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The Hidden Dance of Gut Neurons: Unraveling Neurogenesis and Neuronal Renewal in the Enteric Nervous System

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Commentary

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Abstract

"The findings are noteworthy because they provide a better knowledge of how our stomach neural system works. The continual turnover and regeneration of nerve cells is critical to the health of the stomach. This research may also have implications for understanding and treating diseases involving the neural system of the stomach, such as gastrointestinal problems. Overall, it's an interesting discovery that calls into question our assumptions about nerve cells in the adult stomach and opens up new avenues for future research"

Keywords: The Enteric Nervous System; The Enteric Neural Progenitor Cells; Intestinal Neurons; Neurogastroenterology; Muscle-Myenteric Plexus; Muscularis Macrophages

Commentary

The enteric nervous system (ENS) is a complex network of nerve cells that control different parts of the digestive tract [1]. Up until recently, it was thought that the amount of enteric neurons didn't change as a person aged [2]. But new information shows that healthy people have neurogenesis and neuronal turnover in their ENS [3]. Understanding how neurons die and are restored in the ENS is very important for figuring out what causes digestive problems and coming up with ways to treat them. This article is an overview of a study that looked at how quickly enteric neurons die and whether or not healthy adult mice have enteric neural progenitor cells (ENPCs) [3,4].

Several types of tests were done on healthy adult mice to look at the death of intestine neurons and the birth of new neurons. Researchers marked neurons in the myenteric ganglia of adult mice that were going through apoptosis with an antibody that was specific for cleaved caspase-3. A brightly colored caspase-3/7 substrate was used to show that caspase-3 was working biochemically. Marking cells that were dying in living things with propidium iodide was another way to show that they were there. Neurogenesis was studied by using mice with the gene NOS1- CREERT2. tamoxifen was used to mark the nos1-expressing neurons with tdTomato. The loss of marked neurons and changes in the number of neurons in the myenteric ganglia were looked at using in vivo imaging and immunohistochemistry [3,5].

The results showed that gut neurons die in all healthy adult mice. By making cleaved caspase-3, a large number of myenteric neurons showed signs of death. This result was the same as what other studies had found, which was that young people have dead neurons in their myenteric plexus. Biochemical tests and imaging in vivo also showed that neurons were dying. Even though some groups of neurons were lost, the total number of neurons in the myenteric ganglia stayed about the same. This shows that neurons can be replaced. This study also shows how muscularis

macrophages help get rid of dead neurons and the waste they leave behind. In the muscularis, phagocytic macrophages were found to get rid of cholinergic neurons. Flow cytometry and imaging techniques showed that muscularis macrophages have neural inclusions, which supports their role as active cleaners. Nestin, a cytoskeletal protein that is made by many neural stem cells, was used in the study to find possible ENPCs. Neutrospheres made from the longitudinal muscle-myenteric plexus layer of the small intestine were able to turn into neurons in the lab and then engraft into the myenteric ganglia of adult mice, where they continued to grow and develop. Using a nestin-GFP mutant mouse as a model, researchers looked for possible ENPCs in the body. In the small intestine, nestin-GFP+ cells made a big network in the submucosal zone, the muscular layers, and the myenteric plexus [3,5].

These data tell us a lot about neurogenesis and how often neurons in the ENS of healthy people die and grow back. The fact that enteric neurons die at a high rate shows that new neurons are always needed to keep the ENS running well. The fact that potential ENPCs that make nestin have been found backs up the idea that the adult intestine has a group of neural stem cells that change over time. The fact that muscularis macrophages eat dead neurons and get rid of neuronal trash shows how important immune cells are for keeping the ENS in balance. This process of getting rid of waste may play a role in the development of gut diseases like Parkinson's disease and enteric neuropathies that cause the loss of enteric nerves. Focusing on how the immune system gets rid of waste could help restore neural balance in these cases [5].

We also wonder where ENPCs come from and what they do in the ENS because of this work. More study needs to be done to find out if ENPCs come from local stem cells or from somewhere else, like circulating progenitors or migration from other parts of the nervous system. To understand how ENPCs are controlled and to use their therapeutic potential, it will be important to figure out the molecular markers and signaling pathways that are involved in their selection, proliferation, and differentiation.

In the end, the study shows that the ENS of healthy adults has regeneration and neuronal change. We know more about how the ENS stays healthy and changes now that we know a lot of enteric neurons die, that muscularis macrophages are involved in neuronal clearance, and that possible ENPCs have been found. These results are important for coming up with new ways to treat stomach problems. They also open the door for more research into how neurogenesis works in the ENS and what it means for how it works.

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