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Ellagic acid reduces L-type Ca2+ current and induces negative inotropy through NO-GC-cGMP pathway in rat ventricular myocytes

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Recent evidences have shown that phenolic structures can exert many biological functions. Ellagic acid (EA), a phenolic compound, has been suggested to have cardioprotective effects. In this study we aimed to investigate the effect of EA on cardiac Ca2+ currents and contractility in rat ventricular myocytes and to elucidate the underlying mechanisms of these changes.

All records measured from the freshly isolated ventricular myocytes of rat heart at 36 ± 1 °C by using whole-cell configuration of voltage clamp. Cell shortening was measured by detecting the length of edges with video-based system at 1 Hz frequency of field stimulation. We found that EA dose dependently reduced Ca2+ currents with EC50= 23 nM. EA decreased voltage dependent L-type Ca2+ current density (ICaL) but it didn't affect the inactivation and reactivation parameters. Inhibition of adenylate cyclase (AC) with SQ-22536 (10 μM) and using probucol (antioxidant, 5 μM) had no effect on EA modulation of ICaL. Interestingly, blockage of nitric oxide synthase (NOS) with L-NAME (500 μM) and guanylate cyclase (GC) with ODQ (1 μM) abolished inhibitory effect of EA on ICaL. Moreover, EA dose dependently blunted fractional shorthening of ventricular myocytes.

In conclusion, EA affects ionic and mechanical properties of ventricular myocytes starting at nanomolar concentrations. Our findings indicated that EA suppresses ICaL and exerts negative inotropic effects through activation of NOS-GC-cGMP pathway. Accordingly, EA may be useful in pathophysiological conditions whereby these effects might be favorable such as hypertension and ischemic heart diseases.

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