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

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Abstract

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Background: : Polydatin has anticancer and anti-inflammatory properties, howeve on its putative cardioprotective effects against anticancer therapies. Sunitinib, a rec targeted tyrosine kinases inhibitor, prolongs survival in patients with metastatic ren gastrointestinal stromal tumors, however a dose related cardiotoxicity was well des on the reduction of cytokines and growth factors of polydatin resulting in putative **Methods:** Human fetal cardiomyocytes were untreated (control) or treated for 48 h (50,100,200 and 400 μM) or sunitinib (5,10,25 and 50 μM) alone or combined to po incubation period, we performed the following tests: determination of cell viability, mitochondrial dehydrogenase activity, study of lipid peroxidation (quantifying cellu and 4-hydroxynonenal), intracellular Ca2+ homeostasis. Moreover, pro-inflammato performed (activation of NLRP3 inflammasome; expression of TLR4/MyD88; mTOR transcriptional activation of p65/NF-κB and secretion of cytokines involved in cardi 8, 6). **Results:** Exposure of adult cardiomyocytes to polydatin combined to plasma-

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