0.31–1.64	
Relocated pigs					0.219			0.220
No	25	275	9.1	1.00		1.00	–	
Yes	5	32	15.6	1.72		1.75	0.73–4.19	
Total	30	307	9.8					

* CIR = cumulative incidence ratio; CI = confidence interval; LRT = likelihood ratio test.
† Robust confidence intervals to account for household-level correlation.
‡ Information about number of rooms and latrine use was not obtained in all households.

Received September 19, 2006. Accepted for publication October 26, 2006.

Acknowledgments: We thank the population of Matapalo and local health workers for their support and cooperation.

Financial support: This study was partially supported by research grants P01 AI51976 and U01 AI35894 from the National Institute of Allergy and Infectious Diseases, National Institutes of Health. Research grants from the Wellcome Trust (063109), the Food and Drug Administration (002309), and the Bill and Melinda Gates Foundation (23981) fund ongoing cysticercosis research by the authors.

Disclaimer: The opinions and assertions contained herein are the private ones of the authors and are not to be construed as official or reflecting the views of the United States Department of the Army or Navy or any of the other organizations listed.

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