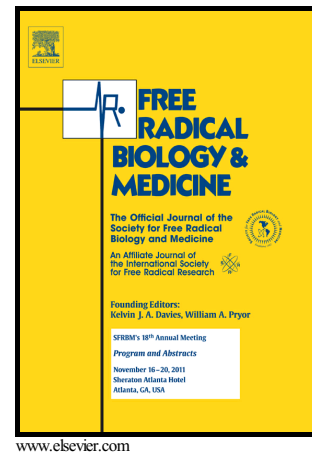


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The proinflammatory effects of macrophage-derived NADPH oxidase function in autoimmune diabetes

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Abstract

Type 1 diabetes (T1D) is an autoimmune disease culminating in the destruction of insulin-producing pancreatic β -cells. While ultimately a T cell-mediated disease, macrophages play an indispensable role in disease initiation and progression. Infiltrating macrophages generate an inflammatory environment by releasing NADPH oxidase-derived superoxide and proinflammatory cytokines. The synthesis of reactive oxygen species (ROS) is acknowledged as putative factors contributing to autoimmunity and β -cell damage in T1D. In addition to direct lysis, free radicals collectively participate in β -cell destruction by providing a redox-dependent third signal necessary for islet-reactive CD4 and CD8 T cell maturation and by inducing oxidative post-translational modifications of β -cell epitopes to further exacerbate autoimmune responses. This review will provide an overview of macrophage function and a synergistic cross-talk with redox biology that contributes to autoimmune dysregulation in T1D.

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