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IN BRIEF

ASTHMA AND ALLERGY

Vitamin D primes neonatal immune system

Vitamin D deficiency during pregnancy has been linked to the development of childhood asthma, but most studies in this area have been observational. To directly examine how vitamin D status in pregnancy affects the neonatal immune system, Hornsby *et al.* analysed cord blood samples from neonates whose mothers had received daily vitamin D3 supplements during pregnancy. Cord blood mononuclear cells (CBMCs) from these neonates showed increased production of pro-inflammatory cytokines following activation with innate stimuli *in vitro*, and increased production of IL-17A in response to polyclonal T cell stimulation. Their CMBCs also produced greater amounts of IL-10 in response to dexamethasone treatment. The authors suggest that strong neonatal immune responses may protect against the development of asthma by improving respiratory health in early life.

ORIGINAL ARTICLE Hornsby, E. *et al.* Vitamin D supplementation during pregnancy: effect on the neonatal immune system in a randomized controlled trial. *J. Allergy Clin. Immunol.* http://dx.doi.org/10.1016/j.jaci.2017.02.039 (2017)

MICROBIOTA

Baby bugs can't stop the thugs...

Newborns are highly susceptible to orally acquired bacterial infections and this is generally attributed to immaturity of their immune system. This study shows that another contributing factor is the neonatal microbiota, which is less effective in mediating colonization resistance. Kim *et al.* colonized adult germ-free mice with caecal contents from neonatal or adult mice; unlike adult microbiota, neonatal microbiota was unable to prevent colonization of the gut by the bacterial pathogens *Salmonella enterica* serovar Typhimurium and *Citrobacter rodentium*. Around 50% of mice colonized with neonatal microbiota died following *S.* Typhimurium infection, whereas all mice colonized with adult microbiota survived. The absence of *Clostridia* species from the neonatal microbiota was shown to account for the lack of colonization resistance and administration of *Clostridia* to neonatal mice protected them against lethal *S.* Typhimurium infection. The authors showed that the neonatal microbiota supports the acquisition of *Clostridia* species before weaning, an event that is crucial for subsequent protection against enteric infections.

ORIGINAL ARTICLE Kim, Y. et *al.* Neonatal acquisition of *Clostridia* species protects against colonization by bacterial pathogens. *Science* **356**, 315–319 (2017)

INNATE IMMUNITY

Alarmins rewire innate immunity in newborns

This study describes an important role for the endogenous alarmins S100A8 and S100A9 in protecting newborn infants from sepsis. Healthy newborns produce extremely high levels of S100 alarmins for the first five days of life; the authors found that these alarmins signal through TLR4 to preactivate MYD88-dependent (but not TRIF-dependent) genes in neonatal monocytes, leaving them refractory to subsequent activation of MYD88. This 'rewiring' of neonatal monocytes prevented hyperinflammatory responses to bacteria without compromising host immunity. In a mouse model of *Staphylococcus aureus*-induced sepsis, S100A9-deficient neonates produced increased levels of pro-inflammatory cytokines, leading to fatal sepsis. In humans, pre-term infants that experienced late-onset neonatal sepsis had lower cord blood levels of S100 alarmins.

ORIGINAL ARTICLE Ulas, T. *et al.* S100-alarmin-induced innate immune programming protects newborn infants from sepsis. *Nat. Immunol.* **18**, 622–632 (2017)