Perinatal brain injury

Preterm birth is an important cause of perinatal brain injury (PBI). Neurologic injury in extremely preterm infants often begins in utero with chorioamnionitis (CHORIO) or inflammation/infection of the placenta and concomitant placental insufficiency. Studies in humans have shown dysregulated inflammatory signaling throughout the placental-fetal brain axis and altered peripheral immune responses in children born preterm with cerebral palsy (CP).

Yellowhair et al., hypothesized that peripheral immune responses would be altered in a well-established rat model of CP. Specifically, theye proposed that isolated peripheral blood mononuclear cells (PBMCs) would be hyperresponsive to a second hit of inflammation throughout an extended postnatal time course. Pregnant Sprague-Dawley dams underwent a laparotomy on embryonic day 18 (E18) with occlusion of the uterine arteries (for 60 min) followed by intra-amniotic injection of lipopolysaccharide (LPS, 4 µg/sac) to induce injury in utero. Shams underwent laparotomy only, with equivalent duration of anesthesia. Laparotomies were then closed, and the rat pups were born at E22. PBMCs were isolated from pups on postnatal day 7 (P7) and P21, and subsequently stimulated in vitro with LPS for 3 or 24 h. A secreted inflammatory profile analysis of conditioned media was performed using multiplex electrochemiluminescent immunoassays, and the composition of inflammatory cells was assayed with flow cytometry (FC). Results indicate that CHORIO PBMCs challenged with LPS are hyperreactive and secrete significantly more tumor necrosis factor α (TNFα) and C-X-C chemokine ligand 1 at P7. FC confirmed increased intracellular TNFα in CHORIO pups at P7 following LPS stimulation, in addition to increased numbers of CD11b/c immunopositive myeloid cells. Notably, TNFα secretion was sustained until P21, with increased interleukin 6, concomitant with increased expression of integrin β1, suggesting both sustained peripheral immune hyperreactivity and a heightened activation state. Taken together, these data indicate that in utero injury primes the immune system and augments enhanced inflammatory signaling. The insidious effects of primed peripheral immune cells may compound PBI secondary to CHORIO and/or placental insufficiency, and thereby render the brain susceptible to future chronic neurological disease. Further understanding of inflammatory mechanisms in PBI may yield clinically important biomarkers and facilitate individualized repair strategies and treatments [1)].

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