Dear Sir:

We are grateful that Kaiser and others responded to our short report.\(^1\) Our findings do not in any way deny a correlation between onchocerciasis and epilepsy in some (but not all) studies of the subject. We also agree with their assessment that additional study is needed to more clearly clarify this complex issue. We believe, however, that results from any correlation must consider confounding variables with other prevalent conditions that cause epilepsy, particularly co-parasitism with *Taenia solium* cysticercosis. In that regard, we are intrigued that Kaiser and others cite the study from southwestern Burundi, whose results show a trend to higher seroprevalence for cysticercal antibodies in 103 patients with onchocerciasis-associated epilepsy compared with controls (11.7% versus 2.8%; \(P = 0.06\)). Many might conclude that those results might well have been significant if the sample were larger.

We recently joined a district health team in a visit to a village in western Uganda in which some 70 persons claiming to be epileptic came out in desperate hope of receiving medical assistance for their condition. This community has been under mass ivermectin treatment for onchocerciasis for more than 10 years, with reported treatment coverages more than 65%.\(^2\) Our recent studies from sentinel communities in Uganda have shown low microfilaria prevalence in children as a result of prolonged annual ivermectin treatments.\(^3\) However, in the village we visited, many of those persons with epilepsy were children less than 10 years of age who were born after commencement of mass treatment. If onchocerciasis is the cause of epilepsy, why does the condition persist as an incident condition in what are areas now under excellent disease control?\(^2,3\) The biological plausibility of onchocerciasis causing seizures has always been in question; the findings that epilepsy persists after the putative cause has been “eliminated as a public health problem”\(^2\) makes that plausibility even more doubtful.

We also need to remember as we attempt to draw conclusions about the role of neurocysticercosis as a cause of seizures that 1) cysticercosis can, like some other infectious diseases, be highly focal and making it difficult to draw inferences easily between villages with similar or dissimilar rates of epilepsy; and 2) results of serologic surveys may not provide a clear picture to the potential for seizures because one neural cyst can result in seizures but have a low predictive value (the Centers for Disease Control and Prevention immunoblot assay for cysticercosis has a 28–72% rate of positivity in persons with one enhancing lesion).\(^4\)

We believe that studies should be broadened to look at other causes of epilepsy, and include sociologic and anthropologic aspects to get a clearer picture of factors responsible. Cysticercosis, in particular, needs to be further investigated, and if its impact is demonstrated, relief needs to be provided for this preventable condition to affected communities.\(^5\) The main purpose of our short report was intended as a call to action to do just that, and to remind readers that taeniasis/cysticercosis is much more common in parts of Africa than recognized, and that any study of epilepsy and/or seizures needs to clearly account for that possibility.

Received September 8, 2008. Accepted for publication September 10, 2008.
REFERENCES


MOSES KATABARWA  
FRANK RICHARDS JR  
The Carter Center  
Atlanta, GA 30307  
E-mail: mkataba@emory.edu  
or rzk5@cdc.gov

MARK EBERHARD  
Centers for Disease Control and Prevention  
Atlanta, GA 30341