Periostin prolongs action potential duration thorough inhibiting voltage-dependent Na^+ channel activity in rat ventricular myocytes

Keisuke Imoto, Muneyoshi Okada, Hideyuki Yamawaki

Lab. Vet Pharmacol., Sch. Vet. Med., Kitasato Univ.

Periostin (POSTN), a matricellular protein is related to structural remodeling of pathological heart. However, it remains to be clarified whether POSTN mediates electrical disorders of heart. Thus, we investigated the effects of POSTN on electrophysiological properties in rat ventricular myocytes. Male Wistar rats were injected with recombinant rat POSTN (64 mg/kg, i.v.) for 24 h. After electrocardiogram was recorded, ventricular myocytes were isolated. Action potential (AP) and voltage-dependent Na^+ channel (Nav) current (I_{Na}) in the isolated ventricular myocytes or neonatal rat ventricular myocytes (NRVMs) were measured by a whole cell patch-clamp technique. The QRS duration was increased in the POSTN-injected rats. The duration and time to peak of AP were prolonged with a decrease in peak amplitude of AP and I_{Na} in the isolated ventricular myocytes from POSTN-injected rats. POSTN (1 μg/ml, 24 h) suppressed the peak amplitude of I_{Na} in NRVMs. The present study for the first time demonstrated that POSTN prolongs the time to peak of AP through inhibiting Nav activity in rat ventricular myocytes. It is suggested that POSTN might cause arrhythmia via prolonging AP duration in ventricular myocytes.