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The good and the bad of a potential Alzheimer's target

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Research in fruit flies has shown that enhancing the production of a protein called neprilysin can reduce the formation of plaques and neuron death associated with Alzheimer's, at the expense of reducing the flies' lifespan.

The buildup of amyloid-beta protein plaques within the brain is a major hallmark of Alzheimer's, and one that is believed to contribute to disease progression. Normally, special enzymes chew up and clear these plaques, and deficiencies in these enzymes are one potential disease cause.

In fact, one major amyloid degrader called neprilysin (NEP) decreases naturally with age and may be the reason the elderly are more at risk for Alzheimer's. Enhancing NEP production might therefore be an attractive therapy, and studies in mice have suggested it has potential. However, no studies have really looked into possible adverse effects of over-activating NEP (after all, nature probably turns it off for a reason).

In this study, research groups led by Koichi Iijima and Kanae Iijima-Ando did just that, using transgenic fruit flies expressing human NEP and/or amlyoid-beta protein. On the positive side, NEP expression did reduce plaque deposits and neuron damage in the flies as expected; on the other hand, NEP also reduced the activity of important neural proteins called CREB proteins and shortened the average lifespan of the flies (normal flies live about 60 days) by about 10 days (although NEP-flies did live longer than those only expressing amyloid protein).

This study illustrates the care that must be taken when considering Alzheimer's treatments, and that it's critical to better understand normal aging when dealing with Alzheimer's or other age-related conditions.

Source: American Society for Biochemistry and Molecular Biology

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