Role of constitutive nitric oxide synthases in the dynamic regulation of the autophagy response of keratinocytes upon UVB exposure

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Abstract

Ultraviolet B (UVB) radiation induces autophagy responses, which play a role in the regulation of the oncogenic processes of irradiated cells. However, the mechanism of autophagy responses post-UVB irradiation remains to be fully elucidated. Previous studies indicate that UVB radiation induces the activation and uncoupling of constitutive nitric oxide synthases (cNOS), which produce nitric oxide and peroxynitrite; both have been shown to regulate autophagy responses. In this study, the UVB-induced autophagy responses were analysed in cell line- and UVB dose-dependent manners, and the role of cNOS in UVB-induced autophagy responses was also studied. Our data showed that UVB induces both autophagosome formation and degradation, and that cNOS is involved in the regulation of autophagy responses post UVB exposure. Both nitric oxide and peroxynitrite, the two products that are produced in cells immediately after UVB exposure, could upregulate autophagy in a dose-dependent manner. Furthermore, cNOS is involved in the UVB-induced downregulation of SQSTM1/p62, a scaffold protein used as a reporter of the autophagy response. However, the cNOS-mediated reduction of SQSTM1/p62 is autophagy-independent post UVB irradiation. Our results indicated that autophagy responses post UVB exposure are a dynamic balance of autophagosome formation and degradation, with cNOS playing a role in the regulation of the balance.

Palabras clave

KeyWords Plus:SENESCENT KERATINOCYTES; IN-VITRO; EXPRESSION; RADIATION; APOPTOSIS; ACTIVATION; DAMAGE

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