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Polydatin reduces cardiotoxicity of tyrosine kinase inhibitor sunitinib by decreasing pro-oxidative stress, pro-inflammatory cytokines and NLRP3 inflammasome expression.

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Abstract

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Background: Polydatin has anticancer and anti-inflammatory properties, however its putative cardioprotective effects against anticancer therapies. Sunitinib, a receptor tyrosine kinase inhibitor, prolongs survival in patients with metastatic renal cell carcinoma and gastrointestinal stromal tumors, however a dose related cardiotoxicity was well described. The aim of this study was to investigate the cardioprotective effects of polydatin on the reduction of cytokines and growth factors of polydatin resulting in putative cardioprotection.

Methods: Human fetal cardiomyocytes were untreated (control) or treated for 48 h with polydatin (50, 100, 200 and 400 μ M) or sunitinib (5, 10, 25 and 50 μ M) alone or combined to polydatin. After 48 h of incubation period, we performed the following tests: determination of cell viability, mitochondrial dehydrogenase activity, study of lipid peroxidation (quantifying cellular malondialdehyde and 4-hydroxynonenal), intracellular Ca^{2+} homeostasis. Moreover, pro-inflammatory cytokines were measured (activation of NLRP3 inflammasome; expression of TLR4/MyD88; mTORC1/2 phosphorylation; transcriptional activation of p65/NF- κ B and secretion of cytokines involved in cardiotoxicity). **Results:** Exposure of adult cardiomyocytes to polydatin combined to plasma-

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