Genetic Endowments, Alzheimer's Disease, and Economic Outcomes

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Extended Abstract: This research examines how genetic propensity for Alzheimer's Disease (AD) is associated with lifecycle health and economic outcomes in the Health and Retirement Study. We consider two measures of genetic predisposition for AD: a polygenic score for AD (hereafter: *AD score*) and a variable indicating the presence of the APOE-ε4 allele. A higher AD score means an individual has an increased genetic risk for AD. Being a carrier of APOE-ε4 allele increases the likelihood of AD three to twelve fold depending on the number of copies possessed by an individual.

We make two broad contributions. One, we show that a genetic propensity for AD predicts a host of economic outcomes above and beyond AD itself, including lower education, worse labor market outcomes and a lack of retirement planning. Two, we find that these outcomes are not limited to individuals who are diagnosed with AD or related dementias—or even to those who exhibit cognitive decline according to standard measures. Together, these findings highlight how the use of genetic data can provide novel insights into AD, including how it manifests among those who are never diagnosed. The crucial idea is that genes that predict AD allow us to observe people who are not diagnosed or who did not exhibit cognitive decline—but might have under different circumstances. Ongoing research will examine whether lack of diagnosis or cognitive decline among people with relatively high genetic propensities for AD can be explained by environmental factors that prevent illness. For example, lower completed education explains relatively worse AD-related outcomes among people with similar genetic propensities for AD. Identifying these protective

or insulating factors could inform research on new treatments. As part of our ongoing agenda, we are examining whether these genetic associations are large enough to provide clinically useful information. Understanding interactions between genetic markers and environmental factors is critical, since combining genetic and environmental factors may help to detect at-risk populations better than using either source of information in isolation.

More specifically, while our research is ongoing, we currently present four main sets of results. First, the AD genetic measures predict cognitive decline as well as diagnosis of a memory-related disease in our sample. Interestingly, carrying the APOE allele increases the probability of memory-related disease diagnosis even after flexibly controlling for measures of cognitive decline.

Second, the genetic measures predict a host of later-life economic outcomes, including increased probability of retirement and lack of financial and later-life planning (e.g., lower probability of holding long-term care insurance, having a will, or designating power of attorney for healthcare). These results hold even among those who do not exhibit cognitive decline as well as those not diagnosed with a memory-related disease.

Third, there is a strong relationship between the AD score and educational attainment, suggesting that some of the relationships we find between the AD genetic measures and economic outcomes may operate through lower educational attainment. This empirical link is, to our knowledge, unique to the literature and merits further exploration as several distinct models could explain it. For example, it could suggest that AD manifests very early, but could also suggest that individuals who inherit AD genes live in households with fewer resources.

Fourth, both educational attainment and genetic endowments for education (i.e., the educational attainment polygenic score, hereafter the EA score) reduce time to diagnosis of a memory-related disease among those who experience cognitive decline, which illustrates selection into diagnosis. On the other hand, educational attainment and the EA score are associated with less cognitive decline and better economic outcomes. Thus, education and genetic endowments for education are protective and/or insulating: they prevent cognitive decline and/or reduce the impact of cognitive decline on economic outcomes.

In subsequent analyses, we will explore other factors that might be protective or insulating, such as proximity to children, having a healthy spouse, access to medical care, parents' experience with memory-related disease and long-term care, and occupation. Together our findings provide a more complete understanding of the ramifications of a genetic propensity for AD, which may capture decline that is otherwise unobserved. Our findings also suggest that there are environmental factors that contribute to lack of cognitive decline and AD diagnosis among those with a relatively high genetic propensity for AD. In other words, there are factors that may actually improve

matters for those who may otherwise have experienced worse outcomes. Understanding these factors is important for guiding effective treatments and interventions.