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Pertussis immunisation in newborn babies

We read with interest the Article by Daan Barug and colleagues,[1] and the accompanying Comment by Kirsten Maertens and Elke Leuridan,[2] on maternal pertussis immunisation combined with delayed primary infant vaccination using reduced doses of acellular pertussis vaccine.

The randomised trial confirms the effectiveness of maternal pertussis immunisation to protect against pertussis infections in infants during the first few months of life via increased transplacental transfer of maternal vaccine-specific antibodies. The results also confirm the interaction between increased concentrations of maternal antibodies and active immunisation in the infant (ie, the blunting effect[3]), and show that antibody concentrations were similar to those in the control group during the first 3 months of life. These results indicate that changing the primary vaccination schedule for infants is feasible.

The findings are useful for health-care experts and policy makers who define vaccination schedules in high-incomes countries. However, some questions need to be answered before the most effective schedule is established.

Immunity against pertussis is multifactorial. In addition to specific antibody production, cellular immunity is involved in the elimination of bacteria that escape humoral defence mechanisms. The concentrations of some IgG antibodies are associated with clinical protection: anti-pertussis IgG concentrations seem to be the most important and are most commonly used as a specific marker of immunity against pertussis.[4] To date, the range of antibody concentrations that effectively confer protection against pertussis in newborn babies and in children has not been established. Previous studies testing different vaccination schedules have not been helpful in this regard because the effectiveness of reducing the number of pertussis cases was not assessed as an outcome. Published trials have also been limited by small sample size.

In terms of immunogenicity of vaccines and schedules, the antibody concentrations observed by Barug and colleagues in newborn babies in the maternal tetanus, diphtheria, and acellular pertussis (Tdap) group at 2 and 6 months of life are similar to those reported by Wood and colleagues[5] in newborn babies who received a monovalent acellular pertussis vaccine at birth. Which of the two schemes is more effective remains unclear. We hypothesise that vaccinating the mother before birth and the child at birth, and delaying subsequent vaccinations, might be the best approach.

These questions could be answered by a large, international, prospective study that overcomes the limitations of previous studies and includes the measurement of antibodies against different pertussis vaccines, antibody responses (concentrations and half-life), and vaccine interference, with follow-up that lasts through infancy and monitors susceptibility to pertussis.

We declare no competing interests.

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Diagnosis and treatment of human sparganosis

We thank M Teresa Galán-Puchades[1] for her comments on our Clinical Picture reporting a 50-year-old woman with a recurrent eyelid swelling.[2] We agree that the exact species of plerocercoid should in most cases be identified by PCR. We had no doubt, however, that our case was caused by plerocercoid larvae belonging to the *Spirometra* genus on the basis of morphological features, although the exact diagnosis of the species by morphology alone can be difficult.[3] Identification can be done by inoculating the larvae into a susceptible host, collecting the adult worm in the intestine, and examining the eggs in the faeces,[3] or alternatively in the current era, by PCR.

The first diagnosed case of sparganosis in a human was identified in Xiamen, China, in 1882, and was found to be caused by *Spirometra mansoni*.[3] Sparganosis has now been reported worldwide, although occurs mainly in Asia, and *S mansoni* is considered to be the predominant species in this region.[4] Although PCR identification was not done in our case, sparganosis in China is commonly perceived to be caused by *S mansoni*, and our parasitologist therefore also concluded that this was the species in our case.

Regarding the treatment, we agree that clinicians such as ourselves should be careful to use albendazole in view of potentially causing neurocysticercosis in a region with endemic cysticercosis 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

See **Online** for appendix

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