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News Section
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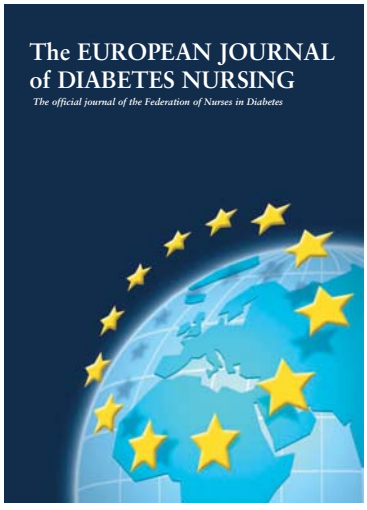
ANNOUNCEMENT

The EUROPEAN JOURNAL of DIABETES NURSING

The official journal of the Federation of European Nurses in Diabetes

FEND and the Publishers, John Wiley & Sons, are delighted to announce this new journal that is due to be launched in Munich at the FEND Annual Conference, 3–4 September 2004.

Under the Editorship of Sarah Hills, Dept. of Internal Medicine, University of Pisa, Italy, the journal will aim to embrace clinical practice, policy, research and systems of care specifically for nurses who work in diabetes across Europe. The journal will be nurse led, multidisciplinary in scope, and will publish a mixture of original research, reviews, case reports, conference reports and very importantly political comment. The journal will be distributed free of charge to all members of FEND, and to all nurses who work in diabetes, or who have an interest in diabetes across Europe, who register to receive it. The journal will also be made available to opinion leaders, academic institutions, and Ministries of Health at a National and European level.



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The Editor-in-Chief welcomes contributions to the journal of all types, whether original research, reviews, case reports, conference reports, conference notices, or comments etc. For further information and a copy of the Guidelines for Authors, please contact:

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European Foundation for the Study of Diabetes
The Research Foundation of the European Association for the Study of Diabetes

Report on an EFSD/MSD Travel Fellowship for Young Scientists 2003

Isabelle Eisele
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Type 2 diabetes mellitus is becoming a worldwide epidemic. The strongest risk factor for developing Type 2 diabetes is obesity. In obesity, white adipose tissue (WAT) mass increases dramatically. The tissue secretes a diverse range of protein factors, termed 'adipokines', some of which are implicated in the pathologies linked to obesity. It is therefore fundamental to unravel the timepoints and quantity of secreted adipokines, as well as their mechanisms of action and interactions with other factors and systems.

A new, rare and interesting tool in the study of human adipocytes is the Simpson Golabi Behmel Syndrome (SGBS) preadipocyte cell strain. It was derived by Dr.Wabitsch and colleagues at the University of Ulm in Germany, who kindly provided it to Prof. Paul Trayhurn at the University of Liverpool, U.K..

The aim of this project was to provide basic information for subsequent studies focused on determining the factors involved in regulating the production of inflammatory and acute phase response adipokines by human adipose tissue.

During my stay at Prof. Trayhurn's lab the handling of the SGBS cell strain was learned, together with some basic molecular biology techniques, and a time course study of adipocyte differentiation performed. Cells and media were harvested as well as pictures taken at different timepoints. RT-PCR was performed for semi-quantiative screening of adipokine expression during the timecourse. Amplified products were sequenced (commercially) to confirm their identity. In addition, there was also the opportunity to examine the DNA microarray technique.

Expression of leptin and adiponectin genes was detected in the SGBS adipocytes. Resistin expression could not be detected, however, strengthening the notion that resistin is playing a different role in humans than in mice (or the site of resistin gene expression is different in the two species).

PAI-1 and angiotensinogen gene expression was evident in both the preadipocytes and the adipocytes – though angiotensinogen seemed to be more highly expressed in the adipocytes. Adipsin mRNA was found in the adipocytes.

Haptoglobin mRNA was exclusively present in the differentiated adipocytes and these data coincide with those of our microarray studies and with those that I found in dog WAT. IL-6 and MT-2A mRNA were found in both the preadipocytes and adipocytes.

No special pattern for TNF-α expression could be observed; we concluded that the preadipocytes and the adipocytes are able to express TNF-α but that this was dependent on stress factors. The C-reactive protein gene was expressed by the adipocytes, but at low levels.

Overall, the SGBS cell line expresses several adipokine genes, including those encoding classical cytokines and acute phase proteins. With these data we affirm that the SGBS cell strain is a good tool for further investigations into the biology of human preadipocytes and adipocytes. It can provide fundamental information for subsequent studies aimed at determining the factors involved in regulating the production of inflammatory adipokines in *vitro* and in *vivo*. This hopefully will help in understanding the role of adipocytes in the pathogenesis of the inflammatory response in Type 2 diabetes mellitus.

Finally, I would like to thank EFSD and MSD for the award of the Travel Fellowship and Prof. Trayhurn and his colleagues in the Neuroendocrine & Obesity Biology Unit at the University of Liverpool for their excellent supervision.