IN BRIEF

IMMUNOMETABOLISM

Macrophages and smaller ages

Lipid-rich diets are associated with impaired glucose metabolism and a reduced lifespan — this study shows that macrophages can contribute to both of these outcomes. Using *Drosophila melanogaster* as a model organism, Woodcock *et al.* found that a lipid-rich diet promoted the activation of macrophages and their expression of the type 1 cytokine Unpaired 3 (Upd3), which signals via the JAK–STAT pathway. Silencing of Upd3 expression in macrophages (but not in other cell types) increased fly longevity in response to a lipid-rich diet, and Upd3-deficient flies fed a control or lipid-rich diet had similar lifespans. Although wild-type *D. melanogaster* showed systemic JAK–STAT activation and developed hyperglycaemia in response to a lipid-rich diet, Upd3-deficient flies were protected against these effects. Macrophage depletion also protected flies on a lipid-rich diet from developing hyperglycaemia and increased their lifespan. A final series of experiments suggested that macrophage uptake of excess lipids via the scavenger receptor Croquemort may trigger a JNK-mediated stress response that leads to the production of Upd3.

ORIGINAL RESEARCH PAPER Woodcock, K. J. *et al.* Macrophage-derived *upd3* cytokine causes impaired glucose homeostasis and reduced lifespan in *Drosophila* fed a lipid-rich diet. *Immunity* http://dx.doi.org/10.1016/j.immuni.2014.12.023 (2015)

INFECTION

The cost of targeting helminths

Laboratory studies have shown that helminths can skew the immune response to increase the susceptibility of the host to co-infecting bacteria or viruses. Therefore, it has been suggested that targeting helminths could be an effective strategy for combating microbial co-infections. In this study, Ezenwa and Jolles assessed how anthelmintic treatment affects the spread of bovine tuberculosis (BTB) in wild populations of African buffalo (*Syncerus caffer*). The authors found that animals treated with an anthelmintic had an increased ability to secrete interferon-γ compared with untreated controls. Treatment with the anthelmintic had no effect on BTB acquisition rates in buffalo, but led to a ninefold decrease in mortality following the acquisition of BTB. However, anthelmintic treatment had a negative effect at the population level as it increased the spread of BTB among buffalo. These findings suggest that anthelmintics can have a positive effect at the individual level, but may ultimately have a detrimental effect at the population level by enhancing pathogen spread.

ORIGINAL RESEARCH PAPER Ezenwa, V. O. & Jolles, A. N. Opposite effects of anthelmintic treatment on microbial infection at individual versus population scales. *Science* **347**, 175–177 (2015)

INFLAMMATION

Dopamine blocks inflammasome activation

Yan *et al.* report that the neurotransmitter dopamine can prevent the activation of the NOD-, LRR- and pyrin domain-containing 3 (NLRP3) inflammasome. Signalling through the dopamine D1 receptor (DRD1) stimulated the production of cyclic AMP, which bound to NLRP3 and promoted its ubiquitylation and subsequent degradation. Notably, dopamine and DRD1 signalling suppressed NLRP3 inflammasome activation in models of neurotoxin-induced inflammation, lipopolysaccharide-induced systemic inflammation and monosodium urate-induced peritonitis. These findings suggest an important anti-inflammatory role for dopamine.

ORIGINAL RESEARCH PAPER Yan, Y. *et al.* Dopamine controls systemic inflammation through inhibition of NLRP3 inflammasome. *Cell* http://dx.doi.org/10.1016/j.cell.2014.11.047 (2014)