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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See **Online** for appendix

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.

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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

in populations with low malaria prevalence. Infected individuals could then be treated with antimalarials. Work has shown that people infected with malaria parasites produce a body odour that is detected by mosquitoes, which results in malaria mosquitoes preferentially feeding on asymptomatic, malaria-infected individuals.[1] We hypothesised that dogs, with their highly advanced sense of smell,[2] could be trained to detect people carrying malaria parasites.

We collected foot odours from Gambian school children aged 5–13 years with and without asymptomatic falciparum malaria. Each child was fitted with a pair of ankle-length nylon socks (Blue Box Disposables, Doncaster, UK) to wear overnight. The diagnostic accuracy of two trained dogs was assessed in a blinded study done 17–20 months after the socks were collected. Sock samples from 30 malaria-positive individuals, assessed by microscopy, and 145 uninfected children who were randomly selected (qPCR negative individuals) were tested (video, tables 1, 2).

Dog L distinguished between sock samples from malaria-infected and uninfected school children (p<0·0001), with a sensitivity of 73·3% (95% CI 54·1–87·7) and specificity of 91·0% (85·2–95·1); the sensitivity of Dog S was 70·0% (50·6–85·3), with a specificity of 90·3% (84·3–94·6%). Both dogs responded in the same way to 141 (80·6%) of 175 samples. Excluding four samples which had only sexual forms, at densities around 100-times lower than asexual forms, the sensitivity of detection was 76·9% (56·4–91·0) for Dog L and 90·3% (84·3–94·6) for Dog S. Agreement among the 145 uninfected samples was 89·7%. Six apparently uninfected samples were indicated as positive by both dogs, more than the expected number given their respective specificities (Fisher's exact test, p=0·0003). This result might have occurred because an uninfected child was sharing a bed with an infected child. With bed sharing common in this community and a parasite prevalence of 8% by microscopy, one would expect 12 of the 145 uninfected children to be sharing a bed with an infected child.

	Indication		Total
	No	Yes	
Dog L			
Uninfected	132 (91·0%)	13 (9·0%)	145
Malaria	8 (26·7%)	22 (73·3%)	30
Dog S			
Uninfected	131 (90·3%)	14 (9·7%)	145
Malaria	9 (30·0%)	21 (70·0%)	30

Data are n (%).

***Table 1*: Malaria sample detection by individual dogs**

	Dog S indication		Total
	No	Yes	
Dog L indication (uninfected samples)			
No	124 (93·9%)	8 (6·1%)	132
Yes	7 (53·9%)	6 (46·1%)	13
Dog L indication (malaria-infected samples)			
No	4 (50·0%)	4 (50·0%)	8
Yes	5 (22·7%)	17 (77·3%)	22

Data are n (%).

***Table 2*: Concordance in sample detection between dogs**

Our findings are consistent with the hypothesis that dogs can distinguish between the smell of asymptomatic malaria-infected and uninfected individuals. This study was the first to use dogs to detect odours identifying individuals infected with malaria. These results are broadly in line with WHO's criteria for the procurement of rapid diagnostic tests, in which the test should be able to detect at least 75% of *Plasmodium falciparum* samples at densities of 200 parasites per µL or greater, with specificity of no less than 90%.[3] In our study, 60% of the samples were below this parasite density. Including only samples with 200 parasites per µL or greater increased the mean sensitivity of both dogs to 81·8% (95% CI 59·1–104·5), above the threshold required for WHO malaria diagnostics.

In the future, malaria detection dogs could be used to detect individuals infected with malaria at ports of entry in countries or regions that are malaria-free or approaching malaria elimination.

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See **Online** for video

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New environmental reservoir of CPE in hospitals

The incidence of multidrug-resistant carbapenemase-producing Enterobacteriaceae (CPE) is steadily increasing, and maximal infection control and prevention strategies should be implemented to limit their spread in health-care settings.[1] WHO guidelines, published in 2017, recommend hand hygiene compliance, screening, contact precautions, environmental disinfection, and educational sessions to prevent the transmission of CPE, and surveillance cultures of the environment are advised to be undertaken with caution.[2] Drains, sinks, and faucets have been identified as vehicles for the transmission of bacteria, mainly *Acinetobacter baumannii* and *Pseudomonas aeruginosa*.[3,4] Based on expert opinion, environmental cultures are most appropriate for analysis if these organisms are involved.[2] To our knowledge, we describe the first detection of CPE in toilet bowls and traps and subsequent hospital-associated transmission of *Citrobacter freundii* producing OXA-48 to four patients in one ward. We also outline the subsequent surveillance and infection control measures undertaken to contain the outbreak.

In a 71-year-old woman, who at the time of infection had recently undergone a pancreatic-oduodenectomy procedure, culture of post-operative wounds and a subsequent rectal swab yielded a *C freundii* producing OXA-48. The second patient, an 87-year-old woman residing in the same two-person room as the first patient, also screened positively for *C freundii* OXA-48 infection. Both patients were not known to be carrying, or at risk of carrying, a CPE. These first cases of *C freundii* OXA-48 in a CPE-negative hospital were unexpected. Weekly rectal screening of all patients residing in the ward for 2 weeks following the identification of the first two infections revealed no other CPE-positive cases.

1 month after discharge of both patients, a 90-year-old man was admitted to the same index room and his urine sample tested positive for *C freundii* OXA-48; the other patient concurrently residing in the same room tested negative for CPE. Our CPE outbreak protocol was initiated and thorough sampling of all high-touch surfaces, medical tools, and toilet water was done. 50 mL of water from the toilet bowl was concentrated through the membrane filter technique, with subsequent culture of the 0·22 µm membrane (Millipore, Merck, Billerica, MA, USA) on a CPE selective agar (Oxoid Oxoid Ltd. Thermo Fisher Scientific, Cambridge, UK). Of the sampled environmental cultures, only the toilet water was found to be positive for *C freundii* OXA-48, with a confluent culture on the membrane. Despite the removal of toilet brushes and daily toilet disinfection, with a final concentration of 5000 ppm chlorine, for 3 consecutive days, the *C freundii* persisted to the same extent. After 3 days of daily pre-cleaning with biguanide/quaternary ammonium (Hexanios, Anios, Lille, France) for 15 mins, followed by disinfection with 2500 ppm peracetic acid for 30 mins, *C freundii* OXA-48 in toilet bowls and traps was succesfully eradicated. Room disinfection was done with 5000 ppm chlorine. All toilet bowls and traps of the ward were screened for CPE. Nine rooms that shared the common waste plumbing tested positive for *C freundii* OXA-48. 15 patients residing in these rooms were subsequently screened for CPE by rectal swab, and one additional patient tested positive for *C freundii* OXA-48. All toilets in the adjacent rooms were cleaned and disinfected with the same procedure on a weekly basis. Follow-up CPE screening of patients and cultures of the toilets remained negative 1 year after the outbreak on this ward.

In conclusion toilet bowls and traps in a hospital environment are a possible source of CPE, which pose a risk for hospital-acquired infections and propagation of outbreaks. Besides patient CPE screening, environmental sampling might allow earlier detection of unexpected reservoirs. Periodic disinfection with peracetic acid was effective in terminating the reported outbreak. The original source of the *C freundii* OXA-48 remains unknown, but because Enterobacteriaceae are predominantly of human origin in hospitals, future guidelines should also consider the disinfection of toilet, sink, and shower drainage systems in CPE isolation rooms. Efforts to redesign waste water drainage systems, toilets, showers, and sinks to minimise the risk of transmission from colonised water reservoirs should be initiated urgently.

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