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33 **RUNNING HEAD: Epilepsy and Cysticercosis in Western Kenya**

34

35 **Abstract**

36 Cysticercosis is the leading cause of acquired epilepsy worldwide and has been shown to be  
37 highly prevalent in pig populations in Western Kenya. We conducted a community-based door-  
38 to-door survey in a region of Western Kenya with a high proportion of pig-keeping households.  
39 Persons with epilepsy (PWE) were determined using a screening questionnaire followed by a  
40 neurologist evaluation. Cysticercosis serum apDia antigen ELISAs and Western blot for LLGP  
41 and rT24h antigen were performed on all PWE and 2% of screen-negative patients. All PWE or  
42 with positive apDia underwent contrast-enhanced brain computed tomography (CT). Of a  
43 sample of 810 village residents, 660 (81%) were present in the homestead of whom 648 (98%)  
44 participated. Of these, 17 were confirmed to have lifetime epilepsy, an estimated crude  
45 prevalence of 2.6%. No humans with (n=17) or without (n=12) epilepsy had serological evidence  
46 of cysticercosis infection. Fourteen PWE and one individual with borderline positive apDia  
47 antigen ELISA underwent brain CT; none had radiographic findings consistent with  
48 neurocysticercosis. Nearly 30% of households kept pigs with 69% always tethered in both wet  
49 and dry seasons. Over 8% (6/72) of pigs had palpable lingual cysts; these pigs all originated from  
50 homesteads with latrines, one-third of which were free-ranging at least some of the time.  
51 Epilepsy prevalence in our study was greater than the national prevalence, but we found no  
52 individuals with epilepsy attributable to cysticercosis. Additional studies are required to identify  
53 causes of epilepsy, human and porcine cysticercosis, the role of spatial clustering, and protective  
54 factors like host-pathogen immunity.

55

56 **1. Introduction**

57 Nearly 65 million people are estimated to have epilepsy worldwide of whom 80% live in  
58 developing countries and between 56 and 75% do not receive appropriate epilepsy treatment  
59 (known as the epilepsy treatment gap).(1-4) Further, mortality rates in persons with epilepsy  
60 (PWE) are approximately 2-3 times higher in low-income countries than in the middle- and high-  
61 income countries.(5) Active epilepsy prevalence in rural coastal Kenya is between 2.9 and 7.8  
62 per 1000 people, nearly 3-6 times higher than the global active epilepsy prevalence,(6, 7) and  
63 over 70% of Kenyan PWE are not receiving appropriate epilepsy treatment.(8)

64 Neurocysticercosis is the leading cause of acquired epilepsy in the developing world and  
65 is due to infection of the brain with the larval form of the pork tapeworm *Taenia solium*.

66 Approximately 50 million people are infected with neurocysticercosis worldwide, and  
67 neurocysticercosis is thought to occur in up to 30% of people with epilepsy or seizures in  
68 endemic regions such as India, Central and South America.(9-11) However, the prevalence of  
69 neurocysticercosis in persons with epilepsy in sub-Saharan Africa varies widely, ranging from  
70 23.2% in an endemic area of Zambia,(12) but only 2.8% in northeastern Tanzania.(13)  
71 Moreover, exposure to other parasites, such as *Onchocerca volvulus* or *Toxoplasma gondii*, is  
72 associated with active epilepsy in sub-Saharan Africa (sSA).(14)

73 In Kenya, little is known about human neurocysticercosis though porcine cysticercosis is  
74 common and has been widely studied.(15, 16) Studies of small-holder pig-keeping communities  
75 in Kenya revealed that 10-17% of pigs had lingual cysticercal cysts.(17, 18) In rural Western  
76 Kenya, the prevalence of porcine cysticercosis by HP-10 Antigen (Ag) ELISA testing was 32.8%  
77 in a rural population(15) and 37% among pigs entering the food chain.(16) Spatial clustering of  
78 cysticercosis has also been demonstrated in several regions throughout the world, which may

79 greatly affect susceptibility of pig-to-human cysticercosis transmission and subsequently  
80 neurocysticercosis prevalence.(20-22) Our objective was to to determine the prevalence of  
81 epilepsy, human neurocysticercosis and human and porcine cysticercosis in a village with  
82 unknown cysticercosis and neurocysticercosis prevalence located within a region of Western  
83 Kenya. This region was of particular interest as it has been previously reported to have a high  
84 proportion of pig-keeping households and elevated prevalence of human and porcine  
85 cysticercosis throughout.(16, 18).

86

## 87 **2. Methods**

88 We conducted a three-stage door-to-door community-based assessment of a typical Western  
89 Kenyan village with a high proportion of pig-keeping households between 25<sup>th</sup> February and 3<sup>rd</sup>  
90 June 2015 to determine the prevalence and association between epilepsy, human and porcine  
91 cysticercosis.

92 **2.1. *Study population and sampling:*** We selected a sub-location (Busibwabo) from a region in  
93 Western Kenya that was roughly a 45km radius semi-circle centered on the town of Busia,  
94 Kenya. This region was chosen as it is representative of the wider Lake Victoria crescent zone  
95 and is known to have a high prevalence of human and porcine cysticercosis based on results of  
96 prior studies.(16, 18) We subsequently identified a village (Bumanyi) within this region with a  
97 high proportion of pig-keeping households based on discussions with local representatives of the  
98 Ministry of Agriculture, traders in pork meat, village elders, and a reconnaissance of the area.  
99 We included all household members irrespective of age or sex, and we did not have any  
100 particular exclusion criteria.

101 **2.2. Human Procedures:** In *Stage 1*, we surveyed the head of the household to answer a  
102 household questionnaire, as well as a 9-question epilepsy screening questionnaire adapted from  
103 Placencia, et al. that was translated into the local languages (Kiswahili and Kiluhya).(24, 25)  
104 This questionnaire reliably detects convulsive epilepsies, but its sensitivity for other epilepsy  
105 types is unknown(26). A caregiver was asked to answer on behalf of children under age 12 years.  
106 A brief health examination was performed on all individuals.

107 In *Stage 2*, all human individuals who screened positive for epilepsy on the epilepsy  
108 screening questionnaire underwent a detailed history and physical examination by a study  
109 neurologist (ACM and DS), as well as phlebotomy and blood testing for detection of Ag  
110 (cysticercal antigen). A random sample of 2% of the individuals who screened negative for  
111 epilepsy also underwent venipuncture.

112 In *Stage 3*, all human individuals with confirmed lifetime epilepsy who were  $\geq 5$  years of  
113 age underwent contrast-enhanced Computed Tomography (CT) of the brain at Aga Khan  
114 Hospital in Kisumu, Kenya. Head CT was done in children  $< 5$  yrs only if they had a focal  
115 neurological deficit or positive cysticercal Ag. Head CT was also performed in individuals who  
116 screened negative for epilepsy but had a positive cysticercal Ag result. Contrast-enhanced CT of  
117 the brain was performed using standard protocols and read by two independent radiologists  
118 blinded to the cysticercal serostatus and presence or absence of epilepsy. One radiologist had  
119 expertise in local epidemiology (PR) and the second was a neuroradiologist with expertise in  
120 East Africa (FM). Any discrepancies would be resolved by a 3<sup>rd</sup> radiologist. However, there  
121 were no discrepancies between the neuroradiologists. CT protocol was performed with and  
122 without contrast, including 8mm slice thickness with a 4mm gap between CT slices in the

123 supratentorial brain region, and 5mm slice thickness with a 2.5mm gap in the infratentorial  
124 region.

125 **2.3. Porcine Procedures:** We examined all pigs in the household. We excluded pigs who were  
126 <3 months of age, pregnant or lactating. We administered a brief health survey and performed a  
127 physical examination and phlebotomy. Physical examination and phlebotomy were undertaken  
128 by trained and experienced animal health technicians. We excluded pigs who were pregnant,  
129 lactating or under 3 months of age and an exam was not performed on these. The ventral surface  
130 of the tongue was examined for the presence of cysticerci after restraining the pig with a snare  
131 behind the canine teeth and using a short stick to open the mouth and cotton gauze to protract the  
132 tongue. Anterior vena cava blood samples were collected.(28)

133 **2.4. Cysticercosis testing:** Blood samples were collected in BD Vacutainer® 10-ml plain tubes  
134 and were transported to the field laboratory on ice where they were centrifuged at 3000 rpm for  
135 20 minutes at room temperature. Sera were then aliquoted into 2ml cryovials and stored at -40°C  
136 between 2 to 5 months until they were transported on dry ice to the International Livestock  
137 Research Institute (ILRI) facility in Nairobi where they were stored at -80°C between 2-16  
138 months prior to laboratory analysis. Ag ELISA was performed at ILRI using the apDia  
139 Cysticercosis Ag ELISA kit, a commercially available enzyme immunoassay for qualitative  
140 determination of viable metacestodes (cysticerci) of *Taenia spp* (ApDia, Turnhout, Belgium).  
141 Cut-off values were calculated according to kit instructions. Subsequently, samples were  
142 transported on dry ice to the Centers for Disease Control and Prevention (CDC) in the United  
143 States where enzyme-linked immunoelectrotransfer blot (EITB) assay developed at the Center  
144 for Disease Control in the US using lentil lectin-bound glycoproteins (LLGP) extracted from *T.*  
145 *solium* cysticerci were performed, with a sensitivity of 99% and specificity of 99% for

146 diagnosing NCC(30) on all human and porcine samples. The EITB detects antibodies to any one  
147 of seven cyst-derived glycoproteins including GP50, GP39-42, GP24, GP21, GP18, GP14 and  
148 GP13. The sensitivity of EITB with two or more intracranial cysts present is 100% with 99%  
149 specificity in serum.(30, 31). rT24h antigen, a recombinant *T. solium* protein antigen, was also  
150 performed at the CDC on all human and porcine samples with a sensitivity of 99% and  
151 specificity of 100% for detection of NCC.(30)

## 152 **2.5. Definition of study outcomes**

153 We defined human and porcine cysticercosis and human neurocysticercosis using the following  
154 definitions:

- 155 1. Epileptic seizure: An epileptic seizure is a clinical manifestation (sudden and transitory  
156 abnormal phenomena) presumed to result from an abnormal and excessive discharge of a  
157 set of neurons in the brain, perceived by the patient or an observer. It might include  
158 alteration of consciousness or motor, sensory, autonomic, or psychic events. Febrile and  
159 eclamptic seizures were excluded from analyses.(32)
- 160 2. Lifetime Epilepsy: Lifetime epilepsy consisted of two or more unprovoked epileptic  
161 seizures in a lifetime. An episode of status epilepticus or multiple seizures occurring in a  
162 24-hour period were considered a single event.(32)
- 163 3. Active epilepsy: A person with active epilepsy is an individual who meets criteria for  
164 lifetime epilepsy *and* has had at least one epileptic seizure in the previous 5 years,  
165 regardless of antiepileptic drug treatment.(32)
- 166 4. Treatment Gap: The proportion of individuals with active epilepsy not currently taking  
167 anti-epileptic drugs.(33)



168 5. Human cysticercosis, neurocysticercosis and neurocysticercosis-related epilepsy:  
169 Standard clinical diagnostic criteria were used for human neurocysticercosis relying  
170 primarily on CT scans and serological testing as this community-based sample did not  
171 have acute complaints. Neurocysticercosis-related epilepsy was defined as an individual  
172 with active epilepsy and probable or definite neurocysticercosis.(34, 35)

173 6. Porcine cysticercosis: was defined as the presence of lingual cysts or positive-Ag on the  
174 ApDia Ag-ELISA.(15)

175 **2.6. Statistical Analyses:** We used each household as the primary sampling unit. We described  
176 the demographic and health characteristics of the human and porcine study participants. We  
177 generated crude, age and age- and sex-adjusted prevalence estimates using an internal reference  
178 population both for active and lifetime epilepsy using binomial regression. All analyses were  
179 conducted in Stata version 14.

180 **2.7. Ethics:** This study was approved by the Institutional Research Ethics Committee (ILRI-  
181 IREC2014-16) and Institutional Animal Care and Use Committee (ILRI-IACUC2014.36) of the  
182 International Livestock Research Institute (ILRI), Kenya and the Human Investigation  
183 Committee (HIC) at Yale University, USA. ILRI IREC is registered and accredited by the  
184 National Commission for Science, Technology and Innovation in Kenya and approved by the  
185 Federalwide Assurance for the Protection of Human Subjects in the USA. The IACUC at Yale  
186 exempted the protocol.

187

### 188 **3. Results**

189 The village was made up of 154 households among which five did not consent to participate and  
190 four were not present for at least two attempted visits. Of 810 village residents, 660 (81%) were

191 present in the household of whom 648 (98%) participated (Figure 1). Median household size was  
192 4 (Interquartile Range [IQR]: 3, 6) and the median number of children per household was 4  
193 [IQR: 3, 7], and 24% grew crops for sale (Table 1). About one-third of adults were married  
194 (32%), and around two-thirds had primary school education or less (73%) and were farmers  
195 (76%).

196 Over 28% (41/145) of households kept pigs, of which 97% (70/72) were the local pig  
197 breed and 58% (41/71) were procured from another village, and 23% (16/71) from another home  
198 in the same village. The median number of pigs in pig-keeping households was 1 [Range:1-7]  
199 and two households had suckling piglets. Less than half (43%) of pigs had been given veterinary  
200 treatment in the prior 12 months (83% of these a general de-wormer) and among those 30 pigs,  
201 the primary treatment provider was a household member (86%) with a minority receiving  
202 treatment from an animal health assistant (6%) or veterinary drug supplier; Table 3.

203 Approximately 70% (52/72) of pigs were always tethered in both wet and dry seasons.  
204 Over 73% (469/643) of individuals reported always using a latrine (Table 1), and significantly  
205 more pig-keeping households having a latrine that was completely closed (33% vs. 16% among  
206 non-pig-keeping households;  $p=0.03$ ). Pig-keeping households were significantly more likely to  
207 keep other livestock: cattle ( $p=0.004$ ), chickens ( $p=0.24$ ) and dogs ( $p=0.001$ ) in the home. Nearly  
208 one-quarter of non-pig keeping households had electricity in the household, while 42% of pig-  
209 keeping households had access to electricity ( $p=0.04$ ) (Table 1).

210 **3.1. Epilepsy:** There were 36 individuals who screened positive for epilepsy and one additional  
211 individual was identified as a PWE by a family member. Of these, 30 were evaluated by a  
212 neurologist of whom 17 were confirmed positive after neurologist review (Figure 1). Alternative  
213 diagnoses included syncope (5), chills (1), fasciculations/muscle fatigue (3), Parkinson's Disease

214 (1), behavioral episodes (1), febrile seizure (1). Crude lifetime epilepsy prevalence was 26.2 per  
215 1000 population (95%CI: 15-42) and age- and sex-adjusted prevalence was 36 per 1000  
216 population (95%CI: 13-59). Crude active epilepsy prevalence was 22 per 1000 population  
217 (95%CI: 13-36) and age-and sex-adjusted prevalence was 36 per 1000 population (95%CI: 15-  
218 57) (Table 2).

219 The median age of PWE was 16 years [IQR: 12, 21] and 53% were female (9/17). The  
220 clinical phenotype of seizures in a majority was generalized tonic-clonic, though 12% (2)  
221 reported focal onset of seizures. The median age of seizure onset was 8 years (IQR: 2, 13) and  
222 only two individuals had onset above 18 years of age. A family history of epilepsy was reported  
223 in 53% (9/17) of PWE (first- and second-degree relatives). A history of malaria was reported in  
224 41% (7/17) and febrile seizures in 18% (3/17). An abnormal neurological exam was reported by  
225 the examining neurologist for 29% (5/17) of PWE and developmental delay in 24% (4/17). The  
226 current epilepsy treatment gap was 71% (12/17) but 59% (10/17) reported having taken AEDs in  
227 the past. Nearly half (47%; 8/17) ever sought treatment for seizures from traditional healers at  
228 least once. Among five PWE currently taking AEDs, three were taking carbamazepine and two  
229 phenobarbital.

230 **3.2. Human and Porcine Cysticercosis:** None of the PWE had serological (n=10) or  
231 radiographic (n=15) evidence of cysticercal infection by Ag-ELISA, (Figure 1, Table 2). Of the  
232 12 randomly sampled individuals without epilepsy for whom serological testing was performed,  
233 two individuals without epilepsy with trace positive Ag-ELISA had CT scans performed. Neither  
234 had radiographic evidence of cysticercal infection and follow-up Western Blot testing was  
235 negative. Over 8% (6/71) of pigs had palpable lingual cysts; these pigs all originated from

236 households with latrines and 33% (2/6) were free-ranging at least some of the time. None (0/53)  
237 of the pigs had serological evidence for cysticercosis (Figure 1).

#### 238 **4. Discussion**

239       Epilepsy prevalence across sSA, including East Africa, varies widely. Our study found a  
240 lifetime epilepsy prevalence of 2.6%, comparable to studies from other countries of sSA(36-39),  
241 higher than others(8, 41-45) attributable to differences in sampling, screening methods and  
242 diagnostic criteria utilized.(46) The treatment gap in our study was 64%, similar to other  
243 treatment gap prevalence reported in the region (67%-79%)(8, 37, 42, 46-49). NCC is thought to  
244 be the most common cause of epilepsy worldwide, however, our study did not find evidence of  
245 NCC among those who screened positive for epilepsy, nor among the sample that screened  
246 negative for epilepsy.(9-11) Prior studies from nearby regions demonstrated the prevalence of  
247 human *Taenia spp.* infection was 19.7% (95% CI 16.7-22.7) and porcine *Taenia spp.* infection  
248 was 17.2% (95% CI 9.1-25.3) using Ag-ELISA.(18) Another study from the same region  
249 reported an even higher porcine cysticercosis prevalence of 37.6% (95% CI 29.3-45.9%) using  
250 the same assay.(16)

251       Despite reports of high prevalence of human *Taenia* infection and porcine cysticercosis in  
252 the region,(16, 18) all PWE nor pig had negative cysticercal serology using either Ag-ELISA or  
253 immunoblot antibody testing in this small study. There may be several reasons for this. First,  
254 Most of the data on NCC and epilepsy come from Latin America, with few studies from Eastern  
255 Africa. One study from rural Tanzania(52) found that definite NCC lesions were present on CT  
256 in only 2.4% of persons with epilepsy compared with 1% of the study population without  
257 epilepsy. A higher prevalence of CT-defined NCC was reported in Rwanda with 7.4% of PWE  
258 having definite NCC lesions on CT.(53) Variability exists across sSA with several factors that

259 may contribute to NCC prevalence across the region. Environmental, animal husbandry,  
260 household and individual risk factors known to affect the prevalence of human and porcine  
261 cysticercosis are similar between the low cysticercosis prevalence village we studied and the  
262 high prevalence surrounding Kenyan,(18, 19) including age and gender distribution, pork  
263 consumption, low household wealth, limited access to and use of latrines, and free-roaming  
264 pigs.(19, 54-56) In our study, all households with pigs in our study were kept in homes with a  
265 latrine,and some evidence suggests that this may decrease the risk of cysticercal  
266 seropositivity(57). Moreover, other causes of epilepsy, such as other endemic infections (i.e.  
267 *Onchocerca volvulus* or *Toxoplasma gondii*) associated with epilepsy in sSA,(14) or traumatic  
268 brain injury or hypoxic brain injury at birth should be considered.

269         Extensive inter-country variability exists in human cysticercosis prevalence ranging from  
270 0 to 21.5% and in porcine cysticercosis (0 to 56.7%) using cysticercal Ag detection.(19, 59) One  
271 potential explanation for the variability of cysticercosis prevalence may be spatial clustering of  
272 infection leading to inter-village variability, a finding that has been reported in endemic regions  
273 worldwide.(19, 20, 22) One recent study in Peru found that strong clustering of porcine  
274 cysticercosis occurs near areas with a high prevalence of tapeworm carriers within rural northern  
275 Peru.(22) Therefore, a better understanding of potential protective factors against cysticercosis  
276 infection and special clustering may inform future refinements to the design of elimination  
277 strategies.(60)

278         Although no pig was antigen nor antibody positive, there were six pigs with palpable  
279 lingual cysts. Palpation of lingual cysts is known to be poorly sensitive for porcine cysticercosis,  
280 thus, our results may have been due to other infectious causes of cysts such as *Taenia hydatigena*  
281 or *Taenia asiatica*(61). We do not expect a false positive lingual cyst finding as all veterinary

282 technicians were trained in porcine lingual palpation, therefore, it is possible that the lingual  
283 cysts detected were from an infection other than *Taenia solium* as the potential for cross-  
284 reactivity between *Taenia* species exists.

285         Limitations to this study included only testing a small proportion of individuals with  
286 serum sample for seral cysticercal Ag or head CT which may have underestimated the true  
287 prevalence of cysticercosis and neurocysticercosis in this single village. Moreover, since we  
288 surveyed only one village in this region of Western Kenya, the findings from this study cannot  
289 be generalized to the district or national level. In addition, more recent guidelines recommend a  
290 cut-off of one year of time to define active epilepsy due to problems of recall over longer periods  
291 of time.(68) In our study, we used a five-year cut-off to define active epilepsy using other  
292 guidelines which may be more susceptible to inaccurate reporting and recall.(32) We did not  
293 collect stool samples as part of the study which may limit the findings of this study.

## 294 **5. Conclusions:**

295         The prevalence of epilepsy and the epilepsy treatment gap were high in a rural village of  
296 Western Kenya. Untreated hypertension was also common in this group. In contrast to regional  
297 estimates, the prevalence of human and porcine cysticercosis in this village was very low and no  
298 individuals with epilepsy were found to have neurocysticercosis on head CT. This may be due to  
299 spatial clustering of infection, partial immunity due to prior infection with other *Taenia spp.*, a  
300 small geographical size sampled, or other unmeasured social and environmental factors.  
301 Moreover, the ecology of free range pig-keeping means that the household level risk associated  
302 with latrine provision may be strongly influenced by provision at broader geographical  
303 scales.(58) Wide scale surveys of infectious causes of epilepsy are required to establish baseline  
304 prevalence of neurocysticercosis in areas of known cysticercosis transmission and to create an

305 indicator for measuring impact of *Taenia* control activities. Future research to better understand  
306 potential protective factors and the impact of spatial clustering on the epidemiology of  
307 cysticercosis may aid in tailoring cysticercosis elimination strategies. In addition, a better  
308 understanding of the infectious and non-infectious causes of epilepsy in developing countries is  
309 an important step toward reducing epilepsy burden.

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543 **Table 1. Household and individual human health and demographic characteristics (n=144).**  
 544  
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	Overall
<b>Household Characteristics</b>	(n=144)
Household Size [median (IQR)]	4 (3, 6)
<b>Latrine</b>	
Household has a latrine [% (n)]	76% (109)
All latrines in household completely closed [% (n)]	20% (29)
<b>Keep Other Animals</b>	
Cattle [% (n)]	62% (89)
Goat [% (n)]	12% (17)
Chicken [% (n)]	88% (126)
<b>Socio-Economic Status</b>	
Electricity [% (n)]	29% (42)
Mains	7% (10)
Solar	19% (28)
<b>Individual Characteristics</b>	n=643
Females [% (n)]	57% (365)
Age in years [median (IQR)]	14 (7, 32)
Child [% (n)]	61% (389)
Married [% (n)]	32% (203)
Years lived in village [median (IQR)]	18 (6,35)
Number of children [median (IQR)]	4 (3, 7)
<b>Education/Occupation Among Adults</b>	
Primary school education or less [% (n)]	73% (185)
Farming as primary occupation [% (n)]	76% (192)
<b>Health and Sanitation</b>	
Worms in feces in last year [% (n)]	27% (156)
Took de-worming medication in last month [% (n)]	2% (14)
Always uses latrine for defecation in last month [% (n)]	73% (469)
Livestock have access to home [% (n)]	75% (484)
<b>Water Source</b>	

Primary water source, wet season [% (n)]	
Well or Borehole	66% (472)
Roof	85% (604)
Primary water source, dry season [% (n)]	
Well or Borehole	61% (439)
River or Spring	87% (619)
Do not treat drinking water [% (n)]	15% (99)

547 **Table 2. Crude and adjusted prevalence of lifetime and active epilepsy, human and porcine**  
548 **cysticercosis\*.**  
549

	Prevalence (per 1000 population) [95% CI]	
<b>Lifetime Epilepsy</b>		
Crude	26	[15, 42]
Crude Female	22	[9, 42]
Crude Male	32	[15, 60]
Age-Adjusted	30	[12, 49]
Age-Sex-Adjusted	36	[13, 59]
<b>Active Epilepsy</b>		
Crude	22	[13, 36]
Crude Female	14	[6, 32]
Crude Male	32	[17, 61]
Age-Adjusted	26	[9, 43]
Age-Sex-Adjusted	36	[15, 57]
<b>Cysticercosis</b>		
<i>Human</i>		
Epilepsy	0	[0, 308]*
Without Epilepsy	0	[0, 265]*
<i>Porcine</i>		
Lingual examination	85	[32, 175]
Serology	0	[0, 67]*

550  
551 \* One-sided, 97.5% confidence interval  
552 \*\*Crude, age and age-and-sex-adjusted prevalence estimates were generated using an internal  
553 reference population as the reference population.  
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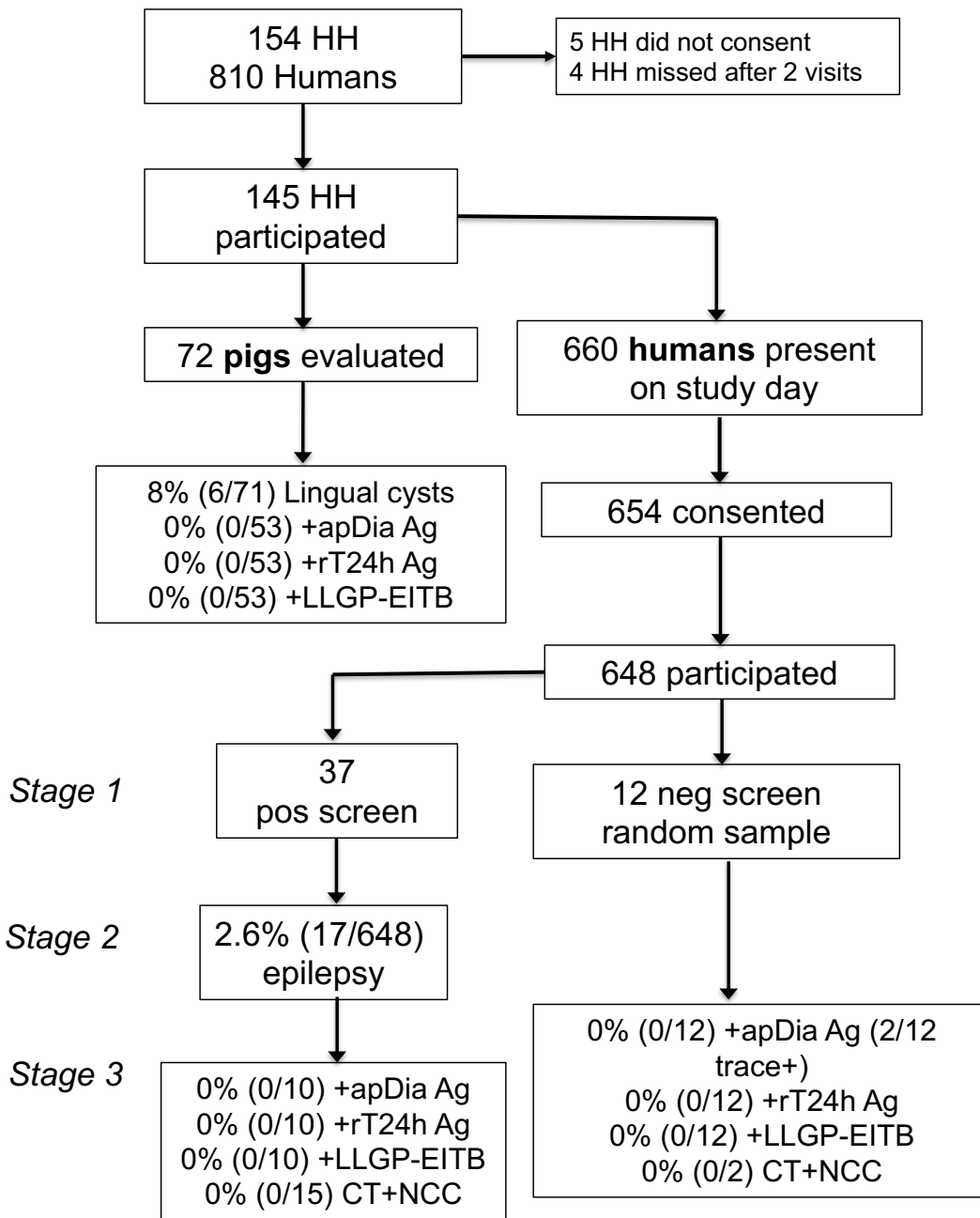


557 **Table 3. Demographic and health characteristics of the porcine population (n=72).**  
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<b>Porcine Population:</b>		
Local Pig Breed [% (n/N)]		97% (70/72)
Age (months) [median (IQR)]		4 (2, 6)
Females [% (n/N)]		55% (40/72)
	Pregnant	18% (7/40)
	Lactating	10% (4/40)
Pig Source [% (n/N)]		
	Another village	58% (41/71)
	Another home, same village	23% (16/71)
	Home	18% (13/71)
	Market	1% (1/71)
Obtained any treatment for pig in prior 12 months [% (n/N)]		43% (30/69)
Treatment type [% (n/N)]		
	De-worming Tablet	83% (25/30)
	Drench	10% (3/30)
	Tablet and Spray	3% (1/30)
	Multivitamin	3% (1/30)
Self or household member was Treatment provider [% (n/N)]		
	Self or Household member	86% (26/30)
Pigs always tethered in dry season [% (n/N)]		72% (52/72)
Pigs always tethered in wet season [% (n/N)]		74% (53/72)
Palpable tongue cyst on examination [% (n/N)]		8% (6/71)
Raise pigs to sell [% (n/N)]		93% (67/72)

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561 **Figure 1. Recruitment and Study Flow.**  
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565 \* Two individuals had trace positive testing on the apDia Cysticercosis Antigen ELISA.  
566 Abbreviations: +CC serology: apDia Cysticercosis Antigen ELISA, LLGP, rT24 Ag above cut-  
567 off value; CT+NCC: Contrast-enhanced Computed Tomography of the Brain consistent with  
568 diagnosis of neurocysticercosis; HH: Household; neg: negative; pos: positive.