**ALLICIN DISRUPTS CARDIAC CAV1.2 CHANNELS VIA TRAFFICKING**

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**Objectives:** Allicin is a potential antiarrhythmic agent. The antiarrhythmic properties of allicin are due to its blockade of different ion channels. L-type calcium (Cav1.2) channel provides a pivotal substrate for cardiac electrophysiologic activities. The mechanism of allicin on Cav1.2 remains unclear. This study aimed to evaluate the potential of allicin on the synthesis and trafficking of Cav1.2 channels.

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

**Results:** CellTiter-Glo assay indicated that with increased allicin concentration, no significant difference of apoptotic toxicity from the actual cell viability was observed (P> 0.05) in all groups, except that the viability in 0.001ug/ml and 0.01ug/ml groups at 24h was increased to 137.37% and 135.96% with significant difference (P < 0.05). Western blot showed no obvious inhibition of allicin on the synthesis of Cav1.2 and confocal laser scanning microscopy revealed trafficking dysfunction of Cav1.2 channels by allicin in primary CMs.

**Conclusions:**This study firstly validated that allicin inhibits cardiac Cav1.2 channels by disrupting Cav1.2 protein trafficking, which may be responsible for its antiarrhythmic benefits.

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