REPLY

Autoimmune cardiac channelopathies: the heart of the matter

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We thank Jin Li and Ange Maguy for their Correspondence (Autoimmune channelopathies: questions remain. *Nat. Rev. Cardiol.* <http://dx.doi.org/10.1038/nrcardio.2017.110>; 2017)¹ on our Review (Lazzerini, P. E. *et al.* Autoimmune channelopathies as a novel mechanism in cardiac arrhythmias. *Nat. Rev. Cardiol.* <http://dx.doi.org/10.1038/nrcardio.2017.61>; 2017)².

Li and Maguy state that the term “autoimmune cardiac channelopathies” was first introduced by Li *et al.* in 2013 (REF. 3). However, we were unable to identify the specific term “autoimmune cardiac channelopathies” in this article³. Conversely, the general term “autoimmune channelopathies” mentioned by Li *et al.* in their paper³ had been well recognized in the field of neuroscience since 2005 (REF. 4). In addition, searches on Google Scholar, PubMed, and

Scopus indicate that the specific term “autoimmune cardiac channelopathies” had not been used before our Review article in *Nature Reviews Cardiology*².

We do not believe that the Figures in our Review are misleading or overemphasize the knowledge in the field. We agree that the use of whole sera containing anti-KCNQ1 autoantibodies³ or anti- $\text{Na}_v1.5$ autoantibodies⁵ to demonstrate interaction with I_{Ks} and I_{Na} , respectively, is not evidence of direct causality in the absence of the use of affinity-purified antibodies. Nevertheless, control sera negative for anti-KCNQ1 autoantibodies and anti- $\text{Na}_v1.5$ autoantibodies was used and showed no electrophysiological effect on I_{Ks} or I_{Na} , respectively, in these studies^{3,5}. Our use of schematic representations of autoantibodies acting on ion channels is intended to communicate the

proposed mechanism of action simply. We hope that our Reply addresses the concerns about misunderstanding expressed by Li and Maguy.

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Competing interests statement

The authors declare no competing interests.