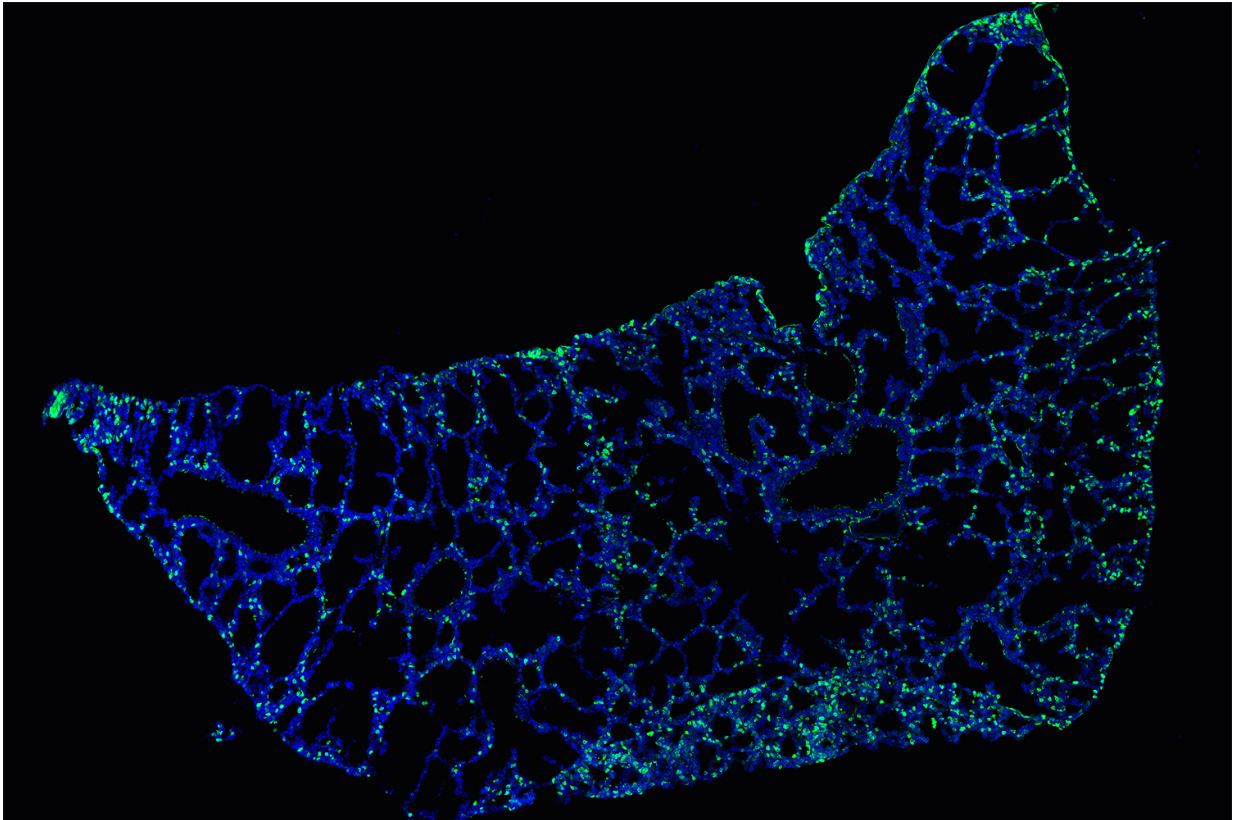


# First breath shapes the lung's immune system

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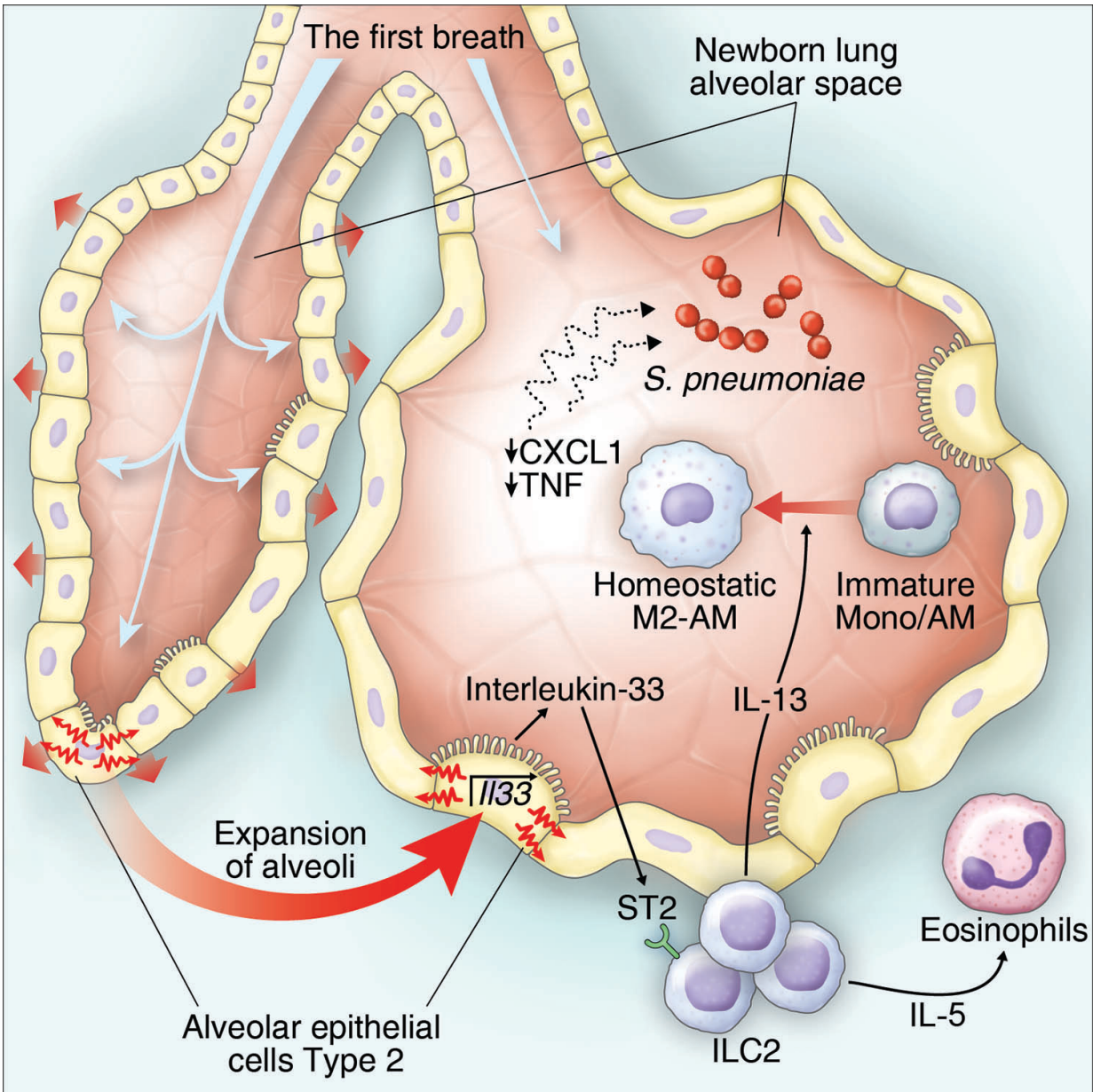
Fluorescent staining of IL-33 in a newborn mouse lung. Credit: CeMM/Simona Saluzzo

The lung is an important interface between the body and the outside environment: with each breath, a surface of roughly 100 square meters exchanges oxygen for carbon dioxide. More than 10,000 liters of air pass adult lungs every day and with this come numerous viruses, bacteria and

pollutants, which need to be prevented from entering the body.

To defend the organism from these intruders, the lungs harbor their own arsenal of highly specialized immune cells that are equipped to maintain the balance between host defense and tissue quiescence. However, how this balanced immune homeostasis in lungs emerged after birth, was largely unexplored. Now, for the first time, the group of Sylvia Knapp, Director of Medical Affairs at CeMM Research Center for Molecular Medicine of the Austrian Academy of Sciences and Professor of Infection Biology at the Medical University of Vienna showed with the help of mouse models that the very first breath of a newborn releases crucial signals that shape the lifelong immunological milieu of lungs.

The study, published in *Cell Reports*, reveals that the mechanical forces of spontaneous ventilation at birth lead to the release of interleukin (IL)-33, a cytokine with a wide-range of effects: So-called "type 2 [innate lymphoid cells](#)" (ILC2s) follow the IL-33 signal and migrate into the [lung](#) tissue, where they release IL-13, another cytokine. This second signal determines the fate of alveolar macrophages by inducing the anti-inflammatory M2 phenotype.



A graphical abstract of the study. Credit: CeMM/Simona Saluzzo

"ILC2-cells are crucial in defending the lungs against parasites or influenza viruses, but little was known about their role in lung homeostasis", first author Simona Saluzzo, PhD Student at CeMM and the Medical University of Vienna, explains. "Now we understand that

right after birth, ILC2 are responsible for the differentiation of [alveolar macrophages](#) into specialized cells that keep the immune system in check and ensure that the lungs stay calm and healthy to ensure proper gas exchange."

These ILC2-induced effects protect the lungs from excessive inflammation to daily encountered environmental triggers - but there is a catch, senior author Sylvia Knapp emphasizes: "We could show in our study that the described mechanisms are crucial in achieving lung quiescence after the first contact with the outside world. However, these processes at the same time increase the susceptibility to bacterial infections, such as caused by pneumococci. In other words: The mechanism that maintains the lung function of gas exchange at the same time explains why bacterial pneumonia is the primary cause of death by an infectious disease in Western countries."

**More information:** "First-breath induced type-2 pathways shape the lung immune environment" *Cell Reports*, [DOI: 10.1016/j.celrep.2017.01.071](#)

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