**Impairment of TRPC1-BK complex in diabetic rat coronary artery**

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**Introduction**: This study aimed to investigate the dysregulation of the transient receptor potential canonical channel 1 (TRPC1) and the large conductance Ca2+-activated K+ (BK) channels complex (TRPC1-BK complex) in diabetic coronary vasculopathy.

**Methods**: DM was induced with streptozotocin (STZ) in male Sprague-Dawley rats. Diabetic and control coronary arteries were respectively achieved 2 months after STZ or vehicle treatment. Using quantitative real-time PCR, immunoblotting, fluorescent assay, patch clamp techniques and vascular tension measurements, we investigated TRPC1 channel and BK channel activities and coronary vasoreactivity in both control and diabetic rats.

**Result**: We confirmed TRPC1 channel and BK channel were physically associated in the coronary artery SMCs of both control and diabetic rats. The expression of TRPC1 channel was significantly increased while BK-β1 subunit was decreased without changing of BK-α subunits, in diabetic coronary artery SMCs. Cytosolic calcium concentrations were higher in diabetic coronary artery SMCs than control group, and pre-incubation with TRPC1 channel blocker SKF96365 decreased cytosolic calcium concentrations in both groups. BK channel current densities were reduced (P<0.05) and the constriction of coronary artery induced by BK inhibitor iberiotoxin was weakened in diabetic groups than control group. The dilatation of coronary artery was stronger in diabetic group than control group in presence of SKF96365.

**Conclusion**: TRPC1 channels and BK channels are co-localized on diabetic coronary artery SMCs. The imbalance of TRPC1-BK complex was due to increased TRPC1 channel expression and decreased BK channel expression in diabetic coronary artery SMCs, in turn contributing to diabetic rat coronary artery dysfunction.