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Alzheimer's disease and cell death: A better understanding

Throughout human development our bodies are constantly balancing out cell death and cell survival. In fact, for a healthy life, damaged cells must die while strong cells survive. Normally our bodies control this by programmed cell death (PCD); however, problems with our natural mechanisms for “maintenance” can occur and cause many diseases. Autophagy is one type of cell death that is characterized by the degrading and recycling of various components of the living cell, and if enough of this degradation occurs, the cell will ultimately die. Deficiencies in levels of this cellular recycling mechanism have been linked to various diseases, including neurodegenerative diseases such as Alzheimer’s disease (AD). Abnormally low levels of Autophagy in Alzheimer’s brains have been shown to cause significant alterations, including an impairment of the protective properties Autophagy has on brain cells. By learning more about how proteins interact in the brain, we can better understand how Autophagy works to protect healthy cells and prevent disease by degrading damaged components. Once we know more about this process, we can potentially find treatments for these diseases by controlling the levels of cell death. Our research will focus on some of the proteins that interact with Beclin, a protein known to induce the Autophagy process. This is important to better understand the role of Beclin, and the proteins it interacts with, in cell death and ultimately in the treatment or prevention of Alzheimer’s disease. Understanding the process of cell death will not only help us with treatment of Alzheimer’s disease, but may also one day help with treatment of various other diseases such as cancer, Batten disease, and Huntington’s Disease.