

## OBESITY

## Training your brain can improve food choice

Regulation of craving (ROC) training — a mechanism-based intervention developed at Yale University, USA, by Hedy Kober and colleagues — improves food choices and reduces calorie intake, according to new research. In the present study, which included 1,528 participants, the authors found that individuals who had received ROC training were more likely than those who had not received the training to choose healthy foods over unhealthy foods.

“In a prior meta-analysis we showed that craving for unhealthy foods is an important mechanism driving eating and weight gain,” explains Kober, the corresponding author. “We also found that we can teach people to use cognitive strategies, which involve thinking about the negative consequences of unhealthy foods, to reduce their craving for unhealthy foods.” With the aim of helping people to improve their eating and food choices, the team used the knowledge they gained during these prior studies to develop ROC training.

The ROC training was developed by combining methods from clinical psychology, experimental studies of emotion regulation

and behavioural economics. Kober and colleagues tested the efficacy of ROC training on multiple dependent variables, including self-reported craving, willingness to pay for food (in \$US), food choices and calorie consumption. “This multi-modal approach allowed us to develop a more robust intervention — drawing insights from different fields — and to validate it on a variety of relevant outcomes,” adds Kober.

The authors report that when faced with unhealthy food options, ROC training increased the participant’s chance of selecting a healthy food choice by 5.4–11.2% and participants who received training indicated that they were willing to pay up to \$1.50 more for healthy foods. “The results of our study show that knowledge alone is not enough and that training in the implementation of knowledge is necessary to help people change their food choices,” concludes Kober.

Alan Morris

**ORIGINAL ARTICLE** Boswell, R. G. et al. Training in cognitive strategies reduces eating and improves food choice. *Proc. Natl Acad. Sci. USA* <https://doi.org/10.1073/pnas.1717092115> (2018)

## CIRCADIAN RHYTHMS

## Circadian protein affects mTORC1 activity

Given that the key circadian proteins (BMAL1, CLOCK, PER and CRY) are nucleus–cytosol trafficking proteins, it has been assumed that they have novel roles in the cytosol. This assumption has been confirmed previously for BMAL1 and CRY1, and a new study has now identified that PER2 has binding partners in the cytosol through which it inhibits mTORC1 (a complex with diurnal oscillations in activity).

Using immunoprecipitation analyses, Yi Liu and colleagues found that PER2 strongly interacts with RAPTOR and mTOR, key components of the mTORC1 complex, in the cytosol. Further analyses revealed that PER2 is a scaffold protein that tethers RAPTOR, TSC1 and mTOR, which results in the activity of the mTORC1 complex being suppressed.

Next, Liu and colleagues used *Per2*<sup>−/−</sup> mice to demonstrate that PER2 is required for protein synthesis and has a role in regulating autophagy. In addition, the researchers noticed that fasting resulted in a significant increase in levels of PER2 in mouse liver, accompanied by inhibition of mTORC1, and found that the injection of glucagon mimicked these effects. In *Per2*<sup>−/−</sup> mice

that were injected with glucagon, the suppression of hepatic mTORC1 activity was abolished. These findings suggest that PER2 is required for the inhibitory effects of glucagon on mTORC1.

“Our study not only finds a novel biological role of PER2 in inhibiting the mTORC1 signalling pathway, but also reveals the molecular mechanism underlying the circadian rhythms of mTORC1 activity and autophagy in mouse liver,” states Liu. “Furthermore, our study suggests the existence of a negative feedback loop between mTORC1 and circadian clock.”

“As protein–protein interactions are mutual, we assume that mTORC1 might also regulate PER2 activity,” says Liu. “For example, one of our future plans is to check whether mTORC1 phosphorylates PER2 to affect its intracellular localization or protein stability.”

Claire Greenhill

**ORIGINAL ARTICLE** Wu, R. et al. The circadian protein Period2 suppresses mTORC1 activity via recruiting Tsc1 to mTORC1 complex. *Cell Metab.* <https://doi.org/10.1016/j.cmet.2018.11.006> (2018)

## IN THE NEWS

## Highlights from SfE BES 2018

This November, Glasgow, UK, hosted the Society for Endocrinology’s annual meeting (SfE BES 2018), which saw endocrinologists from around the world gathering to discuss the latest advances in the field.

The first day opened with a fascinating symposium session on curing diabetes mellitus. Colin Dayan (Cardiff University, UK) outlined the need for immunotherapies for type 1 diabetes mellitus and gave an update on the trials that are currently underway. In addition, advances in clinical islet transplantation and the latest results on low-calorie diets for patients with type 2 diabetes mellitus were discussed.

In a symposium session entitled ‘The most important 9 months; impact of maternal health’, Susan Ozanne (University of Cambridge, UK) gave a detailed account of the effects of maternal diabetes mellitus on the child’s health. 1 in 7 births are affected by gestational diabetes mellitus, and fetuses exposed to maternal diabetes mellitus in the womb seem to be at increased risk of cardiometabolic disease as a result of in utero epigenetic effects.

Ursula Kaiser (Brigham and Women’s Hospital, USA) expertly delivered the first plenary of the conference. Kaiser discussed the neuroendocrine signals that trigger puberty and outlined how the timing of puberty onset can affect the risk of certain diseases (such as obesity and endometrial cancer) later in life.

Robin Peeters (Erasmus Medical Center, Netherlands) gave the Pitt-Rivers lecture, in which he discussed defining optimal thyroid function. Peeters outlined the challenges of defining thyroid functions in a range of situations, including during pregnancy; the fetus is dependent on maternal thyroxine, so maternal thyroid dysfunction can result in negative pregnancy outcomes, such as a preterm birth. On the final day of the conference, Maria-Christina Zennaro (INSERM, France) gave the European Medal Lecture on the molecular mechanisms of primary aldosteronism, which occurs in 11% of patients with hypertension.

SfE BES 2018 was a very enjoyable meeting, and I look forward to SfE BES 2019, which will be in Brighton, UK.

Claire Greenhill

**FURTHER READING** Agha-Jaffar, R. et al. Gestational diabetes mellitus: does an effective prevention strategy exist? *Nat. Rev. Endocrinol.* **12**, 533–546 (2016) | Pozzilli, P. et al. Combination immunotherapies for type 1 diabetes mellitus. *Nat. Rev. Endocrinol.* **11**, 289–297 (2015) | Korevaar, T. I. M. et al. Thyroid disease in pregnancy: new insights in diagnosis and clinical management. *Nat. Rev. Endocrinol.* **13**, 610–622 (2017)