

THE AUTHORS REPLY

We appreciate the points made by Lifson et al. (1) regarding our study (2). We agree that standard definitions, multiple reviewer adjudication, and clinical judgement on the part of reviewers are all necessary for myocardial infarction (MI) adjudication. These were key strengths of our study. We also wholeheartedly agree with Lifson et al. on the importance of reviewing the primary data and not just case report forms. In fact, we avoid case report forms entirely, and reviews are entirely based on primary data.

We agree with Lifson et al. regarding the benefits of being able to categorize events based on degree of diagnostic certainty: We used the categories of definite and probable. We would further highlight the importance of distinguishing primary spontaneous MI events from events that occur secondarily to other clinical syndromes, such as sepsis causing severe hypotension. Secondary MIs are categorized as type 2 MIs according to the universal MI definition (3). They have treatment implications distinct from those of primary MIs, and we have found them to be very common in persons with human immunodeficiency virus (approximately half of all events).

We thank Lifson et al. for sharing their “INSIGHTS” with us.

ACKNOWLEDGMENTS

Conflict of interest: none declared.

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RE: “ESTIMATED RATE OF REACTIVATION OF LATENT TUBERCULOSIS INFECTION IN THE UNITED STATES, OVERALL AND BY POPULATION SUBGROUP”

Tuberculosis (TB) disease can occur soon after new infection (or reinfection) or many years thereafter through reactivation of latent infection (1). Reliable estimates of rates of reactivation are needed to predict the impact of interventions, particularly in low-burden, high-income settings where there may be little ongoing transmission.

We welcome Shea et al.’s (2) recent attempt to directly calculate this rate for the whole of the United States using empirical data. This could have permitted validation of currently used estimates, obtained from mathematical models that fit to country-level historical data (3). Shea et al. reported differential TB reactivation rates by place of birth and human

immunodeficiency virus (HIV) status. If valid, these estimates would also be valuable. However, we have concerns about the assumptions used to derive these metrics from the limited data available. To calculate the rate of TB reactivation in the United States, Shea et al. divided estimates of the number of cases that they attributed to reactivation by estimates of the number of persons considered at risk of reactivation (2).

For the numerator (cases of reactivation TB), they used TB isolates from 2006–2008 with a unique genotype within the national genotyping database. Here, there were substantial missing data, that is, TB cases without a genotype. Only 57% of all TB cases (73% of culture-positive cases) reported