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The relative contributions of disease label and disease prognosis to Alzheimer's stigma: A vignette-based experiment

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ABSTRACT

Background: The classification of Alzheimer's disease is undergoing a significant transformation. Researchers have created the category of "preclinical Alzheimer's," characterized by biomarker pathology rather than observable symptoms. Diagnosis and treatment at this stage could allow preventing Alzheimer's cognitive decline. While many commentators have worried that persons given a preclinical Alzheimer's label will be subject to stigma, little research exists to inform whether the stigma attached to the label of clinical Alzheimer's will extend to a preclinical disorder that has the label of "Alzheimer's" but lacks the symptoms or expected prognosis of the clinical form.

Research questions: The present study sought to correct this gap by examining the foundations of stigma directed at Alzheimer's. It asked: do people form stigmatizing reactions to the label "Alzheimer's disease" itself or to the condition's observable impairments? How does the condition's prognosis modify these reactions?

Methods: Data were collected through a web-based experiment with $N = 789$ adult members of the U.S. general population (median age = 49, interquartile range, 32–60, range = 18–90). Participants were randomized through a 3×3 design to read one of 9 vignettes depicting signs and symptoms of mild stage dementia that varied the disease label ("Alzheimer's" vs. "traumatic brain injury" vs. no label) and prognosis (improve vs. static vs. worsen symptoms). Four stigma outcomes were assessed: discrimination, negative cognitive attributions, negative emotions, and social distance.

Results: The study found that the Alzheimer's disease label was generally *not* associated with more stigmatizing reactions. In contrast, expecting the symptoms to get worse, regardless of which disease label those symptoms received, resulted in higher levels of perceived structural discrimination, higher pity, and greater social distance.

Conclusion: These findings suggest that stigma surrounding pre-clinical Alzheimer's categories will depend highly on the expected prognosis attached to the label. They also highlight the need for models of Alzheimer's-directed stigma that incorporate attributions about the condition's mutability.

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1. Introduction

The classification of Alzheimer's disease (AD) has undergone several transformations. Changes in the 1970s eliminated the distinction between senile dementia, which referred to persons over 60 with memory problems, and Alzheimer's disease, which, at the time, referred to persons whose problems began at an earlier age. Alzheimer's thus shifted from a rare diagnosis that garnered little public attention, to a leading cause of death and therefore a pressing public health issue (Fox, 1989; Chaufan et al., 2012). This first transformation contributed to our current associations between Alzheimer's disease, old age, and severe cognitive impairment (Chaufan et al., 2012). These associations often focus on Alzheimer's disease in its most severe form, depicting persons with Alzheimer's as "empty shells" who experience a "death of the mind" (Van Gorp and Vercruysse, 2012), or Alzheimer's as a "death sentence"(Beard and Neary, 2013).

The classification of Alzheimer's disease is changing again. A

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work group from the National Institute on Aging and Alzheimer's Association has created the category of "preclinical Alzheimer's," characterized by biomarker pathology rather than observable symptoms (Sperling et al., 2011). Similarly, a work group composed primarily of European researchers has proposed a category of "asymptomatic at risk for Alzheimer's disease" based on similar criteria (Dubois et al., 2014). Preclinical Alzheimer's begins with the accumulation of amyloid beta in the brain and ends with the presence of neuro-degeneration and subtle signs of cognitive decline. In this new model, Alzheimer's disease spans from a stage where there is a complete absence of observable cognitive or behavioral symptoms, to later stages marked by behavioral changes, loss of awareness, and difficulty with activities of daily living.

While Alzheimer's first transformation contributed to its image as a severe form of cognitive impairment prevalent among the elderly, preclinical Alzheimer's may undo the cultural association between Alzheimer's and severe cognitive impairment. This may create a new cultural understanding of how to react to persons diagnosed with Alzheimer's. Each research group has recommended that preclinical Alzheimer's should be used only in research settings (Sperling et al., 2011; Dubois et al., 2014). But as policymakers place an increased emphasis on early detection and prevention of dementia through efforts such as the U.S. National Alzheimer's Plan (U.S. Department of Health and Human Services, 2014), the likely migration of preclinical AD categories to a broader range of settings raises questions about how expanding the "AD spectrum" will affect persons and their families.

The expansion of the AD spectrum can help research to prevent the disabling, symptomatic form of the condition (Sperling et al., 2014). Yet this strategy, designed to reduce the burden of disability, may create *spillover stigma*, where public fear, social distance, and negative reactions directed towards persons with symptomatic AD (see Werner, 2014 for a review) could spill over to persons given a preclinical AD diagnosis. Some argue that preclinical AD diagnoses will subject persons to "stigma and discrimination"(Gauthier et al., p.110); others worry that the diagnosis may be "distressing, alarming, and stigmatizing"(Le Couteur et al., 2013, p. 17); still others are concerned that persons testing positive for biomarker-based AD risk will face "stigmatization" (Luck et al., 2012, p. e50792).

Unfortunately, little research exists to inform whether the stigma attached to the label of clinical Alzheimer's will extend to the preclinical label (Gauthier et al., 2013; Le Couteur et al., 2013). Similarly, while there is much speculation that clinical stigma will spillover to at-risk labels for conditions like schizophrenia (Corcoran et al., 2005) or cancer (Lerman and Shields, 2004), the few empirical studies of spillover stigma for these conditions focus on patients' experiences of stigmatization rather than attitudes among members of the general public (e.g., DiMillo et al., 2015; Vodermaier et al., 2010).

Should spillover stigma occur for Alzheimer's, the stigma of Alzheimer's clinical form will encompass a constituency of people who are seemingly well, many of them employed and otherwise engaged in social, cultural and political spaces. Should it not, the experience of stigma might divide the Alzheimer's patient community into factions: those with clinical AD who experience both worse symptoms and greater stigma and those with preclinical AD. These scenarios suggest very different approaches to decisions such as diagnostic disclosure of preclinical AD and public health messaging.

The present study pursues these questions by examining the foundations of stigma directed at Alzheimer's clinical stage. Using a vignette-based experiment with a U.S. general population sample, we randomized participants to read about the same set of behavioral symptoms associated with Alzheimer's but assigned different disease labels and different prognoses. The experimental design allows us to examine *which* aspects of Alzheimer's provoke negative reactions: its behavioral symptoms? The Alzheimer's label? Or, the perceived prognosis?

Disentangling the contributors to Alzheimer's stigma lays the groundwork for understanding if preclinical AD categories will be accompanied by *spillover stigma* from the condition's clinical form. If the *Alzheimer's label* itself is the primary contributor to stigma, then those with preclinical Alzheimer's may be subject to stigma even in the absence of symptoms. In contrast, if symptoms are the primary contributor to stigma, then persons who are labeled but asymptomatic may not experience stigma. Furthermore, if stigma is linked to beliefs that the person's cognitive problems will get worse, communication that the course of preclinical AD varies among patients will be important. Understanding contributors to the stigmatization of persons with Alzheimer's thus helps us anticipate the consequences of transforming the condition to include a "preclinical" stage.

1.1. General framework for stigma

The present study draws on a social-cognitive model of stigma that identifies four components of stigmatization (Fig. 1). First, a signal marks someone as a potential target of negative reactions, such as a mental illness label or a person's appearance; 2) the signal prompts others to apply negative *stereotypes*, cognitive frameworks that give meaning to signals; 3) these *stereotypes* contribute to *affective* responses such as pity or fear; 4) these *affective* responses may escalate into *discrimination* against members of the stigmatized group such as social avoidance (Corrigan, 2000, 2007). Below, we also show how the study's findings can be interpreted through other frameworks such as the approach outlined in Link and Phelan (2001). The present study aims to make two main contributions to empirical research inspired by each framework.

The first contribution is to better understand how mild symptoms of cognitive impairment serve as *signals* triggering a chain of negative reactions. Most research on the *signal* step of stigma has examined disorders associated with fear or dangerousness, presenting vignettes of aberrant behaviors such as delusions, paranoia, or impulsivity in the absence or presence of a disease label and asking respondents to report their and others' expected reactions to the person so depicted (e.g., Angermeyer and Matschinger, 2003; Edens et al., 2004; Murrie et al., 2005). A recent systematic review found that labeling behaviors as schizophrenia or as a mental illness led respondents to view the condition as more serious and the person's social skills as more impaired than when the same symptoms were presented without a disease label (Angermeyer and Matschinger, 1996; Arkar and Eker, 1996; Cormack and Furnham, 1998; Sarbin and Mancuso, 1970; for a review, see Read et al., 2006). Less clear is the relative importance of disease labels versus observable behaviors for memory disorders such as Alzheimer's.

The second contribution the present study aims to make is a better understanding of how a disease's perceived *course* contributes to stigma. Jones (1984) highlights course as one of six underlying dimensions of stigma, defining course as the "pattern of change over time" persons associate with a condition (p. 24). Yet subsequent research has primarily studied disease course as a *dependent* variable, asking: how does manipulating the perceived cause or controllability of a stigmatized condition *affect* persons' perceptions of that condition's course? For instance, Weiner et al. (1988), depicting conditions as having a controllable versus non-controllable onset, found no effect on persons' perceptions of the condition's course as more or less reversible. In contrast, research

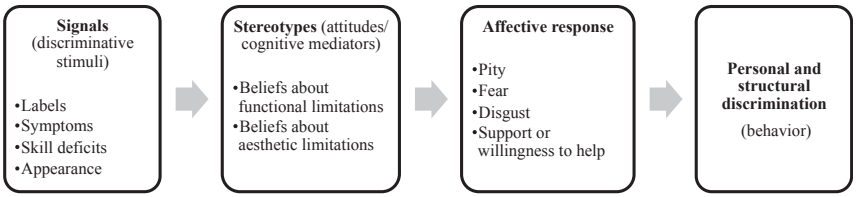

Fig. 1. Social-cognitive framework for Alzheimer's disease stigma (adapted from Corrigan, 2000; Werner et al., 2011).

investigating genetic or neurological "essentialism" has shown that attributing a condition to genetic *causes* results in higher beliefs that the condition will persist throughout a person's life (Phelan, 2005), and researchers have argued that attributing a condition to brain-based causes may have similar effects (e.g., Corrigan and Watson, 2004; Haslam, 2011). The present study reverses this approach. Rather than manipulating the *cause* of a disease and measuring the effect on perceptions of disease course and stigma outcomes, the study manipulates the *course* of a disease and measures the effect of this manipulation on stigma outcomes. In short, we examine disease course as a disease characteristic to manipulate rather than an outcome to be measured.

1.2. Alzheimer's disease-specific stigma

Examining the forms of stigma that the Alzheimer's label fosters or protects against is complicated by the fact that Alzheimer's impairments—deficits in cognition and in social and functional abilities—appear to elicit a different type of stigma than traditional foci of stigma research, stigma that is rooted less in fear of dangerousness or violence and more in harsh judgment on the person with Alzheimer's unkempt appearance, or disgust at or pity for that person (Werner et al., 2010, 2011).

Despite these differences in the cognitive beliefs and emotions that Alzheimer's elicits, Alzheimer's as a condition displays the five interrelated components that Link and Phelan (2001) argue are important for claiming that a characteristic is stigmatized. Alzheimer's is a 1) human difference that 2) people associate with negative attributes such as poor hygiene and disruptiveness in social situations (Werner, 2014). These associations lead persons to 3) separate persons with and without Alzheimer's into "us" versus "them" categories. For instance, research into "anticipatory dementia" describes significant distress among some older adults that normal memory problems associated with aging are an indication of dementia (Cutler and Hodgson, 1996; French et al., 2012). This indicates that people make distinctions between "us"—older adults who sometimes face memory lapses—and "them"—those with an Alzheimer's diagnosis that older adults fear.

As a result of this sharp demarcation, persons with Alzheimer's report 4) an acute sense of losing social status: they begin to fear that others no longer value their company and worry about losing access to social roles (Langdon et al., 2007), which some call "social disenfranchisement"(Beard and Fox, 2008). This status loss can result in discrimination. Finally, persons with Alzheimer's face power disadvantages in guarding themselves against these negative reactions. For instance, qualitative research highlights that persons in the mild to moderate stage of Alzheimer's report feelings of disempowerment when aides or caregivers place restrictions on their movement that seem better suited for persons in later stages (Sabat et al., 2004).

These qualitative studies highlight that Alzheimer's displays important components of a stigmatized condition. However, a recent systematic review notes important shortcomings in existing research on Alzheimer's-directed stigma (Werner, 2014). Importantly, most studies of Alzheimer's use the concept of stigma in a general way and fail to investigate how different *facets* of dementia contribute to stigmatization (e.g., Cheng et al., 2011). The present study adapts the only validated scale that specifically measures AD stigma to study a range of negative reactions (the Family Stigma in Alzheimer's Disease (FS-ADS) Scale). Second, by experimentally manipulating the disease's label and prognosis, the study is the first to examine *which* aspects of dementia elicit stigma at each step of the process.

1.3. The present study

Using a 3 × 3, between-subjects factorial design with a U.S. general population sample aged ≥18, we explore how disease label and disease prognosis contribute to stigma outcomes. We randomize participants to read one of 9 vignettes, each containing the same set of mild stage Alzheimer's disease symptoms. The vignettes vary: 1) the disease label ("Alzheimer's disease" versus "traumatic brain injury" versus no disease label); and, 2) disease prognosis ("symptoms will improve with treatment" versus "with treatment, symptoms will not improve but also not get worse" versus "there is no treatment and symptoms will get worse") attached to the symptoms. We test two hypotheses concerning the effect of disease label and prognosis on different stigma outcomes:

1. Hypothesis #1: the Alzheimer's disease label will lead to higher levels of expected structural discrimination, social distance, and pitying and supportive emotions, but lower levels of negative cognitive attributions and antipathetic emotions than the other two disease labels (TBI and no disease). This hypothesis is based on findings that brain injury evokes low levels of pitying/supportive emotions, and is not a condition for which persons distance themselves socially from the affected persons (Linden et al., 2005). Alzheimer's, in contrast, evokes pitying and supportive emotions and prompts social distance (Werner et al., 2011; Von dem Knesebeck et al., 2014).
2. Hypothesis #2: The above outcomes will hold true only when the Alzheimer's label is combined with the prognosis that the disease will get worse; that is, when the Alzheimer's label is combined with either of the other prognoses (static or will improve), it will no longer provoke higher or lower levels of stigma than the other disease labels. Data show that most persons expect brain-injured patients to fully recover (Ralph and Derbyshire, 2013), while few people expect Alzheimer's patients to fully recover even with treatment (Blay et al., 2008). Thus, we predicted that when Alzheimer's is depicted as having symptoms that stay the same or improve with treatment (as opposed to get worse), there may be few differences between persons' reactions towards those with AD and persons' reactions towards those with TBI and no disease label.

2. Method

2.1. Sample and procedures

The target population was U.S., English-speaking adults, age ≥18

who have never been a primary caregiver of persons with Alzheimer's disease or TBI. Qualtrics Panels recruited the U.S. general population sample. Qualtrics, contracting with 20 online panel providers, maintains a database of 4 million persons who have opted to occasionally participate in survey research and who represent a diverse cross-section of the U.S. population (for examples of use of Qualtrics for general population sampling, see: Cheng, 2014; Tinghog et al., 2013; Wright and Skagerberg, 2012). For general population surveys such as ours, Qualtrics performs random sampling that weights a respondent's probability of being chosen according to U.S. population demographics. The experiment was fielded between September 5th, 2013 and September 13th, 2013. We described the web-based survey as a study of "health beliefs" and did not mention Alzheimer's disease or traumatic brain injury during recruitment or consent. Participants were randomly assigned to one of the nine vignette conditions.

After consent, participants read one of nine vignettes about Mr. Andrews, a man suffering from impairments typical of the mild stage of Alzheimer's disease dementia, who comes to see the doctor with his daughter. Next, participants were given a comprehension check confirming they understood the correct 1) disease label; and, 2) disease prognosis. Participants were given two opportunities to select the correct choice, and if they failed the second attempt, they were taken to the debriefing page of the survey and not included in the final sample. Participants who passed the comprehension check next completed the dependent measures—ratings of the disease's biological/psychological nature; questions about negative attitudes—and controls: measures of Alzheimer's disease knowledge; exposure to persons with AD and TBI; and demographic information.

2.2. Demographic characteristics

Table 1 provides the sample characteristics and compares these to the general U.S. population. Urban versus rural classifications were developed by classifying participant zip codes using the rural urban commuting area (RUCA) classification, combined with the University of Washington's crosswalk file (U.S. Department of Agriculture, 2001). Table 1 shows sample demographics with primary caregivers of a person with AD or TBI included ($N = 900$) and excluded ($N = 789$). Because our study focused on attitudes among members of the general public towards persons with Alzheimer's, all subsequent analyses were run with the caregiver-excluded population. Analyses with the caregivers-included sample showed no substantial change in our results and are available upon request.

Table 1 illustrates that the sample generally resembles the U.S. population with three exceptions. First is that excluding participants under the age of 18 raised the mean age above that of the general U.S. population. Second, the sample was slightly better educated than the U.S. population. Third, our structuring of the race/ethnicity question, which forced participants to exclusively choose Hispanic rather than allowing them to choose Hispanic *and* a racial classification as in the U.S. census, likely undercounted participants who consider themselves Hispanic in our sample. However, the sample's wide age range (18–90), mix of education levels (e.g. 22% of participants' highest level of education was high school or GED), and racial/ethnic diversity (e.g. samples of Asian and African-American participants close to general population levels), gives the present study a more diverse sample than past studies of Alzheimer's stigma, which have enrolled populations such as female, U.S. undergraduates at a 4-year university (Wadley and Haley, 2001), a sample of the UK general public in which 95% were Caucasian (Crisp et al., 2000), or a sample of Hong Kong adults with a mean age of 56 (Cheng et al., 2011).

In addition to standard demographic characteristics, we measured a participant's exposure to a "relative, friend, co-worker, or patient" with AD or TBI. A higher proportion of the sample reported having *any* exposure to a person with AD (53.3%) than TBI (24.3%); a two-sided test of differences in proportions revealed this difference to be significant ($z = 12.10$, $p < 0.001$). However, among those with *any* exposure to a person with AD or TBI, the proportion of persons reporting a given frequency/intensity of exposure did not significantly differ between the two conditions.

All participants completed a modified, 22-question version of

Table 1
Sample characteristics for primary caregivers.

Sociodemographic characteristics	Primary caregivers[a]		United States census (%)
	Included (N = 900)	Excluded (N = 789)	
Age (years), *Mean* (*SD*)	46.9 (16.8)	46.8 (16.8)	37.4
Female	449 (49.9)	385 (48.8)	50.8
Race/ethnicity[b]			
African American	78 (8.7)	62 (7.9)	13.1
Asian	45 (5.0)	40 (5.1)	5.1
Caucasian	694 (77.1)	615 (78.1)	77.8
Hispanic	51 (5.7)	46 (5.8)	16.9
Other/more than one race	28 (3.1)	24 (3.0)	3.8
Did not indicate	4 (0.4)	2 (0.3)	–
Highest level of education[c]			
Less than High School or High School/GED	217 (24.1)	192 (24.3)	42.7
Some College or 2-year College Degree	365 (40.6)	321 (40.7)	26.4
4-year College Degree	211 (23.4)	188 (23.8)	14.1
Masters, Doctoral or Professional degree	107 (10.9)	88 (11.1)	11.1
Urbanicity of zip code of residence			
Urban or metropolitan area	714 (79.3)	621 (78.7)	79.0[d]
Rural or non-metropolitan area	186 (20.7)	168 (21.3)	21.0[d]

Note. All measures, with the exception of Age (years), are reported as the frequency (% of sample). GED = General Educational Development. SD = Standard deviation.

[a] Primary caregiver was assessed by asking: "Do you, or have you, considered yourself the primary caregiver of a person with Alzheimer's?"; "Do you, or have you, considered yourself the primary caregiver of a person with traumatic brain injury?"

[b] The census race/ethnicity groups add up to >100% because the census asks persons separately about their race and then about Hispanic ethnicity; some persons will identify as Caucasian-Hispanic, African-American-Hispanic, etc. In contrast, our survey combined race/ethnicity into one question such that persons chose between Hispanic versus Caucasian, etc.

[c] Masters degrees may include: MA; MSW; M.Ed.; Doctoral degrees may include: PhD, PsyD, DrPH; Professional degrees may include: JD, MD, or MBA.

[d] These data are from the 2000 census, which provides the most recent breakdown of rural versus urban residence.

the Alzheimer's Disease Knowledge Scale (ADKS), with statements about the condition that the respondents mark as "true" or "false." Questions that could be answered with reference to the behavioral symptoms depicted in the vignette were removed from the original, 30-question ADKS (Carpenter et al., 2009). We use the modified ADKS not to test knowledge as a mediator or moderator of stigma outcomes but instead as a check to ensure that the study did not attract respondents particularly knowledgeable about AD. We believe that despite the fact that our 22-question version of the ADKS has not been validated, it is acceptable for this limited purpose of using the ADKS as a check of our recruitment strategy.

2.3. Measures

2.3.1. Vignettes

Participants were randomized to read one of nine vignettes created using a 3 (AD; TBI; no label) × 3 (improve with treatment; static with treatment; no treatment and will worsen) design. For the disease prognosis descriptions (improve; static; worsen), we indicated that symptoms could improve or stay the same *with treatment* (as opposed to spontaneously) to increase the vignettes' believability, particularly for the AD vignette. A U.S. general population survey revealed that 46% believed that Alzheimer's disease has an effective treatment that can slow its progression and alleviate symptoms (Blendon et al., 2012). Surveys from European countries and Brazil find that few persons believe that patients with AD can spontaneously recover (Blay et al., 2008), but that between 24% (Rimmer et al., 2005) and 94% (Blay et al., 2008) of members of the general public believe that AD patients can make at least a partial recovery with treatment. Therefore, our static vignette condition specified that treatment halts the progression of symptoms depicted in the vignette, and our improve condition specified that treatment improves these symptoms.

The vignette symptoms corresponded to mild stage of AD dementia (stage 1) in the Clinical Dementia Rating (CDR) Scale (Hughes et al., 1982) and represented each of CDR's six domains: memory, orientation, judgment and problem-solving, community affairs, homes and hobbies, and personal care. Vignettes were tested for face validity with neurologists and research coordinators familiar with dementia, as well as tested for ease of comprehension with $N = 20$ pilot volunteers whose feedback was incorporated into the final survey. Italicized below are the portions of the vignette that varied between conditions. The electronic supplementary materials highlight the alternate text in each of the nine vignettes.

> Mr. Andrews comes to the doctor's office with his daughter because Mr. Andrews has memory problems that interfere with his daily life. He is having trouble balancing his checkbook, has given up following the stats of his favorite sports teams, and has stopped his long term volunteer job as a crossing guard for the local elementary school. He also takes longer to make decisions than he used to, and he sometimes confuses the facts.
>
> His daughter reports that sometimes Mr. Andrews needs to be reminded to take showers. She says he sometimes gets confused about what day it is and sometimes can't remember how to get home from the post office, a few blocks away.
>
> The doctor does a complete examination of Mr. Andrews. This includes a medical history, memory tests, lab tests, and brain imaging. Based on this information, the doctor diagnoses Mr. Andrews with *Alzheimer's disease*. The doctor tells Mr. Andrews that *treatment can improve his memory problems and functional difficulties.*

2.4. Dependent variables

2.4.1. Disease evaluation

First, participants rated the extent to which vignette's condition had biological or psychological causes (1 = primarily biological; 10 = primarily psychological), an item adapted from Wadley and Haley (2001). Participants also rated the extent to which the condition was a mental illness.

2.4.2. Stigma outcomes

Next, participants completed a modified version of the lay public stigma portion of the Family Stigma in Alzheimer's Disease Scale (FS-ADS), developed and validated by Werner et al. (2011). The FS-ADS was developed based on interviews with adult children who had served as a primary caregiver of a person with AD. These caregivers reported their predictions about stigmatizing attitudes, emotions, and behavioral reactions that members of the general public might have about persons with AD. In the original scale and present study, participants rated items on a 5-point Likert-type scale (1 = Not at all; 3 = somewhat; 5 = to a very great extent).

The original FS-ADS was developed and validated in a caregiver sample reporting on the cognitive attributions, emotional reactions, and behavioral actions that caregivers *perceived* members of the general public would hold toward AD patients. The present study supplements this purpose by examining the extent actual members of the lay public exhibit the attitudes and emotions towards person with AD that caregivers expect, investigating the applicability of the "lay public stigma" portion of this scale to a U.S. general population sample.

We gave participants four of the scales developed by Werner et al. (2011), including items asking about: 1) the degree of structural discrimination they expected the person in the vignette to face (e.g. insurance discrimination; doctors unwilling to provide care); 2) the participant's cognitive attributions about the severity of the vignette character's condition and degree of negative aesthetic attributes (e.g. dirtiness; filth); 3) the person's expectations regarding negative emotions (e.g. pity; compassion; disgust) directed at the vignette character; and, 4) the social distance by friends and relatives the participants expected the vignette character to face (see Electronic supplementary materials). We separated items into those for which it made sense to ask about the participant's own response to the vignette character (e.g. beliefs they have about his appearance and behaviors) and items for which it made sense to ask about the response that participants expected others to have (e.g. friends and family of Mr. Andrews limiting social contact).

2.4.3. Data analyses

After analyzing sample demographics, we used a series of ANOVAs to examine the impact of two main effects—disease label (AD; TBI; no label) and disease prognosis (get worse; static; improve) on the continuous dependent variables. All dependent variables were analyzed for skewness, and each was approximately normally distributed. In addition, for each set of stigma outcomes we performed a principal components analysis using varimax rotation to examine underlying factors. For significant main effects, interactions between the main effect and the demographic variables (e.g. education; race/ethnicity; intensity of exposure to AD and TBI; AD knowledge score) were examined. In addition, effect sizes were analyzed using the partial eta-squared measure of ANOVA effects. These analyses were performed using STATA 13.1 (Statacorp, 2013). Analyses testing the robustness of our results to a sample reweighted to better resemble the educational composition of the general population were performed in R with the assistance of the "Survey" package for handling complex weights (Lumley,

2010). The analyses performed with the reweighted sample are confined to an in-text description in the section discussing the robustness of our results. All other in-text analyses, and the analyses presented in Tables 1–3 and Figs. 2–5, present estimates and standard errors for the original, *unweighted* sample.

2.4.4. Power calculation

A study with a 3 × 3 design with nine groups, eight demographic variables used as interaction terms if a main effect was significant, and seeking a medium effect size of 0.15 in the dependent variables (separate stigma outcomes) (Cohen, 1988), is 90% powered at 750 participants. Therefore, our sample size of 789 participants exceeded 90% power.

3. Results

3.1. Participant flow

A total of 1795 participants clicked on the link in the recruitment email to potentially take the survey. After reading the consent, 682 participants (38.0%) left the page without indicating "yes" or "no" that they were interested in participating in the study; 82 participants (4.6%) explicitly indicated they were not interested in participating, and the remaining 1031 participants (57.4%) gave consent and proceeded with the survey.

After reading the vignette, participants were given a comprehension check that gave them two chances to correctly identify the prognosis attached to the disorder. 139 participants answered incorrectly the first time. Of those 139 persons, 58 persons passed on the second try and 81 persons went on to answer incorrectly a second time and thus fail the comprehension check (7.9% of those who consented/read the vignettes). Those 81 persons were redirected to the debriefing page of the study and thus did not complete any stigma items or subsequent questions. Overall, the comprehension check revealed that almost all participants came away from the vignettes with the correct impression of the disease label and prognosis and thus were properly prepared for the dependent variables. Finally, 50 participants (4.8% of those who consented/read the vignettes) left the study after consenting and passing the comprehension check but without completing all the questions. Overall, the study had 900 full completions (87.3% of those who consented/read the vignette; 50% of those who clicked on the emailed link to the survey), with a total sample size of 789 after excluding self-identified, primary caregivers for a person with AD or TBI from analysis. Table 2 presents the number of participants in each of the nine conditions.

3.2. AD knowledge

In the caregiver-excluded sample, the average score on the modified Alzheimer's disease knowledge scale was 14.7 out of 22 questions ($SD = 3.08$), with a range of 22.7%–100.0% correct. The average percentage of questions correct (66.8) was similar to the average percentage correct for a student sample in the original scale validation paper (67.3), while lower than the average percentage correct for a sample of older adults (80.3) (Carpenter et al., 2009). We interpreted the scores to indicate that study recruitment did not attract a population with knowledge of AD significantly higher than average members of the general population.

Table 2
Number of respondents per vignette condition, grouped by disease label.

Number of respondents, *N* (%)	Disease prognosis description[a]		
	Improve	Static	Worsen
Alzheimer's	92 (11.7)	95 (12.0)	94 (11.9)
Traumatic Brain Injury	77 (9.8)	89 (11.3)	86 (10.9)
No Label	85 (10.8)	91 (11.5)	80 (10.1)

Note.
[a] *Improve* condition specified that symptoms would improve with treatment; Static condition specified symptoms would stay the same with treatment; Worsen condition specified symptoms would worse with treatment.

3.3. Disease evaluation

Performing an ANOVA examining the main effects of disease labels and disease prognosis on how persons evaluated the characteristics of the symptoms, disease label had a significant main effect for both how biological versus psychological a condition's cause was perceived (from 1 = primarily biological to 10 = primarily psychological), $F(2, 788) = 3.00$, $p = 0.05$, and the extent to which the condition was considered a mental illness, $F(2, 788) = 4.38$, $p < .05$. In particular, participants rated the AD-labeled symptoms as having more of a biological cause ($M = 3.60$, $SD = 2.36$) than the TBI-labeled symptoms ($M = 3.89$, $SD = 2.43$) and the same symptoms presented without a disease label ($M = 4.10$, $SD = 2.40$), but also as more of a mental illness ($M = 2.81$, $SD = 1.35$) than TBI ($M = 2.52$, $SD = 1.30$). Table 3 presents means and standard deviations.

3.4. Stigma

We examined four sets of stigma outcomes: structural discrimination, cognitive attributions, emotional reactions, and social distance. All results, both for outcomes with significant main effects and for those without, are presented in Table 3. Table 3 also highlights the number of participants missing data for responses to any of the questions composing each outcome scale. Since very few participants were missing data (less than 0.38% of the sample), participants who failed to answer an outcome variable question were dropped from that analysis. Below, we highlight the most important findings for each outcome.

3.5. Structural discrimination

Structural discrimination included worries that the person in the vignette would face insurance discrimination, discrimination by employers, exclusion from voting, and exclusion from medical decision-making. We examined these items together because a principal components analysis (PCA) using varimax rotation revealed only one factor underlying all seven items, with loading scores ranging from 0.56 (exclusion from voting) to 0.85(discrimination by insurers based on medical records), which exceeds the generally accepted threshold for factor loading.

This analysis revealed a significant main effect of disease prognosis, $F(2, 777) = 20.35$, $p < 0.001$. Participants exposed to the vignette stating that symptoms would *get worse* perceived higher levels of discrimination. In contrast, there was no association between participants' ratings of the degree of structural discrimination and the disease label, $F(2, 777) = 0.35$, $p = 0.73$, and no significant interactions between disease label and disease prognosis, $F(4, 777) = 0.97$, $p = 0.43$ (Fig. 2).

3.6. Cognitive attributions

Principal components analysis revealed two clusters of items for cognitive attributions, which can be thought of as stereotypes about a person's characteristics. Factor one encompassed what we call *negative severity attributions*—thoughts about the severity of the person's impairment, including characteristics such as the

Table 3
Effect of experimental conditions on stigma outcomes.

Stigma outcomes[a]	Disease label, mean (SD)			Prognosis, mean (SD)			Significance (F, p-value|η^2)					
	AD	TBI	No label	Improve	Static	Worsen	Disease		Prognosis		Disease × prognosis	
Disease evaluations												
Biological to psychological rating[b]	3.60 (2.36)	3.89 (2.43)	4.10 (2.40)	4.15 (2.35)	3.68 (2.41)	3.76 (2.42)	3.00, $p = .05$	0.008	2.79, p = .06	0.007	0.11, p = .11	0.0006
Mental illness rating	2.81 (1.35)	2.52 (1.30)	2.82 (1.28)	2.69 (1.25)	2.65 (1.25)	2.83 (1.44)	4.38, p = .01	0.01	1.34, p = .26	0.003	0.55, p = .70	0.003
Institutional reactions												
Structural discrimination	3.31 (0.81)	3.27 (0.89)	3.27 (0.79)	3.10 (0.81)	3.20 (0.80)	3.54 (0.82)	0.35, p = .73	0.0009	20.35, p < .001	0.050	0.97, p = .43	0.005
Cognitive attributions												
Negative severity attributions	2.73 (0.66)	2.71 (0.79)	2.71 (0.80)	2.67 (0.74)	2.69 (0.75)	2.79 (0.76)	0.08, p = .92	0.0002	1.68, p = .19	0.004	1.32, p = .26	0.007
Negative aesthetic attributions	2.19 (0.78)	2.38 (0.90)	2.44 (0.97)	2.29 (0.90)	2.36 (0.85)	2.34 (0.92)	5.94, p = .003	0.02	0.32, p = .73	0.0008	2.61, p = .03	0.01
Emotional reactions												
Antipathy	2.29 (0.81)	2.33 (0.84)	2.32 (0.90)	2.30 (0.89)	2.26 (0.79)	2.38 (0.87)	0.14, p = 0.87	0.0004	1.36, p = .26	0.004	2.83, p = .02	0.01
Supportiveness	3.22 (0.76)	3.26 (0.82)	3.34 (0.86)	3.21 (0.85)	3.30 (0.77)	3.29 (0.82)	1.38, p = .25	0.004	0.76, p = .47	0.002	0.60, p = .66	0.003
Pity	3.26 (0.72)	3.31 (0.78)	3.36 (0.76)	3.21 (0.74)	3.27 (0.72)	3.43 (0.79)	1.39, p = .25	0.004	6.15, p = .002	0.02	0.86, p = .49	0.004
Behavioral reactions												
Social distance	2.61 (0.91)	2.66 (0.97)	2.60 (0.95)	2.54 (0.92)	2.57 (0.89)	2.76 (1.00)	0.25, p = .78	0.0006	4.08, p = .02	0.01	2.14, p = .08	0.01

Note. Data is unweighted. The following variables have missing data (n): structural discrimination ($n = 3$); aesthetic ($n = 3$); antipathy ($n = 3$); pity ($n = 2$); social distance ($n = 2$); severity ($n = 1$); supportiveness ($n = 1$). ***Abbr.*** AD = Alzheimer's disease. TBI = Traumatic brain injury. SD = Standard deviation.
[a] Rated on 5-point Likert scale: (1 = not at all; 2 = very little; 3 = somewhat; 4 = to a great extent; 5 = to a very great extent).
[b] Rated on scale ranging from 1 to 10; 1 = primarily biological; 10 = primarily psychological.

person speaking nonsense or failing at simple tasks. These items had loading scores ranging from 0.53(is dangerous to others) to 0.75(speaks nonsense). Notably, participants were asked to comment on deficits in a person that were *not* discussed in the vignette. Therefore, the *negative severity* items are intended to capture negative beliefs that members of the public form about a person in the absence of specific information that supports these beliefs. There were no significant associations between these

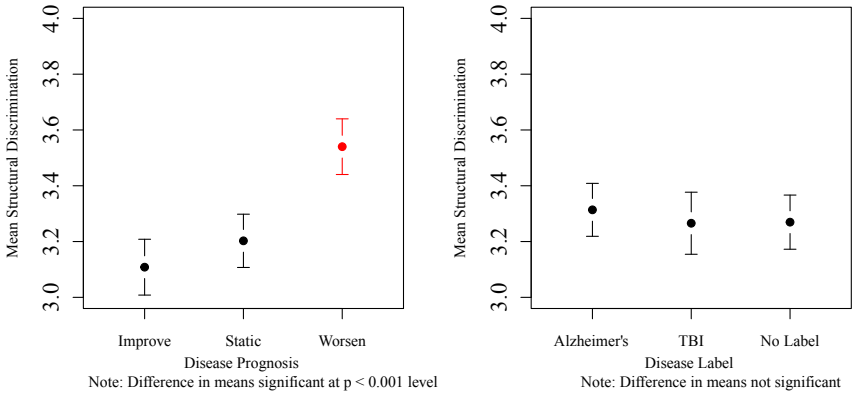

Fig. 2. Independent effects of disease label and prognosis on structural discrimination.

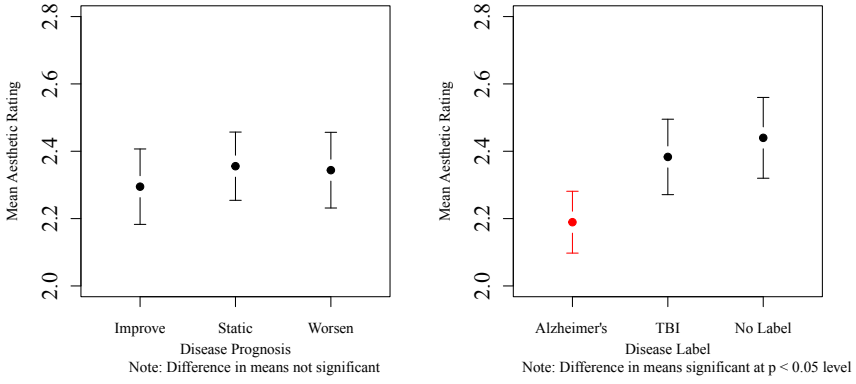

Fig. 3. Independent effects of disease label and prognosis on perceived aesthetics.

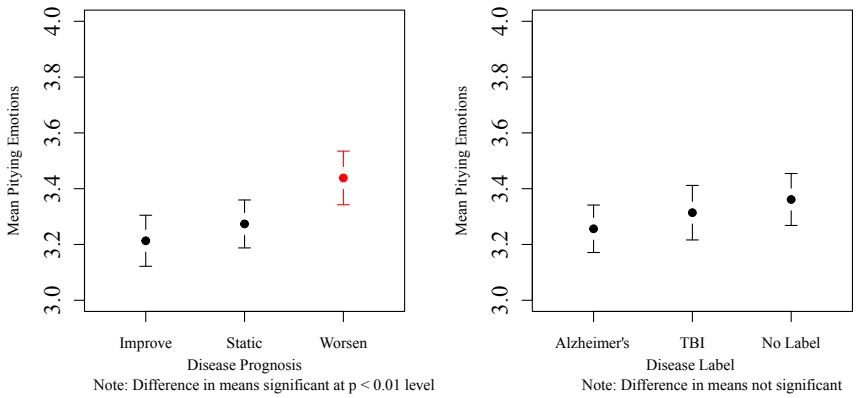

Fig. 4. Independent effects of disease label and prognosis on pitying emotions.

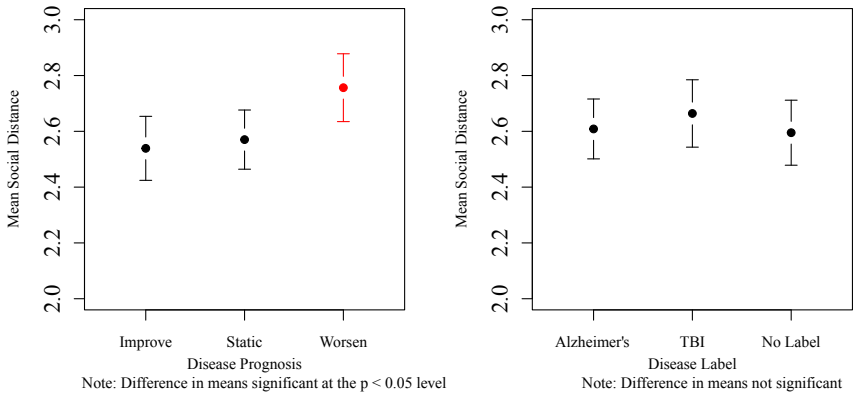

Fig. 5. Independent effects of disease label and prognosis on perceived social distance.

negative severity attributions and the disease label, F (2, 779) = 0.08, p = 0.92 or the disease prognosis, F (2, 779) = 1.68, p = 0.19.

Second were *negative aesthetic attributions*—thoughts about the person's filth, their neglect for self-care, and other features of the person's appearance that may provoke negative judgments in observers, with loading scores ranging from 0.83(looks disgusting) to 0.88(looks filthy). Again, the vignette did not directly discuss any of these symptoms. There was a significant main effect for the disease label, F (2, 777) = 5.94, p < .01 that was modified by a significant interaction between disease label and prognosis, F (4, 777) = 2.61, p < .05. More specifically, the AD label appeared protective against participants viewing the person in the condition as having neglectful or filthy aesthetic attributes (M = 2.19, SD = 0.78), as compared to the same person/set of symptoms with a TBI label (M = 2.38, SD = 0.90) or no label (M = 2.44, SD = 0.97) (Fig. 3).

Examining the distribution of negative aesthetic reactions, we see within the AD label that 11.7% of participants score “1” on the subscale—in other words, on all five items asking the participant about negative aesthetic attributes for the vignette character with Alzheimer's, such as an unkempt appearance, this subset of participants indicated that they did “not at all” hold that view of the person. Another 32.7% of participants in the AD label condition had a subscale between 1.2 and 2.0, indicating that for most items, they believed the vignette character displayed the negative aesthetic attributes “not at all” or “very little.” To account for distinct categories of respondents—those who very much disagreed with the negative aesthetic attributes; those who weakly endorsed the attributes; those who endorsed the attributes more strongly—we divided the aesthetic scale into tertiles (1 ≤ 2.2; 2 = 2.2–2.8; 3 ≥ 2.8) to form an ordinal dependent variable. An ordinal logistic regression with this dependent variable confirmed the results of the ANOVA that treated the aesthetic scale as a continuous variable. Giving the vignette symptoms *no label* as opposed to an *Alzheimer's disease* label significantly predicts a harsher judgment of the aesthetics of the person depicted in the vignette ($OR_{Nodiseaselabel}$ = 2.15, p < .01), though there was no significant difference between the AD and TBI labels ($OR_{TBI\ label}$ = 1.45, p = .17). This confirms that the AD label, rather than promoting harsh judgments that a person is unkempt or neglectful about his or her appearance, appears protective against those stigmatizing judgments relative to unlabeled symptoms.

3.7. Emotional reactions

Stigma items measuring emotional reactions are divided into three subscales: *antipathy* (e.g. disgust; repulsion; fear), *supportiveness* (e.g. concern; compassion; willingness to help), and *pity* (e.g. sympathy; sadness; pity) on the basis of a principal components analysis that found three distinct factors. What we call *antipathy* items had factor loading ranging from 0.70(uneasiness) to 0.87(disgust); *supportiveness* items had factor loadings ranging from 0.71(concern) to 0.89(willingness to help); *pity* items had factor loadings ranging from 0.63(sympathy) to 0.75(pity).

There were no significant main effects for *antipathy* or *supportiveness*. However, *pity emotions* displayed a significant main effect of prognosis, F (2, 778) = 6.15, p < .01. Across all three disease

labels, respondents assigned the vignette that the symptoms would get worse displayed higher levels of perceived pity. There was no significant main effect of disease label, $F(2, 778) = 1.39$, $p = 0.25$ and no significant interaction, $F(4, 778) = 0.86$, $p = 0.49$ (Fig. 4).

There *was* a significant interaction between prognosis and age, $F(3, 743) = 4.16$. $p < 0.01$, meaning that for older age groups, the prognosis that the symptoms will get worse is associated with the highest level of pitying emotions. In contrast, for the youngest participants—aged 18–29—pity does not seem sensitive to the condition's prognosis. In sum, and especially for older participants, a worse prognosis, in addition to predicting higher expected structural discrimination regardless of disease label, also predicted higher levels of pitying emotions across all three disease labels.

3.8. Behavioral reactions

Behavioral reactions were focused on social distance, and included items that measured beliefs that others would limit interactions with Mr. Andrews and avoid him. A principal components analysis revealed one factor with loading scores ranging from 0.82(keep Mr. Andrews away from public places) to 0.91(keep away from Mr. Andrews). There was a significant main effect of prognosis on levels of perceived social distance, $F(2, 778) = 4.08$, $p < .05$. Those whose vignette reported that the symptoms would *get worse* predicted significantly higher levels of social distance ($M = 2.76$, $SD = 1.00$) than the static ($M = 2.57$, $SD = 0.89$) and improve ($M = 2.54$, $SD = 0.92$) prognoses (Fig. 5). There was no significant main effect of disease label, $F(2, 778) = 0.25$, $p = 0.78$, nor was there a significant interaction between disease label and prognosis, $F(4, 778) = 2.14$, $p = 0.08$. In sum, just as the worsen prognosis was associated with higher levels of expected discrimination and higher levels of pity, it was also associated with higher levels of expected social distance regardless of which disease label the symptoms received.

3.9. Robustness of our findings to a more representative educational distribution

Previous research suggests that persons with lower levels of education might possess more stigmatizing views of certain mental illnesses (Rabkin, 1980), while other research finds no such patterns (e.g., Phelan, 2005). Because our sample contained an over-representation of those with some college/a 2-year degree and those with a 4-year college degree, we performed an analysis to test the generalizability of our results to a more educationally representative population. To do so, we used the frequency of each education category in the U.S. census, as reported in Table 1, to construct survey weights using a raking procedure that uses iterative proportional fitting (Lumley, 2010).

The weighted sample's education level more closely resembled the census distribution, with 45.3% receiving a less than high school or high school/GED education (compared to 42.7% in the census), 28.0% receiving some college or a 2-year college degree (compared to 26.4% in the census), 14.9% receiving a 4-year college degree (compared to 14.1% in the census), and 11.8% receiving a master's or professional degree (compared to 11.1% in the census). We then reran our analyses of stigma outcomes on the weighted sample; however, rather than ANOVA's, we used a generalized linear model and then a Rao-Scott likelihood ratio test, since maximum likelihood estimation produces biased results for the weighted dataset we used (Lumley, 2010). The stigma outcomes remained significant in the reweighted sample: a worsen prognosis was associated with significantly higher levels of structural discrimination, $F(2, 777) = 27.67$, $p < 0.001$; significantly higher levels of pity, $F(2, 778) = 4.90$, $p < 0.05$, and a trend towards a significant association for higher social distance, $F(2, 778) = 5.68$, $p = 0.09$. Likewise, the Alzheimer's label remained significantly protective against negative aesthetic reactions, $F(2, 777) = 16.24$, $p < 0.001$. As with the unweighted sample, the other stigma outcomes showed no main effects of disease or prognosis. These results with the reweighted sample enhance the generalizability of our findings to persons with a more representative range of education levels.

4. Discussion and conclusion

The present study experimentally investigated the relative contributions of disease label (AD; TBI; no label) and disease prognosis (improve; static; worsen) to stigmatizing reactions towards the symptoms of early-stage Alzheimer's. The results reveal that disease prognosis was a more significant contributor to greater stigma than the Alzheimer's disease label. Expecting the symptoms to get worse, *regardless of disease label*, led to significantly higher levels of expected structural discrimination, pity, and social distance.

These results show that other studies *measuring* the stigmatization of Alzheimer's might be capturing results driven more by expected deterioration than the disease label itself. Indeed, when the Alzheimer's disease label had an effect, the label seemed to protect persons against stigmatizing reactions rather than exacerbate stigma. More specifically, the AD label did not lead to higher levels of expected structural discrimination, social distance, pitying or supportive emotions, or antipathetic emotions. Its only effect was to lead respondents to make *less* negative aesthetic judgments about the person in the vignette relative to unlabeled symptoms. These findings highlight that in contrast to our predictions that assigning an AD label to a set of symptoms would exacerbate some stigma outcomes and protect against others, the Alzheimer's label had few associations on the outcomes of interest. Interestingly, viewing AD as more of a mental illness with more of a biological cause than TBI did not produce significant differences in stigma outcomes between the two labels.

The study's main finding—that perceived symptom prognosis has a greater effect on stigma than the Alzheimer's disease label—suggests that the negative reactions that persons with mild AD provoke might stem *not* from the disease label itself but instead from the *expected prognosis* embedded in that label. In other words, the stigma may arise from the *prospect* of further deterioration rather than from the degree of present impairment. These findings have implications for the cultural framing and classification of AD and for theoretical models of stigma.

4.1. Implications for cultural framings of Alzheimer's and the experience of stigma for persons in the preclinical categories

Qualitative research has shown that cultural framings of Alzheimer's overwhelmingly focus on the condition's most severe stages. As Fox (1989) argues, “the words ‘Alzheimer's disease’ conjure up images of a hideous, debilitating condition”(p. 58), and other research highlights media framings of Alzheimer's as a state of total incapacity (Behuniak, 2011, p. 84) or of persons with Alzheimer's as “the living dead”(Aquilina and Hughes, 2006). These analyses help contextualize our experimental findings that many stigma outcomes are driven more by the Alzheimer's label's strong cultural association with further deterioration than with the label itself. These past studies illustrate the *means* by which the AD label has become linked to images of decline. Our present study shows the *consequences* of the assumed link between the AD label and severe future decline. These combined accounts of the *cause* and *consequences* of linking AD to future deterioration points to the importance of a cultural reframing of AD that focuses on its range of

severity. In addition, future research should examine whether there is a "tipping point" for Alzheimer's stigmatization: do negative reactions to those with Alzheimer's increase linearly as the impairments become more severe or display a pattern of relatively stable levels of negative reactions until the severity reaches a threshold, after which negative reactions sharply increase?

Applied to preclinical AD, our findings suggest that the more that the preclinical AD label is associated with future, severe deterioration, the more patients with this label may experience certain stigma outcomes. Calibrating a proper framing of preclinical AD will be complex. While the preclinical label will signify the risk of cognitive decline, it will also first be given to patients in the context of clinical trials testing whether a drug can alter the course of cognitive decline (Sperling et al., 2014). These drug trials will shape the public's understanding of preclinical AD, making the drug a direct contribution to the cultural framing of AD's preclinical stages, much as the U.S. public's understanding of hypertension and hypercholesterolemia was shaped by drug trials aimed at those disease categories (Greene, 2007).

4.2. Implications for theoretical models of disease stigma

The present findings suggest that stigma incorporates not only information about a person's present state but also about his or her future. Past vignette-based studies of AD-associated stigma present the diagnosis without referencing possible improvement or deterioration (e.g. Cheng et al., 2011; Wadley and Haley, 2001). This makes it difficult to disentangle the relative contributions of present symptoms versus possible future course to the stigmatizing reactions that past research has uncovered. Our findings about perceived deterioration leading to significantly higher levels of expected discrimination, pity, and social distance point to the theoretical importance of frameworks for stigma that incorporate attributions about the condition's mutability/improvability. For instance, theories of stigma that incorporate ideas about the *essentialism* of a stigmatized category (Haslam, 1998; Haslam and Ernst, 2002) suggest a promising direction for future frameworks directed at understanding AD stigma. Research inspired by these frameworks should take two steps. First, the present study highlights the usefulness of *manipulating* perceived disease course rather than only measuring disease course as a dependent variable. Future research should examine how manipulating *both* the depicted cause (biogenetic vs. behavioral) and the disease course affects stigmatizing reactions towards Alzheimer's, since our vignettes were deliberately silent on the cause of Mr. Andrews' condition. Second, the present study presented participants with information about a *likely* disease course the person would face. Yet as Jones (1984) notes, many disease's expected course are highly uncertain, an observation that holds true for Alzheimer's. As a result, future research should also analyze the effect of noting an *uncertain* disease course in addition to improve, static, and worsen.

4.3. Limitations

Several limitations are worth noting. First, the vignette specified symptoms at one severity level of Alzheimer's disease (CDR stage 1) so that we could isolate the effect of the disease label and disease prognosis on stigma outcomes. However, the impact that prognosis has on stigma outcomes may vary based on the level of cognitive and functional abilities the person still stands to lose. Future research should examine how these outcomes vary across a broader range of disease severity levels, as well as how the emergence of psychotic and other psychiatric symptoms in later stages of dementia affect the outcomes of interest. Second, although we screened persons out who failed to correctly identify the vignette's prognosis, there is still a possibility that most persons understand AD as having a "worsen" prognosis and would respond to the stigma questions with this in mind. While acknowledging this limitation, we also note that it makes it less likely for us to find the significant effects the present study documents, since those in the "static" and "improve" conditions would go into the stigma questions with beliefs similar to those in the "worsen" condition. This makes it more difficult for us to show significant differences between the three groups. Third, the vignette depicted a person (Mr. Andrews) of a single gender and an unspecified race/ethnicity. Future research should thus examine whether our findings extend to reactions to female AD patients and AD patients of different races or ethnicities. Fourth, although Table 3 notes the effect sizes for each of the stigma outcomes, a statistically significant effect is distinct from an effect that makes a tangible difference in the lives of AD patients. Future research is needed to better map levels of stigmatizing attitudes to the tangible effect these attitudes have on discrimination towards and devaluation of AD patients. Fifth, although we believe the main finding of our study can help us anticipate how members of the U.S. general public might react to people given a preclinical AD label, the present study did not directly measure reactions towards preclinical AD as few members of the public have heard of the label. As the public becomes more aware of preclinical AD through developments such as large-scale drug trials and public health campaigns, future research will need to confirm our extrapolation of the present results to the new disorder.

We report the relative contribution of disease label and disease prognosis to stigmatizing reactions to early-stage Alzheimer's impairments. Expectations of future decline, rather than the AD label itself, lead to negative reactions. As neuroscience researchers in fields other than Alzheimer's develop pre-clinical categories that capture persons at elevated risk for severe future problems—for example, prodromal psychosis or biomarkers that capture risk for major depression or Parkinsons disease—future research should explore how to balance accurate information about a person's elevated risk with the need to minimize stigma attached to expectations of future decline.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.socscimed.2015.08.031.

References

Angermeyer, M.C., Matschinger, H., 1996. The effect of diagnostic labelling on the lay theory regarding schizophrenic disorders. Soc. Psychiatry Psychiatr. Epidemiol. 31 (6), 316–320.

Angermeyer, M.C., Matschinger, H., 2003. The stigma of mental illness: effects of labelling on public attitudes towards people with mental disorder. Acta Psychiatr. Scand. 108 (4), 304–309.

Aquilina, C., Hughes, J.C., 2006. The return of the living dead: agency lost and found? In: Hughes, J.C., Louw, S.J., Sabat, S.R. (Eds.), Dementia: Mind, Meaning, and the Person. Oxford University Press, pp. 143–161.
Arkar, H., Eker, D., 1996. Attitudes towards mental disease: impact of type of attributed cause. Turk. J. Psychiatry 7, 191–197.
Beard, R.L., Fox, P.J., 2008. Resisting social disenfranchisement: negotiating collective identities and everyday life with memory loss. Soc. Sci. Med. 66 (7), 1509–1520.
Beard, R.L., Neary, T.M., 2013. Making sense of nonsense: experiences of mild cognitive impairment. Sociol. Health & Illn. 35 (1), 130–146.
Behuniak, S.M., 2011. The living dead? The construction of people with Alzheimer's disease as zombies. Ageing & Soc. 31 (01), 70–92.
Blay, S.L., Furtado, A., Peluso, É.T.P., 2008. Knowledge and beliefs about help-seeking behavior and helpfulness of interventions for Alzheimer's disease. Aging & Ment. Health 12 (5), 577–586.
Blendon, R.J., Benson, J.M., Wikler, E.M., Weldon, K.J., Georges, J., Baumgart, M., Kallmyer, B.A., 2012. The impact of experience with a family member with Alzheimer's disease on views about the disease across five countries. Int. J. Alzheimer's Dis. e903645.
Carpenter, B.D., Balsis, S., Otilingam, P.G., Hanson, P.K., Gatz, M., 2009. The Alzheimer's disease knowledge scale: development and psychometric properties. Gerontol. 49 (2), 236–247.
Chaufan, C., Hollister, B., Nazareno, J., Fox, P., 2012. Medical ideology as a double-edged sword: the politics of cure and care in the making of Alzheimer's disease. Soc. Sci. Med. (1982) 74 (5), 788–795.
Cheng, S.-T., Lam, L.C.W., Chan, L.C.K., Law, A.C.B., Fung, A.W.T., Chan, W., , et al.Chan, W., 2011. The effects of exposure to scenarios about dementia on stigma and attitudes toward dementia care in a Chinese community. Int. Psychogeriatr. 23 (09), 1433–1441.
Cheng, Z.H., 2014. Asian Americans and European Americans' stigma levels in response to biological and social explanations of depression. Soc. Psychiatry Psychiatr. Epidemiol. 1–10.
Cohen, J., 1988. Statistical Power Analysis for the Behavioral Sciences, second ed. Lawrence Earlbaum Associates, Hillsdale, NJ.
Corcoran, C., Malaspina, D., Hercher, L., 2005. Prodromal interventions for schizophrenia vulnerability: the risks of being "at risk." Schizophr. Res. 73 (2–3), 173–184.
Cormack, S., Furnham, A., 1998. Psychiatric labelling, sex role stereotypes and beliefs about the mentally ill. Int. J. Soc. Psychiatry 44 (4), 235–247.
Corrigan, P.W., 2000. Mental health stigma as social attribution: implications for research methods and attitude change. Clin. Psychol. Sci. Pract. 7 (1), 48–67.
Corrigan, P.W., 2007. How clinical diagnosis might exacerbate the stigma of mental illness. Soc. Work 52 (1), 31–39.
Corrigan, P.W., Watson, A.C., 2004. At issue: stop the stigma: call mental illness a brain disease. Schizophr. Bull. 30 (3), 477–479.
Couteur, D.G.L., Doust, J., Creasey, H., Brayne, C., 2013. Political drive to screen for pre-dementia: not evidence based and ignores the harms of diagnosis. BMJ 347 f5125–f5125.
Crisp, A.H., Gelder, M.G., Rix, S., Meltzer, H.I., Rowlands, O.J., 2000. Stigmatisation of people with mental illnesses. Br. J. Psychiatry 177 (1), 4–7.
Cutler, S.J., Hodgson, L.G., 1996. Anticipatory dementia: a link between memory appraisals and concerns about developing Alzheimer's disease. Gerontol. 36 (5), 657–664.
DiMillo, J., Samson, A., Thériault, A., Lowry, S., Corsini, L., Verma, S., Tomiak, E., 2015. Genetic testing: when prediction generates stigmatization. J. Health Psychol. 20 (4), 393–400.
Dubois, B., Feldman, H.H., Jacova, C., Hampel, H., Molinuevo, J.L., Blennow, K., , et al.Pasquier, F., 2014. Advancing research diagnostic criteria for Alzheimer's disease: the IWG-2 criteria. Lancet Neurol. 13 (6), 614–629.
Edens, J.F., Desforges, D.M., Fernandez, K., Palac, C.A., 2004. Effects of psychopathy and violence risk testimony on mock juror perceptions of dangerousness in a capital murder trial. Psychol. Crime Law 10 (4), 393–412.
Fox, P., 1989. From senility to Alzheimer's disease: the rise of the Alzheimer's disease movement. Milbank Q. 67 (1), 58–102.
French, S.L., Floyd, M., Wilkins, S., Osato, S., 2012. The Fear of Alzheimer's Disease Scale: a new measure designed to assess anticipatory dementia in older adults. Int. J. Geriatr. Psychiatry 27 (5), 521–528.
Gauthier, S., Leuzy, A., Racine, E., Rosa-Neto, P., 2013. Diagnosis and management of Alzheimer's disease: past, present and future ethical issues. Prog. Neurobiol. 110, 102–113.
Greene, J., 2007. Prescribing by Numbers: Drugs and the Definition of Disease. Johns Hopkins University Press, Baltimore.
Haslam, N., Ernst, D., 2002. Essentialist beliefs about mental disorders. J. Soc. Clin. Psychol. 21 (6), 628–644.
Haslam, N.O., 1998. Natural kinds, human kinds, and essentialism. Soc. Res. 65 (2), 291–314.
Haslam, N., 2011. Genetic essentialism, neuroessentialism, and stigma: commentary on Dar-Nimrod and Heine (2011). Psychol. Bull. 137 (5), 819–824.
Hughes, C.P., Berg, L., Danziger, W.L., Coben, L.A., Martin, R.L., 1982. A new clinical scale for the staging of dementia. Br. J. Psychiatry 140 (6), 566–572.
Jones, E.E., 1984. Social Stigma: The Psychology of Marked Relationships. W.H. Freeman.
Langdon, S.A., Eagle, A., Warner, J., 2007. Making sense of dementia in the social world: a qualitative study. Soc. Sci. Med. 64 (4), 989–1000.
Lerman, C., Shields, A.E., 2004. Genetic testing for cancer susceptibility: the promise and the pitfalls. Nat. Rev. Cancer 4 (3), 235–241.
Linden, M.A., Rauch, R.J., Crothers, I.R., 2005. Public attitudes towards survivors of brain injury. Brain Inj. 19 (12), 1011–1017.
Link, B.G., Phelan, J.C., 2001. Conceptualizing stigma. Annu. Rev. Sociol. 27 (1), 363–385.
Luck, T., Luppa, M., Sieber, J., Schomerus, G., Werner, P., König, H.-H., Riedel-Heller, S.G., 2012. Attitudes of the german general population toward early diagnosis of dementia – results of a representative telephone survey. PLoS One 7 (11), e50792.
Lumley, T., 2010. Complex Surveys: A Guide to Analysis Using R. Wiley, Hoboken, N.J.
Murrie, D.C., Cornell, D.G., McCoy, W.K., 2005. Psychopathy, conduct disorder, and stigma: does diagnostic labeling influence juvenile probation officer recommendations? Law Hum. Behav. 29 (3), 323–342.
Phelan, J.C., 2005. Geneticization of deviant behavior and consequences for stigma: the case of mental illness. J. Health Soc. Behav. 46 (4), 307–322.
Rabkin, J.G., 1980. Determinants of public attitudes about mental illness: summary of the research literature. Attitudes Ment. Ill Res. Perspect. 15–26.
Ralph, A., Derbyshire, C., 2013. Survivors of brain injury through the eyes of the public: a systematic review. Brain Inj. 27 (13–14), 1475–1491.
Read, J., Haslam, N., Sayce, L., Davies, E., 2006. Prejudice and schizophrenia: a review of the "mental illness is an illness like any other" approach. Acta Psychiatr. Scand. 114 (5), 303–318.
Rimmer, E., Wojciechowska, M., Stave, C., Sganga, A., O'Connell, B., 2005. Implications of the Facing Dementia Survey for the general population, patients and caregivers across Europe. Int. J. Clin. Pract. 59, 17–24.
Sabat, S.R., Napolitano, L., Fath, H., 2004. Barriers to the construction of a valued social identity: a case study of Alzheimer's disease. Am. J. Alzheimer's Dis. Dement. 19 (3), 177–185.
Sarbin, T.R., Mancuso, J.C., 1970. Failure of a moral enterprise: attitude of the public toward mental illness. J Consult. Clin. Psychol. 35 (2), 159–173.
Sperling, R.A., Aisen, P.S., Beckett, L.A., Bennett, D.A., Craft, S., Fagan, A.M., , et al.Phelps, C.H., 2011. Toward defining the preclinical stages of Alzheimer's disease: recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. Alzheimer's Dement. J. Alzheimer's Assoc. 7 (3), 280–292.
Sperling, R.A., Rentz, D.M., Johnson, K.A., Karlawish, J., Donohue, M., Salmon, D.P., Aisen, P., 2014. The A4 study: stopping AD before symptoms begin? Sci. Transl. Med. 6 (228), 228fs13–228fs13.
Statacorp, 2013. Stata Statistical Software: Release 13. StataCorp LP, College Station, TX.
Tinghög, G., Andersson, D., Bonn, C., Böttiger, H., Josephson, C., Lundgren, G., , et al.Johannesson, M., 2013. Intuition and cooperation reconsidered. Nature 498 (7452), E1–E2.
U.S. Department of Agriculture, Economic Research Service, 2001. ERS/USDA Briefing Room: Measuring Rurality: Rural-Urban Commuting Area Codes. Available at: http://www.ers.usda.gov/briefing/Rurality/RuralUrbanCommutingAreas/.
U.S. Department of Health and Human Services, 2014. National Plan to Address Alzheimer's Disease: 2014 Update. Available at: http://aspe.hhs.gov/daltcp/napa/NatlPlan2014.pdf.
Van Gorp, B., Vercruysse, T., 2012. Frames and counter-frames giving meaning to dementia: a framing analysis of media content. Soc. Sci. Med. (1982) 74 (8), 1274–1281.
Vodermaier, A., Esplen, M.J., Maheu, C., 2010. Can self-esteem, mastery and perceived stigma predict long-term adjustment in women carrying a BRCA1/2-mutation? Evidence from a multi-center study. Fam. Cancer 9 (3), 305–311.
Von dem Knesebeck, O., Angermeyer, M.C., Lüdecke, D., Kofahl, C., 2014. Emotional reactions toward people with dementia – results of a population survey from Germany. Int. Psychogeriatr. 26 (03), 435–441.
Wadley, V.G., Haley, W.E., 2001. Diagnostic attributions versus labeling impact of alzheimer's disease and major depression diagnoses on emotions, beliefs, and helping intentions of family members. J. Gerontol. Ser. B Psychol. Sci. Soc. Sci. 56 (4), P244–P252.
Weiner, B., Perry, R.P., Magnusson, J., 1988. An attributional analysis of reactions to stigmas. J. Personal. Soc. Psychol. 55 (5), 738–748.
Werner, P., 2014. Stigma and Alzheimer's disease: a systematic review of evidence, theory, and methods. In: The Stigma of Disease and Disability: Understanding Causes and Overcoming Injustices. American Psychological Association, Washington, DC, US, pp. 223–244.
Werner, P., Goldstein, D., Buchbinder, E., 2010. Subjective experience of family stigma as reported by children of Alzheimer's disease patients. Qual. Health Res. 20 (2), 159–169.
Werner, P., Goldstein, D., Heinik, J., 2011. Development and validity of the Family Stigma in Alzheimers Disease Scale (FS-ADS). Alzheimer Dis. Assoc. Disord. 25 (1), 42–48.
Wright, D.B., Skagerberg, E.M., 2012. Measuring empathizing and systemizing with a large US sample. PLoS One 7 (2), e31661.