which somewhat contradicts the biological hypothesis of the authors.

The paper poses many pertinent questions, as to whether there is a strong link in literature or a potential research question as to whether atopy is associated with a chronic bacterial disease like leprosy. If so, this can have therapeutic repercussions. This raises the fundamental conundrum as to whether we start screening all our atopy cases for leprosy or vice versa. The practical connotations of this study need a valid, statistical, and evidence-backed backbone as the suggestion of atopy with leprosy is fraught with confounders.

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Author's reply

Dear Editor,

We appreciate the opportunity to respond to the letter to the editor[1] regarding some critical observations about the methodology and the inferences drawn from our results.[2]

First, the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire is an accepted and validated instrument worldwide and the scientific literature has shown high sensitivity, specificity and positive predictive value for the epidemiological diagnosis of allergic diseases, and it is known for its good reproducibility and validity in previous studies and its reliability in telephone interviews.[3,4] The great advantage of ISAAC is that it allows the comparison of data between studies from around the world. Although initially designed for children and adolescents, it is possible to use ISAAC for adults as well.[3–5] Nevertheless, we know that this questionnaire evaluates symptoms related to allergic diseases and that it is not possible to be certain that all patients are truly atopic (allergic), and classifying allergic diseases based solely on questionnaire responses may not fully capture the complexity and heterogeneity of these conditions that are known to have various genotypes and phenotypes. To accurately characterise the atopic state, it would be necessary to confirm it with laboratory data, such as immunoglobulin E (IgE) levels. Therefore, we acknowledge the limitation of our study regarding the confirmation of the atopic status of the patients, since it is epidemiological in nature. We encourage new studies on the association between atopic diseases and leprosy that include laboratory data.

Regarding the sample size, it is important to note that we worked with a convenience sample of patients treated at the University Hospital of the Federal University of Sergipe. The number of leprosy patients is limited, even in an endemic

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area. Regardless, we obtained a significant and substantial sample that allowed us to make inferences based on our findings. About the relationship between the number of cases and controls, we acknowledged this as a limitation of our study and justified it due to the difficulty in recruiting household contacts who have prolonged contact with the cases (leprosy patients) and are not genetically related to them. Recognising that this is one of the strengths of this study in comparison with other studies in the literature, it is important to remember that leprosy is a disease that carries a significant negative stigma throughout history, and many patients and their families do not want to disclose that they have this disease, affecting the recruitment of controls.

Finally, we recognise the complexity of the immune response present in leprosy in its various clinical forms and the evolution in identifying new types of immune responses involved in the disease (Th9, Th17, Th22 and Treg). However, the cited study itself states that despite the new types of immune responses involved, the classical immunological paradigm of the interaction between Th1 and Th2 lymphocytes exists and an immunoregulation between them is still recognised.[6]

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