

significantly associated with higher Ct value samples for both E-gene and S-gene RT-PCRs (appendix). To explain our observation, we hypothesised that samples with higher Ct values might have gained detection efficiency through an RNA carrier effect in pools from the other negative samples with potentially higher cellular RNA content. We are currently addressing this interesting question in a further study, as well as whether different swab collection systems affect this phenomenon. The concern raised by Lee and colleagues that this phenomenon might cause false-positive results is not supported by our data obtained with now more than 3900 pools assessed in our institution since March, 2020.

In a broader context, several distinct steps contribute to accurate test results. Major contributing factors are adequate sample collection, quality of swabs, transport media, efficient nucleic acid extraction from a sufficient amount of material, and a highly sensitive detection method. All these steps need to be optimised and validated within the laboratory to obtain optimal pool testing efficiency and accuracy.

Owing to highly diverse laboratory settings, it might be difficult to harmonise worldwide pool testing protocols for SARS-CoV-2. However, we would be grateful if national authorities could guide SARS-CoV-2 pool testing procedures as has been done for blood donor pool testing in Germany<sup>4</sup> and recently been announced by the US Food and Drug Administration for SARS-CoV-2.<sup>5</sup>

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## Macrolide consumption and resistance in *Mycoplasma genitalium*

Dorothy Machalek and colleagues published a systematic review and meta-analysis<sup>1</sup> detailing the alarming increase in the prevalence of mutations associated with macrolide resistance in *Mycoplasma genitalium*. Machalek and colleagues hypothesised that this increase and large heterogeneity in prevalence between countries could be due to differences in national protocols for sexually transmitted infection treatment or differences in the consumption of macrolides at the population level. To test this hypothesis, we used Spearman's correlation to assess the association between the country-level prevalence estimates of resistance that Machalek and colleagues generated and national macrolide consumption. The prevalence of resistance was defined as the prevalence of resistance-conferring mutations in the 23S ribosomal RNA gene at positions 2058 or 2059, in all isolates that were successfully characterised. Country-level macrolide

consumption data were obtained from IQVIA (IQVIA, Danbury, CT, USA). IQVIA uses national sample surveys to generate estimates of antimicrobial consumption, reported as defined daily doses per 1000 population per year.<sup>2</sup> We calculated the median year of data collection for the studies that contributed to resistance prevalence estimates shown by Machalek and colleagues. Our measure of antimicrobial consumption was taken from 1 year before the year used to provide resistance prevalence.<sup>3</sup> For the sensitivity analysis, we repeated the analyses using average macrolide consumption for 3 years before the median year used to provide resistance estimates.

Data on macrolide consumption were available for 18 of 21 countries with macrolide resistance estimates. Considerable differences in macrolide consumption and resistance were evident, and both positively correlated with antimicrobial consumption in the preceding 1 year ( $p=0.51$ ;  $p=0.032$ ; appendix p 1) and the preceding 3 years ( $p=0.49$ ;  $p=0.038$ ). Low prevalence of resistance was seen in Belgium (6.5%) and France (11.3%) relative to high levels of macrolide consumption. However, these prevalence estimates were based on data collected between 2003 and 2016. More recent publications,<sup>4,5</sup> not included in the systematic review, found that the prevalence of macrolide resistance was 74% in Belgium (in the general population) and 58% in France (in the pre-exposure prophylaxis cohort).

Although these results could be due an ecological inference fallacy, taken together with similar findings from other bacteria such as *Streptococcus pneumoniae* and *Neisseria gonorrhoeae*, they suggest that the promotion of macrolide stewardship in the general population and core groups should be considered as a strategy to counter the further emergence of macrolide resistance in *M genitalium*.<sup>3</sup>

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See Online for appendix

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## Why we should stop using the word leprosy

We were gratified that Cynthia Butlin and Diana Lockwood read and commented on our Personal View about the need to stop using the word leprosy.<sup>1</sup> However, we would like to correct a misunderstanding. We were not advocating for the Portuguese term hanseníase or the English equivalent, Hansen's disease, to be used globally as a substitute term. Indeed, we wrote that "This term might not be the most suitable for the rest of the world, meaning that each country might need a debate about suitably dignified terminology in their own language and in accordance with best practice." We also acknowledged clearly the issue of Hansen's unethical actions and cited WHO guidelines for naming diseases.<sup>2</sup>

Hansen's name has been adopted by people affected in several countries, including Brazil, Japan, Portugal, and the USA, to dignify and destigmatise their condition and not to honour Hansen.<sup>3</sup> As we stated in our Personal View, Hansen's disease has become "a term already used by many people affected worldwide as an empowering instrument". Our main point was that dignified language must be adopted in clinical practice to counteract the stigma and discrimination, and that this language must be chosen in consultation with people affected by the disease. We are glad that our Personal View has served to bring this principle to the forefront of one of the most stigmatising diseases in the history of medicine, and we hope that the endpoint will be to discontinue use of the term leprosy in anything other than a historical context.

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In their Correspondence,<sup>1</sup> Cynthia Butlin and Diana Lockwood put forth a passionate argument against use of the terms leprosy or Hansen's disease, and propose using mycobacterial neurodermatitis instead. Similar points were raised on the Leprosy Mailing List forum,<sup>2–4</sup> where I had expressed a few reservations, which I reiterate and add other points.

First, the term mycobacterial neurodermatitis can be easily confused with neurodermatitis, which is a group of skin diseases caused by habitual rubbing and scratching, with the prototype being lichen simplex chronicus. Indeed, a Google search for the term neurodermatitis returns results for neurodermatitis. Neurodermatitis is defined by Merriam-Webster dictionary as "a dermatosis caused by or related to psychosomatic or neurogenic factors".<sup>5</sup> Further, Lexico (a collaboration between Dictionary.com and Oxford University Press), defines neurodermatitis as simply neurodermatitis.<sup>6</sup> None of these are accurate descriptions of leprosy.

I routinely see patients with leprosy when practicing as a dermatologist in the city of Mumbai, India. Many patients with leprosy search for information about their disease on the internet. Most people in India (a country with a major disease burden from leprosy) are comfortable with Hindi language or regional languages, and not English. Perhaps in India, use of the term mycobacterial neurodermatitis would not serve a purpose, and an alternative Hindi term might have to be developed. The same could be true for other low-income and middle-income countries.

I feel that any attempt to reduce the stigma related to leprosy or skin diseases should take into account the behavioural immune system<sup>7</sup> and research which explores the impact of these psychological mechanisms on the attitudes of the public towards patients with leprosy or skin diseases.

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