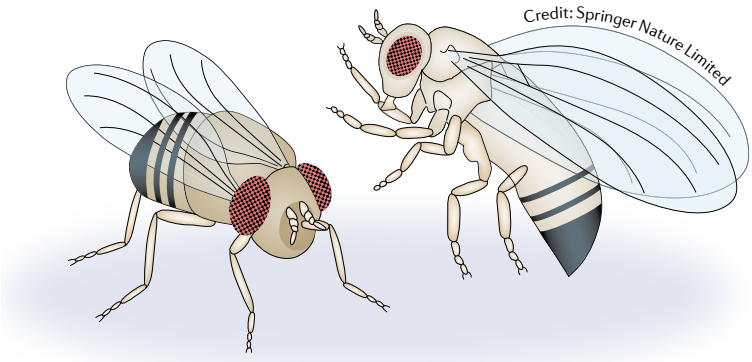

neurons, and silencing fpC1 neurons reduced the aggression induced by high-intensity activation of CAP neurons. Thus, fpC1 neurons in female flies seem to be functionally analogous to MAP neurons in male flies.

Previous work has shown that social isolation increases aggressiveness in flies. Here, suppressing CAP neuron activity in socially isolated (SI) flies of either sex reduced aggressive approaches and attacks compared with control SI flies. Even weak activation of CAP neurons in SI flies of either sex elicited both approach and attack behaviours. Moreover, the responses of MAP neurons or fpC1 neurons to optogenetic activation of CAP neurons were greater in SI flies than in group-housed flies. Therefore, social isolation boosts aggressiveness, potentially by strengthening the functional connection between CAP neurons and downstream MAP or fpC1 neurons.

Overall, this study provides evidence that CAP neurons in both sexes control the appetitive phase of aggressive behaviour (approach), whereas MAP neurons in males, or fpC1 neurons in females, regulate sexually dimorphic consummatory (attack) behaviours.

Natasha Bray

ORIGINAL ARTICLE Chiu, H. et al. A circuit logic for sexually shared and dimorphic aggressive behaviors in *Drosophila*. *Cell* https://doi.org/10.1016/j.cell.2020.11.048 (2020)

stimulus in an aversive or appetitive manner, respectively. Furthermore, in mice trained to identify sweet and bitter tastants (indicating their choice by licking to the left or right of the stimulus-delivery spout), direct activation of *Sst*-expressing or *Calb2*-expressing rNST neurons was reported by the animals as a bitter or sweet stimulus, respectively.

The discovery that rNST neurons are essential for the representation of bitter and sweet tastes suggested this region as a possible site of cross-modulation between the two pathways. Indeed, the responses of *Calb2*-expressing rNST neurons to a sweet tastant were suppressed when it was mixed with a bitter tastant. Using fluorescent tracers, the authors showed that neurons in the gustatory cortex region encoding bitter taste (GCbt) and in the central amygdala (CeA, a region important for encoding sensory valance) project to rNST, suggesting that these regions might exert top-down control over taste responses.

Patch-clamp recordings and retrograde viral tracing showed that *Sst*-expressing rNST neurons receive direct excitatory inputs from GCbt, whereas *Calb2*-expressing neurons receive indirect inhibitory input from GCbt, via CeA. Optogenetic activation of GCbt neurons increased *Sst*-expressing neuron activity, whereas activation of GCbt or of GCbt–CeA projections reduced the activity of *Calb2*-expressing rNST neurons. This showed that combined positive and negative feedback pathways from the cortex to rNST enhance aversive responses to bitter tastants, while suppressing appetitive responses to any accompanying sweet stimuli. The importance of this feedback for the modulation of sweet taste perception was confirmed by pharmacological and optogenetic silencing of CeA inputs to the rNST, which eliminated the suppression of appetitive responses to sweet tastants by bitter stimuli.

This study dissects a neural circuit through which top-down control mechanisms can modify an innate behaviour, revealing the brainstem to be a key site for such modulation.

Katherine Whalley

ORIGINAL ARTICLE Jin, H. et al. Top-down control of sweet and bitter taste in the mammalian brain. *Cell* **184**, 257–271 (2021)

IN BRIEF

CELLULAR NEUROSCIENCE

Nose-to-tail transcriptomics

Primary cultures of rat hippocampal neurons were microdissected to separate individual dendritic processes and somas. In the three types of GABAergic interneuron studied, transcriptome-based clustering analysis revealed that dendritic expression profiles differed from those of somatic compartments. Dendrites typically contained around 4,000 mRNA species, some of which encoded dendritic proteins, suggesting local translation. Indeed, newly made proteins were detected in neurites, suggesting that, as in excitatory neurons, local protein synthesis also occurs in inhibitory neurons.

ORIGINAL ARTICLE Perez, J. D. et al. Subcellular sequencing of single neurons reveals the dendritic transcriptome of GABAergic interneurons. *eLife* https://doi.org/10.7554/eLife.63092 (2021)

NEUROIMMUNOLOGY

COVID crossing

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infects cells via its spike protein, S1, binding to angiotensin-converting enzyme 2 (ACE2). Here, crossing of radiolabelled S1 (I-S1) into the brains of mice was shown to occur by adsorptive transcytosis and was increased by its co-injection with ACE2. Lipopolysaccharide-induced elevation of plasma cytokine levels, similar to that observed in some SARS-CoV-2 cases, also increased I-S1 brain entry and resulted in variable distribution across brain structures. Intranasally administered I-S1 was detected in all tested brain regions, especially the olfactory bulb and hypothalamus, but entered the brain with lower efficiency than systemically administered I-SI. This study supports a mechanism by which SARS-CoV-2 could enter the brain.

ORIGINAL ARTICLE Rhea, E. M. et al. The S1 protein of SARS-CoV-2 crosses the blood–brain barrier in mice. *Nat. Neurosci.* https://doi.org/10.1038/s41593-020-00771-8 (2020)

PAIN

A painless STING

STING is an intracellular sensor of DNA expressed in nociceptors that, when activated, induces expression of type 1 interferons (IFN1). Knockout of STING or IFN1 receptors selectively from mouse peripheral sensory neurons increased sensitivity to nociceptive stimuli and increased intrinsic excitability. Conversely, central administration of a STING agonist resulted in reduced nociceptor action potential firing and anti-nociceptive effects in mice and non-human primates. Thus, STING-mediated IFN1 induction is an important regulator of nociception in rodents and non-human primates.

ORIGINAL ARTICLE Donnelly, C. R. et al. STING controls nociception via type I interferon signalling in sensory neurons. *Nature* https://doi.org/10.1038/s41586-020-03151-1 (2021)

NEUROGENETICS

Double trouble

Inherited and de novo genetic factors (a major source of which are tandem repeats) are thought to contribute to autism spectrum disorder (ASD). Here, the authors developed a method to identify de novo tandem-repeat mutations (TRMs) in whole-genome sequencing data from probands with ASD and their unaffected siblings. Probands showed more TRMs than unaffected siblings, and TRMs in probands were enriched in genomic regions associated with fetal brain development. Certain TRMs may therefore contribute to risk of ASD.

ORIGINAL ARTICLE Mitra, I. et al. Patterns of de novo tandem repeat mutations and their role in autism. *Nature* **589**, 246–250 (2021)