

Letter to the Editor

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Letter by Villa Abrille et al Regarding Article, “Hyperactive Adverse Mechanical Stress Responses in Dystrophic Heart Are Coupled to Transient Receptor Potential Canonical 6 and Blocked by cGMP-Protein Kinase G Modulation”

To the Editor:

The recent article by Seo et al,¹ published in *Circulation Research*, presented many interesting aspects of the myocardium from mice lacking transient receptor potential canonical channels TRPC3 or TRPC6, from dystrophic animals, and their interaction with cGMP. These authors measured the muscle response to stretch, widely known as slow force response (SFR).

We would like to comment only on one aspect of this study that, although it might sound trivial at first glance, it unnecessarily confuses a well-established mechanical response. The authors arbitrarily changed the widely accepted denomination of SFR^{2–14} by the term stress-stimulated contractility without any explanation to justify the change.

The SFR or Anrep effect was described by Glen von Anrep in 1912¹⁵ in a whole heart preparation and later on found in isolated cardiac preparations by Parmley and Chuck,¹⁶ Lakatta and Jewell,¹⁷ who called this mechanism slow increase in develop tension, and a few years later (1982) by Allen and Kurihara¹⁸ who in addition demonstrated that this slow phase of tension increase after a change in length was because a progressive increase in the calcium transient. The scientific community has accepted the term SFR to identify this powerful intrinsic heart mechanism to adapt cardiac output to changes in hemodynamic conditions, mechanism that is being exhaustively analyzed because of the similarity of its intracellular signaling pathway to that leading to myocardial hypertrophy.¹⁹

We are sure that the authors from this prestigious University where we all 3 spend unforgettable times doing research can be more creative than changing the name to the SFR.

Disclosures

None.

María Celeste Villa-Abrille
Néstor Gustavo Pérez
Horacio Eugenio Cingolani

*Centro de Investigaciones Cardiovasculares
Facultad de Ciencias Médicas de La Plata
UNLP-CONICET, Argentina*

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(*Circ Res*. 2015;116:e11. DOI: 10.1161/CIRCRESAHA.114.305543.)

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DOI: 10.1161/CIRCRESAHA.114.305543