In our opinion, the extent to which dysglycaemia is causally associated with tuberculosis remains unanswered. Dr Ugarte-Gil and colleagues, with no control group, cannot answer that question.¹ We note that the prevalence of diabetes in, for example, their South African TB patients is not dissimilar to that reported in the general population of South Africans aged over 30 years.² In fairness, their TB cohort are at the lower end of this age distribution.

However, it seems that the authors are in a position to remedy two problems with the diabetes-tuberculosis literature.

First, they report collecting but, as is common, do not present data on dysglycaemia at treatment completion. An HbA1c at this time point (ideally later) could unpick which of their patients with ‘newly diagnosed DM’ have diabetes versus stress hyperglycaemia. This assumes that, after few months of effective treatment, the stress response settles and patients regain most TB associated weight loss. We note (Table 4), that median HbA1c was already beginning to normalise in ‘New DM’ patients after a fortnight of TB treatment.

Second, the diabetes-tuberculosis literature, almost universally, dichotomises or (as here) aggregates dysglycaemia into discrete categories. For an inherently continuous variable, this makes no sense. If dysglycaemia is causally associated with tuberculosis, a substantial burden of attributable disease may be driven by the (larger) group of individuals with modest elevations in their blood sugar.³

We would, therefore, be interested to see both data on dysglycaemia at treatment completion and the distributions of glycaemic control in these TB patients from four very different settings. We encourage the authors to make this information available, either in their response to this letter or in future publications.

The authors declare no relevant conflicts of interest.

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² Bertram MY, Jaswal AVS, Wyk VP Van, Levitt NS, Hofman KJ. The non-fatal