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Letter to Editor

hERG Gene: Towards the Pathophysiology Beyond Long QT

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hERG is human-ether-a-go-go-gene which codes for potassium channel. Mutation of the hERG gene decreases the efflux of potassium ions which lead to hyperpolarization of the cardiac cell causes prolonged QT syndrome [1]. Not only it causes QT prolongation, it sometimes increases effluxing more potassium ion which may lead to QT shortening. Beyond causing cardiac arrhythmia the expression of hERG gene in different cells of our body may introduce other diseases. It's overexpression cause disturbance in the cell cycle and boosts the expression biomarker for cancer. In MCF-7(breast cancer cell), the expression of hERG gene on this cell may develop cancer [2]. Experiments in gastric and ovarian carcinoma highlighted the overexpression of hERG gene in the S phase, whereas while interfering with G2 or M phase it produces endometrial cancer [3]. hERG expression with other biomarkers (TNFRI or CXCR4) produces neurological disorder. In pancreatic alpha and beta cells, the expression of hERG gene has been reported. Pharmacological antagonism of the channel in these cells appears to enhance glucose and arginine-induced insulin secretion and repress glucagon secretion under low glucose conditions by modulating transmembrane calcium fluxes. In the case of hepatic cells, the overexpression of hERG gene on the HepG2 cell cause E4031 inhibition, which leads to cell proliferation and fibrosis. Moreover, overexpression of hERG gene on LDL receptor cause decreases the metabolism lead to hyperlipidemia [4]. Thus, here we considered these other functions of hERG, particularly their impact on diseases beyond cardiac arrhythmia.

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