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IN BRIEF

BREAST CANCER

Inhibiting fatty acid oxidation blocks tumour growth

Expression of the oncogenic transcription factor MYC is increased in triple-negative breast cancer (TNBC). Although MYC has been shown to alter metabolism during tumorigenesis, the role of MYC in TNBC metabolism has not been established. Camarda *et al.* now report that fatty acid oxidation (FAO) is upregulated in a mouse model of MYC-overexpressing TNBC, as well as in tumours from patients with breast cancer. Inhibition of FAO, using the carnitine palmitoyltransferase 1 inhibitor etomoxir, blocked tumour growth in both a MYC-driven transgenic TNBC mouse model and in a patient-derived xenograft model of MYC-overexpressing TNBC.

ORIGINAL ARTICLE Camarda, R. *et al.* Inhibition of fatty acid oxidation as a therapy for MYC-overexpressing triple-negative breast cancer. *Nat. Med.* **22**, 427–432 (2016)

METABOLIC DISEASE

Beige cells secrete thermogenic factor

Activation of thermogenesis in brown and beige fat has emerged as a potential strategy to improve metabolic health. Now, Svensson *et al.* identify SLIT2, a member of the SLIT extracellular protein family, as a PR domain zinc finger protein 16 (PRDM16)-regulated secreted factor from beige fat cells. Upon secretion, SLIT2 is processed proteolytically to release a plasma-soluble carboxy-terminal protein (SLIT2-C). SLIT2-C promoted adipocyte thermogenesis through induction of protein kinase A (PKA) signalling, resulting in augmented energy expenditure and improved glucose homeostasis when injected into diet-induced obese mice.

ORIGINAL ARTICLE Svensson, K. J. *et al.* A secreted Slit2 fragment regulates adipose tissue thermogenesis and metabolic function. *Cell Metab.* **23**, 454–466 (2016)

OVARIAN CANCER

Cyclic peptide inhibits metastases

The glycoprotein prosaposin has previously been demonstrated to inhibit tumour metastasis in lung and breast cancer models through stimulation of monocyte production of the antitumorigenic protein thrombospondin 1 (TSP1). Here, Wang *et al.* report that a cyclic peptide derived from the active sequence in prosaposin promotes tumour regression in a patient-derived tumour xenograft model of metastatic ovarian cancer. Analysis of tumour tissue from 134 patients with serous ovarian cancer revealed that 97% of the tumours expressed CD36, the receptor that mediates the pro-apoptotic activity of TSP1.

ORIGINAL ARTICLE Wang, S. *et al.* Development of a prosaposin-derived therapeutic cyclic peptide that targets ovarian cancer via the tumor microenvironment. *Sci. Transl Med.* **8**, 329ra34 (2016)

ANTICANCER DRUGS

Tumour-specific angiogenesis inhibition

Existing antiangiogenic drugs, which target vascular endothelial growth factor (VEGF) or its receptor VEGFR2, disrupt normal vasculature as well as tumour vasculature. Here, Al-Hilal *et al.* report that the prion-like protein doppel is expressed in the blood vessels of human cancer tissues, but not in normal tissues. *In vivo* and *in vitro* studies demonstrated that doppel promotes tumour endothelial cell (TEC) angiogenesis, through interaction with VEGFR2. An orally active glycosaminoglycan, LHbisD4, specifically binds to doppel, causing constitutive internalization of the doppel–VEGFR2 complex, resulting in inhibition of VEGF signalling and suppression of tumour growth in mouse xenograft models.

ORIGINAL ARTICLE Al-Hilal, T. A. *et al.* Targeting prion-like protein doppel selectively suppresses tumor angiogenesis. *J. Clin. Inves.* **126**, 1251–1266 (2016)

ERRATUM

Inhibiting fatty acid oxidation blocks tumour growth

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The name of the first author of the paper, Roman Camarda, was provided incorrectly in the article text and the reference. This has now been corrected in the online version.