

IN BRIEF

GUT–BRAIN COMMUNICATION**Permitting pain**

Neuropathic pain is a common adverse effect of chemotherapeutic agents, such as oxaliplatin. The authors here showed that antibiotic-mediated eradication of the gut microbiota protects mice against both oxaliplatin-induced inflammation in the dorsal root ganglia and mechanical hyperalgesia. The permissive effect of the gut microbiota on chemotherapy-induced pain was found to be mediated by the interaction of microbiota-derived lipopolysaccharides with haemopoietic cells expressing Toll-like receptor 4.

ORIGINAL ARTICLE Shen, S. *et al.* Gut microbiota is critical for the induction of chemotherapy-induced pain. *Nat. Neurosci.* <http://dx.doi.org/10.1038/nn.4606> (2017)

NEURONAL NETWORKS**Individual arrangements**

Functional connectivity analyses of group data have revealed several large-scale distributed brain networks, including the default network (DN), frontoparietal control network (FPN) and dorsal attention network (dATN); however, few studies have examined network organization within individual subjects. The authors performed multiple functional MRI scans in four individuals and found that the DN, FPN and dATN could each be fractionated into two parallel and spatially juxtaposed, but dissociable, networks that were distributed across the cortex.

ORIGINAL ARTICLE Braga, R. M. & Buckner, R.L. Parallel interdigitated distributed networks within the individual estimated by intrinsic functional connectivity. *Neuron* **95**, 457–471 (2017)

NEURODEGENERATIVE DISEASE**A programmed killer**

Neuronal loss is a hallmark of Alzheimer disease (AD), but the mechanisms through which the neurons die are unclear. Here, the authors described evidence for the activation of necroptosis — a programmed form of necrosis that has been implicated in multiple sclerosis and amyotrophic lateral sclerosis — both in the post-mortem human AD brain and in a mouse model of AD that features neuronal loss. Inhibiting necroptosis in neurons from AD mice *in vitro* or *in vivo* reduced neuronal death, suggesting that inhibition of necroptosis could be examined as a possible future therapeutic strategy for AD.

ORIGINAL ARTICLE Caccamo, A. *et al.* Necroptosis activation in Alzheimer's disease. *Nat. Neurosci.* <http://dx.doi.org/10.1038/nn.4608> (2017)

PSYCHIATRIC DISORDERS**Acute actions**

Selective serotonin reuptake inhibitors (SSRIs) are commonly used antidepressants; however, their mechanism of action is elusive. The authors here show that activation of serotonin 5-HT_{1B} receptors on cholecystinin-expressing interneurons (CCK neurons) in the dentate gyrus underlies the effects of acute SSRI treatment on depressive-like behaviours in mice, whereas 5-HT_{2A} receptors mediate the effects of chronic SSRI treatment. Chemogenetic inhibition of CCK neurons had an acute antidepressant effect, suggesting that this neuronal population might be a viable target for the development of improved antidepressants.

ORIGINAL ARTICLE Medrihan, L. *et al.* Initiation of behavioral response to antidepressants by cholecystinin neurons of the dentate gyrus. *Neuron* <http://dx.doi.org/10.1016/j.neuron.2017.06.044> (2017)