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Inflammatory Mediators and Fibroblasts

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Fibroblasts are mesenchymal cells that produce and release extracellular matrix proteins such as collagens, laminins and proteoglycans during both tissue development and in response to tissue injury. In terms of tissue repair following injury, excessive healing of the injury can lead to tissue scarring. Fibroblasts have also been shown to affect neighboring cells through the release of cytokines, growth factors, and/or differentiation factors. Despite this information, little is understood about the fibroblast response to diseases characterized by acute inflammation. Necrotizing enterocolitis (NEC), is a devastating disease in response to microbial infiltration which mostly affects the gastrointestinal tract of premature infants. NEC results in nearly 2% of deaths of newborns annually. Survivors of NEC display excessive scarring in the intima of the intestinal epithelium and develop strictures leading to intestinal blockage. To that end, in the current study, we examined the fibroblast response following exposure to the following inflammatory cytokines, interferon gamma (INF-γ), interleukin-6, interleukin-8 and the anti-inflammatory cytokine, interleukin-10. Additional inflammatory mediators *N*α-Formyl-L-methionyl-phenyalanine (fMLP), Lipopolysaccharides ((LPS), 4524 [*E. coli*], 2515 [*E. coli*], *Salmonella minnesota*) were also exposed to fibroblasts. Fibroblast morphology shows that 4524 resulted in substantial growth with cells displaying a spindle shape while exposure to INF-γ resulted in reduced growth with cells displaying less of the spindle morphology, but with enlarged nuclei. Other inflammatory mediators displayed minimal differences. We examined CD36 in fibroblasts exposed to 4524 and INF-γ. Expression was reduced in INF-γ-exposed cells suggesting that CD36 may be involved in cellular over-growth of fibroblasts exposed to LPS. Taken together, excessive growth and activation of fibroblasts may contribute to acute inflammatory conditions like NEC.

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