Magnitude of the Disease Burden from Neurocysticercosis in a Developing Country

Caryn Bern,¹ Hector H. Garcia,³ Carlton Evans,⁵ Armando E. Gonzalez,⁴ Manuela Verastegui,³ Victor C. W. Tsang,¹ and Robert H. Gilman^{2,3} From the ¹Division of Parasitic Diseases, Centers for Disease Control and Prevention, Atlanta, Georgia, and ²Johns Hopkins University School of Hygiene and Public Health, Baltimore, Maryland; ³Universidad Cayetano Heredia and ⁴Facultad de Medicina Veterinaria, Universidad Nacional Mayor de San Marcos, Lima, Peru; and ⁵University of Cambridge Clinical School, Cambridge, United Kingdom

Cysticercosis contributes to higher epilepsy rates in developing countries than in industrialized ones, yet no estimate exists for the associated burden of disease. We used epidemiological data on neurocysticercosis in Peru to calculate the burden of disease and applied our model to the other countries of Latin America where neurocysticercosis is endemic to determine a regional estimate. Analysis of 12 population-based community studies demonstrated that neurocysticercosis was endemic in highland areas and high jungles, with seroprevalences from 6% to 24%. In one community, the adult seizure disorder rate was 9.1% among seropositive persons versus 4.6% among seronegative persons; we used this difference for estimates. On the basis of average prevalence rates in areas of endemicity of 6%–10%, we estimated that there are 23,512–39,186 symptomatic neurocysticercosis cases in Peru. In Latin America, an estimated 75 million persons live in areas where cysticercosis is endemic, and ~400,000 have symptomatic disease. Cysticercosis contributes substantially to neurological disease in Peru and in all of Latin America.

Cysticercosis is a frequent infection in developing countries where pigs are raised [1]. Population-based studies show substantially higher epilepsy rates in developing countries than in industrialized countries, a difference attributed in part to neurocysticercosis [2, 3]. Nevertheless, no estimate of the disease burden due to cysticercosis exists. In this article we estimate, for the first time, the burden of morbidity attributable to cysticercosis in a developing country.

Cysticercosis is the larval tissue stage of the pork tapeworm *Taenia solium*. When humans consume undercooked cysticercotic pork, the larva attaches to the gut wall and grows into the intestinal tapeworm. The life cycle is maintained when pigs eat human feces containing tapeworm eggs and develop porcine cysticercosis, a process facilitated by husbandry practices in villages where pigs are free-roaming scavengers and human fecal contamination is widespread. These life-cycle stages are benign in comparison with those of human cysticercosis, defined by the presence of *T. solium* larvae in human tissues, which

Clinical Infectious Diseases 1999: 29:1203-9

occurs when people ingest tapeworm eggs from food, drink, or soil contaminated by the feces of a tapeworm carrier.

Seizures are the most common manifestation of symptomatic human cysticercosis [4–6]. The disease may also be associated with headache, hydrocephalus, chronic meningitis, or symptoms due to a space-occupying central nervous system (CNS) lesion [7]. Isolated nonneurological manifestations, such as ocular or dermal cysts, account for <5% of cases of symptomatic disease [8]. We therefore use the term *neurocysticercosis* interchangeably with the phrase *symptomatic human cysticercosis*.

The interval from infection to onset of symptoms is lengthy. In a case series of British soldiers infected in Asia, the median time from infection to appearance of first symptoms was 4 years [9]. The proportion of persons whose infections progress to symptomatic disease is unknown. In a substantial proportion of persons in clinical trials, all or some cysts spontaneously disappear [10]. Studies have found that anthelminitics hasten the disappearance of active parenchymal lesions [11]. Current data are inadequate for estimating the proportion of cases in a population in which complete resolution of seizures or other neurological symptoms occurs, with or without treatment, or the average duration of symptomatic disease.

Until recently, studies of cysticercosis generally focused on clinical case series of patients with seizures or other neurological symptoms [6, 12, 13]. Epidemiological reviews of cysticercosis are often biased by the lack of generalizability of such case series and by inappropriate comparisons between data from case series and data from community studies.

Received 11 March 1999; revised 24 June 1999.

Financial support: Many of the studies of the Cysticercosis Working Group in Peru cited in this article were supported by National Institutes of Health grant 1-UO1-AI-35894-01; Fogarty International Center training grant 5D43TW0001008; Federal Drug Administration grant FD-R-001107-01; and Fogarty International Center collaborative award TW0059801A1.

Reprints or correspondence: Dr. Caryn Bern, Division of Parasitic Diseases (MS F-22), Centers for Disease Control and Prevention, 4770 Buford Highway NE, Atlanta, GA 30341 (cxb9@cdc.gov).

^{© 1999} by the Infectious Diseases Society of America. All rights reserved. 1058-4838/1999/2905-0015\$03.00

Diagnosis of neurocysticercosis, especially in populationbased studies, is complex. No diagnostic test identifies all cases of cysticercosis, and each test identifies a somewhat different group of individuals (figure 1) [14]. Computerized tomography (CT) indicates structural disease but misses brain stem and intraventricular neurocysticercosis lesions and, if not performed with enhancement, misses those that are isodense. In addition, the accuracy of CT diagnosis is extremely dependent on the judgement of the reader, which may be highly variable [14].

Magnetic resonance imaging (MRI) is superior to CT but is rarely available in countries where neurocysticercosis is endemic. In population-based studies, it has been noted that a proportion of asymptomatic individuals have CT images suggestive of neurocysticercosis [15, 16]. Many of these abnormalities probably represent asymptomatic neurocysticercosis, but some may be CNS lesions unrelated to neurocysticercosis. Neither CT nor MRI is practical for large-scale populationbased studies of neurocysticercosis.

A recently developed serological test, the electroimmunotransfer blot (EITB) [17], is currently the most practical screening tool for use in populations. In clinical evaluations, the specificity of the EITB was 100%, whereas the sensitivity for detection of cases with multiple intracranial cysticerci was 90%–100% [18, 19]. However, the EITB demonstrated only 65% sensitivity in individuals with a single intracranial cyst (72% for an enhancing lesion and 40% for a calcified cyst) [18]. Thus, an unknown proportion of seizures due to neurocysticercosis will occur in EITB-seronegative individuals who have a single cyst or who no longer have active disease but have residual lesions in crucial areas of the brain.

Conversely, it is common to encounter asymptomatic EITBseropositive individuals in community studies. As for many other infectious diseases, seropositivity is generally interpreted to mean subclinical infection. However, in some cases, asymptomatic seropositivity might mean that the individual was exposed to *T. solium* and successfully developed protective immunity. For our estimates, we have therefore focused not on the overall infection burden, but on the burden of symptomatic neurocysticercosis.

We reviewed all human cysticercosis studies carried out by the Cysticercosis Working Group in Peru [13, 14, 20–26] to discern patterns of seropositivity and disease, to map areas of endemicity in the country, and to estimate the burden of symptomatic neurocysticercosis disease. Finally, to place the Peru-

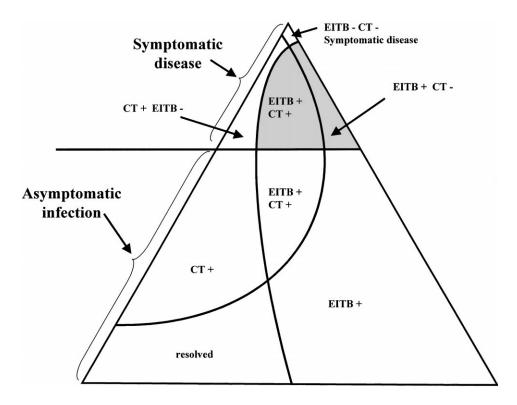


Figure 1. Diagram of relationship between cysticercosis disease or infection and diagnostic test results (+, positive; -, negative). Lower part of triangle represents persons with asymptomatic cysticercosis infection, whereas upper part represents those who have symptomatic neurocysticercosis disease. Some infections and some symptomatic cases involve positive electroimmunotransfer blot serology (EITB) only or positive computerized tomography (CT) of the brain; some involve both or neither. Our disease burden estimate corresponds to shaded area, representing only EITB-positive symptomatic cases (see text for full explanation).

vian data in its global context, we reviewed recent studies from other geographic areas.

Methods

From 1988 to 1998, the Cysticercosis Working Group in Peru conducted 12 surveys of communities and 4 studies among series of patients in Lima. We reviewed published data and analyzed unpublished data to collate all available information on human and porcine seroprevalence in Peru, determined with use of the EITB. On the basis of these data, we partitioned Peru into areas in which neurocysticercosis is endemic and nonendemic and calculated the populations in those areas [27]. To estimate the disease burden, we made the following assumptions.

(1) We included only the population living currently in the area where neurocysticercosis is endemic, though a substantial percentage of those in the area where it is nonendemic (especially in Lima) spent their childhood and youth in areas of endemicity. We have further assumed that in large urban areas neurocysticercosis is nonendemic, though some person-to-person transmission clearly occurs in these settings.

(2) We included only the population aged \geq 15 years, because seropositivity rates tend to increase with age [20, 28] and because seizures due to neurocysticercosis characteristically begin in early adulthood [29].

(3) We constructed our model using 3 alternative prevalence estimates: 6%, 8%, and 10%. The seroprevalence in areas of endemicity was often above all 3 of these estimates. Some village study areas were chosen because of reported high endemicity, which could lead to a bias to higher rates. However, the large-scale studies in Huancayo and Andahuaylas were conducted in highland areas selected without prior knowledge of neurocysticercosis prevalence, and they included both towns and villages. In both studies, the median seroprevalence was well above 10%. We believe that our seroprevalence estimates are likely to be conservative.

(4) To estimate the proportion of seizure disorders attributable to neurocysticercosis, we used the difference in rate between EITB-positive and EITB-negative persons aged ≥ 15 years in the Andahuaylas survey. In this study, medical residents in neurology interviewed all urban participants regarding any history of seizures. Our calculations counted only EITB-positive symptomatic neurocysticercosis (the shaded area in figure 1) and probably resulted in an underestimate.

(5) Case series demonstrate that seizure disorders account for 69%–96% of cases of symptomatic neurocysticercosis [6, 9, 12]. We used a median estimate of 80%.

Our direct estimate relied on the seropositivity rate and the difference in seizure disorder rates between seropositive and seronegative persons. As a cross-check on this estimate, we also calculated the disease burden on the basis of the rates of seizure disorder in several reported population-based studies in Andean countries [2, 3, 30]. We used case-series data to estimate the percentage of adult seizure disorders due to neurocysticercosis. We multiplied this proportion by the population-based seizure disorder rate to calculate the number of seizure disorders expected to be from neurocysticercosis. Finally, to place the Peruvian data in its global context, we reviewed recent data from other countries. To estimate the disease burden for Latin America, the epidemiological model developed for Peru was applied to the rural adult populations of El Salvador, Honduras, Guatemala, Mexico, Nicaragua, Panama, Bolivia, Brazil, Colombia, Ecuador, and Venezuela. We chose these countries because of their reported high endemicity of cysticercosis [1].

Results

Community studies. The Cysticercosis Working Group in Peru conducted serosurveys of people in 12 communities over the past 10 years (table 1). Two, in Huancayo and Andahuaylas, were large-scale studies of highland communities. In Huancayo, the seroprevalence ranged from 7% to 17%, depending on the group of villages, whereas in Andahuaylas, both urban and rural populations had a seroprevalence of ~12%. All but 2 of the other studies (Saylla, Lima shantytown 1) involved population-based village samples. The disease was uniformly endemic in the highlands below 4000 m and in the high jungle areas. The human seroprevalence in these areas was consistently above 6% and often above 10%.

In a highland village well-known for its pork dishes and its free-range pigs (Saylla), we found 24% seroprevalence in members of a mothers club and their relatives; however, this sample was not strictly population-based [25]. We documented only one coastal area with endemic disease (Piura), but data from the coast were more limited than from other regions; we assumed that on the remainder of the coast cysticercosis is nonendemic. In surveys from a sheep-raising community above 4000 m, from the low jungle, and from Lima, the human seroprevalence was never >1%.

Medical residents interviewed 1290 of the 1529 serosurvey participants from the town of Andahuaylas. Among participants aged ≥ 15 years, 14 (9.1%) of 154 seropositive persons reported a history of seizures, compared with 48 (4.6%) of 1033 seronegative persons (relative risk, 1.9; P < .05).

Porcine seroprevalence was measured in 10 communities with endemic disease, a highland pig market, and a commercial slaughterhouse in Lima (table 1). In all but 1 of the locations where disease is endemic, the porcine seroprevalence was at least 25% and as high as 68%. Among Lima slaughterhouse pigs raised in a highly controlled, sanitary environment, no infected animals were detected. In population-based surveys in areas of endemicity where most pigs were free-range, the ratio of human to porcine seroprevalence varied from 1 : 2 to 1 : 8. The single exception was in Monterredondo, where the porcine seroprevalence was 25% lower than the human seroprevalence.

Clinical series. During the same time period, we studied 4 series of neurology patients in Lima hospitals (table 2). Although cysticercosis is not endemic in Lima, each clinical series demonstrated substantial rates of seropositivity (6%–18%). The rates were always higher among patients with a history of con-

Site	Department	Proportion (%) seropositive		Prevalence ratio	
		Humans	Pigs	(humans : pigs)	Reference
Large-scale surveys					
Andahuaylas					
Urban	Apurimac	198/1529 (12.9)	_	_	
Rural	Apurimac	418/3443 (12)	36/1345 (27.0)	1:2	UD
Huancayo					
Quilcas	Junin	275/1631 (16.9)	364/538 (67.7)	1:4	[24], UD
San Pedro	Junin	45/446 (10.1)	45/94 (47.9)	1:5	[24], UD
Canchayllo	Junin	33/468 (7.1)	3071 (42.3)	1:6	[24], UD
Coto coto market	Junin	_	40/77 (51.9)	_	[24], UD
Village or community surveys					
Monterredondo	Piura	78/489 (16)	13/98 (13.0)	1:0.8	[24]
Pomabamba	Ancash	15/112 (13.4)	_	_	[21]
Haparquilla	Cuzco	14/108 (13)	51/110 (46.0)	1:4	[25]
Saylla	Cuzco	24/99 (24)	19/53 (36.0)	1:1.5	[31]
Maceda	San Martin	30/421 (8)	57/133 (43)	1:5	[25]
Churusapa	San Martin	9/134 (6.7)	43/87 (49)	1:7	[21, 24]
La Matanza	Piura	a	6/20 (30)	_	[26]
Tupac Amaru	Junin	2/309 (0.6)	_	_	[21]
Urban shantytown 1	Lima	1/98 (1)	—	_	[24]
Urban shantytown 2	Lima	0/250 (0)	—	_	[24]
Villages	Iquitos	0/200 (0)	—	_	UD
Slaughterhouse	Lima	_	0/42 (0)	_	[32]

 Table 1.
 Studies of cysticercosis seroprevalence among communities in Peru (1988–1998), as determined by electroimmunotransfer blot assays.

NOTE. UD, unpublished data (HH Garcia).

^a Data for humans are listed in table 2.

vulsive disorder or a clinical or radiological diagnosis of neurocysticercosis. In the series of 946 patients whose serum was submitted for EITB testing, 18% were seropositive, reflecting the selection of patients for whom the clinical suspicion of cysticercosis was high. Each additional factor (i.e., for persons with epilepsy who were born in Lima, patients with other neurological disorders who were born outside Lima, and persons with epilepsy who were born outside Lima) was associated with higher seroprevalence (9%, 17%, and 31%, respectively).

In the village of La Matanza, where neurocysticercosis is endemic, the seroprevalence among individuals with a seizure history was 34% (table 2), a rate similar to that among persons with epilepsy who were born outside Lima in the case series of 946 patients [23].

Cysticercosis disease burden. On the basis of our data review, we constructed a map delineating the areas where neurocysticercosis is endemic, comprising the highlands below 4000 m, the high jungle, and the northern coast. Using 1993 census data, we estimated that 10.5 million Peruvians live in areas of endemicity, of whom 6.3 million are aged ≥ 15 years (table 3). We estimate that between 376,000 and 627,000 Peruvians are EITB-seropositive. Using the difference between seizure disorder rates among seropositive and seronegative residents of Andahuaylas, we estimate that 5% of seropositive persons have a convulsive disorder attributable to neurocysticercosis. This leads us to estimate that between 19,000 and 31,000 Peruvians have neurocysticercosis-associated seizures and that between 23,500 and 39,000 have symptomatic neurocysticercosis.

An alternative estimate calculated with use of population-

based epilepsy rates in Latin America yielded the following. The epilepsy rate in Andean countries is $\sim 17-20$ per 1000, compared with 3–6 per 1000 in industrialized countries [2, 3, 30]. Studies suggest that the proportion of adult seizure disorders attributable to neurocysticercosis in areas of endemicity in Latin America is $\sim 25\%-50\%$ [22, 34]. We therefore assumed that, in areas of endemicity, $\sim 4-5$ epilepsy cases per 1000 are due to neurocysticercosis. We applied this rate to the population living in areas of endemicity in Peru, resulting in an estimate of 25,079–31,349 cases of epilepsy due to neurocysticercosis, a result quite consistent with our first set of calculations.

Brief review of recent cysticercosis survey data from other countries. Recently, a number of studies of cysticercosis prevalence have been conducted in various countries. Community surveys show that EITB-determined seroprevalence rates among humans in rural Mexico, Guatemala, Honduras, Ecuador, and Bolivia range from 10% to 23% [15, 34–36], and that porcine prevalence ranges from 25% to 39% [37–39]. In Mexico, community surveys of 2524 people of all ages found that 12 (5.6%) of 42 seropositive individuals reported a history of seizure disorder, compared with 30 (1.3%) of 2310 seronegative individuals [28], a difference similar to that found in Andahuaylas.

In China, Rwanda, India, Colombia, and Ecuador, seroprevalence among persons with seizure disorders ranged from 20% to 47% [15, 36, 40, 41]. In Ecuador, 54% of epilepsy patients and 14% of residents of a community where cysticercosis is endemic had abnormalities evident on CT that were consistent with neurocysticercosis [15].

CID 1999;29 (November)

Subjects	Proportion (%) of patients seropositive	Reference
Neurology outpatients in Lima		[22]
With seizure disorder	22/189 (12)	
Without seizure disorder	8/309 (3)	
With seizure disorder, born outside Lima	19/101 (19)	
Neurology inpatients and outpatients (adults)	172/946 (18)	[23], UD
With epilepsy, born in Lima	13/144 (9)	
Without epilepsy, born in Lima	7/112 (6.3)	
With epilepsy, born outside Lima	94/299 (31.4)	
Without epilepsy, born outside Lima	49/282 (17.4)	
Neurology inpatients in Lima	21/173 (12)	[13]
With NCC diagnosed by CT	11/14 (79)	
With other diagnosis made by CT	4/28 (14)	
With normal CT findings	1/14 (7)	
Self-referred persons in La Matanza, Piura	_	[26]
With history of seizures	14/41 (34.1)	
With history of headache or dizziness	1/8 (12.5)	
With history of passing proglottids	11/23 (47.8)	
Endoscopy patients in Lima	2/223 (1)	[13]
Non-neurology inpatients in La Merced, Junin	0/26 (0)	[26]

 Table 2.
 Seroprevalence of cysticercosis in case series from Peru.

NOTE. NCC, neurocysticercosis; CT, computerized tomography; UD, unpublished data (HH Garcia).

If the epidemiology of cysticercosis in the rural areas of the other developing countries of Latin America is similar to that in rural Peru, then there are 75 million people living in areas of endemicity and ~400,000 cases of symptomatic neurocysticercosis in Latin America alone.

Discussion

This study is the first attempt to present a comprehensive picture of the disease burden caused by cysticercosis in one developing country. We calculated conservatively that \sim 30,000 Peruvians have symptomatic neurocysticercosis, a finding supported by the consistency of data from multiple studies and the agreement between results from 2 different methods of estimation. It is clear that cysticercosis is a common infection in much of Peru, where half the population lives under conditions in which *T. solium* transmission is an everyday occurrence.

In these areas, nearly one-tenth of the people are infected by the time they reach adulthood, and one in 200 have seizures associated with neurocysticercosis. The disease burden for other countries of Latin America has not been studied, but if the prevalence rate in the rural populations of other Latin American countries where neurocysticercosis is endemic is similar to that found in Peru, then neurocysticercosis accounts for ~400,000 of the extant cases of symptomatic neurological disease.

Neurocysticercosis is a chronic disease associated with substantial morbidity and high social and economic costs. In rural villages, seizure disorders are poorly understood. Sufferers may be stigmatized and unable to work or participate fully in communal life [42]. Seizure disorders raise the risk of injuries; in New Guinea, the introduction of cysticercosis was followed by an epidemic of serious burns when convulsions caused people to fall into open cooking fires [43]. In order to manage cysticercosis effectively, health care providers must understand the disease, and patients need access to medical care that is often lacking. Even where medical care is available, the costs of treating neurocysticercosis (whether borne by the individual or by the government) are considerable in a developing country.

Porcine cysticercosis also entails important economic losses. In Peru, cysticercotic pigs are confiscated by meat inspectors without compensation. Small producers therefore sell cysticercotic pigs clandestinely at a discounted price rather than take a complete loss, a practice which helps to maintain the parasite life cycle [44].

As our data demonstrate, cysticercosis is not homogeneously distributed in a country of cysticercosis-endemicity. The transmission of cysticercosis appears to be primarily a rural phenomenon, sustained by free-range pigs and poor sanitary conditions. This heterogeneity is clear in our review: in Lima, where the disease is not endemic, even poor communities have a low seroprevalence, whereas in endemic communities, $\geq 10\%$ of the adult population are seropositive.

As Latin America becomes increasingly urbanized and the number of free-range pigs declines, the frequency of cysticercosis may gradually fall. However, the reservoir of infected people and pigs is large, so this will be a slow process, and seizures from neurocysticercosis will continue to be a common diagnosis for rural migrants in urban hospitals. In addition, migrants with tapeworms may import cysticercosis into unlikely settings, such as upper-class urban homes in regions of endemicity and industrialized countries. A striking instance of imported cysticercosis occurred in an orthodox Jewish community in Brooklyn, New York, where >1% of people tested seropositive [45]. Infection was associated with having a Latin American immigrant working in the home [46].

Porcine seroprevalence reflects recent infection, and porcine serosurveys are the fastest and least expensive way to document the level of ongoing transmission. Pigs are infected early in life [39] and are usually slaughtered at or before age 1 year. In our survey in an urban commercial pig slaughterhouse, no animals had cysticercosis. In contrast, in areas where the disease is endemic, the seroprevalence among pigs was almost invariably more than twice that among the corresponding human population.

The sole exception to this pattern was in Monterredondo, where the community had begun to grow rice 3 years before and had made a collective decision to tether pigs to protect the rice fields. The introduction of rice meant that for the first time the benefit accrued by tethering pigs outweighed the cost of feeding them. At the time of our survey, nearly all pigs were aged younger than 3 years, had never been free-ranging, and thus had a relatively low seropositivity rate. The seroprevalence among humans reflected the cumulative effect of past exposure.

An understanding of cysticercosis transmission in commu-

 Table 3.
 Calculation of disease burden associated with neurocysticercosis (NCC) in Peru.

Variable	Calculated value(s)		
Population of Peru (1993) ^a	22,704,204		
Population in areas of endemicity ^b	10,449,649		
Population aged >15 years in areas of endemicity	6,269,789		
Estimated range of cysticercosis seroprevalence, % ^c	6	8	10
No. of seropositive persons aged >15 years	376,187	501,583	626,979
With NCC-associated seizure disorders ^d	18,809	25,079	31,349
With other clinical manifestations of NCC ^e	4702	6370	7837
Total with symptomatic NCC	23,512	31,349	39,186

^a 1993 Peru census data [27].

^b Based on population of departments where NCC is endemic (or districts, where departments were partitioned).

^c Seroprevalence in areas of endemicity, based on findings of population-based community studies (see Methods and Results).

^d Equals 5% of no. of seropositive persons aged >15 years in areas of endemicity. Rate of 5% is based on difference in rates of seizure disorder between seropositive and seronegative persons in a neurological survey in Andahuaylas.

^e Equals 25% of the no. of people with NCC-associated seizure disorders (based on [6, 9, 12, 33]).

nities is the essential step to designing control programs; at the same time, studies in neurological clinical settings are useful. They allow analysis of a group in which morbidity and infection are concentrated and therefore provide an indication of human disease in a region. However, it is important not to be misled by case-series data. All our neurological case series were conducted in Lima but demonstrated seropositivity rates similar to those in areas where neurocysticercosis is endemic. The high seroprevalence in these case series is a direct result of the association of cysticercosis infection with neurological abnormalities.

Our data are consistent with observations from other Latin American countries in which the disease is endemic, where $\geq 25\%$ of cases of adult-onset epilepsy are associated with neurocysticercosis [4, 47]. It is important to note that these findings are not in conflict with the observation that the majority of seropositive individuals are asymptomatic. To take an example from the Andahuaylas survey, 9.1% of seropositive individuals (14/154) had seizures, compared with 4.6% of seronegative persons, but >90% of seropositive persons had no symptoms suggestive of neurocysticercosis. However, the converse calculation yields the following: among persons reporting a history of seizures, 23% (14/62) tested seropositive, a rate consistent with clinical case-series data.

Neurocysticercosis could be eradicated with use of the following strategies and available tools: improvements in sanitation, better pig husbandry, (possibly) mass drug treatment of pigs, and targeted or mass treatment of humans [37, 48]. A porcine vaccine against oncosphere antigens is under development and shows promise (M. Verastegui and A. Gonzalez, Lima, Peru, unpublished data). Demonstration intervention projects are under way in Peru and other parts of Latin America.

This review confirms that cysticercosis is a major public health problem that warrants serious disease control efforts, with the eventual goal of eradication.

Acknowledgments

We thank Peter Schantz for helpful comments on the manuscript and Mary Bartlett for editorial assistance.

References

- Craig PS, Rogan MT, Allan JC. Detection, screening and community epidemiology of taeniid cestode zoonoses: cystic echinococcosis, alveolar echinococcosis and neurocysticercosis. Adv Parasitol 1996; 38:169–250.
- Placencia M, Shorvon SD, Paredes V, et al. Epileptic seizures in an Andean region of Ecuador: incidence and prevalence and regional variation. Brain 1992; 115:771–82.
- Senanayake N, Roman GC. Epidemiology of epilepsy in developing countries. Bull World Health Organ 1993;71:247–58.
- Del Brutto OH, Wadia NH, Dumas M, Cruz M, Tsang VC, Schantz PM. Proposal of diagnostic criteria for human cysticercosis and neurocysticercosis. J Neurol Sci 1996;142:1–6.
- Carpio A, Escobar A, Hauser WA. Cysticercosis and epilepsy: a critical review. Epilepsia 1998; 39:1025–40.
- Monteiro L, Coelho T, Stocker A. Neurocysticercosis—a review of 231 cases. Infection 1992; 20:61–5.
- King CH. Cestodes (tapeworms). In: Mandell GL, Bennett JE, Dolin R, eds. Mandell, Douglas and Bennett's principles and practice of infectious disease. 4th ed. New York: Churchill Livingstone, 1995:2544–53.
- 8. Del Brutto OH. Neurocysticercosis. Curr Opin Neurol 1997;10:268-72.
- Dixon HBF, Lipscomb FM. Cysticercosis: an analysis and follow-up of 450 cases. London: Privy Council, Medical Research Council, 1961:1–58.
- Padma MV, Behari M, Misra NK, Ahuja GK. Albendazole in neurocysticercosis. Natl Med J India 1995;8:255–8.
- Baranwal AK, Singhi PD, Khandelwal N, Singhi SC. Albendazole therapy in children with focal seizures and single small enhancing computerized tomographic lesions: a randomized, placebo-controlled, double blind trial. Pediatr Infect Dis J **1998**; 17:696–700.
- Del Brutto OH, Santibanez R, Noboa CA, Aguirre R, Diaz E, Alarcon TA. Epilepsy due to neurocysticercosis: analysis of 203 patients. Neurology 1992;42:389–92.
- Garcia HH, Martinez M, Gilman R, et al. Diagnosis of cysticercosis in endemic regions. Lancet 1991;338:549–51.
- Garcia HH, Herrera G, Gilman RH, et al. Discrepancies between cerebral computed tomography and western blot in the diagnosis of neurocysticercosis. Am J Trop Med Hyg 1994; 50:152–7.

- Cruz I. Epidemiology of neurocysticercosis in Ecuador. In: Garcia HH, Martinez SM, eds. *Taenia solium* taeniasis/cysticercosis. 2nd ed. Lima: Editorial Universo S.A., 1999:289–96.
- Garcia-Noval J, Silva FdM, Moreno E, et al. Epidemiologia de la taeniasis/ cisticercosis en dos comunidades de Guatemala. Guatemala City, Guatemala: Universidad de San Carlos de Guatemala, 1996:1–24.
- Tsang VC, Brand JA, Boyer AE. An enzyme-linked immunoelectrotransfer blot assay and glycoprotein antigens for diagnosing human cysticercosis (*Taenia solium*). J Infect Dis **1989**;159:50–9.
- Wilson M, Bryan RT, Fried JA, et al. Clinical evaluation of the cysticercosis enzyme-linked immunoelectrotransfer blot in patients with neurocysticercosis. J Infect Dis 1991;164:1007–9.
- Diaz JF, Verastegui M, Gilman RH, et al. Immunodiagnosis of human cysticercosis (*Taenia solium*): a field comparison of an antibody–enzymelinked immunosorbent assay (ELISA), an antigen-ELISA, and an enzymelinked immunoelectrotransfer blot (EITB) assay in Peru. Am J Trop Med Hyg **1992**;46:610–5.
- Moro PL, Guevara A, Verastegui M, et al. Distribution of hydatidosis and cysticercosis in different Peruvian populations as demonstrated by an enzyme-linked immunoelectrotransfer blot (EITB) assay. The Cysticercosis Working Group in Peru (CWG). Am J Trop Med Hyg 1994;51: 851–5.
- Diaz F, Garcia HH, Gilman RH, et al. Epidemiology of taeniasis and cysticercosis in a Peruvian village. Am J Epidemiol 1992;135:875–82.
- Garcia HH, Gilman R, Martinez M, et al. Cysticercosis as a major cause of epilepsy in Peru. Lancet **1993**;341:197–200.
- Garcia HH, Gilman RH, Tovar MA, et al. Factors associated with *Taenia* solium cysticercosis: analysis of nine hundred forty-six Peruvian neurologic patients. Am J Trop Med Hyg **1995**; 52:145–8.
- Garcia HH, Gilman RH, Gonzalez AE, Tsang VCW, Verastegui M. Epidemiology of *Taenia solium* infection in Peru. In: Garcia HH, Martinez SM, eds. *Taenia solium* Teniasis/cysticercosis. 2nd ed. Lima: Editorial Universo S.A., **1999**:297–305.
- Garcia HH, Gilman RH, Gonzalez AE., et al. Human and porcine *Taenia* solium infection in a vilage in the hihglands of Cuzco, Peru. Acta Tropica 1999; 73:31–6.
- Garcia HH, Gilman RH, Tsang VC, Gonzalez AE. Clinical significance of neurocysticercosis in endemic villages. Trans R Soc Trop Med Hyg 1997;91:176–8.
- Instituto Nacional de Estadistica e Informatica. Datos del Censo Nacional de 1993. Lima: Gobierno de Peru, 1997.
- Schantz PM, Sarti E, Plancarte A, et al. Community-based epidemiological investigations of cysticercosis due to *Taenia solium:* comparison of serological screening tests and clinical findings in two populations in Mexico. Clin Infect Dis **1994**; 18:879–85.
- Monteiro L, Nunes B, Mendonca D, Lopes J. Spectrum of epilepsy in neurocysticercosis: a long-term follow-up of 143 patients. Acta Neurol Scand 1995; 92:33–40.
- Zuloaga L, Soto C, Jaramillo D, Mora O, Betancur C, Londono R. Prevalence of epilepsy in Medellin, Colombia, 1983. Bol Oficina Sanit Panam 1988;104:331–44.
- 31. Garcia HH, Araoz R, Gilman RH, et al. Increased prevalence of cysticercosis

and taeniasis among professional fried pork vendors and the general population of a village in the Peruvian highlands. Am J Trop Med Hyg **1998**; 59:902–5

- Gonzalez AE, Cama V, Gilman RH, et al. Prevalence and comparison of serologic assays, necropsy, and tongue examination for the diagnosis of porcine cysticercosis in Peru. Am J Trop Med Hyg 1990;43:194–9.
- 33. Martinez MA, Martinez SM, Padilla C, et al. Clinical aspects and unsolved questions in cysticercosis. In: Garcia HH, Martinez SM, eds. In: Garcia HH, Martinez SM, eds. *Taenia solium* taeniasis/cysticercosis. 2nd ed. Lima: Editorial Universo S.A., **1999**:149–62.
- Sarti E, Schantz PM, Plancarte A, et al. Prevalence and risk factors for *Taenia* solium taeniasis and cysticercosis in humans and pigs in a village in Morelos, Mexico. Am J Trop Med Hyg **1992**; 46:677–85.
- Garcia-Noval J, Allan JC, Fletes C, et al. Epidemiology of *Taenia solium* taeniasis and cysticercosis in two rural Guatemalan communities. Am J Trop Med Hyg **1996**; 55:282–9.
- 36. Tsang VCW, Garcia HH. Immunoblot diagnostic test (EITB) for *Taenia solium* cysticercosis and its contribution to the definition of this under-recognized but serious public health problem. In: Garcia HH, Martinez SM, eds. *Taenia solium* taeniasis/cysticercosis. 2nd ed. Lima: Editorial Universo S.A., 1999:245–54.
- Allan JC, Velasquez-Tohom M, Fletes C, et al. Mass chemotherapy for intestinal *Taenia solium* infection: effect on prevalence in humans and pigs. Trans R Soc Trop Med Hyg **1997**;91:595–8.
- Rodriguez-Canul R, Allan JC, Dominguez JL, et al. Application of an immunoassay to determine risk factors associated with porcine cysticercosis in rural areas of Yucatan, Mexico. Vet Parasitol 1998; 79:165–80.
- Sakai H, Sone M, Castro DM, et al. Seroprevalence of *Taenia solium* cysticercosis in pigs in a rural community of Honduras. Vet Parasitol 1998;78: 233–8.
- Palacio G, Jimenez I, Garcia HH, et al. Neurocysticercosis in persons with epilepsy in Medellin, Colombia. Epilepsia 1998;39:1334–9.
- Newell E, Vyungimana F, Geerts S, Van Kerckhoven I, Tsang VC, Engels D. Prevalence of cysticercosis in epileptics and members of their families in Burundi. Trans R Soc Trop Med Hyg 1997;91:389–91.
- Placencia M, Farmer PJ, Jumbo L, Sander JW, Shorvon SD. Levels of stigmatization of patients with previously untreated epilepsy in northern Ecuador. Neuroepidemiology 1995; 14:147–54.
- Bending JJ, Catford JC. Epidemic of burns in New Guinea due to cerebral cysticercosis [letter]. Lancet 1983; 1:922.
- Cysticercosis Working Group in Peru. The marketing of cysticercotic pigs in the Sierra of Peru. Bull World Health Organ 1993;71:223–8.
- Moore AC, Lutwick LI, Schantz PM, et al. Seroprevalence of cysticercosis in an Orthodox Jewish community. Am J Trop Med Hyg 1995; 53:439–42.
- Schantz PM, Moore AC, Munoz JL, et al. Neurocysticercosis in an Orthodox Jewish community in New York City. N Engl J Med 1992;327:692–5.
- Sarti E, Schantz PM, Plancarte A, et al. Epidemiological investigation of *Taenia solium* taeniasis and cysticercosis in a rural village of Michoacan state, Mexico. Trans R Soc Trop Med Hyg 1994;88:49–52.
- Schantz PM, Cruz M, Sarti E, Pawlowski Z. Potential eradicability of taeniasis and cysticercosis. Bull Pan Am Health Organ 1993; 27:397–403.