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New study finds key mechanism in calcium regulation

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All living cells keep their cellular calcium concentration at a very low level. Since a small increase in calcium can affect many critical cellular functions (an elevated calcium concentration over an extended period can induce cell death), powerful cellular mechanisms ensure that calcium concentration quickly returns to its low level.

It is known that impairments of cellular calcium regulation underlie almost all neurodegenerative diseases. For example, age-related loss of calcium regulation was shown to promote cell vulnerability in Alzheimer's disease.

In a study recently published in the Journal of Neuroscience, Hebrew University of Jerusalem researchers, along with others from Israel and the US, presented their findings of a previously undescribed cellular mechanism which is essential for keeping cellular calcium concentration low. This mechanism operates together with other already characterized mechanisms.

Dr. Shirley Weiss and Prof. Baruch Minke of the Hebrew University's Institute of Medical Research Israel-Canada (IMRIC) and the Edmond and Lily Safra Center for Brain Sciences (ELSC) characterized this mechanism using photoreceptor cells of the fruit fly, which is a powerful model for studying basic biological processes.

They found that a protein-designated calphotin (a calcium buffer) operates by sequestering elevated calcium concentration. Genetic

elimination of calphotin led to a light-induced rise in cellular calcium for an abnormally extended time, leading to retinal photoreceptor degeneration in the fruit flies.

The researchers stress that this kind of research, leading to a better understanding of the fundamental mechanisms underlying cellular calcium regulation, is critical for the development of new drugs and treatments for neurodegenerative diseases.

More information: Compartmentalization and Ca2 Buffering Are Essential for Prevention of Light-Induced Retinal Degeneration, Shirley Weiss, Elkana Kohn, Daniela Dadon, Ben Katz, Maximilian Peters, Mario Lebendiker, Mickey Kosloff, Nansi Jo Colley, and Baruch Minke, *The Journal of Neuroscience*, October 17, 2012. 32(42):14696 –14708

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