

Research Article

# The Framing of “Alzheimer’s Disease”: Differences Between Scientific and Lay Literature and Their Ethical Implications

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## Abstract

**Background and Objectives:** The meaning of Alzheimer’s disease (AD) is changing in research. It now refers to a pathophysiological process, regardless of whether clinical symptoms are present. In the lay literature, on the other hand, AD is understood as a form of dementia. This raises the question of whether researchers and the lay audience are still talking about the same thing. If not, how will these different understandings of AD shape perspectives on (societal) needs for people with AD?

**Research Design and Methods:** We use framing analysis to retrieve the understandings of the term AD that are upheld in the research literature and in national Dutch newspaper articles. We make explicit how the framings of AD steer our normative attitudes toward the disease.

**Results:** In the analyzed research articles, AD is framed as a pathological cascade, reflected by biomarkers, starting in cognitively healthy people and ending, inevitably, in dementia. In the lay literature, AD is used as a synonym for dementia, and an AD diagnosis is understood as an incentive to enjoy “the time that is left.”

**Discussion and Implications:** The two different uses of the term AD in research and in the lay literature may result in misunderstandings, especially those research framings that falsely imply that people with AD biomarkers will inevitably develop dementia. Adoption of the research understanding of AD in clinical practice will have normative implications for our view on priority setting in health care. For example, it legitimizes biomarker testing in people without dementia as improving “diagnostic” certainty.

**Keywords:** Biomarkers, Dementia, Ethics, Framing analysis, Metaphor

For decades, Alzheimer’s disease (AD) was uniformly defined as a form of dementia, characterized by loss of memory and cognitive abilities that interferes with daily life (McKhann et al., 1984). In 2007, it was first proposed to expand the diagnosis of AD to “earlier stages” in the research context (Dubois et al., 2007).

After decades of research efforts failed to find a cure for AD dementia, it is now believed that the pathophysiology of AD is too advanced in people with dementia for pharmaceutical interventions to have any clinical effect. Therefore, researchers now aim to prevent or delay dementia by intervening earlier, *before* the onset of symptoms (Spertling

et al., 2014). Clinical trials have been set up accordingly, grounded on the hypothesis that dementia is the end-stage result of an underlying pathophysiological “cascade” of brain changes that starts years to decades earlier with the accumulation of the proteins amyloid- $\beta$  and tau (Jack et al., 2018). Following this “amyloid cascade” hypothesis, participant recruitment has focused on people without dementia who have abnormal levels of amyloid- $\beta$  and tau in cerebrospinal fluid (CSF) or on a Positron Emission Tomography (PET) scan—also known as biomarkers of AD. Depending on whether research participants have no or only mild cognitive impairment, they are labeled in the context of these trials as having “preclinical” AD (Sperling et al., 2011) or “prodromal” AD (Dubois et al., 2014), respectively. To make research language on AD uniform, several proposals were published from 2007 onward to define AD as a pathophysiological process irrespective of clinical symptoms, all based on this “amyloid cascade” hypothesis (Dubois et al., 2007; Jack et al., 2018).

However, the “amyloid cascade” hypothesis received strong criticism when clinical trials showed that removing amyloid- $\beta$  did not delay, let alone arrest, cognitive decline (Panza et al., 2019). Moreover, around 25%–45% of those who die in old age without cognitive impairment were found to have abnormal levels of amyloid- $\beta$  in their brain (Rabinovici & Jagust, 2009). Not everyone with “preclinical” or “prodromal” AD will thus develop dementia; they are only at increased risk of developing the disease. Even so, the pathophysiological definition of AD, based on the amyloid cascade hypothesis, is now gradually moving from research toward clinical practice, where biomarkers are used to “diagnose” AD in people without dementia (de Wilde et al., 2019; Frisoni et al., 2017; Johnson et al., 2013).

In the lay literature, on the other hand, people with AD are typically described as having severe dementia, as mere reflections of the persons they used to be (Clarke, 2006), or as “the living dead” (Behuniak, 2011). Because the term AD is so commonly used as a synonym for dementia, it also serves as a metaphor for forgetfulness (Zeilig, 2013). Compelling stories of people with AD in the media are known to have constructed an understanding of AD as being a “monster” or an “enemy.” These framings of AD as a personal or a societal threat have been criticized for infusing stigma (Aquilina & Hughes, 2006; George & Whitehouse, 2014). Some advocacy groups are trying to counter this negative imagery. Alzheimer Europe, for example, provided guidelines for reflection on the portrayal of dementia in which they warn against alarmist and frightening framings (Alzheimer Europe, 2017). Despite these efforts, AD is one of the most feared diseases of aging (Harvard School of Public Health and Alzheimer Europe, 2011). It is currently unknown whether the rise of the pathophysiological definition of AD may have affected the use of the term AD as a synonym to dementia in the public debate as it also includes cognitive healthy individuals.

The parallel existence of these two different ways of using the term AD—as a pathophysiological process and as a form of dementia—raises two questions. First, does the term AD have the same meaning in the research and lay literature? Different perspectives on the disease can exist in parallel and support each other, but a fundamentally different use of the term AD by different parties may result in miscommunication. Second, if the pathophysiological understanding of AD as currently used in research becomes more dominant in clinical practice, as predicted in the research literature (Frisoni et al., 2017), how will this change the role of AD in our societies and our normative attitudes toward it? For a start, defining AD based on biomarkers instead of clinical symptoms will drastically expand the AD population. To illustrate, around half of those who are 75 years old and have mild cognitive impairment are estimated to have biomarkers of AD (Jansen et al., 2015). Given the current absence of treatment, more people will thus have an AD diagnosis for a longer period of time. This raises new ethical concerns, especially with regard to those who will never develop dementia (Schermer & Richard, 2019). Depending on the country, an AD diagnosis may affect the ability to apply for long-term care insurance, for example, or the right to hold a driver’s license. Cognitive healthy people have also been found considering fundamental life changes after receiving positive AD biomarker results, such as selling their house, even though their risk to develop dementia remained uncertain (Largent et al., 2020).

The way we write and talk about AD, the words we use, and the stories we tell, that is, the way in which we frame AD, influence the way in which the disease is understood and experienced. Hence, framings are not value-free. They link a concept to the sentiments and attitudes that are attached to the applied frame (Van Gorp & Vercruyse, 2012), for example, those linked to the framing of AD as an “enemy.” Consequently, different framings of the same concept may trigger different emotional and behavioral responses (Thibodeau & Boroditsky, 2011). For this reason, alarming framings of AD have also been used strategically to secure research funding (Fox, 2000).

Framings—and metaphors in particular—may have such a normative impact because we use them to make sense of the world around us, as famously argued by Lakoff and Johnson (1980). By making an implicit comparison with a more familiar concept, the metaphor serves as a model for how the abstract concept can be understood (Lakoff & Johnson, 1980). Particularly in science, which often revolves around making sense of abstract phenomena, metaphorical framings are crucial for theory building and hypothesizing (Knudsen, 2003). Framings of AD as a cascade in science are therefore not mere rhetorical embellishments; they are not interchangeable with any other metaphor. Rather, they reflect how the author understands AD, namely *as being like* a cascade.

In this article, we analyze the framings of AD applied in the international research literature and in Dutch national newspapers to retrieve the understandings of the term upheld in both literatures. Together, the results of these framing analyses will show whether the research understanding of AD is congruent with the use of the term in the lay literature. Second, by explicating the aspects of AD that are either emphasized or neglected by the employed framings, we analyze how the employed framings of AD steer the audiences' normative attitudes toward the disease. In the discussion, we further elaborate on the impact and consequences of our findings.

## Method

### Article Selection

**Research articles defining AD as a pathophysiological process**  
 Since 2007, six research articles have been published that propose pathophysiological disease criteria for AD for the context of research. They were written by two groups of researchers that are being referred to as the International Working Group (IWG; Dubois et al., 2007, 2014, 2016) and the National Institute on Aging–Alzheimer's Association (NIA-AA) working group (Albert et al., 2011; Jack et al., 2018; Sperling et al., 2011). Despite slight differences between these approaches, both the IWG and NIA-AA argue that AD should be (re)defined based on biomarkers of amyloid- $\beta$  and tau instead of clinical symptoms (Jack et al., 2018). This pathophysiological definition of AD has subsequently been adopted in a series of clinical trials (e.g., the A4 study; Sperling et al., 2014), in research policy documents (European Medicines Agency, 2018), and in a guideline for the clinical use of amyloid PET imaging (Johnson et al., 2013). The set of research articles that are analyzed in this paper represents a specific and prominent movement in AD research, primarily focused on clinical trials and diagnostic technologies, and does not necessarily represent the entire AD research field. This research movement is particularly interesting when analyzing AD framings because it explicitly aims to *redefine* AD and has thereby created the current unique situation of having two different definitions of AD for either research or clinical practice. Other research views on AD do not redefine the concept of the disease. Moreover, when analyzing the impact of an AD definition on priority setting in research and health care, this research movement is particularly interesting because it is endorsed by the NIA and the AA in the United States, which are among the main funding bodies for AD research.

### Lay literature: national newspaper articles

We focus on Dutch national newspaper articles to retrieve and analyze the AD framings that are central in the lay literature. First, we retrieved all articles that mentioned the term "Alzheimer" published between

July 2013 and July 2018 in the four most read national newspapers in the Netherlands (*NRC Handelsblad*, *De Volkskrant*, *Algemeen Dagblad*, and *De Telegraaf*) via the international newspaper database LexisNexis. To optimize the sensitivity of our search, the term "Alzheimer" was our only search criterion. Second, search results were screened to remove duplicates. Articles less than 200 words (e.g., short anecdotes) and articles off-topic were excluded from the analysis. Articles were labeled off-topic when removal of the term AD would not fundamentally change the article's content, such as articles on a healthy diet that was claimed to lower one's risk for AD among several other diseases such as cardiovascular disease and cancer.

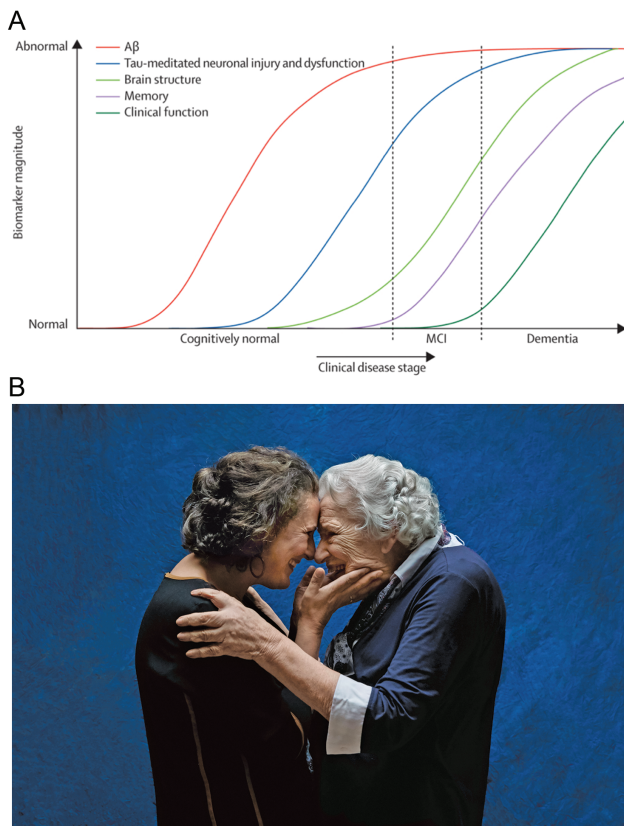
### Framing Analysis

Framings steer the audience's understanding of a concept by making certain aspects of it more salient than others (Entman, 1993). A concept can be framed by linking it to moral appeals, arguments, analogies, metaphors, images, or the wider context in which a term is mentioned. For example, the metaphorical framing of AD as a "silver tsunami" emphasizes the high (and overwhelming) number of older people who are expected to have AD in the future.

Given the specific role for metaphors in the construction of scientific understanding through conceptual models (Boyd, 1993), we focus on recurrent metaphors for AD as a pathophysiological process in the analyzed research literature. Metaphors that are routinely used for the same abstract scientific phenomenon, such as AD, within scientific communities are known as "structural metaphors" and are believed to reflect the conceptual model according to which the phenomenon is understood in that community (Lakoff & Johnson, 1980). Scientists themselves may not be aware that their research jargon consists in part of structural metaphors once the jargon is conventionalized, but, even so, analyzing structural metaphors for AD as a pathophysiological process may provide insight into the conceptual model for AD that has become increasingly prominent in research.

Different methodologies exist to analyze framings. Generally, they aim to provide insight into the author's or speaker's understanding of a certain issue, event, or concept, or into how framings steer the audience's understanding and normative attitudes toward it, or both (Thibodeau & Boroditsky, 2011; Van Gorp & Vercruyssen, 2012). We aim to do both.

Our framing analyses were set up as a qualitative content analysis with the help of Qualitative Solutions and Research NVivo 11 data analysis software. Research and lay literature were analyzed separately. First, recurring framings of AD were identified in the texts. In our analyses, we included framings in the form of single wordings, including analogies and metaphors (e.g., amyloid- $\beta$  "burden"), sentences (e.g., a newspaper title "Promising



**Figure 1.** (A) Reprinted with permission of the copyright owner: the “amyloid cascade hypothesis” pathway that aims to represent the disease model of Alzheimer disease (AD), an adjusted figure from Sperling et al. (2011) (first published in the work of Jack et al. (2010)). (B) By Cigdem Yuksel (published in the *Volkskrant* among other newspapers): a picture of a daughter and her mother, who has AD. They had a difficult relationship. The daughter feels closer to her mother now that dementia has made her less strict. These—very different—images typically represent the meaning of AD in the context of the scientific literature and the lay literature.

drug slows down Alzheimer”), or illustrations that emphasize a certain aspect of AD (Figure 1). Structural (recurrent) framings formed the central coding themes in our analysis and were adjusted when new framings came up during the analysis process. Identified framings were discussed with a second interpreter and adjusted when needed, based on consensus, to minimize bias. Central framings were selected in consultation with all authors. Lastly, following the method of qualitative content analysis, we formed clusters of the themes of framings that reflect a similar understanding of AD.

### Normative Analysis

We explicate how the framings of AD in the research literature and in the general media steer the readers’ views on the needed health care and research efforts by defining the disease or disease population and by emphasizing certain aspects of AD rather than others.

## Results

### Framing of AD in the Research Literature on Disease Criteria

In the analyzed research articles, AD is framed as essentially a pathophysiological process. For example, the claim that amyloid PET imaging increases diagnostic certainty implies that this biological insight offers relevant information about whether someone has AD (Dubois et al., 2007, 2014; Jack et al., 2018; Sperling et al., 2011). Similarly, the suggested analogy between cancer treatment in presymptomatic stages and the hope for future AD treatment in people with “preclinical” AD (Sperling et al., 2011) implies that AD is a pathophysiological process that is ongoing in persons without cognitive impairments. In what follows, we describe the two structural metaphorical frames that represent the disease model for AD that is upheld in research: AD as a biological continuum and AD as a cascade of biomarker abnormalities eventually leading to “full-blown” dementia.

### AD as a biological continuum

In all the analyzed research articles, AD is described as a biological “continuum”: a sequence of abnormalities in a series of biomarker levels that reflects the “underlying pathophysiology” of AD (Albert et al., 2011, p. 271), “AD pathophysiology” (Dubois et al., 2014), or simply “AD” (Jack et al., 2018). The understanding of AD as a continuum is a conceptual break from the binary understanding of AD according to which one either has it or one does not have it. AD is here defined by static biomarker measures of amyloid- $\beta$  and tau in CSF or on amyloid PET imaging that are believed to indicate subsequent phases of a pathological process that eventually may lead to “full-blown dementia” (Dubois et al., 2007, p. 736). This “AD continuum” starts when cognitively healthy individuals show “biomarker ‘evidence’” of AD (Sperling et al., 2011, p. 282), that is, abnormal amyloid- $\beta$  levels. These individuals are referred to as having “preclinical AD” (Sperling et al., 2011).

Framing the presence of positive biomarkers as “one of the earliest measurable stages of AD” (Sperling et al., 2011, p. 287), as “evidence” of AD (Sperling et al., 2011, p. 282), or as the main criterium for a diagnosis of ‘pre-clinical AD’ imply that these biomarkers *are* AD. This understanding of AD is, in fact, already implied in the use of the term “biomarker” itself because a “marker” per definition is a “distinctive feature or characteristic indicative of a particular quality or condition” (Oxford Dictionary, 2019). On the other hand, framings of positive biomarkers as being the “biological footprint” of AD (Dubois et al., 2007, p. 741), “a hallmark feature” (Albert et al., 2011, p. 274), and “a biological or molecular ‘signature’ of AD” (Dubois et al., 2016, p. 294) imply that AD is an unidentified pathology and that biomarkers are merely indicative of its presence. In either case, these framings imply that AD is present



once its biomarkers turn abnormal, irrespective of clinical symptoms.

The terminology used to refer to AD biomarkers is generally negative. Increased levels of amyloid- $\beta$  in the brain are, for example, being referred to as amyloid- $\beta$  “load” (Sperling et al., 2011), an “abnormality” (Jack et al., 2018), a “burden” (Dubois et al., 2016), or as a “pathological state” (Jack et al., 2018).

#### AD as a cascade from biomarker abnormalities to full-blown dementia

The nomenclature that is suggested in these articles implies a cascade model, a model of one-way inevitable progression within individuals from being cognitively healthy and having positive AD biomarkers to developing dementia. The term “preclinical” AD (Sperling et al., 2011), for example, suggests that this asymptomatic stage will be followed by “clinical AD.” Similarly, those with mild cognitive impairment and positive biomarkers of AD are labeled as “prodromal” AD (Albert et al., 2011; Dubois et al., 2007, 2014, 2016, p. 735; Jack et al., 2018; the latter uses “prodromal” between brackets). After all, more generally, the term “prodromal” denotes the period between the appearance of mild symptoms and the full manifestation of a disease (Oxford Dictionary, 2019).

Future worsening of cognitive abilities is also implied in the title and illustration of this biomarker-based disease model for AD: the “amyloid cascade hypothesis” (Figure 1A; Jack et al., 2018). A cascade is, after all, a metaphorical model of a waterfall consisting of several small streams that fall down a rocky slope. The understanding of AD as being a “cascade” recurs in phrases such as “the presence of markers of ‘upstream’ Ab [amyloid- $\beta$ ] accumulation is associated with markers of ‘downstream’ pathological change [...] Ab accumulation is sufficient to incite the downstream pathological cascade of AD” (Sperling, 2011, p. 284, emphasis added).

Relatively little attention is given to the last stage of the disease continuum in the analyzed articles, namely that of “full-blown dementia” (Dubois, 2007, p. 736). The analyzed articles are, after all, written for prevention research which is motivated by the belief that the “critical opportunity for potential intervention” has already passed in those with dementia (Sperling, 2011, p. 281).

#### Framings of AD in Dutch National Newspapers

Our search retrieved 1,571 newspaper articles that mentioned “Alzheimer.” After removing duplicates, articles less than 200 words and articles off-topic, 42 articles remained (Figure 2). The newspaper article references can be found in Supplementary Table 1. The resulting frames are described below.

#### AD as a synonym for dementia, breaking down one’s life

In the majority of the analyzed newspaper articles, the word “Alzheimer” is used interchangeably with “dementia.” This

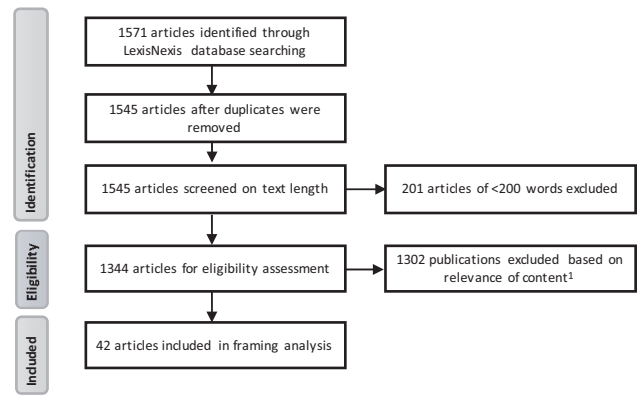


Figure 2. Article eligibility screening.

understanding of AD as a clinical disease is typically reflected in anecdotes about individuals realizing that a person had AD after he or she had demonstrated abnormal behavior. For example, an anecdote about a mother who serves her guests thick pieces of raw beef on a plate instead of cookies while explaining that she goes to a new bakery<sup>1</sup> suggests that in this article, AD equals the presence of severe cognitive symptoms associated with dementia.

The progression of the disease is described as a severe worsening of cognitive abilities, as watching someone “losing” his personality until he becomes something “human-like that makes unrecognizable sounds, and only eats, sleeps, befouls himself and wakes up again.”<sup>2</sup>

Receiving a diagnosis of AD is described by patients and their caregivers as a “destructive” experience<sup>3</sup> that brings anger, sadness, and despair to all involved.<sup>4</sup> This is in line with the framing of AD as something that “demolishes”<sup>4</sup> or “breaks down”<sup>5,6</sup> a person’s life. From this understanding of AD, it is not surprising that caring for a loved one with AD is described as exhausting<sup>7,8</sup> and that the move to a care home is considered to be an inevitable step for someone with AD. For loved ones, this move is accompanied by feelings of guilt<sup>9</sup> because they do not want “to put them away.”<sup>10</sup> The willingness to care for a loved one with AD is illustrated in Figure 1B.

Occasionally, it is explained in the lay articles that a cascade of biological processes leads to AD, but then this cascade hypothesis is often criticized as well.<sup>11</sup> More importantly, these biological explanations for cognitive symptoms are mostly framed as processes *leading to* AD rather than being AD itself. In other newspaper articles, AD is framed as a biological process mirroring cognitive decline as simultaneous processes. This is reflected in explanations such as “Alzheimer develops if the brain protein beta-amyloid is not being removed.”<sup>12</sup>

#### Alzheimer as a worst-case future scenario?

For healthy people, mainly children of people with AD, AD represents a frightening future scenario of developing severe dementia. They describe how experiences of forgetting little things, such as car keys, reinforce their fear of developing

AD and motivate them to visit a physician.<sup>13</sup> Repeatedly, AD is mentioned in personal stories of people who are setting up advanced directives in case they might—at some point in the future—suffer severely from AD or in stories about the difficulties that physicians have handling such requests from a legal perspective.<sup>14</sup>

The frame of AD as a worst-case future scenario might be tempered by the hope for future treatment. This is reflected in newspaper headlines such as “Promising drug slows down Alzheimer”<sup>15</sup> or “Preventive Alzheimer drug on the way”<sup>16</sup> or the suggestion that a healthy lifestyle helps to prevent AD.<sup>17–20</sup> Then again, these optimistic signals are toned down by the emphasis in other newspaper articles on the lack of progress in Alzheimer research over the last decades<sup>21,22</sup> and worries about insufficient attention for the care of current Alzheimer patients in the allocation of (research) resources.<sup>23</sup>

### AD as a threat to society

Estimations of the current number of Alzheimer patients in the Netherlands—ranging from 140,000<sup>14</sup> up to 256,000 people on a population of around 17 million—are often mentioned at the start of newspaper articles, emphasizing the magnitude of the “societal problem” that is AD. Estimations of the “exploding”<sup>24</sup> number of AD patients that is expected in the future—ranging from 300,000,<sup>25</sup> 538,000,<sup>26</sup> or even 1 in 5 people in 2040<sup>27</sup>—strengthen the frame of AD as a societal threat, similar to phrases such as “Alzheimer threatens to suppress the Dutch healthcare system”<sup>28</sup> and “Alzheimer makes increasingly more victims in the Netherlands.”<sup>29</sup> In combination with raised concerns about inadequate health care support at home<sup>30</sup> or in care homes,<sup>31</sup> this paints a frightening image. While some researchers suggest that the only solution for this “doom scenario”<sup>32</sup> lies in more research investments, others criticize the way in which AD has been “blown up” to a national disaster.<sup>33</sup>

### Normative Implications of the Identified Framings

The cascade and continuum framings of AD employed in the research articles imply that cognitively healthy people have AD—hence, *are diseased*—once their biomarker levels pass the set threshold for positivity. Furthermore, an understanding of AD as a pathophysiological cascade steers into thinking about solutions for AD in the form of interventions targeted at abnormal biomarkers that are applied as early in the disease cascade as possible. This explains that for prevention researchers “the neurobiological advantage of earlier intervention within this cascade is clear” (Dubois et al., 2007, p. 735).

In the analyzed newspaper articles, AD is portrayed as a devastating disease—a synonym to dementia—that starts with forgetting little things and ends, inevitably, in a stage of being mentally and physically dependent on the continuous care of others. This framing of AD implies that

receiving an AD diagnosis equals having a severe cognitive impairment and facing a future of progressive decline. The hope for a future treatment for AD is framed as ambiguous, while health care resources are described as falling short.

### Discussion

There is a strong movement in AD research toward metaphorically portraying and defining AD as a pathophysiological continuum or cascade. This framing implies that people with positive biomarkers may be unaware of having (preclinical) AD and, hence, are unaware of facing a future of dementia until they undergo biomarker testing. It should be noted here that many of those who have biomarkers of AD will actually never develop dementia (Richard et al., 2013).

Framing of biomarker testing as a *diagnostic* tool for AD in line with its pathophysiological understanding tends to legitimize its use in clinical practice in people without dementia who want to know if they have AD (Boenink, 2018), a population that is increasingly seeking medical attention (Gruters et al., 2019; Le Couteur et al., 2013). The importance of offering more *diagnostic* certainty is currently an important motivation to test biomarkers in people without dementia in specialized memory clinics (de Wilde et al., 2019) and to endorse this practice in clinical practice guidelines (Johnson et al., 2013). The argument that biomarker results may increase *diagnostic* certainty does not hold when applying a definition of AD as a form of dementia, according to which a diagnosis revolves around the presence of clinical symptoms rather than biomarkers. From that perspective, the added value of biomarkers can only be framed in terms of its *prognostic* value, that is, to what extent biomarkers can predict the onset of dementia. However, the *prognostic* value of biomarkers is generally considered to be too poor for clinical practice (Bunnik et al., 2018; Petersen et al., 2018).

The framing of biomarker testing as an improvement of AD “diagnostics” in people without dementia also has implications for priority setting in research and health care; it steers toward widespread clinical adoption of biomarker tests. This would require enormous financial investments (Wimo, 2018). Given the scarcity of health care resources, this framing distracts attention from the need of psychosocial support for those already having dementia or their caregivers (Jongsma & Sand, 2017; Leibing, 2015). Moreover, framings of “full-blown” dementia as a stage beyond hope may exacerbate the relative quiescence of research efforts directed at the care for and support of dementia patients. This framing also leaves little room for societal factors that may drive AD, such as lifestyle and education, a viewpoint that may yield major health benefits (Lock, 2013). As the authors of the pathophysiological definition of AD, the Alzheimer’s Association therefore seems to take a normative stance on priority setting in research, in line with its past (Fox, 2000). Whether and how the

commercial interest of pharmaceutical companies plays a role in encouraging biomarker testing in clinical practice is not clear.

Furthermore, our analysis of Dutch newspaper articles shows that the traditional definition of AD as a form of dementia has been preserved. This is in line with previous research results finding that people with AD are typically portrayed in a severe stage of dementia rather than in a stage in which they can still act (relatively) independently (Clarke, 2006). When interpreting these results, it should be kept in mind that we only analyzed newspaper articles, which may not reflect the lay literature in its totality, and focused on the Netherlands, while understandings of AD may differ between countries. Framings in newspaper articles might be influenced by scientists or pharmaceutical companies who may have an interest in telling a certain story about AD, for example, on a promising line of research. Even so, newspapers are an important and trusted source of information in the Netherlands and a platform for prominent opinion formers and may therefore reflect and influence the readers' perspective on AD. Future research may involve comparing our findings with AD framings employed on other forums—for example, used by a specific community of older people—or in other countries.

We found that a diagnosis of AD is described in the lay literature as demolishing a person's life, knowing that memories and the ability to act independently will be lost. Popular framings of AD steer one into believing that when one receives a diagnosis of AD, one should set up advanced directives and try to enjoy the time that is left before moving into a nursing home. This portrayal of AD in the lay literature provides insight into the potential negative consequences of providing people with a "diagnosis" of AD in the absence of an effective treatment. People may start to see themselves as sick or others may start to treat them that way (Alzheimer Europe, 2017). These consequences are especially worrisome for those receiving a "preclinical" or "prodromal" diagnosis of AD who may never develop dementia.

In combination with the emphasis on the large number of people having AD in the Netherlands and the prediction of a further increase in the future, the popular framing of AD steers one into thinking that a large policy strategy should be set up to ensure suitable care for people with dementia and their caregivers (Cuijpers, 2016). It steers in an opposite direction in terms of priority setting in research and health care compared to the AD framing in research; it emphasizes the need to invest in practical and psychological support related to dementia care rather than in "earlier" diagnosis based on biomarkers.

The parallel existence of these two prominent framings of AD—as a pathophysiological process and as a synonym for dementia—may lead to miscommunication. Whereas the researchers' framing focuses on those who are (relatively) healthy and at risk of developing dementia, the popular framing of AD focuses on those who are in the later

stages of dementia. Clearly, these two understandings of the term AD are incongruent: people who have biomarkers of AD but no dementia have AD according to the research definition, while they do not fall within the understanding of AD that is upheld in the lay literature. The translation of the research framing of AD into clinical practice may lead persons with subjective cognitive decline or mild cognitive impairment to be told that they have AD which, for them, implies that they have dementia and will face a bleak future of deterioration, in line with the lay framing of AD, which they are familiar with, conveys. Many research models are not incongruent with a lay understanding of AD, such as those reflecting an understanding of AD as a result of the biological aging process (Kaeberlein, 2019).

Little is known about how this information can be safely conveyed in clinical practice, but modes and methods for the safe and responsible communication of AD biomarkers to people without dementia have been developed for the research context (Grill et al., 2013; Milne et al., 2017). Research guidelines for disclosing AD biomarker results to participants recommend organizing elaborate education sessions and to refrain from framing biomarkers as "diagnostic" tests for AD to avoid the (anticipated) misinterpretation that having abnormal results means that you will, undoubtedly, develop dementia (Grill et al., 2013; Harkins et al., 2015). Researchers are well aware of the uncertainties around the clinical interpretation of biomarker results (Lock, 2013). Still, 19 of the 50 cognitively healthy research participants who received education sessions on the meaning of biomarker results (and were screened beforehand on anxiety and depression) did not understand that elevated amyloid levels reflect an increased *but uncertain* risk of developing dementia (Mozerky et al., 2018). These recent findings suggest, in view of our results, that the educational efforts to explain the pathophysiological understanding of AD might not be able to overwrite the persistent and longstanding framing of AD as a synonym to dementia in the lay literature nor people's own experiences with AD as a form of dementia.

Moreover, our results suggest and illustrate how the risk of miscommunication may be increased by the framings of AD in research jargon that may generate false expectations. The analyzed research literature recurrently states that amyloid- $\beta$  is possibly "not causal in AD pathogenesis" (Jack et al., 2018, p. 536) and not everyone with positive biomarkers will develop dementia, yet the employed framings of AD suggest otherwise. For example, the framings of amyloid- $\beta$  as the "underlying disease" (Dubois et al., 2016) or the "pathophysiology" and cognitive decline as "clinical consequences of the disease" (Jack et al., 2018, p. 535) do suggest that the biomarker is causally linked to dementia. Similarly, the nomenclature "preclinical" and "prodromal" AD also suggest that developing "clinical" AD is the logical next disease stage. These framings leave no room for the significant possibility that a person's cognitive functioning will remain intact, let alone

improve, even in the presence of AD biomarkers (Boenink, 2010). These AD framings thus *falsely* imply an inevitable future cognitive worsening. This is especially worrying because having such expectations is known to create expectations of discrimination, higher pity, and social distance (Johnson et al., 2015) and thereby to contribute to stigma. This means that people may be harmed by potentially misleading framings of AD that are widespread in current research jargon. Therefore, they should not be employed in the communication toward the lay audience, in research, media performances, and elsewhere. Future research will have to prove whether this could help to prevent miscommunication.

This is not to say that preserving the current understanding of AD as a pathological process in research, and thereby maintaining the current status quo of two different uses of the term AD, is desirable. The previously discussed disadvantages of the current research framings of AD might be avoided by referring to biomarkers as positive or negative amyloid- $\beta$  or tau results without equating it to “Alzheimer’s pathology” or “AD.” Interestingly, this alternative was suggested in the most recent NIA-AA proposal for research criteria for AD (Jack et al., 2018). Following this alternative, the term AD could (again) refer to a clinical diagnosis of dementia and these biomarkers as “risk factors” for the disease. It does justice to the fact that not all persons with positive biomarkers will develop dementia; it steers away from the negative consequences of providing (relatively) healthy people with an AD diagnosis and it is in line with the meaning of AD upheld in the lay literature. Therefore, we support this proposal to refrain from defining AD by biomarker results, in research and elsewhere.

## Conclusions

Our results show two different understandings of AD that exist in parallel. In research, the term AD refers to a pathophysiological continuum or cascade that starts with abnormal biomarkers in cognitively healthy individuals and ends in “full-blown” dementia. In the lay literature, on the other hand, the term AD is used as a synonym for (severe) dementia. Due to these differences in understanding AD, miscommunication may arise when researchers explain biomarker results to people without dementia, in a research setting, clinical practice, or the media. Researchers should be aware that participants’ understandings of AD may be fundamentally different and that research framings of AD could be confusing. Therefore, they should explain the meaning of biomarker results in terms that align with the public understanding of AD and hence in terms of being a risk factor rather than a disease. This will also avoid burdening those who will never develop dementia with the potential negative psychological and social consequences of receiving an AD diagnosis.

Our results imply that the gradual integration of the pathophysiological (research) definition of AD in clinical practice tends to legitimize widespread biomarker testing by appeal to the importance of improving “diagnostic certainty.” This focus on AD biomarker testing in people without dementia may have undesirable implications for priority setting in research and health care, especially in the absence of effective treatment, because it distracts attention from those with dementia. We therefore plead in favor of preserving the definition of AD as a synonym to dementia in clinical practice.

## Supplementary Data

Supplementary data are available at *The Gerontologist* online.

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## Conflict of Interest

None declared.

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