

Cellular response to infrared radiation involves retrograde mitochondrial signalling

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Abstract

Infrared A radiation (IRA) is a major component of sunlight. Similar to ultraviolet (UV) B and UVA, IRA induces gene transcription. In contrast to the UV response very little is known about the IRA response. In the present study, IRA-induced expression of matrix metalloproteinase-1 (MMP-1) was found to be mediated by the formation of intracellular reactive oxygen species (ROS). Staining of IRAirradiated cells with MitoSox revealed an increase in mitochondrial superoxide anion production and treatment of fibroblasts with the mitochondrial targeted antioxidant MitoQ completely abrogated the IRA, but not the UVB or UVA1, response. ROS relevant for IRA-induced signaling originated from the mt electron transport chain, because (i) chemical inhibition of the electron transport chain prevented IRA, but not UVB or UVA1, radiation-induced MMP-1 expression, (ii) rho0 fibroblasts specifically failed to increase MMP-1 expression in response to IRA, and (iii) peroxisome proliferator-activated receptor gamma coactivator-1 (PGC-1) overexpressing fibroblasts with increased electron transport chain content were hypersensitive to IRA radiation-induced gene expression. Thus, IRA, in contrast to UV, elicits a retrograde signaling response in human skin.