IN BRIEF

METABOLISM

LSD1 tunes adipose tissues

In response to environmental cues, adipose tissues adapt their metabolism to maintain energy balance. This study shows that Lys-specific histone demethylase 1 (LSD1) promotes oxidative metabolism and the formation of brown-fat-like adipocytes in white adipose tissue (WAT). Following cold exposure or β3-adrenergic treatment, LSD1 levels were increased in mouse WAT; this enhanced mitochondrial activity in differentiated adipocytes. LSD1, together with nuclear respiratory factor 1 (NRF1), activated genes involved in oxidative phosphorylation and mitochondrial biogenesis. Moreover, transgenic expression of LSD1 in mice led to the formation of metabolically active brown-fat-like adipocytes in WAT, enhanced oxidative capacity and increased energy expenditure, which limited weight gain in response to a high-fat diet. Thus, LSD1 regulates thermogenesis and oxidative metabolism in adipose tissue.

ORIGINAL RESEARCH PAPER Duteil, D. *et al.* LSD1 promotes oxidative metabolism of white adipose tissue. *Nature Commun.* http://dx.doi.org/10.1038/ncomms5093 (2014)

CELL CYCLE

Making the spindle checkpoint strong

Spindle checkpoint signals (generated by checkpoint proteins, including MAD1 and the RZZ (Rod–Zw10–Zwilch) complex) arrest mitosis until all kinetochores are correctly attached to spindle microtubules, whereupon checkpoint proteins are removed in a dynein-dependent manner. Matson and Stukenberg report that the centromeric protein CENPI is required for the stable association of MAD1 and RZZ with kinetochores. CENPI cooperated with Aurora B in the recruitment of MAD1 and RZZ. Moreover, CENPI prevented their premature removal by dynein in an Aurora B-independent manner. Thus, CENPI and Aurora B create and maintain a robust checkpoint signal until all spindle attachments are correctly formed.

ORIGINAL RESEARCH PAPER Matson, D. R. & Stukenberg, P. T. CENP-I and Aurora B act as a molecular switch that ties RZZ/Mad1 recruitment to kinetochore attachment status. *J. Cell Biol.* **205**, 541–554 (2014)

CELL MIGRATION

Dynamic junctions during group travel

While studying adherens junctions and one of their components, N-cadherin, in primary astrocytes, Peglion *et al.* noted that they flowed continuously from the front to the back of leader cells during collective cell migration. Furthermore, N-cadherin was trafficked to the front edge of leading cells, where it was incorporated into new junctions. How is this polarized recycling of N-cadherin regulated? p120 catenin, which interacts with N-cadherin and is required for its stability at the leading edge of migrating cells, was phosphorylated at Thr310 by GSK3β at the rear of cells but not at the front. This phosphorylation event reduced N-cadherin–p120-catenin interactions, which explains how N-cadherin is free for recycling at the cell rear but incorporated into adherens junctions at the cell front.

ORIGINAL RESEARCH PAPER Peglion, F. *et al.* Adherens junction treadmilling during collective migration. *Nature Cell Biol.* http://dx.doi.org/10.1038/ncb2985 (2014)