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Acute post-ischemic GPER1 activation protects the myocardium against ischemia/reperfusion injury by reducing mitochondrial protein ubiquitination, histone acetylation and calpain10 levels and inhibition of the mPTP opening

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We recently found that acute pre-ischemic estrogen-induced cardioprotection against ischemia/reperfusion injury was mainly mediated via G protein-coupled estrogen receptor1 (GPER1) activation but not through classical estrogen receptors: alpha (ERα) and beta (ERβ). We investigated whether acute post-ischemic estrogen (PI-E2) treatment can also induce cardioprotective effect via GPER1 activation in the intact animal subjected to ischemia/reperfusion injury. Male and ovariectomized female were subjected to 35 min of the Left Anterior Descending (LAD) artery occlusion, followed by 180 min reperfusion. An E2 bolus (50 mg/kg) or PBS (same volume) was applied via the femoral vein 5 min before reperfusion and a GPER1 antagonist, G15, was given 10 min before E2. Myocardial infarct size was assessed by TTC staining method. Mitochondrial $Ca2^{+}$ retention capacity (CRC) required to induce Mitochondrial Permeability Transition Pore (mPTP) opening was assessed after 10 min reperfusion. Expression of ubiquitinated; acetylated; calpains1 and 10 proteins were measured by Western Blot in mitochondrial fractions. We found that PI-E2 treatment reduced myocardial infarct size and increased mitochondrial CRC. PI-E2 treatment reduced mitochondrial protein acetylation, ubiquitination and also calpain10 levels in mitochondrial fractions as compared to control, respectively. Interestingly, all these of E2 effects were abolished by addition of G15. Acute PI-E2 treatment induces cardioprotection against ischemia/reperfusion injury via GPER1. PI-E2 effects through GPER1 involve the reduction mitochondrial proteins acetylation, ubiquitination, and calpain10 levels and is associated with the inhibition of the mPTP opening.

Biography

Jean C Bopassa has completed his PhD at the age of 31 years from Claude Bernard University, Lyon1, France and postdoctoral studies from Harvard University and University of California at Los Angeles. Currently, he is an Assistant Professor in the Department of Physiology, in the school of medicine at UTHSCSA. He has published more than 17 papers in reputed journals and has been serving as an editorial board member of several reputed journal.

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