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The elderly are not only at greater risk of developing Alzheimer's disease (AD), but are also less likely to receive appropriate therapies for AD, potentially resulting in poorer clinical outcomes. Over the last decade, there has been a significant increase in the number of clinical trials for AD aimed at slowing or preventing cognitive decline. Most of these trials have failed to demonstrate statistically significant improvements in cognitive outcomes, largely due to the absence of sensitive and specific outcome measures that reliably reflect the cognitive effects of potential treatments. Currently available measures of cognition include scales that primarily reflect episodic memory (i.e. memory tests), which have been found to be poor predictors of disease progression. Although there are tests of processing speed and executive function that are sensitive to the cognitive deficits observed in AD, these tests have not been used to assess the effect of potential therapies in a placebo controlled design. Thus, new measures of cognition must be developed in order to obtain a more complete picture of the cognitive deficits associated with AD and the effect of potential therapies. The proposed work aims to address this gap by developing a battery of measures that will permit sensitive assessment of cognitive change with the use of placebo control conditions. We will take advantage of the high availability of longitudinal data on older adults collected over the past several decades as part of the Health and Retirement Study (HRS), and will recruit a sample of healthy elderly adults to whom we will administer the new battery of tests of cognition to evaluate the ability of these tests to capture the cognitive deficits associated with AD. During the initial phase of data collection, we will also evaluate the stability of the battery of tests over time and identify factors that predict the stability of the test battery over time. We will then administer the battery of tests to a sample of elders with mild cognitive impairment (MCI), AD, and healthy controls. We hypothesize that the battery of tests will be highly sensitive to cognitive change across diagnostic groups and that these changes will correlate with biomarkers of neuropathology (i.e. amyloid beta and tau pathology). In the second phase of data collection, we will administer the battery of tests to a subset of participants who will then be randomized to receive either a placebo or an active treatment for AD. We hypothesize that the placebo and treatment groups will differ on cognitive outcomes, as well as biomarkers of AD neuropathology. Finally, we will examine the association between changes in cognitive performance and incident dementia. This work is an important step in identifying new and more sensitive tests of cognitive change that can be used to assess the 82157476af