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Short communication

Does educational attainment shape reactions to genetic risk for Alzheimer's disease? Results from a national survey experiment

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ABSTRACT

While higher education is associated with healthy lifestyles and health literacy, it remains unclear whether education shapes reactions to varying levels of genetic risk for Alzheimer's disease (AD). In this study, participants (N = 701) in the National Genetic Risk Survey Experiment (NGRISE) received a hypothetical genetic risk assessment for AD (ranging from 20 to 80% lifetime risk) and then completed items on their cognitive (perceived threat to health), emotional (general negative affect), and anticipated behavioral (seek information, improve health behaviors, engage in public or private civic action) reactions to this risk. Individuals with a college education showed reactions to increasing genetic risk approximately twice or several times as strong relative to those of individuals with lower (high school, HS) education. In fact, behavioral reactions do not significantly increase with AD risk among those with HS education. Some educational differences in risk response widen at older ages.

Educated individuals live longer, healthier lives across diverse measures of well-being, physical functioning, and disease burden (Cutler and Lleras-Muney, 2010; Mirowsky and Ross, 2003). However, research on educational health disparities typically focuses on how educated individuals have fewer health problems or manage existing ones, not on how they might negotiate specific risks associated with possible future disease onset. Because contemporary health and insurance providers often monitor, describe, and regulate health in terms of portfolios of specific risks for major diseases, these risks no longer sit at the margins of everyday life as they once did prior to advances in medical and genetic technology (Almeling and Gadarian, 2014; Timmermans and Buchbinder, 2010). Individuals possess an unprecedented level of awareness about their own genetic and lifestyle vulnerabilities for certain major diseases such as cancer, heart disease, or dementia, and often are told by providers to mitigate or manage associated risks. Therefore, understandings, motivations, or actions organized around specific health risks – as distinct from general maintenance of a healthy lifestyle – comprise a potentially important yet largely unexplored pathway linking education to health across the contemporary life course.

As a prime example, Alzheimer's disease (AD) is a leading cause of death in the United States and accounts for up to four-fifths of all dementia cases (Alzheimer's Association, 2016). Dementias produce staggering economic, medical and social costs worldwide estimated at $604 billion annually (Wimo et al., 2013). While AD presently is without any known cure, public awareness campaigns for AD have placed an emphasis on managing disease onset and severity, as AD risk may be manageable to some extent through lifestyle choices (Baumgart et al., 2015).

Recommended preventative measures against AD, such as physical activity, healthy diet, and routine cognitive and social stimulation, are notably more common among those with higher levels of education (e.g., Bennett et al., 2003; Cutler and Lleras-Muney, 2010; Stern, 2012). Physical activity, which may be especially preventative, is linked to education independently of important pre-educational differences such as early-life cognition and parental social class (Clouston et al., 2015; Cutler and Lleras-Muney, 2010). While education is associated with healthy lifestyles, it remains unclear whether education influences the perception and management of Alzheimer's risk itself. Maintaining

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health in the presence of known risk departs substantially from routinized health lifestyles, in terms of the nature and frequency of health perceptions, behaviors, and emotional states (Cockerham, 2005, 2013) – which makes educational differences in living with risk important to examine in their own right.

The ascertainment of genetic risk for major diseases such as Alzheimer's is increasingly common. A number of AD risk alleles (e.g., APOE-e4) have been identified, and numerous individuals have elected to undergo AD genetic testing (Ashida et al., 2010; Chao et al., 2008; Green et al., 2009; Rahman et al., 2012; Roberts et al., 2004). These studies often find that education is associated with undergoing testing or deciding to learn test results.

However, studies have not resolved how education shapes reactions to AD genetic risk information itself. Genomic research tends to view education as a demographic control variable, rather than as a factor that might fundamentally shape how individuals react to – and subsequently manage – genetic risk. Living with genetic risk is a liminal state in which one is a "patient-in-waiting" and is motivated to manage any associated uncertainty about future health to the extent possible (Almeling and Gadarian, 2014; Timmermans and Buchbinder, 2010). At the same time, however, individuals greatly differ in resources for living with risk, which should fundamentally alter how one's reactions to genetic risk information unfold.

Education provides a bundle of diverse health resources, making it a compelling potential explanation for why individuals react differently to genetic risk. As education increases, individuals generally show greater material (income, wealth, insurance), behavioral (healthy lifestyles), psychological (e.g., conscientiousness, sense of control), cognitive (general and specific problem-solving skills), social (e.g., social support and networks, lower occupational stress and work flexibility), and cultural (e.g., exposure to health norms) resources for maintaining good health (e.g., Cockerham, 2013; Cutler and Lleras-Muney, 2010; Mirowsky and Ross, 2007). Taken together, these bundled resources seen with higher education should change the apparent salience, threat, manageability, and importance of AD genetic risk, thus vitally altering how individuals respond to and manage a given level of AD risk. Education is also linked to health literacy itself, though this may partly reflect differences in cognition prior to schooling (Murray et al., 2011).

AD is most common at older ages (Alzheimer's Association, 2016; Tschanz et al., 2012). In line with this fact, public surveys consistently show that individuals associate dementia with aging (e.g., Cahill et al., 2015). Therefore, age may further enable or activate AD risk response across levels of education, as it may make AD risk seem more salient.

1. Overview of the present study

In this study, we use national vignette data to generate new insights about educational differences in reactions to Alzheimer's genetic risk. By varying risk randomly across respondents, we help resolve the previously obscured roles of educational attainment and participant age in shaping risk reactions. Our primary hypothesis is that education is linked to strengthened reactions to Alzheimer's risk, and our secondary hypothesis is that age will serve to further increase these educational differences.

Relative to prior work, our randomized vignette approach overcomes limitations with sample selection, lack of risk variation, and omitted health variables. Patients who participate in genomic disclosure studies usually are "already part of the medical system," in that they have enrolled in a study for health reasons or have been referred on the basis of neurological screening or preexisting conditions (Almeling and Gadarian, 2014). Analysis of patient populations is at odds with devising preventative efforts against Alzheimer's in the general population. Moreover, patient studies involve disclosures regarding specific genes (e.g., APOE alleles) and their associated lifetime risk estimates, which inhibits drawing conclusions about how reactions vary across *levels* of risk. Finally, omitted health variables are an important drawback of non-experimental studies, as health risks and states are mutually correlated and difficult to isolate specifically. Toward this end, our vignette data target Alzheimer's risk in particular.

Our research design analyzes in-the-moment reactions to hypothetical genetic risk. We argue that these momentary reactions provide useful benchmarks for how people *assign meaning* to risk, by revealing how individuals direct their attention and subjectively experience various thoughts and emotions about given health states (Almeling and Gadarian, 2014; Ashida et al., 2010; Stivers and Timmermans, 2016; Timmermans and Buchbinder, 2010). How individuals react in the moment to risk disclosure may form a basis for any short- or long-term changes in distress or preventative health behaviors (Chao et al., 2008; McBride et al., 2010).

2. Materials and methods

2.1. Participants and procedure

In January 2011 the second and third authors commissioned the nonpartisan research firm YouGov (YG) to run the National Genetic Risk Survey Experiment (NGRISE), by recruiting a nationally representative sample of respondents for an online survey. NGRISE obtained an AAPOR Category 3 response rate of 39% (a standard response rate for national public opinion polls) and YG studies generally yield estimates similar to those obtained by telephone surveys (Almeling and Gadarian, 2014). The University of California-Berkeley's Committee for the Protection of Human Subjects approved NGRISE. Respondents provided informed consent.

After a self-reported AD diagnosis screen (those with AD were screened out) and a series of demographic and health questions, respondents viewed an Alzheimer's disease vignette ($N = 701$) constructed using language from the Mayo Clinic and National Institute on Aging. They were told: "Alzheimer's disease is a progressive brain disorder that slowly destroys memory and thinking skills and, eventually, the ability to carry out daily activities. Genetic, environmental, and lifestyle factors may increase or decrease a person's chances of developing Alzheimer's disease. Alzheimer's disease affects many women and men in America each year, and it is more common in people over the age of 60. There are no treatments for Alzheimer's disease, but some medications may keep symptoms from getting worse for a limited time." Following this, they were randomized to different levels of genetic risk (20%–80%, increments of 10%; $N = 100–101$ per risk treatment) and viewed a prompt that said "Now imagine that you take a genetic test, and it reveals that you have a [Risk Percentage] chance of developing Alzheimer's disease at some time in your life." Each respondent also saw a pictogram (shaded human figure) illustrating the risk level. Following this, respondents reported their reactions to their risk.

2.2. Measures

Reaction Scales (Cognitive, Emotional, and Behavioral). Similar to previous work (e.g., Almeling and Gadarian, 2014; Ashida et al., 2010; Chao et al., 2008; Green et al., 2009; Rahman et al., 2012), respondents reported their cognitive, emotional and anticipated behavioral reactions to genetic risk. For cognitive appraisals, they were asked whether they viewed their risk as "life-threatening," "dangerous," "serious," or signifying that they are "likely to develop" the disease (*anticipated threat to health* scale; Cronbach's