JAK-STAT, 4:1–10, 2015

ISSN: 2162-3996 online
DOI: 10.1080/21623996.2015.1084084

REVIEW

Mitochondrial STAT3 and reactive oxygen species: A fulcrum of adipogenesis?

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The balance between cellular lineages can be controlled by reactive oxygen species (ROS). Cellular differentiation into adipocytes is highly dependent on the production of ROS to initiate the process through activation of multiple interlinked factors that stimulate mitotic clonal expansion and cellular maturation. The signal transducer and activator of transcription family of signaling proteins have accepted roles in adipogenesis and associated lipogenesis. Non-canonical mitochondrial localization of STAT3 and other members of the STAT family however opens up new avenues for investigation of its role in the aforementioned processes. Following recent observations of differences in mitochondrially localized serine 727 phosphorylated STAT3 (mtSTAT3-pS727) in preadipocytes and adipocytes, here, we hypothesize and speculate further on the role of mitochondrial STAT3 in adipogenesis.

KEYWORDS. adipogenesis, mitochondria, signal transducer and activator of transcription 3, reactive oxygen species, STAT3

INTRODUCTION

Adipose tissue (majorly but not solely composed of adipocytes and endothelial cells) as an endocrine organ is vitally important to homeostasis through the release of hormone cytokines like leptin, adiponectin and resistin as well as its involvement in steroid metabolism.[1,2] Imbalances in adipose tissue are largely detrimental to human health e.g. obesity.[3-5] Understanding the developmental and maintenance factors in adipocyte biology are essential.

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Received June 9, 2015; Revised August 7, 2015; Accepted August 11, 2015.
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