Cell Communication and Signaling BioMed Central

Meeting abstract

Open Access

Adipocytokines – mediators of fat tissue linking obesity and cancer

J Ratke*, B Niggemann, KS Zänker and K Lang

Address: Institute of Immunology, Witten/Herdecke University, Witten, Germany
* Corresponding author

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):A36 doi:10.1186/1478-811X-7-S1-A36

This abstract is available from: http://www.biosignaling.com/content/7/S1/A36

Obesity is a dramatically increasing public health problem worldwide. Traditionally, fat tissue was considered to be solely an energy storage depot. However, recent studies have shown that adipose tissue exerts important endocrine functions, which are predominantly mediated by a network of various soluble factors derived from adipocytes. New evidence has come to light elucidating a modulatory role of this adipocytokines in the regulation of cancer development. For example, adipocytokines such as leptin were shown to have an effect on breast cancer progression. In this study we have investigated the impact of leptin on the proliferation and migration of colon carcinoma cells. Treatment of human SW480 colon carcinoma cells with leptin resulted in a significant increase of the proliferation. In parallel, using our unique 3D cell migration assay and time-lapse video microscopy, leptin strongly stimulated the spontaneous migratory activity from 29% locomoting cells to 52%. This leptin-induced migration resulted in an activation of various transcription factors such as Stat-3 and c-Jun. Accordingly the phosphorylation of Stat-3 was accompanied by an increase of SOCS-3, its negative feedback regulator. Furthermore, using a Stat-3 specific inhibitor inhibited the leptin-induced migration. Understanding the impact of different adipocytokines on tumour migration and the underlying signal transduction mechanisms is mandatory for the future development of cancer therapy.

Acknowledgements

This work was supported by the Fritz-Bender Foundation, Munich, Germany.