

# Study reveals immune cells that drive gastrointestinal disease in babies

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A Yale-led research team has identified the immune cells that drive the inflammation observed in necrotizing enterocolitis (NEC), a severe gastrointestinal complication that often affects infants born prematurely.

Writing in the *Journal of Experimental Medicine*, a team led by Yale's Liza Konnikova describes how a type of innate immune [cells](#) known as monocytes flock to the intestine of babies with NEC and cause severe inflammation.

While previous studies have noted several [cell types](#) involved in the dysregulation caused by the [gastrointestinal disease](#), this is the first time scientists have used single-cell techniques to study it.

"NEC is a very severe [disease](#); about 40% of babies who have it don't survive, and those who do survive have very significant lifelong complications," said Konnikova, an assistant professor of pediatrics and obstetrics, gynecology, and reproductive sciences at Yale School of Medicine and senior author of the study. "This disease has been studied for probably over 100 years, but with the advent of single-cell technology, people have been able to study this and other diseases much better.

"What we identified is a particular subtype of monocyte, or [immune cells](#), that are enriched in patients with NEC and are likely either causing the disease or making the disease much worse."

Because of these findings, researchers now have a better understanding of the immune dysregulation that occurs in patients with NEC and potential therapeutics that might be used to treat it.

In forthcoming research, Konnikova and her team will explore biomarkers associated with the disease, therapeutic targets, as well as the role of the adaptive immune system in NEC.

**More information:** Oluwabunmi O. Olaloye et al, CD16<sup>+</sup>CD163<sup>+</sup> monocytes traffic to sites of inflammation during necrotizing enterocolitis in premature infants, *Journal of Experimental Medicine*

(2021). [DOI: 10.1084/jem.20200344](https://doi.org/10.1084/jem.20200344)

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