tuberculosis in high-risk migrants during post-migration follow-up, which is currently done by Canadian, USA, and Australian border authorities and agencies. Chan and colleagues[10] selected 20 articles, which described 222 375 high-risk persons out of 8 355 030 migrants processed between 1981 and 2014. The high-risk definition was based on different methods: in some of the studies chest radiography alone was used to define high risk, whereas others included sputum smears, sputum culture, and tuberculin skin testing. The pooled cumulative incidence of tuberculosis post-migration was 2794 (95% CI 2179–3409) per 100 000 high-risk migrants, much higher than the tuberculosis rates in the migrant-receiving countries. Eight studies described the tuberculosis incidence found at first post-migration assessment (pooled cumulative incidence 3284 [95% CI 2173–4395] per 100 000 persons). An interesting finding of the study was the higher yield rate for the combined chest radiography and smear microscopy screening by comparison with chest radiography and sputum culture. It was noted that the yield of positive cases from continuing assessments fell off markedly compared with the first assessment done after arrival.

This study is new and important, showing the vulnerability of high-risk migrants (with incidence rate ratios in high-risk migrants ranging from 102 [95% CI 75–128] in the UK to 416 [314–525] in the USA compared with the general population) and the need for systematic post-migration screening, eventually complementing a more sensitive pre-migration one. More needs to be known about the cost-effectiveness and advantages of the various screening approaches available. Nevertheless, this study should stimulate all recipient countries to develop improved tuberculosis screening of migrants within sensitive health systems, respecting human rights, and preventing stigma. Only by efficient screening and treatment of migrants on arrival will the general population be protected from rising tuberculosis incidence.[11]

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Global perspectives on maternal immunisation

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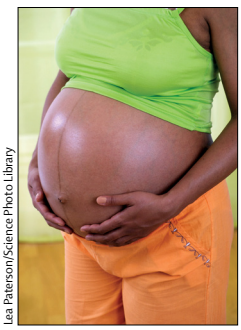
Although considerable progress has been made globally to reduce under-5 mortality, advances in the reduction of neonatal deaths in the first year of life have been slower. The annual deaths of almost 700 000 infants younger than 1 month are attributed to infectious diseases such as pneumonia and sepsis, and these account for about 28% of neonatal deaths under the age of 1 month.[1] Additionally, many more newborn babies, with immature immune systems, have long-term morbidity from infections early in life.[2] Sustainable Development Goal 3·2 calls for the end of preventable deaths of newborn babies and children younger than 5 years by 2030, with all countries aiming to reduce neonatal mortality to at least 12 per 1000 livebirths.[3] To reach these goals, effective strategies to protect infants in the first weeks of life will be essential.

Maternal immunisation during pregnancy is one such strategy, with the untapped potential of providing protection to the infant through the transfer of maternal antibodies across the placenta.[4] These antibodies persist in the circulation after birth and can protect the infant



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against infectious diseases during the first months of life. This strategy could potentially provide additional benefit by preventing infectious causes of preterm births and stillbirths, and by protecting pregnant women.

The effectiveness of maternal immunisation has been shown by the Maternal and Neonatal Tetanus Elimination programme,[5] which has reduced the burden of neonatal tetanus in low-income and middle-income countries. Additionally, increasing evidence shows the safety and efficacy of vaccines against influenza and pertussis for the protection of pregnant women and their infants.[6,7] The success of these programmes has stimulated the development of new vaccines for maternal immunisation against group B streptococcus and respiratory syncytial virus, among others. A respiratory syncytial virus vaccine has been advanced to phase 3 clinical trials (ClinicalTrials.gov NCT02624947) in pregnant women and could represent the first vaccine explicitly approved for use in pregnant women to protect newborn babies. This novel development shows that maternal immunisation is gaining recognition as an important strategy that could combat neonatal morbidity and mortality.

However, further research is needed regarding the underlying mechanisms of maternal immunisation. The development of safe and efficacious vaccines for use during pregnancy requires an improved understanding of the unique immunobiology of pregnancy, and of the fetus and neonate, as described in the first paper of this Series by Arnaud Marchant and colleagues.[8] Subsequently, this knowledge could be used to optimise vaccine formulations, timing, and dosing of immunisation during pregnancy and for diverse populations.

Marchant and colleagues' paper[8] outlines the process by which a broad community of experts and stakeholders collaborated to identify the important knowledge gaps in immunobiology that are most relevant for the advancement of maternal immunisation. The subsequent papers identify knowledge gaps most important for pertussis and influenza,[9] and discuss group B streptococcus and respiratory syncytial virus vaccine development for maternal immunisation.[10] Together, these papers provide a timely landscape review that serves to inform the design and development of safe and effective vaccines for use in pregnancy to potentially reduce global infant mortality. Maternal immunisation vaccine development and implementation programmes could be further strengthened by the inclusion of future research that is geared towards closing such knowledge gaps.

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Zika enhancement: a reality check

The recent Zika virus epidemic in Brazil was associated with microcephaly and fetal malformation. Severe cases of CNS malformation in northeastern Brazil have raised questions about why infection in pregnant women was apparently more severe in this region. We wonder whether antibody-dependent enhancement (ADE) of infection might have contributed to the high incidence of congenital Zika syndrome.

Two populations of dengue virus antibodies, monotypic and multitypic, participate in dengue virus intrinsic and extrinsic ADE.[1] The polyclonal antibodies induced after a single dengue virus infection enhance a second dengue