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Roles of I(f) and Intracellular Ca^{2+} Release in Spontaneous Activity of Ventricular Cardiomyocytes During Murine Embryonic Development

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Wang et al. and the Editor were informed that findings presented in the above paper had originally been published in two papers from 2008 (Rapila et al. and Korhonen et al.). These earlier papers characterized the pacemaking mechanisms in detail (Rapila et al.) and based on that data built a mathematical model recapitulating these mechanisms (Korhonen et al.). Further, it was mentioned that data presented by Wang et al. overlap data presented in these papers, and some of the experiments shown in Rapila et al. had been duplicated with the same conclusions without citing the original papers.

Wang et al. reviewed these comments, and agreed that the publications by Rapila et al. and Korhonen et al. in 2008 are highly related to their own work, and should be cited in an erratum. Further, Wang et al. clarified the findings of Rapila et al., Korhonen et al., and how those findings differed from that presented by Wang et al.

Rapila et al. found that voltage-gated calcium channels and Ca2+ release from intracellular stores coexisted and operated in pacemaking in mouse cardiomyocytes during embryonic days 9–11. They further found that both Ryanodine (Ry)- and inositol-3-phosphate (IP3) receptors triggered intracellular Ca2+ release, activated Na+/Ca2+ exchanger (NCX) and thus regulated beating frequency. With mathematical modeling, Korhonen et al. presented how Ca2+ releases from the sarcoplasmic reticulum (SR) triggered APs via NCX and AP activated voltage-activated Ca2+ intrusion to the cell. Wang et al. had the similar observation that Ca2+ releases from SR via NCX determined the occurrence of APs, and still had novel findings. Wang et al. focused more on the developmental changes of the pacemaking mechanism in embryonic ventricular cells of early and late stages. Their data indicated that RyRs and IP3Rs (early stage, embryonic days 10–12.5) or RyRs (late stage, embryonic days 16.5–18.5)-mediated ICR triggered APs by activating NCX, whereas I(f) only regulated the firing rate.

Wang et al. have revised the second paragraph of the introduction of their publication to include the works by Rapila et al. and Korhonen et al.; the corrected material and references are shown below.

In embryonic ventricular cells, I(f) was found to be relevant to spontaneous electrical activity by an indirect evidence that loss of pacemaker potency is in association with a loss of I(f) current [Yasui et al., 2001; Lakatta and DiFrancesco, 2009]. In recent years, Ca2+ cycling-driven events, referred to as an intracellular "Ca2+ clock," have provided new insight into the mechanisms that drive pacemaker function and control heart rate. It is suggested that the periodic oscillations of local intracellular Ca2+ originated from sarcoplasmic reticulum is critical for the initiation of spontaneous activity of cardiomyocytes during early murine cardiogenesis and adult rabbit sinoatrial nodal cells [Viatchenko-Karpinski et al., 1999; Sasse et al., 2007; Rapila et al., 2008; Korhonen et al., 2008]. Ryanodine receptors (RyRs) and inositol triphosphate receptors (IP3Rs)-mediated ICR activated the Na–Ca exchanger current (INa/Ca), and subsequently induced membrane potential oscillations, which repeatedly draw membrane potentials toward threshold to fire cardiomyocytes of embryonic Day 9.5 [Sasse et al., 2007; Rapila et al., 2008; Korhonen et al., 2008]. Mery et al. [2005] reported that intracellular Ca2+ oscillations supported by IP3-sensitive stores constituted a pacemaking mechanism in early cardiac development using a-MHC positive cardiac cell derived from embryonic stem cells. In adult sinoatrial nodal cells, CAMP/PKA-dependent local ICR through RyRs permited the occurrence of action potentials (APs) and determines its beating rate [Bogdanov et al., 2001; Vinogradova et al., 2006].

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