

Researchers reveal a new mechanism that regulates intestinal stem cells

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Confocal microscopy images (wide shot and a close-up) of a drosophila posterior midgut, showing the different intestinal cell types (stem cells in green, differentiated enterocytes in blue and enteroendocrine cells in red). Credit: Foundation for Research and Technology - Hellas

Adult stem cells have attracted great scientific interest because of their ability to self-renew and differentiate into other cell types. In an <u>article</u> published in *Nature Communications*, researchers reveal a new mechanism that regulates the proliferation and differentiation of intestinal stem cells.



Their research highlights the important role of epigenetic regulation of a micro-RNA during the differentiation process of intestinal stem cells into mature enterocytes. Epigenetic modifications affect <u>gene expression</u> without altering the DNA sequence. They can be influenced by environmental factors, lifestyle and aging and are often reversible.

The researchers, from the National Kapodistrian University of Athens (NKUA), the Institute of Molecular Biology and Biotechnology (IMBB) of the Foundation for Research and Technology-Hellas (FORTH), and Harvard University, discovered that, during aging, epigenetic deregulation leads to malfunction of stem cells and disruption of the cellular integrity of the gut.

The researchers used genetic lineage-tracing methods to study the function of intestinal stem cells of Drosophila, an experimental model organism that continues to provide groundbreaking basic research. By mining gene expression databases, they also showed that these epigenetic mechanisms are evolutionarily conserved in humans.

The results of the study could be used to develop targeted treatments for intestinal diseases or nutritional interventions to ameliorate intestinal dysfunction due to aging.

More information: Zoe Veneti et al, Polycomb-mediated silencing of miR-8 is required for maintenance of intestinal stemness in Drosophila melanogaster, *Nature Communications* (2024). DOI: 10.1038/s41467-024-46119-9

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