ABSTRACT

Increased use of pyrethroids and the exposure to pyrethroids for pregnant women and children have raised the concerns over the potential effect of pyrethroids on developmental cardiotoxicity and other abnormalities. The purpose of this study was to investigate whether long term perinatal deltamethrin exposure altered embryonic cardiac electrophysiology in mice. Pregnant mice were administered with 0 or 3 mg/kg of deltamethrin 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. Deltamethrin exposure resulted in increased mortality of pregnant mice and decreased viability of embryos. Moreover, deltamethrin slowed the maximum depolarization velocity (Vmax), prolonged the action potential duration (APD) and depolarized the maximum diastolic potential (MDP) of embryonic cardiomyocytes. Additionally, perinatal deltamethrin exposure decreased the mRNA expression of Na^+ channel regulatory subunit Navβ1, inward 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, deltamethrin administration did not significantly alter the regulation of β-adrenergic or muscarinic receptor on embryonic cardiomyocytes. In conclusion, deltamethrin exposure at perinatal stage significantly alters mRNA expression of embryonic cardiac ion channels and therefore influences embryonic cardiac electrophysiological properties. This highlights the need to understand the persistent effects of pyrethroid exposure on cardiac function during embryonic development due to potential for cardiac arrhythmogenicity.

Keywords: action potential; deltamethrin; developmental cardiotoxicity; embryonic cardiomyocytes; pyrethroid.