caused by *Taenia solum*. We did a craniocerebral CT in our patient and found no evidence of brain abnormalities, implying no infection with *T solum* or *S mansoni*. In our case, the worm was completely removed during the operation and no remaining scolex was found on subsequent examination under an operating microscope. From this examination we also confirmed that no other worms were present.

Sparganosis can cause multiple lesions in the body, which are prone to recurrence.[3,5] Our patient, however, rejected our proposal for a general examination, and antiparasitic therapy was therefore initiated to control possible larvae elsewhere in the body.

We declare no competing interests.

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New conceptual framework for tuberculosis transmission

We read with interest the Comment by Onisillos Sekkides[1] and the accompanying articles on the necessity of increasing the understanding of tuberculosis transmission dynamics to design more effective control strategies. This need is particularly acute in HIV-endemic settings, in which many individuals are coinfected with both pathogens.

In sub-Saharan Africa, populations are extremely mobile, and this mobility increases the complexity of the transmission dynamics of infectious diseases. We propose a new conceptual framework that includes mobility for understanding and modelling tuberculosis transmission in sub-Saharan Africa. Our framework includes three transmission pathways: first, resident-to-resident transmission (ie, individuals acquire tuberculosis in their home community from other residents); second, visitor-caused transmission (ie, individuals acquire tuberculosis in their home community from a resident of another community); and third, travel-related transmission (ie, individuals acquire tuberculosis in another community). Within this framework, a country-level epidemic is conceptualised as a series of mobility-linked microepidemics.

The importance of each pathway can be determined by constructing country-level maps of HIV and tuberculosis, and by identifying large-scale mobility networks. This requires detailed spatial epidemiological data on HIV and tuberculosis, and population-level mobility data. Many countries in sub-Saharan Africa have HIV-testing data that can be used to map their epidemic; as an example, data from Malawi are shown in the appendix. Data needed to map tuberculosis epidemics exist for some countries in sub-Saharan Africa. However, mobility data are extremely scarce; travel data reveal spatial patterns (appendix), but not networks. Mobility networks can be identified by analysing large datasets of call-detail records from mobile phones.[2] This approach has been used to determine the importance of visitor-caused and travel-related transmission of malaria.[3] Applying this approach to tuberculosis could result in a greater understanding of transmission.

As Sekkides[1] discussed, mathematical models are used to predict the effect of tuberculosis control strategies. We believe that there is a need to develop a new generation of models that include mobility-driven transmission; current tuberculosis models are based on models developed by Blower and colleagues[4,5] almost 25 years ago. New models should include visitor-caused and travel-related transmission pathways, and realistic representations of large-scale mobility networks. We predict that these more realistic models will show that reducing transmission in a hotspot (an area of high transmission), without also preventing visitor-caused and travel-related transmission, is an ineffective control strategy. More importantly, we predict that modelling mobility-driven transmission will lead to the design of more effective tuberculosis control strategies.

We declare no competing interests.

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Trained dogs identify people with malaria parasites by their odour

Eliminating malaria would be simpler if a non-invasive method was available for detecting infected individuals