An estimated 15% of all neonatal deaths globally were due to sepsis in 2018 (WHO). Sepsis in infants refers to an infection involving the bloodstream shortly after birth. The liver plays a critical role in this pathology. NFkB signaling has been demonstrated to have a key function in coordinating the innate immune response to infection. The NFkB subunit p65 (RelA) contains a DNA binding domain and can translocate into the nucleus upon canonical activation of the NFkB pathway, acting as a key transcriptional regulator of both anti- and pro-apoptotic genes.

In adult mice, administration of LPS induces pro-inflammatory cytokines and anti-apoptotic response genes in the liver, mediated by NFkB activation. Newborn mice, however, exhibit delayed p65 presence in the nuclei of liver cells. LPS-induced hepatic apoptosis in neonates has not been characterized in a murine model.

We hypothesized that delayed p65 translocation could blunt the anti-apoptotic response to LPS, and lead to increased hepatic cell death for 3-day old (p3) mice.

**RESULTS**

**Fig 1: Adult liver nuclear p65, delayed nuclear p65 in p3 liver after 5 mg/kg LPS, WB**

**Fig 2: Adult and p3 anti-apoptotic genes, qPCR**

**Fig 3: Adult and p3 pro-apoptotic genes, qPCR**

**Fig 4: Adult vs p3 liver apoptosis, TUNEL staining**

**CONCLUSION**

We compared adult and p3 mice in nuclear p65 presence over a time-course of LPS exposure, gene expression of anti-apoptotic and pro-apoptotic markers, and TUNEL staining. We observed delayed nuclear p65 in the p3 mouse liver, blunted anti-apoptotic gene expression, and a delayed increase in pro-apoptotic gene expression after 24 hours of LPS.

TUNEL staining revealed an increased number of apoptotic cells in the p3 liver after 24h LPS compared to adults. These data support our hypothesis.