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Human eosinophils modulate peripheral blood mononuclear cell response to Schistosoma manson i adultworm antigen in vitro. Tweyongyere R, Namanya H, Naniima P, Cose S, Tukahebwa EM, Elliott AM, Dunne DW, Wilson S. *Parasite Immunol. 2016 May 12. doi: 10.1111/pim.12336. [Epub ahead of print]*

Abstract

High numbers of eosinophils are observed in parasitic infections and allergic diseases, where they are proposed to be terminally differentiated effector cells that play beneficial role in host defense, or cause harmful inflammatory response. Eosinophils have been associated with killing of schistosomulae in vitro, but there is growing evidence that eosinophils can play additional immuno-regulatory role. Here we report results of a study that examines peripheral blood mononuclear cell (PBMC) cytokine responses to S. mansoni adult worm antigen (SWA) when stimulated alone or enriched with autologous eosinophils. Production of the Th-2 type cytokines interleukin (IL)-4, IL-5 and IL-13 were lower (p=0.017, 0.018 and <0.001 respectively) in PBMC + eosinophil cultures than in PBMC-only cultures stimulated with SWA. Substantial levels of IL-13, IL-10, interferon gamma (IFNγ) and tumor necrosis factor alpha (TNFα) were recorded in cultures of eosinophils, but none of these cytokines showed significant association with the observed eosinophil-induced drop in cytokine responses of PBMC. Transwell experiments suggested that the observed effect is due to soluble mediators that downmodulate production of Th-2 type cytokines. This study shows that eosinophils may down-modulate schistosome-specific Th-2 type cytokine responses in S. mansoni infected individuals. The mechanism of this immune-modulation remains to be elucidated.

Early cranial ultrasound findings among infants with neonatal encephalopathy in Uganda: an observational study. Cally J. Tann, Margaret Nakakeeto, Cornelia Hagmann, Emily L. Webb, Natasha Nyombi, Flaviah Namiiro, Kelly Harvey-Jones, Anita Muhumuza, Kathy Burgoine, Alison M. Elliott, Jennifer J. Kurinczuk, Nicola J. Robertson & Frances M. Cowan. *Pediatric Research* (2016) doi:10.1038/pr.2016.77

Background: In sub-Saharan Africa, the timing and nature of brain injury and their relation to mortality in neonatal encephalopathy (NE) are unknown. We evaluated cranial ultrasound (cUS) scans from term Ugandan infants with and without NE for evidence of brain injury.

Methods: Infants were recruited from a national referral hospital in Kampala. Cases (184) had NE and controls (100) were systematically selected unaffected term infants. All had cUS scans <36 h reported blind to NE status.

Results: Scans were performed at median age 11.5 (interquartile range (IQR): 5.2–20.2) and 8.4 (IQR: 3.6–13.5) hours, in cases and controls respectively. None had established antepartum injury. Major evolving injury was reported in 21.2% of the cases vs. 1.0% controls ($P < 0.001$). White matter injury was not significantly associated with bacteremia in encephalopathic infants (odds ratios (OR): 3.06 (95% confidence interval (CI): 0.98–9.60). Major cUS abnormality significantly increased the risk of neonatal death (case fatality 53.9% with brain injury vs. 25.9% without; OR: 3.34 (95% CI: 1.61–6.95)).

Conclusion: In this low-resource setting, there was no evidence of established antepartum insult, but a high proportion of encephalopathic infants had evidence of major recent and evolving brain injury on early cUS imaging, suggesting prolonged or severe acute exposure to hypoxia–ischemia (HI). Early abnormalities were a significant predictor of death.