Travellers' Tales

Dr Chen Bing, Lecturer in Medicine at the Obesity Biology Unit of the University of Liverpool, used a Biochemical Society travel grant to visit the 4th Cachexia Conference in Tampa, FL, USA. The Conference has been a key component of the communication among scientists as well as clinicians studying the causes, mechanisms and treatment of cachexia.

My overall research theme centres on the regulation of energy balance and body weight. My current research is mainly directed to characterize the molecular mechanisms underlying the pathogenesis of cancer cachexia. Cachexia is a complex metabolic disorder characterized by progressive weight loss with reductions in both skeletal muscle and adipose tissue. Cachexia affects most cancer patients and is associated with increased morbidity and mortality. Despite its clinical importance, the pathogenesis of cachexia is poorly understood. Since adipose tissue emerges as an important organ in the control of energy homoeostasis, extensive fat depletion in cachexia links to hyperlipidaemia and insulin resistance, as well as complicating anti-tumour therapies. My work focuses on identification of the key cachexia mediators and their roles in the modulation of adipose tissue mass and function. Understanding the pathogenesis of fat loss is essential for the development of better treatments for the cachexia syndrome.

The conference was well organized, with a focus on understanding the pathogenesis of tissue wasting which occurs in association with malignancy and chronic diseases. This conference has provided me with up-to-date information through lectures, posters and discussions with other participants, including leading scientists from all over the world. These interactions have not only facilitated the cross-fertilization of original ideas, but also led to new collaborations. There is no doubt that attending this conference has been beneficial for me to develop a research programme in the UK at the forefront of cachexia research.

At the conference, I presented my latest work as a poster: '*Mechanisms of adipose atrophy in cancer cachexia*'. Profound loss of adipose tissue is a hallmark of cancer cachexia, but the underlying mechanisms remain elusive. My group examined cellular and molecular characteristics of white fat in mice with cancer cachexia, induced by transplanting MAC16 tumour into the flank of NMRI mice. Adipose tissue morphology was examined using light and electron microscopy, the mRNA levels of the key adipogenic factors were quantified by real-time PCR, and protein was analysed by Western blotting.

We saw that adiposity was markedly reduced in MAC16 mice compared with pair-fed and freely fed controls. Adipose tissue from MAC16 mice contained adipocytes that were shrunken and heterogeneous in size. Increased fibrosis was evident by strong collagen-fibril staining in the tissue matrix. Ultrastructure of 'slimmed' adipocytes revealed severe delipidation and modifications in cell membrane conformation. Neither apparent infiltration of mononuclear cells nor increased expression of cytokines was found. There were major reductions in mRNA levels of adipogenic transcription factors including C/EBP (CCAAT/enhancer binding protein) α and β, PPARγ (peroxisome-proliferator-activated receptor γ), and SREBP-1c (sterol-regulatory-element-binding protein-1c) in adipose tissue, which was accompanied by reduced protein content of C/EBPα and SREBP-1. mRNA levels of SREBP-1c targets, fatty acid synthase, acetyl-CoA carboxylase, stearoyl-CoA desaturase-1 and glycerol-3-phosphate acyltransferase, also decreased, as did GLUT4 (glucose transporter-4) and leptin. The MAC16 tumour induces significant remodelling of adipose tissue and suppression of the adipogenic factors. These findings suggest that the tumour has an inhibitory effect on adipocyte development and lipid-storing capability which may underlie adipose atrophy in cancer cachexia.

During my time in Tampa, I had a half day to explore the waterfront of St Petersburg before catching my flight back to the UK after the conference. It was such a pleasant walk from tree-shaded downtown streets to Tampa Bay where the pier complex is a distinctive landmark. Just to the north of here is the Museum of Fine Arts located in a Mediterranean-style villa. I was very impressed by the collections which start from ancient Greek and Roman sculpture to the major periods of European art and then 19th and 20th Century American works. There were also Asian, African and Native American arts and crafts being exhibited. I wished that I could spend more time there.

The travel grant covered the air ticket and the accommodation, and I thank the Society for giving me this opportunity to participate in the meeting. ■

Guidelines for applications

The rules and regulations for applying for a Travel Grant can be viewed in full at www.biochemistry.org, where you can also obtain an application form. See page 51 of this issue.