Cyclooxygenase-2-Derived Prostacyclin Protective Role on Endotoxin-Induced Mouse Cardiomyocyte Mortality

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Résumé en anglais	Cardiovascular dysfunction characterizes septic shock, inducing multiple organ failure and a high mortality rate. In the heart, it has been shown an up-regulation of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2) expressions with subsequent overproduction of nitric oxide (NO) and eicosanoids. This study is focused on the links between these products of inflammation and cell loss of mouse cardiomyocytes during treatment by the Salmonella typhimurium lipopolysaccharide (LPS) in presence or in absence of NOS or COX inhibitors. LPS induced RelA/NF-κB p65 activation, iNOS and COX-2 up-regulations, resulting in NO and prostacyclin releases. These effects were reversed by the NO-synthase inhibitor and increased by the specific COX-2 inhibitor. Immunostainings with FITC-conjugated anti-Annexin-V and propidium iodide and caspase 3/7 activity assay showed that cardiomyocyte necrosis was inhibited by L-NA during LPS treatment challenge, while apoptosis was induced in presence of both LPS and NS-398. No effect on LPS cellular injury was observed using the specific cyclooxygenase-1 (COX-1) inhibitor, SC-560. These findings strongly support the hypothesis of a link between iNOS-dependent NO overproduction and LPS-induced cell loss with a selective protective role allotted to COX-2 and deriving prostacyclins.
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