How the brain compensates for sensory loss and points to its early evolutionary roots

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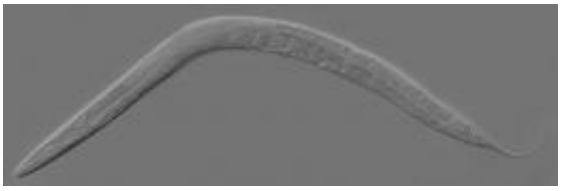

Caenorhabditis elegans. Image: Wikipedia.

The human brain has the remarkable capacity to respond to sensory loss by boosting the remaining functioning senses. Through a compensation mechanism in the brain, known as cross-modal plasticity, some senses are enhanced following the loss of other sensory input, such as the improvement of hearing in people who are blind.

So far, this mechanism has been studied in humans and other mammals, under the assumption that it is a function of complex brains consisting of billions of cells, such as in humans. Now, a new international study led by researchers at The Hebrew University of Jerusalem, revealed that the brain's compensatory mechanism is a basic feature that also exists in less complex nervous systems. The study has also exposed the way the mechanism works in the brain through an inter-sensory signaling system.

The research, which was published in the journal *PLOS Biology*, was carried out jointly at the Institute for Medical Research Israel-Canada

(IMRIC) at the Hebrew University's Faculty of Medicine, in collaboration with the MRC Laboratory for Molecular Biology in Cambridge, UK; and the Fred Hutchinson Cancer Research Center in Seattle, United States.

"One of the most fascinating capabilities of the brain is the ability to compensate for the loss of sensory input. We can learn a lot from how a relatively simple nervous system is able to exercise a brain function as sophisticated as this. In this research, we revealed an upper bound for the neural complexity required for a compensating mechanism like this, allowing us to examine and understand more easily how it works, all the way from the molecular level to the behavioral level," said Dr. Ithai Rabinowitch, who led the research during his work in the Department of Medical Neurobiology at the Hebrew University.

To better understand how cross-modal plasticity functions, the research team examined an organism with a substantially less complicated nervous system than is found in humans—the roundworm C. elegans. It is one millimeter long, feeds on bacteria, and its nervous system has only 302 neurons (compared to 100 billion in the human brain).

The researchers examined the relationship between the loss of the sense of touch and the possible enhancement of the sense of smell. To do this they focused on worms with a genetic mutation that eliminates their sense of touch.

They discovered that C. elegans mutants that cannot sense touch to the body exhibit an improved sense of smell. They were able to pinpoint this change in sensory performance to a change in strength of a specific synapse in the olfactory circuit.

"We were able to reverse these effects by artificially stimulating the touch neurons and by engineering a new synapse into the olfactory

circuit," Dr. Rabinowitch explained. "We've still got a long way to go, but we can already think of future applications for treating unwanted side effects following the loss of sensory input."

This research adds to a series of studies investigating the role of neuropeptides in the brain's inter-sensory signaling, and expands the knowledge about the cellular and molecular processes underlying cross-modal plasticity. These results may also point to the ancient evolutionary roots of this compensation mechanism, now that it has been revealed in a much less developed system than our own.

More information: Ithai Rabinowitch et al. Neuropeptide-Driven Cross-Modal Plasticity following Sensory Loss in Caenorhabditis elegans, *PLOS Biology* (2016). DOI: 10.1371/journal.pbio.1002348

Provided by Hebrew University of Jerusalem