populations is required for the precise determination of the additional causal polymorphisms present in patients with RA or SLE.

In conclusion, we confirm that *TNFAIP3* is a genetic risk factor for the development of both SLE and RA in the Japanese population. Although the nonsynonymous SNP rs2230926 is a strong causal variant candidate in this region, a search for additional causal variants in *TNFAIP3* is required.

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AUTHOR CONTRIBUTIONS

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. Kochi had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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