**PHENYTOIN INHIBITS THE EXPRESSION OF CARDIAC CAV1.2 CHANNELS VIA DISRUPTING SYNTHESIS AND TRAFFICKING**

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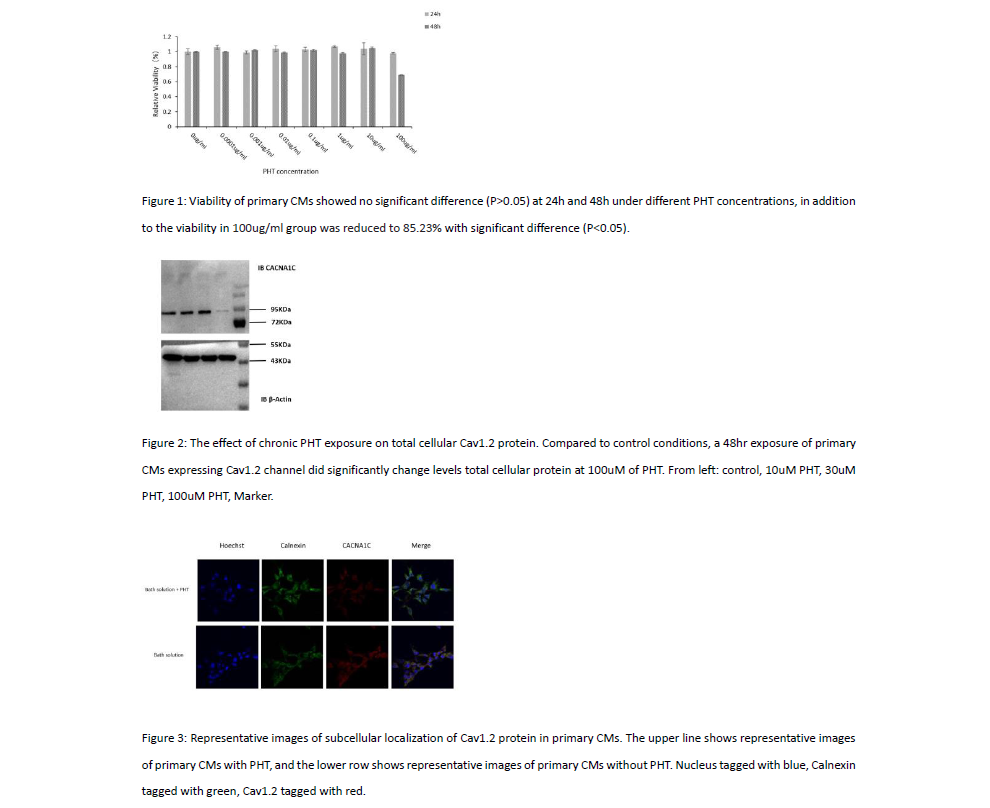
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**Objective:** Antiepileptic drug phenytoin (PHT) is related to pro-arrhythmic events. L-type calcium (Cav1.2) channel significantly contributes to normal cardiac rhythm. The potential of PHT on Cav1.2 remains unclear. This study aimed to evaluate the potential of PTH on the synthesis and trafficking of Cav1.2 channels.

**Method:** Primary cardiomyocytes (CMs) of neonatal SD rat were acutle digested by trypsin and type II collagenase and incubated exposed to PHT for 48 hours. Cell Titer-Gloassay was used to measure cell viability. Western blotting and confocal laser scanning microscopy were applied to evaluate the effects of PHT on the expression of Cav1.2 channel protein in primary CMs.

**Results:** In CellTiter-Glo assay, with increased PHT concentration, no significant difference from the actual cell viability was observed (P>0.05) except that the viability in 100ug/ml group at 48h was reduced to 85.23% (P<0.05). Western blotting and confocal laser scanning microscopy indicated significant inhibition of L-type calcium channels by PHT in primary CMs.

**Conclusion:** This study validated the potential of PHT to inhibit the expression of cardiac Cav1.2 channels for the first time. This may be responsible for its pro-arrhythmic effects.

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