The frequencies of both mutations, and hence the frequencies of the corresponding lineages in the study by Valvatne et al., are in line with spoligotyping data from another study that analysed strains from the same year and sample location, and found that 4.8% and 48.4% of isolates belonged to the CAS lineage and the EAI lineage, respectively.6

**Transparency declarations**
None to declare.

**References**

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Sir,

We thank Köser et al.1 for their valuable comments on our paper2 and note with interest the association of specific single nucleotide polymorphisms (SNPs) with *Mycobacterium tuberculosis* strain lineages; the mutation at position –46 in the oxyR–ahpC intergenic region with the Central Asian (CAS) lineage and the C to T mutation in oxyR with East African–Indian (EAI) lineage strains. This corroborates some of our own recent observations3 in a study conducted in a country that neighbours Myanmar (India), where we showed by SNP analysis of the pncA gene (confering resistance to pyrazinamide) a CAS lineage-specific silent mutation, S65S, which is observed for the majority of CAS lineage isolates.

There seems to be a somewhat mixed view with regard to the role of the frequent mutation in the oxyR–ahpC intergenic region at position –46. In a study by Baker et al.,4 among 378 strains, the oxyR–ahpC mutation was present in 23.7% of isoniazid-resistant isolates and 7.5% of susceptible isolates. Although a phylogenetic marker for a subgroup of *M. tuberculosis* strains originating on the Indian subcontinent (CAS), this marker is strongly associated with isoniazid resistance and the katG 315Thr mutation.5

Indeed, oxyR is a pseudogene, and in our study from Myanmar we interestingly observed that a synonymous polymorphism is more frequently observed in isoniazid-resistant isolates than in multidrug-resistant isolates (*P*<0.001). This finding needs to be verified in studies from other geographical areas.

**Transparency declarations**
None to declare.

**References**