Amyloid peptide regulates calcium homoeostasis and arrhythmogenesis in pulmonary vein cardiomyocytes.

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BACKGROUND: Amyloid peptides modulate cardiac calcium homoeostasis and play an important role in the pathophysiology of atrial fibrillation. Pulmonary veins (PVs) are critical in the genesis of atrial fibrillation and contain abundant amyloid peptides. Therefore, the purpose of this study is to investigate whether amyloid peptides may change the PV electrical activity through regulating calcium homoeostasis.

METHODS AND RESULTS: The channel and calcium-handling protein expressions, intracellular calcium and ionic currents were studied in isolated rabbit PV cardiomyocytes in the presence and absence (control) of beta-amyloid (Aβ(25-35)) for 4-6 h, using Western blot analysis, indo-1 fluorimetric ratio and whole-cell patch clamp techniques. Aβ(25-35) decreased the expressions of Ca(V) 1.2, total or Ser16-phosphorylated phospholamban (p-PLB), p-PLB/PLB ratio, sodium/calcium exchanger, but did not change ryanodine receptor, sarcoplasmic reticulum (SR) ATPase and K(+) channel proteins (Kir2.1, Kir2.3, Kv1.4, Kv1.5 and Kv4.2). Aβ(25-35) -treated cardiomyocytes had smaller calcium transient, SR calcium store, L-type calcium current and sodium/calcium exchanger current than control cardiomyocytes. Moreover, Aβ(25-35) -treated cardiomyocytes (n = 20) had shorter 90% of the action potential duration (82 ± 3 vs. 93 ± 5 ms, P < 0.05) than control cardiomyocytes (n = 16).

CONCLUSION: Aβ(25-35) has direct electrophysiological effects on PV cardiomyocytes.


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