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Effects of Isoflavones on the Release of Inflammatory Mediators by Cigarette Smoke in Airway Epithelial Cells

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Introduction. Chronic obstructive pulmonary disease (COPD) is characterized by airway inflammation, and is associated with cigarette smoking. The morbidity and mortality of COPD patients remain high, despite the currently available pharmacological treatments.
Aims. The present study investigated the potential of isoflavones in reducing airway inflammation.
Methods. Inflammation was induced in human bronchial epithelial BEAS-2B cells by exposure to cigarette smoke medium (CSM). These cells were incubated with or without different isoflavones [daidzein, genistein, genistin, glycetin and puerarin], the anti-inflammatory glucocorticoid, dexamethasone, or the extracellular signal-regulated kinase (ERK) inhibitor, U0126. The amounts of the inflammatory mediators, interleukin (IL)-8 and monocyte-chemotactic protein-1 (MCP-1), in the culture medium was measured with enzyme immunoassays.
Results. CSM (4%) stimulated the release of IL-8 and MCP-1 by BEAS-2B cells after 24 hours. Dexamethasone (1 μmol/L) and U0126 (10 μmol/L) inhibited the cigarette smoke-induced release of these inflammatory mediators. Among the five isoflavones tested, only genistein, at 3 and 10 μmol/L, inhibited the release of IL-8 from BEAS-2B cells. Genistein (10 μmol/L) also reduced the MCP-1 release from the cells.
Discussion. The findings, therefore, suggest that genistein has an anti-inflammatory effect on airway epithelial cells. In view of the involvement of ERK in cigarette smoke-induced inflammation, it is possible that genistein may cause inhibition of this enzyme to reduce airway inflammation.

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Investigation of the Antiarrhythmic Mechanism of the Multi-herbal Medicine Xing Su Ning

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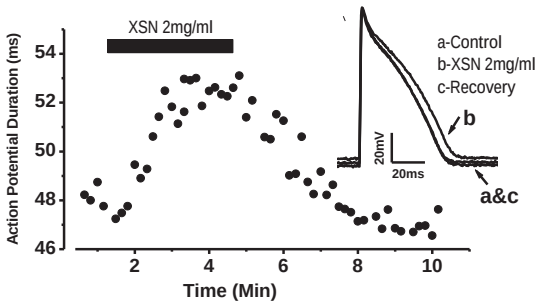
Introduction. Xing Su Ning (XSN) is a multi-herbal Chinese medicine produced by Momentum Pharmaceutical Co. Ltd., which is sold in China since 2005 for treating cardiac ventricular arrhythmia, especially arrhythmias induced by cardiac ischemia and viral myocarditis.
Aims. To discover the cellular electrophysiological mechanism of the actions of XSN in treating cardiac arrhythmia.
Methods. Whole-cell patch-clamp techniques (1) were used to record action potentials and whole cell current in isolated adult rat ventricular myocytes. The myocytes were continuously perfused with physiological solution without or with XSN.
Results. XSN at 2mg/ml significantly prolonged the action potential Duration (APD) as shown in the Figure: control was 45.4ms±4.9 and with XSN 2mg/ml was 52.2ms±4.5 (P<0.01, n=7). At this concentration XSN did not have significant effect on the resting potential or the amplitude of the action potential. The effect of XSN is reversible upon the washout of the medicine.



Discussion. XSN Prolongs APD, an action that increases the effective refractory period which suppress tachyarrhythmias caused by reentry mechanisms. XSN displays the property of Class III antiarrhythmic drugs, such as amiodarone, without adverse reactions. Further studies on the effect of XSN at various concentrations on action potential and ionic channels, i.e. potassium channels, will be carried out.

(1) Ma Y-L et al (2006) European Journal of Pharmacology. 545:87-92