

## In the news

### FROM AACR 2017

At the 2017 American Association for Cancer Research (AACR) annual meeting in Washington D.C., USA, the tripartite association between obesity, inflammation and cancer was a recurring theme.

The concept that obesity-driven changes in the adipose microenvironment can influence cancer development and progression is increasingly recognized. Seminal talks from Liza Makowski (University of North Carolina, USA) and Andrew Dannenberg (Weill Cornell Medical College, New York, USA) explored the putative roles of macrophage infiltration and chronic low-grade inflammation in the adipose microenvironment during obesity as pro-tumorigenic mechanisms in breast cancer. The systemic

metabolic dysfunction that underpins the adipose–cancer link was also explored; Philipp Scherer (University of Texas Southwestern, USA) outlined the contributions of adipocytes in cancer and presented work on the emerging role of the adipocyte–myofibroblast transition in breast cancer. In addition, the metabolic and transcriptional differences between subcutaneous and visceral adipose tissues were illustrated by Cornelia Ulrich (University of Utah, USA), who showed that visceral adiposity is associated with advanced stage and inflammation in colorectal cancer.

Advances in prevention research were also showcased; Dipali Sharma (Johns Hopkins School of Medicine, Maryland, USA) stressed the

importance of defining molecular targets that mediate obesity-driven cancer and highlighted that alterations in the adipocyte secretome, particularly leptin, can promote breast cancer. The favourable effects of exercise and weight-loss interventions on serum inflammatory markers in patients with cancer were also presented.

Despite these associations, the reversibility of the obesity–cancer link is controversial; in an excellent forum session, researchers discussed the potential for weight-loss strategies to prevent obesity-driven breast cancer and the possibility that epigenetic reprogramming of adipocytes during obesity might limit such interventions.

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