

Meeting abstract

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## The circadian rhythm of primary dermal fibroblasts affects infrared-A-induced gene expression

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from 12th Joint Meeting of the Signal Transduction Society (STS). Signal Transduction: Receptors, Mediators and Genes Weimar, Germany. 29–31 October 2008

Published: 26 February 2009

*Cell Communication and Signaling* 2009, **7**(Suppl 1):A55 doi:10.1186/1478-811X-7-S1-A55

This abstract is available from: <http://www.biosignaling.com/content/7/S1/A55>

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Until now the susceptibility of human skin to detrimental effects of solar radiation (e.g. photoaging) has mostly been assigned to the type of radiation and to the skin type. We have raised the question whether the circadian rhythm plays a role in the susceptibility of human dermal fibroblasts (HDF) to IRA (760–1440 nm).

IRA accounts for one third of the solar energy reaching the earth's surface and penetrates deeply into the human skin. Previous studies of our group have shown that IRA in HDF induces a retrograde signaling cascade involving mitochondrial reactive oxygen species (ROS) and, via involvement of the MAP-kinases ERK 1/2, alters gene expression. Among the genes regulated is matrix metalloproteinase 1 (MMP-1), the dominant enzyme in terms of collagen degradation in the dermal extracellular matrix.

The hypothalamic suprachiasmatic nucleus is the centre of circadian rhythms in mammals. It is reset daily by light and regulates the circadian clocks of the peripheral tissues. Clock genes drive both rhythms. In vitro, synchronicity is lost in cell culture but synchronizers such as high serum concentrations can reinduce molecular oscillations of clock genes, resynchronizing the circadian rhythm of cultured cells.

To address the role of circadian rhythm in susceptibility towards IRA induced changes in gene expression HDFs from various donors were treated with a serum shock and

irradiated 24 h, 30 h and 36 h later with 360 J/cm<sup>2</sup> IRA under temperature controlled conditions. Realtime PCR results show an increased susceptibility to IRA peaking 30 h after serum shock compared to control cells.

Our findings for the first time show that the susceptibility of skin cells towards a part of natural sunlight is affected by the circadian rhythm. Further analysis of clock genes (PER1, BMAL1) at the time of irradiation will provide insight into the relationship between the circadian rhythm and IRA induced changes in gene expression.