Results: Overall, 13% of students reported consuming energy drinks on a weekly basis. A further 18% indicated that they consume less than a cup a week of these types of drinks, while just over two-thirds (69%) reported that they do not consume energy drinks. In multivariable analysis, weekly consumption of energy drinks was independently associated with male gender, lower socio-economic position (SEP), having at least $40 per week of spending money; high intakes of fast food and other sugar-sweetened beverages, and short sleep duration. There was no independent association with body weight or other demographic characteristics (i.e. year level, geographic location), eating behaviours (i.e. consumption of vegetables, fruit, snack foods or fruit juice), or physical activity and television viewing habits.

Conclusion: While the majority of adolescents do not consume energy drinks, regular consumption is more prevalent among low SEP males and appears to cluster with other unhealthy behaviours. Given there is growing evidence of the potential health harms of energy drinks for young people, improved education is needed along with restrictions on the marketing and sale of these products to adolescents.

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Role of amygdala NPY in the development of stress-induced obesity

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Neuropeptide Y (NPY) is one of the most powerful orexigenic peptides known, exerting critical feeding related functions in the hypothalamus. However, NPY is also present in extra hypothalamic nuclei where it is modulated in response to metabolic and other physiological perturbations, but far less is known about these NPY populations and their influence on energy homeostasis regulation. Now we have identified that NPY neurons in the central amygdala (CeA) are responsible for an exacerbated response to a combined stress and high fat diet intervention leading to accelerated obesity development. Employing CeA NPY neuron specific AAV-NPY overexpression models we were able to replicate the obese phenotype seen in the combined stress/HFD mouse model, which was prevented by the selective ablation of NPY from these neurons. Furthermore, by selectively activating NPY neurons of the CeA via DREADD we mapped the projections of these neurons to the Arc and PVN, known nuclei to be critical for energy homeostasis control. Moreover, using food intake and energy expenditure as the physiological readout we demonstrated that selective activation of CeA NPY neurons results in a robust increase in food intake and decrease in EE which is entirely dependent on the presence of NPY. Mechanistically it is the failure of insulin to no longer control these NPY neurons under combined stress/HFD conditions that leads to accelerated obesity. Taken together this study has uncovered a previously unknown feeding stimulatory pathway that is activated under conditions of stress in combination with calorie dense food.

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Brain control of blood glucose

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Abstract not available.

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AgRP neurons require carnitine acetyltransferase (Crat) to regulate metabolic flexibility and peripheral nutrient partitioning

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Osteoglycin, a novel coordinator of bone and glucose homeostasis

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The importance of coordinating whole body energy balance is known, however, the mechanism controlling and integrating the energy homeostasis of individual tissues during weight change is unclear. Employing transcriptomic and proteomic analysis of Y1 receptor deficient osteoblasts combined with the generation of a genetically modified mouse model we have identified a novel circuitry that is responsible for coordinating bone accretion with changes in energy balance and identify the NPY controlled release of osteoglycin from osteoblasts as the critical component. Osteoglycin acts to suppress bone formation when energy levels are low and to modulate whole body energy supplies by altering glucose uptake through changes in insulin secretion and sensitivity, as well as by altering food intake through central signalling. Human studies show that osteoglycin is associated with BMI and lean mass as well as changes in weight, BMI and glucose levels as a result of the negative energy balance elicited by gastric surgery. Thus, we identify osteoglycin as a novel factor facilitating matching of bone acquisition to alterations in energy status.

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