

# Why are drug trials in Alzheimer's disease failing?

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An Editorial in this week's *Lancet* discusses the poor record of drug trials in Alzheimer's disease, following the dumping of semagacestat on the phase 3 scrapheap of other failed disease-modifying drugs for the condition.

Meta-analysis suggests some animal models inaccurately predict drug efficacy, while other problems could be poor methodology in animal studies or use of models that don't accurately reflect [disease progression](#) in humans. The Editorial says: "Current treatment targets patients with symptomatic Alzheimer's disease. But perhaps the disease is being treated too late, when damage is irreparable?"

The best time to treat Alzheimer's disease is likely to be before [memory loss](#) and tissue destruction occurs, but this is hard to model in animals. That means identifying people at risk of developing the disease, perhaps because of a [genetic predisposition](#) or by measuring biomarkers, such as the recently reported cerebrospinal fluid measurement of a mix of amyloid  $\beta$ 1-42 and phosphorylated  $\tau$  protein."

It concludes: "Drug-industry scientists are failing themselves if their animal studies are poorly done or use the wrong model, and their companies are failing academics who do their phase 3 trials with them, trial participants, and shareholders. Perhaps the problem is 'translational research' itself: a phrase much bandied around, but does anyone know what it really means, let alone how to do it?"

Provided by Lancet

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