Protecting against Alzheimer’s disease with immunotherapy

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A neuronal protein known as ankyrin G (ankG) evokes the production of antibodies which may reduce the level of beta-amyloid and its related neurological diseases reports a study published online this week in Molecular Psychiatry. The work suggests that ankG may be a potential target for immunization against Alzheimer’s disease (AD).

Taking advantage of naturally occurring immune responses, immunotherapy targeting pathological brain aggregates could help to modify the disease course of AD. Antonella Chadha Santuccione and colleagues found that AD patients who are immunopositive for ankG antibodies displayed stable cognitive functions when compared to the immunonegative AD patients. This suggests that endogenous ankG antibodies represent a protective mechanism that slows down the progression of the disease. To test the role of the immune response against ankG and the subsequent effect on beta-amyloid, the authors vaccinated AD-like mice with ankG, and found that immunization significantly reduces the presence of beta-amyloid plaques and improves dendritic spine structure. Further experiments showed that ankG is likely released through exosomes, and may accelerate the clearance of plaques by promoting the interaction of beta-amyloid and microglia in the brain.

This data helps us further understand the complex nature of AD development and points to ankG as a new and relevant key player in the mechanisms underlying the disease. The results suggest that ankG can function in humans by triggering an immune response and generating
ankG-reactive antibodies that act on beta-amyloid. They also provide evidence for a potential and crucial immunotherapy targeted to protect against AD.

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