Infant immunity, though fleeting, found to be strong

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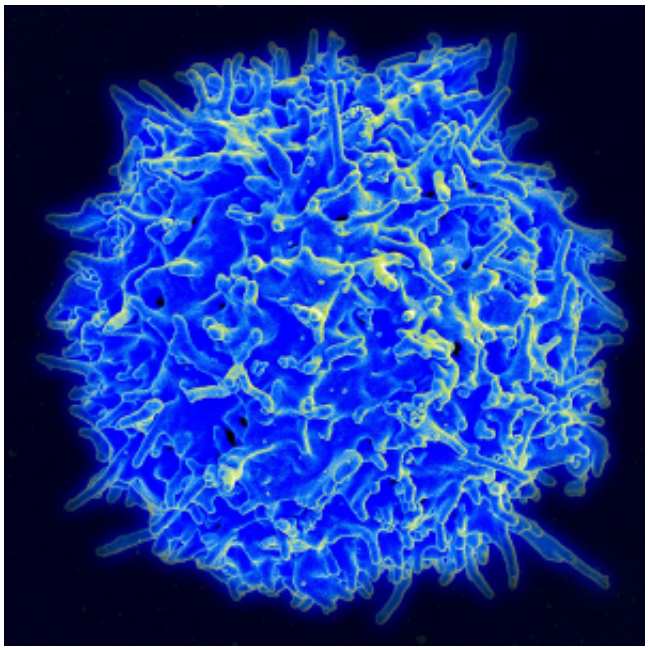

Scanning electron micrograph of a human T lymphocyte (also called a T cell) from the immune system of a healthy donor. Credit: NIAID

(Medical Xpress)—"Forgetful" immune systems leave infants particularly prone to infections, claims a new Cornell study. Upending the common theory that weak immune cells are to blame, the study has found that infants' immune systems in mice actually respond to infection with more speed and strength than adults. Yet the immunities they create fail to last.

Published in the *Journal of Immunology* in May, the discovery suggests a new angle immunizations could take to better protect infants and children from infectious diseases.

Infectious disease accounts for more than one-third of infant deaths worldwide, according to the World Health Organization. Immunizations protect people by "teaching" immune systems to remember pathogens. But infant immunities rapidly wane, often requiring extra booster shots after an initial vaccination.

"The perfect vaccine would be a single dose given at birth that generates long-lasting immunity," said immunologist Brian Rudd at the College of Veterinary Medicine, the study's lead author. "No such vaccine exists because we haven't understood why infants rapidly lose immunities. Our finding could change the way we immunize infants and ultimately lead to more effective ways of enhancing immunity in early life."

Immunity against most microbes depends on forming memory T cells that remember specific pathogens and can rapidly respond to future infections. Adults almost always generate large numbers of effective memory T cells during infection, around 10 percent of which stay in a long-lived memory pool to rapidly respond next time.

Rudd found that newborn T cells generated in response to infection met dramatically different fates. When met with the same pathogen, newborn immune systems in mice made T cells that responded more rapidly to infection than adult cells, but quickly became terminally differentiated, never making it into the memory pool. This disproves the common theory that newborn immune cells have a weak or suboptimal response to infection.

"Surprisingly, we found that newborns' cells actually responded more vigorously to infection compared to adults," said Rudd, assistant professor of immunology. "We also found that newborns' cells go through their lifespans more quickly and die off sooner, before they can give rise to memory T cells and remember what they've learned. So the immune system is forced to start the learning process over again when infected by the same pathogen later in life."

Rudd's lab is adjusting the expression of different

proteins in different-aged T cells to determine how developmental variation in these factors influences memory cell behavior and fate. The researchers are also performing genomewide analyses of different-aged T cells to find the genes that code these differences.

"We hope to find a way to make neonatal cells behave more like adult cells in how they learn from vaccines and respond to infection," said Rudd. "Knowledge gained from these studies could be used to design more effective therapeutic interventions and vaccines that can be safely administered in early life."

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