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IN BRIEF

CIRCADIAN RHYTHMS

Not a morning person? Blame it on your genes

If you enjoy getting up in the mornings, chances are you have one of 15 loci identified in a new genome-wide association study (GWAS). The investigators performed a GWAS on samples from 89,283 individuals who reported that they preferred to wake early in the day, referred to as 'morningness'. Women were more likely to be a morning person than men (48.4% versus 39.7%) and morningness increased with age; from 24.2% of people aged <30 years to 63.1% of people aged >60 years. Of the 15 loci identified, seven were located close to genes known to be involved in circadian rhythms. The investigators found that morningness was also associated with insomnia, increased BMI and depression, but in Mendelian randomization analyses, no causal relationship was identified.

ORIGINAL ARTICLE Hu, Y. *et al.* GWAS of 89,283 individuals identifies genetic variants associated with self-reporting of being a morning person. *Nat. Commun.* http://dx.doi.org/10.1038/ncomms10448 (2016)

NEUROENDOCRINOLOGY

Glucose availability modulates neurogenesis

Low glucose conditions can enhance the proliferation of neural stem cells (NSCs) and promote neurogenesis in mice, according to new research. NSCs form neurospheres in culture, the size and number of which are indicative of NSC proliferation. Twice as many neurospheres formed in low glucose conditions than in high glucose conditions. These neurospheres also expressed increased levels of *Hes-1*, a marker of differentiated NSCs. As CREB and Sirtuin 1 (Sirt1) are involved in nutrient sensing, the investigators hypothesised that these proteins might regulate NSC proliferation. In low glucose conditions, CREB displaces Sirt1 from the *Hes-1* promoter, which leads to increased expression of *Hes-1* and consequently NSC proliferation. Conversely, in high glucose conditions, Sirt1 occupies the promoter and prevents transcription of *Hes-1*. Interestingly, the team found that in calorie-restricted mice, this 'molecular switch' is activated in hippocampal cells, which increases *Hes-1* expression. These results suggest that in states of nutrient excess NSC renewal might be impaired.

ORIGINAL ARTICLE Fusco, S. *et al.* A CREB-Sirt1-Hes1 circuitry mediates neural stem cell response to glucose availability. *Cell Rep.* **14**, 1–11 (2016)

ADIPOSE TISSUE

New pathway in BAT activation?

Inactivating upstream stimulatory factor 1 (USF1) can activate brown adipose tissue (BAT) and increase energy expenditure, which leads to improved cardiometabolic parameters. In *Usf1* knockout mice, clearance of triglycerides from the plasma is enhanced and these lipids directed to BAT. Increased BAT thermogenesis and upregulation of mitochondrial respiratory chain complexes then facilitate the breakdown of these triglycerides. Moreover, plasma levels of HDL cholesterol are also increased in these mice. Importantly, the investigators identified a polymorphism in human *USF1* that is associated with improvements in insulin sensitivity, lipid profiles and atherosclerosis. The team hope that USF1 might be a new therapeutic target for the treatment of cardiometabolic disease.

ORIGINAL ARTICLE Laurila, P-P. *et al.* USF1 deficiency activates brown adipose tissue and improves cardiometabolic health. *Sci. Transl. Med.* **323**, 323ra13 (2016)