"Deep amplicon sequencing for culture-free prediction of susceptibility or resistance to 13 anti-tuberculous drugs." A. Jouet, C. Gaudin, N. Badalato, *et al. Eur Respir J* 2021; 57: 2002338.

When tracing back source data in response to a reader's request for details of the study, the authors of the abovementioned paper detected a problem with available reference phenotypic data for the comparison with the Deeplex Myc-TB results presented in table 3. This involved mostly (expected) susceptible phenotypes, for which hardly any discordant mutations were found by Deeplex testing, and the problem therefore went unnoticed in the original report.

After correction, specificities and sensitivities are only slightly changed compared to previous values for most drugs involved. The overall sensitivity and specificity excluding uncharacterised mutations are 95.2% and 95.0%, respectively (*versus* 95.3% and 97.4% previously). The slight decrease in overall specificity is mostly a result of the lower specificity (68%) for ethionamide. Importantly, in all discordant cases with resistance predictions by Deeplex Myc-TB *versus* a susceptible phenotype for this drug, well-known ethionamide resistance mutations (*ethA* indels and common *fabG1/inhA* mutations, such as C-15T, see table S6) were detected, clearly supporting defective reference phenotypic results, known to be frequent especially for this drug. When excluding ethionamide, the overall sensitivity and specificity excluding uncharacterised mutations are 95.4% and 97.1%.

Table 3 in the manuscript has now been corrected to address this issue, as have supplementary tables S6–S10.

As an additional reflection of these amendments, related sentences in the abstract now read:

On 429 isolates, the assay predicted 92.2% of 2369 first- and second-line phenotypes, with a sensitivity of 95.2% and a specificity of 95.0% (95.4% and 97.1%, respectively, when excluding ethionamide). 83 out of 102 (81.4%) residual discrepancies with phenotypic results involved pyrazinamide, ethambutol and ethionamide, and low-level rifampicin or isoniazid resistance mutations, all notoriously prone to phenotypic testing variability. Only two out of 96 (2.1%) resistance phenotypes undetected by Deeplex Myc-TB had known resistance-associated mutations by WGS analysis outside Deeplex Myc-TB targets.

Also, some data in the Results section entitled "Phenotype prediction on isolates *versus* WGS and phenotypic testing" have been updated; corresponding sentences now read:

Deeplex Myc-TB drug susceptibility predictions based on these 2403 variants were compared with pDST results. In this set, 268 isolates were phenotypically resistant to at least one drug, including 156 MDR and six extensively drug-resistant isolates, resulting in 696 resistant and 1558 susceptible phenotypes. Of these 2254 phenotypes, 2080 (92.3%) were predicted by Deeplex Myc-TB with a mean sensitivity of 95.2% and a mean specificity of 95.0% (table 3 and supplementary table S6). The remaining 174 phenotypes (7.7%) could not be predicted due to the presence of mutations uncharacterised in the variant database. (…) The proportion of resistant phenotypes accurately predicted as resistant by Deeplex Myc-TB was >90% (90.7% for streptomycin to 100% for amikacin) for most individual drugs, except for pyrazinamide (85.7%) and kanamycin (88.9%, but reflecting nine isolates only) (table 3). (…) Of the 630 resistant phenotypes with Deeplex Myc-TB predictions (*i.e.* not uncharacterised), only 30 (4.8%) were predicted as susceptible due the absence of resistance-associated mutation in the Deeplex Myc-TB targets. (…) Likewise, *fabG1* L203L was the sole established resistance-associated mutation (for an isoniazid-resistant phenotype) detected by WGS outside of the assay's targets in 66 phenotypes uncharacterised by Deeplex Myc-TB. Of the 1450 susceptible phenotypes with prediction, only 72 (5.0%) were discordantly predicted as resistant. They all involved ethambutol and *embB* mutations, ethionamide and *ethA* frameshift-causing indels (mechanistically expected to cause ethionamide resistance [30]), or known low-level isoniazid/ethionamide (*inhA* S94A, *ahpC* G-48A, *fabG1* C-15T), rifampicin (*rpoB* L452P, H445N and D435Y) or streptomycin (*gidB* A138V) resistance mutations, all notoriously associated with poor phenotypic reproducibility (note S4 in the supplementary material) [3, 27, 31–33]. When excluding ethionamide, which is especially prone to such phenotypic testing errors, the overall sensitivity of Deeplex Myc-TB phenotype predictions was 95.4% and the overall specificity was 97.1%, when uncharacterised mutations were not considered (table 3). (…).

When doing so for Deeplex Myc-TB predictions, the diagnostic performance could be further improved, with proportions of uncharacterised phenotypes reduced to 0.5–2.6% for rifampicin, ethambutol and pyrazinamide, at the cost of a single incorrect prediction of a susceptible phenotype for each of these drugs (note S5 in the supplementary material and supplementary table S10).