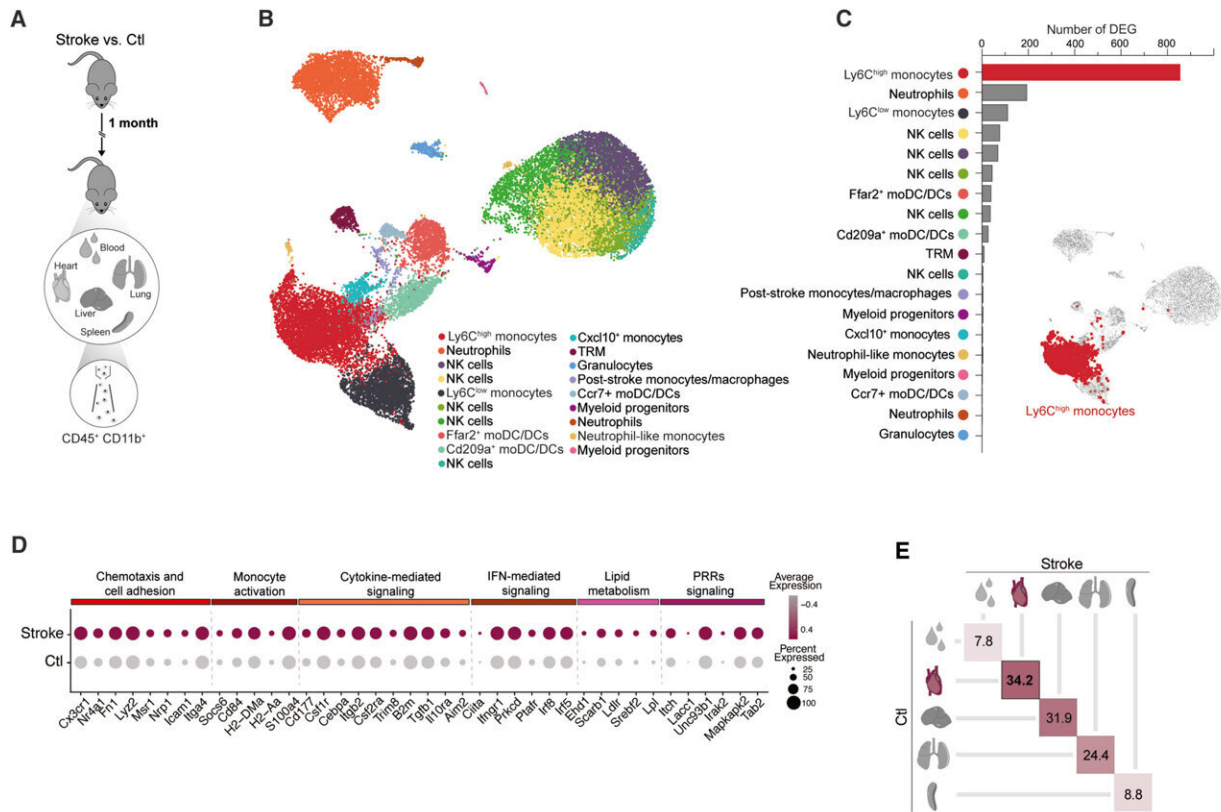


# Brain-heart axis study finds strokes change epigenetics of immune system

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Stroke induces long-term inflammatory changes in systemic monocytes/macrophages. Credit: *Cell* (2024). DOI: 10.1016/j.cell.2024.06.028

A stroke not only causes acute damage to the brain, but can also have long-term health implications for other organs—such as the heart.

"However, there has been little research to date on the effects of brain injuries on systemic immunity," says Professor Arthur Liesz from the Institute for Stroke and Dementia Research (ISD) at LMU University Hospital. Liesz is the principal investigator of a new study that was recently [published](#) in the journal *Cell*.

The researchers worked on the hypothesis that the high rate of comorbidities that develop after a stroke could have a common immunological cause. And they actually managed to find it: The origin of the dysfunctions in other parts of the body lies in the immunological memory of the blood-forming cells in bone marrow.

Using single-cell sequencing techniques, Liesz and his team demonstrated the presence of permanent proinflammatory changes in the transcriptome of certain immune cells (monocytes/macrophages) in several organs. In other words, certain gene segments are transcribed differently there after the stroke, which unbalances the proteome. These epigenetic modifications occur most frequently in the heart, where they can cause scarring and impair pumping function.

"We managed to identify the protein IL-1b as the main culprit for the [epigenetic modifications](#) that affect immunological memory after a stroke," says Liesz.

## **Promising therapeutic approaches on the horizon**

The researchers demonstrated in a [mouse model](#) the connection between modified blood formation in [bone marrow](#) through overexpressed IL-1b and cardiac dysfunctions. Moreover, they showed that blocking IL-1b and inhibiting migration of the proinflammatory cells to the heart both successfully prevented cardiac problems after a stroke.

"These findings are hugely significant, as they open up the promise of

effective therapeutic approaches for the prevention of secondary cardiac conditions after a [stroke](#)," says Liesz. The authors of the study believe that the epigenetic mechanisms they described for the reprogramming of the immune system in the [brain](#)-heart axis will create a new framework for explaining the development of various IL-1b-mediated comorbidities.

**More information:** Alba Simats et al, Innate immune memory after brain injury drives inflammatory cardiac dysfunction, *Cell* (2024). [DOI: 10.1016/j.cell.2024.06.028](https://doi.org/10.1016/j.cell.2024.06.028)

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