

Effects of Perinatal Deltamethrin Exposure on Electrophysiological Properties of Embryonic Ventricular Cardiomyocyte

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Citation: KIBU Conference (2017). Innovative Research and Knowledge for Global Competitiveness and Sustainable Development. Proceedings of 2nd Interdisciplinary International Scientific Conference 14 – 15 June 2017. Kibabii University Main campus, Bungoma Kenya ISBN: 978-9966-59-011-4

Abstract

Pyrethroid insecticides are among of the most commonly used residential and agricultural insecticides. Based on the increased use of pyrethroids and recent studies showing that pregnant women and children are exposed to pyrethroids, there are concerns over the potential for developmental cardiotoxicity and other abnormalities. However, there have been relatively few studies on the developmental cardiotoxicity of pyrethroids. The purpose of this study was to investigate whether perinatal deltamethrin exposure altered mice embryonic cardiac electrophysiology. Pregnant mice were administered 0 or 3 mg/kg of DM by gavage daily from gestational day (gd) 10.5 to gd 17.5. Whole cell patch-clamp technique was used in electrophysiological study, and real time RT-PCR was applied to analyze the molecular changes for the electrophysiological properties. DM exposure resulted in increased mortality of pregnant mice and decreased viability of embryos. Moreover, DM slowed the maximum depolarization velocity (V_{max}), prolonged the action potential duration (APD) and depolarized the maximum diastolic potential (MDP) of embryonic cardiomyocytes. Additionally, perinatal DM exposure decreased the mRNA expression of N^+ channel regulatory subunit $Nav\beta 1$, inwardly rectifier K^+ channel subunit $Kir2.1$, and delayed rectifier K^+ channel subunit $MERG$ while the L-type Ca^{2+} channel subunit, $Cav1.2$ expression was increased. On the contrary, DM administration did not significantly alter the β -adrenergic or muscarinic receptor activities on embryonic cardiomyocytes. In conclusion, developmental DM exposure altered mRNA expression of embryonic cardiac ion channels therefore impacting embryonic cardiac

electrophysiological properties. This highlights the need to understand the persistent effects of pyrethroid exposure on cardiac function during development due to potential for cardiac arrhythmogenicity.

Keywords: *Pyrethroid, Deltamethrin, Embryonic cardiomyocytes, Action potential, Developmental exposure.*