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**Authors' response**

Sir,

Thank you for your interest in the article titled "Effect of vitamin D deficiency on the metabolic

profile of women with polycystic ovary syndrome" by Tandon *et al*¹, which was recently published in IJMR. I appreciate the thoughtful remarks and would like to provide further clarification on the discussed limitations.

It is important to note that the study's main objective was to evaluate the effect of low levels vitamin D deficiency on metabolic parameters, without claiming a causative role in the development of polycystic ovarian syndrome (PCOS). Vitamin D deficiency as rightly pointed out is quite prevalent in India and is commonly found in both healthy individuals as well as those with various diseases². While it may not necessarily be symptomatic or associated with specific conditions, investigating its potential role in PCOS is still relevant as insulin resistance is the cornerstone for the pathophysiology of PCOS and it is postulated that vitamin D deficiency affects insulin resistance^{3,4}.

The primary concern raised is the absence of a control group, both healthy and diseased, matched for age and sex, which limits the significance and interpretation of the study findings. As already mentioned, we acknowledge the importance of having a control group for better understanding the correlation between vitamin D deficiency and high-density lipoprotein (HDL) levels. In future studies, including such control groups will allow for a comprehensive evaluation of the association between vitamin D deficiency, metabolic parameters, and PCOS.

The reference to a bidirectional two-sample Mendelian randomization (MR) study is informative⁵. However, these studies may not capture the full complexity of the relationship between vitamin D deficiency and PCOS due to the multifactorial nature of this disorder. The presence of conflicting results in previous studies that either support or refute the association between vitamin D deficiency and PCOS further emphasizes the complexity of the condition.

The concern regarding the lack of measurement for calcium levels is valid. Given the role of vitamin D in regulating calcium homeostasis, assessing calcium levels in relation to vitamin D deficiency would provide valuable insights into the potential implications on calcium metabolism in PCOS patients.

Lastly, the manifestation of symptoms related to vitamin D deficiency in women with PCOS is an important aspect to consider. PCOS being a heterogeneous disorder symptoms such as tiredness, exhaustion, mood swings, increased susceptibility to infections, and hair loss would not be specific

to just vitamin D deficiency alone but could also be present due to PCOS alone given the psychosocial implications of the disorder. However, we admit that in this retrospective data set, we did not analyze these symptoms even if present to correlate it with symptoms of vitamin D deficiency.

In conclusion, we appreciate the thoughtful discussion on the limitations of the study. Addressing these issues in future research endeavors would strengthen the conclusions and enhance our understanding of the relationship between vitamin D deficiency and PCOS. While the findings of the study suggest an association between vitamin D deficiency and HDL levels, as mentioned further studies are required to understand the molecular mechanism of this association.

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