

of mathematical modelling in formulating performance targets for tuberculosis (TB) control. Furthermore, they appropriately highlight that such models serve as simplifications of a far more complex reality, in which *M. tuberculosis* is transmitted in heterogeneous fashion. They mention two key factors – case density and transmission saturation – that contribute to such heterogeneity. However, there are many more, including nosocomial transmission clusters,¹ strains of different fitness,² social determinants of TB transmission³ and complex interactions with the HIV co-pandemic.⁴ Ultimately, no model can account for all potentially relevant aspects of TB transmission. Thus, we need simple models capable of distilling key components of transmission dynamics into clear messages. However, more complex models can be created to try to show us where – and to what degree – simple models may go wrong. Models exploring case density and transmission saturation could have an important role to play in this regard, and we welcome such efforts.

Ultimately, we must also remember that mathematical models are but one component of a broader TB research agenda that is sorely in need of expansion.⁵ While refining our models, we must not lose sight of the fact that approaches over the past 20 years have failed to stem the tide of ongoing TB transmission and that a broad-based, concerted effort – including an expanded research agenda, relentless improvements in case detection and development of better tools for TB diagnosis and treatment – will be required to meet current goals for TB control. Over the next 20 years, the value of TB mathematical models may be measured less by their ability to accurately describe the dynamics of TB transmission, and more by their power to galvanize support and inform appropriate policy. ■

Author reply

Marais & van Helden provide an important historical context for the role

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