30 - Title: Understanding the Gliogenesis of Necrotizing Enterocolitis

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BACKGROUND
Necrotizing enterocolitis (NEC) is an intestinal disease primarily affecting preterm infants, leading to gut inflammation and systemic progression. A major long-term consequence is neurodevelopmental impairment, including decreased white matter and brain volume. Although the exact mechanisms are unclear, NEC activates microglia, triggering cytokine production that disrupts oligodendrocyte (OL) and oligodendrocyte precursor cell (OPC) development. Existing NEC animal models lack disease severity control. Our lab developed a mouse model using different concentrations of dextran sodium sulfate (DSS) to study NEC severity and its neurodevelopmental effects. We hypothesize that increasing NEC severity will correlate with greater OPC, mature OL (mOL), and white matter loss.

METHODS
P6 and P21 mouse brain slices underwent immunohistochemistry from varied NEC severities (0.25–2% DSS), formula control, and breastmilk (BM) control groups. OPCs and mOLs were quantified in the hippocampal CA1 and corpus callosum (CC), and myelination was measured via MBP fluorescence. Data were analyzed using one-way ANOVAs.

RESULTS
P6 mice showed graded OL lineage loss, with significant reductions in OPC and mOL density in severe NEC. P21 mice had decreased mOL density and increased OPC percentages in non-BM groups, alongside CC white matter loss.

CONCLUSION
Graded NEC severity alters OL lineage and white matter, contributing to long-term neurodevelopmental deficits. Breastfeeding mitigates these effects.