

AD: Are we intervening too late?

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The debate by Emery and Ames is an interesting and important discussion. While Ames correctly emphasizes that pharmacological interventions in people with mild cognitive impairment have so far failed, Emery tries to take us a step back and suggests the interventions even earlier. She focuses on depression which is known to predict dementia and points to several manipulations which may perhaps delay or prevent this conversion. While theoretically appealing, there are no data to support this hypothesis. Neither such data will be forthcoming. It is unimaginable to conduct a controlled study in which some depressed patients will be treated for a long time, while other will serve as non-treated controls. However, this may be a mute point because, in any case, people who are depressed should be treated for their depression, whether by drugs or otherwise. It is quite conceivable, however, that people who are depressed should be treated more aggressively for any comorbid cardiovascular disease, as well as encouraged to engage in a cognitively stimulating activity, as well as physical activity, even lacking evidence-based proof that those will reduce or delay their chances of becoming demented.

The association of depression, and particularly late-life depression, with dementia is intriguing. Emery suggests

several possible explanations for this relationship, and future research should be directed at elucidating each.

Ames reviews the present knowledge concerning pharmacological interventions in early dementia. Lacking understanding of each process leading to Alzheimers disease, these followed various hypotheses: antioxidant manipulator (vitamin E), cholinesterase inhibitors or anti-amyloid interventions. All of which have failed. As discussed by Emery, late-life dementia is the final result of several converging processes and if this is the case, interventions against specific components are unlikely to yield positive results. Interventions using several drugs simultaneously are technically challenging and prohibitively expensive, and thus unlikely to be conducted.

No attempts have been done so far to enhance endogenous processes which must exist to compensate for neurodegenerative processes. Too little is known about the plastic changes in the brain, which limit the extent of damage. Cognitive training may be one such intervention, but it is completely unknown which type of training should be most successful, and whether it should be individually targeted.

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