Wagener, J., Malireddi, S. R.K, Lenardon, M.D., Köberle, M., Vautier, S., MacCallum, D.M., Biedermann, T., Schaller, M, Netea, M.G., Kanneganti, T-D., Brown, G.B., Brown, A.J.P. & Gow, N.A.R. (2014). Fungal chitin dampens inflammation through IL-10 induction mediated by NOD2 and TLR9 activation. *PLoS Pathogens* 10(4): e1004050.

Chitin is an essential structural polysaccharide of fungal pathogens and parasites, but its role in human immune responses remains largely unknown. It is the second most abundant polysaccharide in nature after cellulose and its derivatives today are widely used for medical and industrial purposes. We analysed the immunological properties of purified chitin particles derived from the opportunistic human fungal pathogen Candida albicans, which led to the selective secretion of the anti-inflammatory cytokine IL-10. We identified NOD2, TLR9 and the mannose receptor as essential fungal chitin-recognition receptors for the induction of this response. Chitin reduced LPS-induced inflammation in vivo and may therefore contribute to the resolution of the immune response once the pathogen has been defeated. Fungal chitin also induced eosinophilia in vivo, underpinning its ability to induce asthma. Polymorphisms in the identified chitin receptors, NOD2 and TLR9, predispose individuals to inflammatory conditions and dysregulated expression of chitinases and chitinase-like binding proteins, whose activity is essential to generate IL-10-inducing fungal chitin particles in vitro, have also been linked to inflammatory conditions and asthma. Chitin recognition is therefore critical for immune homeostasis and is likely to have a significant role in infectious and allergic disease.