

September 2022

# Prevalence of Alzheimer's Disease in Adults in the United States

Margaret George and Erica Bassett



# Summary

Alzheimer's disease (AD) has become increasingly prevalent among adults in the United States, with the CDC estimating that AD affects 5.4 million people in the US. As a neurodegenerative disease, AD leads to life-threatening physical and cognitive effects for adults ages 65 and older. Many risk factors contribute to the possibility of an individual developing AD in the United States. Some of these risk factors an individual can somewhat control, including wellness choices and the quality of one's social relationships. Other factors are more challenging for an individual to control, such as one's genetic history and environmental factors like air pollution. The prevalence of AD ultimately results in high mortality and morbidity rates, substantial economic losses, and caregiver burden in the US. Considering that AD currently has no cure, nationwide efforts such as research into wellness

programs may help alleviate this issue.

## Key Terms

**Alzheimer's Disease (AD)**—A degenerative brain disease and the most common form of dementia.<sup>1</sup>

**Alzheimer's Disease Continuum**—The gradual progression of Alzheimer's Disease from preclinical AD (where no symptoms manifest) to severe dementia (where symptoms interfere with daily living). In the mild stage, most individuals can function independently with difficulty in everyday tasks. In the moderate stage, individuals experience memory and language challenges and may need assistance with tasks like dressing. In the severe stage, individuals require regular care and have significant memory and language challenges. The length of each stage depends on several factors, such as age, sex, and genetics.<sup>2,3</sup>

**Apolipoprotein-e4 (APOE-e4)**

**Allele**—One variation of the Apolipoprotein-e gene, which involves the metabolism and

repairing of cell membranes in the central and peripheral nervous systems. This allele most likely contributes to plaque buildup and neurofibrillary tangles, both risk factors for developing Alzheimer's Disease.<sup>4</sup>

**Atherosclerosis**—"The build-up of fats, cholesterol and other substances in and on your artery walls (plaque), which can restrict blood flow."<sup>5</sup>

**Beta-amyloid Proteins (A $\beta$  protein)**—A complex protein found on the surface of cells throughout the body. This protein assists in neural growth and repair. Corrupted forms of this protein destroy neurons and ultimately contribute to Alzheimer's Disease.<sup>6, 7</sup>

**Chronic Illness**—Medical conditions that last one year or more and require ongoing medical attention. Examples of chronic illnesses include arthritis, asthma, diabetes, and heart disease.<sup>8</sup>

**Cortisol**—The body's primary stress hormone. Cortisol increases glucose levels and, when at chronically high levels, can alter the body's immune

system response and increase the risk of various diseases.<sup>9</sup>

**Dementia**—"A usually progressive condition (such as Alzheimer's disease) marked by the development of multiple cognitive deficits such as memory impairment, aphasia, and the inability to plan and initiate complex behavior."<sup>10</sup>

**Disease**—"A condition that impairs normal functioning and is typically manifested by distinguishing signs and symptoms."<sup>11</sup>

**Disorder**—"An abnormal physical or mental condition."<sup>12</sup>

**Gray Matter**—Tissue that makes up the outermost layer of the human brain. This brain tissue exists along the brain, brain stem, and spinal cord. Gray matter is dense in unmyelinated axons (creating its pinkish gray tone), whereas white brain matter is composed of myelinated axons. Gray matter contains networks of neurons that are responsible for essential motor functions.<sup>13, 14</sup>

**Morbidity**—The condition of ill health or disease.<sup>15</sup>

**Neurofibrillary Tangles**—Abnormal accumulations of tau proteins inside neurons, which damages neural networks and prevents the brain from communicating properly. The abnormal accumulations most often reside in brain areas involved in memory.<sup>16</sup>

**Neuroinflammation**—An inflammatory response in the brain or spinal cord. While low levels of neuroinflammation can benefit the body's immune system and brain function, severe neuroinflammation can lead to tissue damage, stress, and cell death.<sup>17, 18</sup>

**Pathology**—“The study of the essential nature of diseases and especially of the structural and functional changes produced by them.”<sup>19</sup>

**Resistiveness**—Any opposition to care, typically during an encounter between a caregiver and care recipient.<sup>20</sup>

**Risk Factor**—“A factor, such as a habit or an environmental condition, that predisposes an individual to develop a particular disease.”<sup>21</sup>

**Tau Protein**—A protein that helps stabilize the structure of a neuron and facilitate the transportation of nutrients and other essential molecules. In unhealthy amounts, tau proteins cling to one another instead, creating protein clumps known as neurofibrillary tangles. These neurofibrillary tangles, made up of tau proteins, disrupt neural functions and are a risk factor for AD.<sup>22, 23</sup>

## Context

**Q: What is Alzheimer's, and how does it relate to dementia?**

**A:** Alzheimer's disease (AD) is a neurodegenerative disease that targets specific brain regions related to memory and executive function. For instance, AD damages the hippocampus and the nearby entorhinal cortex, both involved in forming and storing memories.<sup>24</sup> AD also targets the frontal lobe, an area responsible for judgment and behavior, and areas of the cerebral cortex responsible for language, reasoning, and behavior.<sup>25</sup> Although AD damages critical brain areas, the disease does not directly kill an individual. Instead, complications from the decline in brain function lead to death. Some examples of these complications include vascular changes, infections, injuries from falls, and malnutrition.<sup>26, 27</sup>



Although AD manifests itself in various ways, behavioral and cognitive symptoms generally characterize it.<sup>28</sup> For example, common cognitive symptoms of AD include forgetfulness, disorientation, confusion, and difficulty staying focused. Individuals with AD may also exhibit behavioral or physical symptoms such as wandering, self-harm, inability to swallow or feed oneself, resistiveness, and agitation.<sup>29</sup> Other general AD symptoms include difficulty staying focused, being increasingly overwhelmed, inability to understand instructions, and difficulty finding the right words.<sup>30</sup> Because of these symptoms, individuals with AD often require increased attention or medical care.

A genetic mutation creating excessive production of beta-amyloid proteins causes Alzheimer's disease. This overproduction of proteins leads to amyloid plaques in blood vessels, which limit blood flow to critical areas of the brain and subsequently destroy brain cells.<sup>31</sup>

<sup>32</sup> These amyloid plaques are specific to Alzheimer's disease and distinguish AD from other types of dementia. An additional cause of Alzheimer's disease is abnormal tau protein production. This protein is a key component of microtubules, which transport nutrients between nerve cells. Thus, abnormal tau protein production threatens microtubule structures and increases the risk of AD.<sup>33</sup>

While Alzheimer's is the most common type of dementia,<sup>34</sup> dementia is an umbrella term for memory loss and other impairments to an individual's cognitive function. Dementia leads to decreased ability to perform activities of daily living.<sup>35</sup> The term refers to several types, such as Lewy

body dementia, frontotemporal dementia, mixed dementia, Parkinson's disease dementia, Alzheimer's disease, and others.<sup>36, 37</sup>

### ***Q: How is Alzheimer's diagnosed?***

**A:** The stages of Alzheimer's disease, from the mildest to the most severe stages, are termed as follows: cognitively normal, significant memory concern, early mild cognitive impairment, mild cognitive impairment (MCI), mild dementia, late mild cognitive impairment, and full Alzheimer's disease.<sup>38</sup> Alzheimer's disease is often diagnosed during the mild dementia stage, in which it becomes evident to family and doctors that the patient has "significant trouble with memory and thinking that impacts daily functioning."<sup>39</sup> Computed tomography (CT) scans can be used to diagnose Alzheimer's disease definitively, or the disease can be suspected based on medical history,

characteristic changes in thinking, physical examinations, and laboratory tests.<sup>40</sup>

Identifying the disease is arduous, particularly since Alzheimer's results in several complications that may hide the disease. The plaques indicative of Alzheimer's disease are not fatal in and of themselves. Still, the nerve connections that AD destroys in the brain make ordinary tasks difficult and unfamiliar. Complications are common among patients with Alzheimer's, as neurodegeneration leads to physical effects. Many persons whose underlying cause of death is Alzheimer's die from a complication due to Alzheimer's.<sup>41</sup> In 2021 alone, over 120,000 deaths were attributed to AD.<sup>42</sup> Complications may include vascular changes, aspiration pneumonia, bedsores, sepsis infections from undiagnosed urinary tract infections, infections in general, injuries from falls, malnutrition, and dehydration.

### ***Q: What are the risk factors for developing Alzheimer's disease?***

**A:** Many risk factors contribute to the possibility of an individual developing AD, some of which an individual can somewhat control (modifiable factors) and some which an individual cannot control (nonmodifiable factors). Nonmodifiable risk factors, which increase an individual's risk of developing Alzheimer's, include age, sex, family history, race, and genetics. The research discusses extensively how nonmodifiable risk factors correlate with Alzheimer's.<sup>43</sup> However, several studies suggest a strong correlation between increased risk of AD and the presence of somewhat-modifiable factors, such as chronic high blood pressure,<sup>44, 45, 46</sup> various heart diseases,<sup>47, 48</sup> high cholesterol,<sup>49, 50</sup> and diabetes.<sup>51, 52, 53</sup> For instance, chronic high blood pressure promotes plaque buildup, known as Atherosclerosis, which can kill

cortical tissue and ultimately lead to cognitive impairment.<sup>54</sup>

Simultaneously, regular exercise, a low-salt diet, and other health-promoting treatments can treat chronic high blood pressure.<sup>55</sup> This brief will further discuss modifiable risk factors for AD in the Contributing Factors section.

***Q: Who is most likely to be affected by Alzheimer’s disease?”***

**A:** AD is much more likely to affect those who are older; popular statistics indicate that once an individual reaches the age of 65, their risk of developing Alzheimer’s doubles every five years.<sup>56</sup> Additionally, nearly two-thirds of those diagnosed with AD in the United States are women.<sup>57</sup> Recent studies indicate women may be more likely to develop AD because women tend to live longer than men, and the hormone changes that occur in many women’s bodies due to aging may make females more susceptible to

developing the amyloid plaques characteristic of AD.<sup>58</sup> For women or men who carry the apolipoprotein-e4 (APOE-e4) allele, the risk of developing AD (and developing AD earlier) is higher than for individuals who do not carry the allele.<sup>59, 60</sup>



2 in 3 people with Alzheimer's Disease are women

Race has also been a noted nonmodifiable risk factor in the development of AD; studies indicate that in the United States, African American and Latinx individuals are more likely to develop AD than individuals of European or American heritage.<sup>61, 62</sup> There are many theories for why this is the case, including the hypothesis that African American and Hispanic individuals are more likely to carry the APOE-e4 allele that leaves them more susceptible to developing AD.<sup>63,</sup>

<sup>64</sup> However, we note that there are significant criticisms of the designs



used to determine statistics on AD, the most notable being that medical research in the past has not appropriately accommodated minorities and, as a result, may lead to misdiagnosis and mistreatment in medicine.<sup>65</sup>

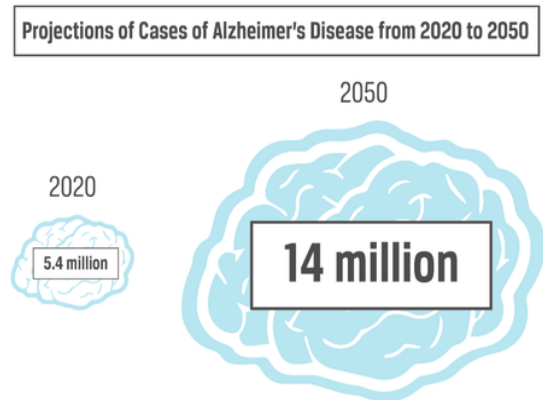
Given the complex nature of mental health issues, it is unclear how much mental health issues such as depression independently correlate with increased risk of AD.<sup>66</sup>

<sup>67</sup> However, one study notes that patients with severe depression were 2.7 times more likely to be diagnosed with dementia in the next 3 years.<sup>68</sup>

***Q: Has Alzheimer’s become more prevalent in recent years?***

**A:** Since its discovery in 1906, Alzheimer’s disease has become increasingly prevalent among adults in the United States. Today, the CDC estimates that 5.4 million people in the US have Alzheimer’s disease.<sup>69</sup> Meanwhile, a report from the Alzheimer’s

Association estimates that 6.5 million adults over the age of 65 have AD in the United States and reports that each year, 100,000 people develop AD.<sup>70</sup> Some studies estimate that by 2050, 14 million people will have AD.<sup>71, 72, 73</sup> Because research methods to identify and diagnose AD is improving, Alzheimer’s disease most likely will continually become more prevalent in the future.



Alzheimer’s disease is currently the sixth-leading cause of death in the United States and is the fifth-leading cause of death for those ages 65 and older,<sup>74</sup> killing more than breast cancer and prostate cancer combined each year.<sup>75</sup> By 2040, approximately one in five Americans will be age 65 or older, rising from

one in eight in 2000.<sup>76</sup> Current projections estimate that in 2050, 1.6 million people in the US could die due to AD, a number that is nearly triple the number of recorded AD deaths in 2010.<sup>77, 78</sup>

Interestingly, research continues to debate whether the actual risk of developing AD will increase, decrease, or remain constant in the US. Several studies indicate that in the last 25 years, the actual risk of developing Alzheimer's disease has either remained stable or declined in the United States and in other Western countries.<sup>79</sup> Researchers have attributed the decline to several factors, such as improvements in education and more excellent controls over cardiovascular risk factors.<sup>80</sup> Other studies argue that the risk has held constant in the last 20 years and will continue to do so.<sup>81</sup> Overall, the risk of AD may be declining or holding constant, AD's prevalence in the US continues to rise as more adults age into their senior years.

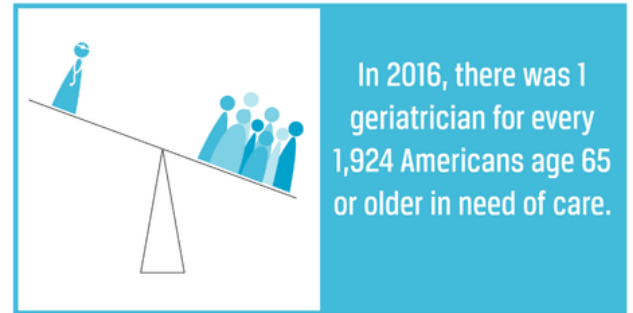
### ***Q: Is there a cure or treatment for Alzheimer's?***

**A:** There is currently no sure way to prevent or cure this disease.<sup>82</sup> Current management of the disease includes five FDA-approved drugs, many of which temporarily improve cognitive symptoms for patients.<sup>83</sup> There are currently no drugs that are FDA approved to treat behavioral and psychiatric symptoms that often develop in patients with Alzheimer's disease.<sup>84</sup> Despite the spending of billions of dollars on research, no cure or permanent means for slowing down the disease exists.<sup>85</sup> However, the identification of varying degrees of Alzheimer's disease has encouraged more significant attention to preventative measures.

### ***Q: How does this issue look today?***

**A:** There is still room for improvement in the care and resources provided for Alzheimer's

patients and their caregivers, as well as the efficiency with which healthcare professionals detect and diagnose the disease. Currently, outside of research settings, many individuals who should be diagnosed with Alzheimer’s disease are not getting the diagnosis they need—being underdiagnosed, misdiagnosed, or undiagnosed because physicians avoid prematurely diagnosing Alzheimer’s disease without a full diagnostic workup.<sup>86</sup> In 2016, there was “one geriatrician for every 1,924 Americans age 65 or older in need of care.”<sup>87</sup> Far from the recommended ratio of 1 geriatrician per 3 or 4 patients, this 2016 figure indicates that the United States health system does not have enough trained geriatric healthcare professionals to meet the need of the increasing prevalence of Alzheimer’s disease in America.<sup>88, 89</sup>



SOURCE: "Current Geriatrician Shortfall," The American Geriatrics Society, accessed December 4, 2018, [https://www.americangeriatrics.org/sites/default/files/inline-files/Current-Geriatrician-Shortfall\\_0.pdf](https://www.americangeriatrics.org/sites/default/files/inline-files/Current-Geriatrician-Shortfall_0.pdf).

## Contributing Factors

### *Lifestyle Choices*

Lifestyle choices, such as physical activity and stress management, contribute to the prevalence of AD because these behaviors impact an individual’s risk of developing AD. Recent research demonstrates that key factors contributing to the development of chronic illnesses such as Alzheimer’s include “high blood pressure, tobacco smoking and second-hand smoke exposure, high body-mass index (BMI), physical inactivity, alcohol use, and diets low in fruits and vegetables and high in sodium and saturated fats.”<sup>90</sup> While not the only risk factor for AD, lifestyle choices makeup approximately 35% of dementia cases and thus are

important modifiable risk factors.<sup>91</sup> Furthermore, addressing these lifestyle choices can seriously reduce individual and societal AD risk, given the prevalence of these choices in the US. One report estimates that reducing these modifiable behaviors even by 10% to 25% can prevent as many as 3 million cases of AD worldwide.<sup>92</sup> Thus, these modifiable risk factors impact the prevalence of AD in the US.



### ***Physical Inactivity***

Physical inactivity has long-term impacts on cognitive activity and the risk for AD. In the US, over 60% of adults do not engage in the recommended amount of daily physical activity, which is either 30 minutes of moderate activity (such as brisk walking) or 15–20 minutes

of strenuous activity (such as jogging or running).<sup>93</sup> Research demonstrates that physical activity, even regular walking, is associated with greater gray matter in several brain areas, improving brain function. Physical activity also increases the size of the hippocampus—a region of the brain responsible for memory formation.<sup>94</sup> One study examined AD patients who did not exercise regularly and AD patients who did regular exercise. The researchers found that AD patients who did not regularly exercise had a 1.5% decline in the size of their hippocampus, while AD patients who did regular exercise had a 2% increase in the size of their hippocampus.<sup>95</sup> While not a perfect solution, the increased size of certain brain areas, including the hippocampus, leads to improved cognitive function. Other studies support the correlation between regular physical activity and improved memory and cognitive function.<sup>96</sup> One study noted that 1 year of regular, moderately intense

exercise (40 minutes a day, 3 days a week) increased spatial memory in healthy older adults and led to reductions in memory impairment.<sup>97</sup> <sup>98</sup> For US adults, regular physical exercise can significantly reduce the risk of AD and improve the quality of life for individuals with AD. One study from 2020 estimates that individuals who exercise regularly reduce their risk of developing AD by approximately 45%.<sup>99</sup> Therefore, physical activity substantially impacts one's risk for and experience with Alzheimer's.

### ***Poor Stress Management***

Prolonged stress poses an additional risk to the development and severity of AD in adults.<sup>100</sup> In recent surveys, only 28% of adults in the US reported managing their stress well through mediums like listening to music, exercising, meditating, and spending time with loved ones.<sup>101</sup> In a 2019 poll, 55% of adults in the US said they experienced stress during "a lot of the day" compared to 35% of adults globally.<sup>102</sup> Research demonstrates that chronic strain

drives the progression of several diseases and worsens symptoms because of increased cortisol levels.<sup>103, 104, 105,</sup> <sup>106</sup> This behavior occurs for cardiovascular diseases, cancers, neurodegenerative diseases such as AD, and other conditions. These various diseases, alongside exacerbating stress and cortisol, then disrupt healthy stress circuits, increasing stress and leading to further complications such as depression, anxiety, and aggressive behavior.<sup>107</sup> As for AD, studies with mice and rats reveal that exposure to stressors increased the production of A $\beta$ , leading to greater amyloid plaque buildup and neurodegeneration.<sup>108, 109</sup> These elevations persisted for up to 12 months after researchers exposed the animals to stress.<sup>110, 111</sup> In a similar pattern, tau protein tangles associated with AD risk were also exacerbated by prolonged stress, leading to increased production of A $\beta$  and eventual neurodegeneration. Thus, prolonged strain alters the

body's normal processes and increases the risk of several diseases, including AD.

Further research demonstrates how stress rapidly progresses AD and other dementias by elevating levels of cortisol.<sup>112</sup> Although research has yet to determine if effective stress management decelerates the progression of AD, several studies note that it is still beneficial to individuals with AD.<sup>113</sup> For instance, stress management may lower stress hormones and amyloid production, thus slowing cognitive decline.<sup>114, 115, 116</sup> Considering that the prevalence of anxiety is reportedly higher in elderly adults than in younger age groups (approximately 15% of all older adults globally),<sup>117</sup> poor stress management may increase the severity and prevalence of AD for adults in the US.<sup>118</sup>

### ***Social Interaction***

Alongside lifestyle choices, one's quality of personal relationships impacts one's overall cognitive health and risk of AD. Generally,

supportive relationships and a rich social network—networks in which an individual maintains long-term, positive, and supportive relationships with others—are key factors to improved cognitive function and lower cognitive decline in older adults.<sup>119, 120, 121</sup> One study found that healthy social networks disrupt the relationship between cognitive function and dementia, where cognitive function remained higher for participants who reported strong social networks.<sup>122</sup>

Unfortunately, research on the exact relationship between social networks and biological processes surrounding dementia risk is inconclusive. However, one study reported a statistically significant relationship between social network quality and the density of neurofibrillary tangles, where the decreased quality of relationships correlated with increased tangle density and increased AD risk.<sup>123</sup> Therefore, strong social networks are a factor in the prevalence of AD.



Extensive research supports a negative correlation between the risk of AD and the extent of one's contact with loved ones.<sup>124, 125, 126</sup> Similarly, feelings of loneliness are associated with an increased risk of dementia, including AD.<sup>127, 128</sup> In one study, elderly participants who reported satisfaction in their relationships had a 23% reduced risk of AD, and participants who reported “[emotionally] receiving

more than they gave” had a 53% reduced risk of AD.<sup>129</sup> These findings have serious implications for reducing the prevalence of AD in the United States. However, we note that measures of healthy social networks are inconsistent in existing research. For instance, studies may utilize marital status, frequency of contact with loved ones, perceptions of relationship quality, number of loved ones, overall satisfaction, or a combination of these measures.<sup>130</sup> Therefore, while the quality of relationships influences dementia risk, these individual measures of healthy social networks may influence overall risk at varying levels.

### ***Environmental Factors***

Environmental factors such as air pollution contribute to the prevalence of AD because such external factors influence one's internal processes, including one's nervous system. More specifically, exposure to toxic air pollutants leads to AD by accelerating A $\beta$

accumulation<sup>131</sup> and prompting other AD-specific processes.<sup>132, 133, 134</sup> Air pollution includes various particles, such as particulate matter (PM), gases (e.g., ground-level ozone, carbon monoxide), organic compounds, metals, etc.<sup>135, 136</sup> These particles can have effects on an individual at all stages of human development, culminating in various diseases.<sup>137</sup> For example, PM is made of organic and inorganic materials and can originate from road and agricultural dust, refineries, power plants, mobile sources, construction, and other common sources.<sup>138</sup> PM is associated with respiratory damage and brain inflammation, leading to neuron damage and an increased risk of AD (along with other conditions, such as cardiovascular diseases).<sup>139, 140, 141</sup> Overall, an estimated 135 million Americans live in regions with unhealthy levels of ozone and air pollution.<sup>142</sup> Given that an estimated 67 million tons of pollution were emitted in the US in 2021, air pollution continues to be relevant to the issue of AD.<sup>143</sup>

Exposure to toxic air pollutants leads to AD through several processes—namely, by accelerating A $\beta$  accumulation,<sup>144</sup> prompting abnormal production of the tau protein, and inducing neuroinflammation and oxidative stress.<sup>145, 146, 147</sup> Each of these processes is a key indicator of AD risk, as discussed earlier. One study suggested that exposure to air pollutants may lead to premature aging and disease progression.<sup>148</sup> For individuals in urban areas, these effects are particularly prevalent.<sup>149, 150</sup> For instance, one study found that participants in urban areas, where air pollutants are more prevalent, had more abnormalities in expressing the APOE-e4 allele and producing the tau protein. Of these individuals, 51% also had A $\beta$  plaque buildup.<sup>151, 152</sup> Therefore, the prevalence of air pollution in the US correlates with the prevalence of AD in the US, given pollutants' effects on an individual's internal processes.



# Consequences

## *High Mortality and Morbidity Rate*

The prevalence of Alzheimer's in the US leads to an increase in morbidity and mortality rates because AD and other dementias directly lead to life-threatening medical complications. This process is known as the Alzheimer's disease continuum, where the degenerative changes made to the brain as a result of Alzheimer's disease cause problems with memory and eventually lead to physical disability.<sup>153, 154</sup> The AD continuum begins with the phase of preclinical Alzheimer's, which manifests no symptoms, and continues until the individual has severe Alzheimer's disease, where the patient needs around-the-clock care to complete tasks involved in daily living—a process spanning an average of 4–8 years.<sup>155, 156</sup> An individual with Alzheimer's may experience complications such as immobility, malnutrition, and

swallowing disorders. Alzheimer's may also compromise an individual's immune system response, leading to conditions like pneumonia, which is commonly identified as the immediate cause of death among older Americans with dementia.<sup>157</sup>

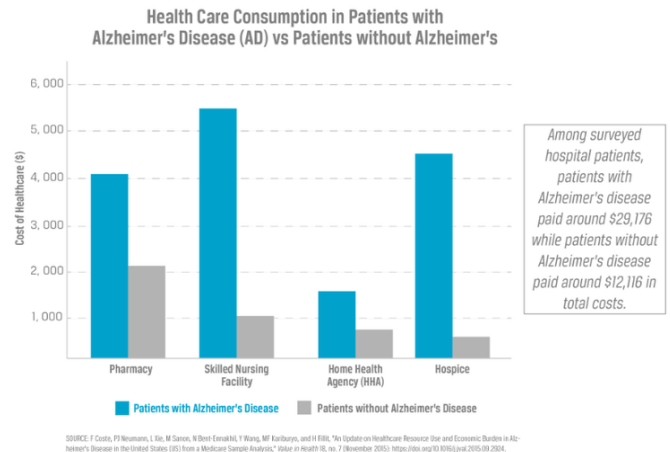
In recent years, the prevalence of Alzheimer's in the US has led to increased deaths due to AD. In the United States in 2000, there was a noted 17.6 deaths due to AD per 1,000 deaths; in 2018, there were 37.3 AD deaths per 1,000 deaths.<sup>158</sup> Instead of concluding that this trend is evidence of AD becoming more deadly, it would be more appropriate to associate this trend with our increased understanding of AD and its effects on the human body. Increased medical understanding has likely led to an increase in pronouncing AD as the cause of death on death certificates.<sup>159</sup>



## Negative Economic Impact

The prevalence of Alzheimer's also leads to negative economic impacts, as the public typically funds long-term care and healthcare payments in the form of Medicare, Medicaid, and Social Security. In a Medicare sample analysis conducted in 2015, patients with Alzheimer's had significantly more costs per patient-per-year health care consumption in inpatient facilities than patients without Alzheimer's. One study found that older patients with AD incurred higher annual healthcare costs in several categories: hospice care, inpatient services, pharmacy, emergency, ambulatory care, and others. For instance, older patients with AD incurred total annual costs

of approximately \$29,176, whereas older patients without AD incurred an average of \$12,116.<sup>160</sup>



Alzheimer's patients also incurred significantly higher inpatient, pharmacy, and total costs.<sup>161</sup> One report estimated that the cost of caring for individuals with Alzheimer's in 2020 was \$305 billion. Of this amount, \$206 billion, or 67%, is expected to be covered by Medicare and Medicaid. Another report estimates that by 2050, expenses due to Alzheimer's (such as expenses for trained medical care and medication) will increase Medicare and Medicaid costs by over 330%.<sup>162</sup> This increase indicates that without a successful cure, the annual out-of-pocket payments for

Alzheimer's and dementia care in the US are projected to reach \$1.1 trillion in total by 2050.<sup>163</sup> These statistics point, not only to a rise in the disease itself, but also to a rise in the cost of care and the need for trained medical staff to meet the growing demand.

### ***Caregiver Burden***

Caregivers to those with Alzheimer's are also negatively affected when the disease progresses because they bear much of the economic and emotional burden of care. Economic burden relates to the financial costs and hardships of caring for an individual with ASD.<sup>164, 165</sup> In the United States, 83% of older adults (with and without AD) are looked after by a friend or family member rather than a paid caregiver.<sup>166</sup> In 2015, this percentage equated to approximately 15 million family members and other unpaid caregivers, who provided an estimated 18.1 billion hours of unpaid care to individuals with Alzheimer's and other

dementias.<sup>167</sup> These hours equate to an economic burden valued at over \$244 billion.<sup>168</sup> These hours may be spent providing direct and indirect care, such as cooking for the patient, administering medications, and assisting the patient around the house.<sup>169</sup> Surveyed family caregivers reported opting to provide care for various reasons, including keeping the loved one with dementia at home and close, particularly when the loved one was a spouse or partner.<sup>170</sup> Caregiving can be a rewarding experience, as it allows the caregiver to maintain close intimacy with a family member or friend near the end of the family member's life.<sup>171</sup> However, many caregivers describe their experience as distressful, particularly when the patient resists caregiver assistance. This resistiveness is a well-known behavioral and psychological symptom of Alzheimer's disease, in addition to agitation and aggression.<sup>172</sup> In 2015, nearly 60% of Alzheimer's and dementia caregivers rated the emotional stress of

caregiving as high or very high, 40% reported symptoms of depression, and 36% of caregivers die before the person they are caring for.<sup>173</sup> When compared to caregivers of patients with schizophrenia (20%), caregivers of patients with dementia report a higher prevalence of depression (30% to 40%).<sup>174</sup> Caregivers for individuals with dementia lose an estimated 2.4–3.5 hours of sleep each week due to prolonged stress and concern for their loved ones with dementia.<sup>175</sup> Due to the physical and emotional toll of caregiving, AD and dementia caregivers in the United States have approximately \$9.7 billion in additional health care costs, which include medication, exams, Medicare expenses, and others. Forty-one percent of caregivers have a household income of \$50,000 or less, meaning total annual costs of approximately \$29,000 makes up a significant amount of yearly expenses.<sup>176</sup>



## Practices

### *Wellness Programs*

Beyond just physical activity, wellness is defined by the Global Wellness Institute as “the active pursuit of activities, choices, and lifestyles that lead to a state of holistic health.” Overall wellness is crucially impacted by the cultural, physical, and social environments in which we live.<sup>177</sup> In the past few decades, researchers have examined the relationship between positive wellness choices and Alzheimer’s disease risk. For instance, a 2006 study enrolled participants in wellness activities to promote self-control, physical exercise, mental stimulation, and social interaction.<sup>178</sup> Compared to a control

group, participants involved in the art programs showed better health, higher morale, fewer depressive symptoms, and greater protection against dementia and cognitive decline.<sup>179</sup> Just a year into the study, groups involved showed stabilization and improvement in cognitive decline, indicating a decreased severity of AD symptoms and a potentially decreased risk of Alzheimer's disease.

In 2018, the Alzheimer's Association announced its latest clinical study, The US Study to Protect Brain Health Through Lifestyle Intervention to Reduce Risk (US POINTER). This 2-year nationwide clinical study examines exactly how lifestyle choices protect cognitive function and memory in older adults.<sup>180</sup> Modeled after a 2014 clinical trial in Finland, POINTER tests a combination of wellness practices, including a healthy diet, physical activity, cognitive exercises, and social activities, and their effect on AD risk.<sup>181</sup> The study, which

officially began in 2019, enrolled 2,000 older American adults (60–75 years old) who may be at risk for dementia.<sup>182</sup> Participants are provided resources and tools to complete the 2-year lifestyle program and are required to receive regular checkups and evaluations on their physical and mental health. These evaluations include measures for episodic memory, executive function, processing speed, the severity of dementia risk, functional ability, and other measures.<sup>183</sup> While this study will not be completed until 2024 due to the COVID-19 pandemic, US POINTER represents the first nationwide study on the relationship between Alzheimer's disease, brain health, and wellness choices.<sup>184</sup> <sup>185</sup> Given the study's emphasis on including sufficient numbers of Black, Hispanic, Asian, and Native Americans as study participants, these results will provide a unique perspective on the prevalence of AD in the US.<sup>186, 187</sup>

# Endnotes

1. "Alzheimer's Disease," Merriam-Webster, accessed February 1, 2021, <https://www.merriam-webster.com/dictionary/Alzheimer%27s%20disease>.
2. 2022 Alzheimer's Disease Facts and Figures (Chicago: Alzheimer's Association, 2022), <https://www.alz.org/media/Documents/alzheimers-facts-and-figures.pdf>.
3. Reisa Sperling et al., "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," *Alzheimers Dement* 7, no. 3 (May 2011): 280–92, <http://doi.org/10.1016/j.jalz.2011.03.003>.
4. "Apolipoprotein E4 - An Overview," ScienceDirect, accessed July 29, 2022, [https://www.sciencedirect.com/topics/neuroscience/apolipoprotein-e4#:~:text=4%20Apolipoprotein%20E,clearance%20\(Leoni%2C%202011\)](https://www.sciencedirect.com/topics/neuroscience/apolipoprotein-e4#:~:text=4%20Apolipoprotein%20E,clearance%20(Leoni%2C%202011)).
5. "Arteriosclerosis/Atherosclerosis," Mayo Clinic, Mayo Foundation for Medical Education and Research, April 24, 2018, <https://www.mayoclinic.org/diseases-conditions/arteriosclerosis-atherosclerosis/symptoms-cause/s/syc-20350569>.
6. David Goodsell, "Amyloid-beta Precursor Protein," PDB-101, July 2006, <https://pdb101.rcsb.org/motm/79>.
7. M. Paul Murphy and Harry LeVine III, "Alzheimer's Disease and the Amyloid-Beta Peptide," *Journal of Alzheimer's Disease* 19, no. 1 (January 2010): 311–323, <https://doi.org/10.3233/JAD-2010-1221>.
8. "Chronic Diseases and Conditions," Department of Health, New York State, November 2021, <https://www.health.ny.gov/diseases/chronic/>.
9. "Chronic Stress Puts Your Life at Risk," Mayo Clinic, July 8, 2021, <https://www.mayoclinic.org/healthy-lifestyle/stress-management/in-depth/stress/art-20046037>.
10. "Dementia Definition & Meaning," Merriam-Webster, accessed February 1, 2021, <https://www.merriam-webster.com/dictionary/dementia>.
11. "Disease Definition & Meaning," Merriam-Webster, accessed December 8, 2020, <https://www.merriam-webster.com/dictionary/disease>.
12. "Disorder Definition & Meaning," Merriam-Webster, accessed August 3, 2022, <https://www.merriam-webster.com/dictionary/disorder>.
13. Anthony A. Mercadante and Prasanna Tadi, "Neuroanatomy, Gray Matter," StatPearls, National Library of Medicine, accessed August 3, 2022, <https://www.ncbi.nlm.nih.gov/books/NBK553239/#:~:text=%5B1%5D%20Grey%20matter%20makes%20up,concentration%20of%20neuronal%20cell%20bodies>.
14. Olivia Guy-Evans, "Grey Matter in the Brain," Simple Psychology, October 11, 2021, <https://www.simplypsychology.org/what-is-grey-matter-in-the-brain.html>.
15. "Morbidity Definition & Meaning," Merriam-Webster, accessed August 3, 2022, <https://www.merriam-webster.com/dictionary/morbidity>.
16. "What Happens to the Brain in Alzheimer's Disease?" National Institute on Aging, accessed August 3, 2022, <https://www.nia.nih.gov/health/what-happens-brain-alzheimers-disease#:~:text=Neurofibrillary%20tangles%20are%20abnormal%20accumulations,to%20the%20axon%20and%20dendrites>.
17. Damon DiSabato, Ning Quan, and Jonathan P. Godbout, "Neuroinflammation: The Devil is in the Details," *Journal of Neurochemistry* 139, no. 2 (October 2016): 136–153, <https://doi.org/10.1111/jnc.13607https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5025335/#:~:text=Neuroinflammation%20is%20defined%20as%20an,oxygen%20species%2C%20and%20secondary%20messengers>.
18. Sarah Moore, "What Are the Types of Neuroinflammation?" News-Medical, February 6, 2020, <https://www.news-medical.net/life-sciences/What-are-the-Types-of-Neuroinflammation.aspx>.
19. "Pathology Definition & Meaning," Merriam-Webster, accessed December 8, 2020, <https://www.merriam-webster.com/dictionary/pathology>.

20. Pamela Spigelmyer et al., "Resistiveness to Care as Experienced by Family Caregivers Providing Care for Someone with Dementia," *Journal of Nursing Scholarship* 50, no. 1 (January 2018): 36–46, <http://dx.doi.org/10.1111/jnu.12345>.
21. "Risk Factor Definition & Meaning," Collins Online Dictionary, accessed August 3, 2022, <https://www.collinsdictionary.com/us/dictionary/english/risk-factor>.
22. "What Happens to the Brain in Alzheimer's Disease?" National Institute on Aging.
23. "Tau," Alzheimer's Association, <https://www.alz.org/media/Documents/alzheimers-dementia-tau-ts.pdf>.
24. "Areas of the Brain Affected by Alzheimer's and Other Dementias," MyHealth.Alberta, Government of Alberta, June 16, 2021, <https://myhealth.alberta.ca/Health/Pages/conditions.aspx?hwid=tp12408>.
25. "What Happens to the Brain in Alzheimer's Disease?" National Institute on Aging.
26. "Alzheimer's Disease - Symptoms and Causes," Mayo Clinic, accessed August 3, 2022, <https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/symptoms-causes/syc-20350447>.
27. "Complications of Alzheimer's Disease (AD)," Healthline, August 16, 2016, <https://www.healthline.com/health/alzheimers-disease-complications#outlook>.
28. Zeinab Breijyeh and Rafik Karaman, "Comprehensive Review on Alzheimer's Disease: Causes and Treatment," *Molecules* 25, no. 24 (December 2020): 5789, <http://doi.org/10.3390/molecules25245789>.
29. Ladislav Volicer, Elizabeth A. Bass, and Stephen L. Luther, "Agitation and Resistiveness to Care Are Two Separate Behavioral Syndromes of Dementia," *Journal of the American Medical Directors Association* 8, no. 8 (October 2007): 527–32, <http://doi.org/10.1016/j.jamda.2007.05.005>.
30. "What Is Mild Cognitive Impairment Due to Ad? Catch It Early," Biogen, accessed February 5, 2021, [https://www.catchitearly.com/?cid=PPC-GOOGLE-Condition\\_Education\\_Unbranded\\_Phrase~S~PH~UB~NER~HCP~CON-information%2Bon%2Balzheimer%27s%2Bdisease-NA-p57945664092&gclid=EAlaIqobChMIuHWkIT7gIVfAytBh0iGQy8EAAYAiAAEgJpB\\_D\\_BwE&gclid=aw.ds](https://www.catchitearly.com/?cid=PPC-GOOGLE-Condition_Education_Unbranded_Phrase~S~PH~UB~NER~HCP~CON-information%2Bon%2Balzheimer%27s%2Bdisease-NA-p57945664092&gclid=EAlaIqobChMIuHWkIT7gIVfAytBh0iGQy8EAAYAiAAEgJpB_D_BwE&gclid=aw.ds).
31. James M. Ellison, "The History of Alzheimer's Disease," BrightFocus Foundation, accessed December 11, 2020, <https://www.brightfocus.org/alzheimers/article/history-alzheimers-disease>.
32. Henry Querfurth and Frank LaFerla, "Alzheimer's Disease," *New England Journal of Medicine* 362, (January 2010): 329–344, <http://doi.org/10.1056/NEJMra0909142>.
33. James M. Ellison, "The History of Alzheimer's Disease."
34. "Alzheimer's & Brain Research Milestones," Alzheimer's Association, accessed August 4, 2022, [https://www.alz.org/alzheimers-dementia/research\\_progress/milestones](https://www.alz.org/alzheimers-dementia/research_progress/milestones).
35. "What Is Dementia?" Centers for Disease Control and Prevention, April 5, 2019, <https://www.cdc.gov/aging/dementia/index.html>.
36. "Dementia," Mayo Clinic, accessed May 26, 2022, <https://www.mayoclinic.org/diseases-conditions/dementia/symptoms-causes/syc-20352013>.
37. "Types of Dementia," Alzheimer's Association, accessed May 26, 2022, <https://www.alz.org/alzheimers-dementia/what-is-dementia/types-of-dementia>.
38. Farheen Ramzan et al., "A Deep Learning Approach for Automated Diagnosis and Multi-Class Classification of Alzheimer's Disease Stages Using Resting-State fMRI and Residual Neural Networks," *Journal of Medical Systems* 44, no. 2 (February 2020): 1–16, <http://doi.org/10.1007/s10916-019-1475-2>.
39. "Alzheimer's Stages: How the Disease Progresses," Mayo Clinic, Mayo Foundation for Medical Education and Research, April 19, 2019, <https://www.mayoclinic.org/diseases-conditions/alzheimers-disease/in-depth/alzheimers-stages/art-20048448#:~:text=Alzheimer's%20disease%20is%20often%20diagnosed,Memory%20loss%20of%20recent%20events>.
40. "What Is Dementia?" Alzheimer's Association, accessed March 23, 2021, <https://www.alz.org/alzheimers-dementia/what-is-dementia>.

41. Cox Media Group National Content Desk and Debbie Lord, "How Does Alzheimer's Disease Kill You?" Boston 25 News, August 9, 2017, <https://www.boston25news.com/news/how-does-alzheimers-disease-kill-you/372114840/#:~:text=Alzheimer's%20disease%20destroys%20nerve%20connections,is%20what%20leads%20to%20death.>
42. "2021 Alzheimer's Disease Facts and Figures," *Alzheimer's and Dementia* 17, no. 3 (March 2021): <https://doi.org/10.1002/alz.12328>.
43. "Causes and Risk Factors for Alzheimer's Disease," Alzheimer's Association, 2020, <https://www.alz.org/alzheimers-dementia/what-is-alzheimers/causes-and-risk-factors>.
44. Dara Dickstein et al., "Role of Vascular Risk Factors and Vascular Dysfunction in Alzheimer's Disease," *Mount Sinai Journal of Medicine* 77, no. 1 (January/February 2010): 82–102, <http://doi.org/10.1002/msj.20155>.
45. Lenore Launer et al., "Midlife Blood Pressure and Dementia: The Honolulu-Asia Aging Study," *Neurobiology of Aging* 21, no. 1 (January/February 2000): 49–55, [http://doi.org/10.1016/S0197-4580\(00\)00096-8](http://doi.org/10.1016/S0197-4580(00)00096-8).
46. Toshiharu Ninomiya et al., "Midlife and Late-Life Blood Pressure and Dementia in Japanese Elderly: The Hisayama Study," *Hypertension* 58, no. 1 (July 2011): 22–28, <http://doi.org/10.1161/hypertensionaha.110.163055>.
47. M. Panpalli Ates et al., "Analysis of Genetics and Risk Factors of Alzheimer's Disease," *Neuroscience* 325, no. 14 (June 2016): 124–131, <http://doi.org/10.1016/j.neuroscience.2016.03.051>.
48. J. A. Luchsinger et al., "Aggregation of Vascular Risk Factors and Risk of Incident Alzheimer Disease," *Neurology* 65, no. 4 (August 2005): 545–551, <http://doi.org/10.1212/01.WNL.0000172914.08967.dc>.
49. Elisabet Barbero-Camps et al., "Cholesterol Impairs Autophagy-Mediated Clearance of Amyloid  $\beta$  while Promoting its Secretion," *Autophagy* 14, no. 4 (June 2018): 1129–1154, <http://doi.org/10.1080/15548627.2018.1438807>.
50. Julie Zissimopoulos et al., "Sex and Race Differences in the Association Between Statin Use and the Incidence of Alzheimer Disease," *JAMA Neurology* 74, no. 2 (February 2017): 225–232, <http://doi.org/10.1001/jamaneurol.2016.3783>.
51. J. Janson et al., "Spontaneous Diabetes Mellitus in Transgenic Mice Expressing Human Islet Amyloid Polypeptide," *Proceedings of the National Academy of Sciences of the United States of America* 93, no. 14 (July 1996): 7283–7288, <http://doi.org/10.1073/pnas.93.14.7283>.
52. George A. Edwards III et al., "Modifiable Risk Factors for Alzheimer's Disease," *Frontiers in Aging Neuroscience* 11 (2019): 146, <http://doi.org/10.3389/fnagi.2019.00146>.
53. A. Ott et al., "Diabetes Mellitus and the Risk of Dementia: The Rotterdam Study," *Neurology* 53, no. 9 (December 1999): 1937–1942, <http://doi.org/10.1212/wnl.53.9.1937>.
54. M. Panpalli Ates et al., "Analysis of Genetics and Risk Factors of Alzheimer's Disease."
55. "High Blood Pressure (Hypertension)," Mayo Clinic, accessed May 26, 2022, <https://www.mayoclinic.org/diseases-conditions/high-blood-pressure/diagnosis-treatment/drc-20373417>.
56. "Alzheimer's Disease - Causes," NHS, accessed August 7, 2022, <https://www.nhs.uk/conditions/alzheimers-disease/causes/>.
57. "Why Does Alzheimer's Disease Affect More Women Than Men? New Alzheimer's Association Grant Will Help Researchers Explore That Question," Alzheimer's Association, accessed August 7, 2022, [https://www.alz.org/blog/alz/february\\_2016/why\\_does\\_alzheimer\\_s\\_disease\\_affect\\_more\\_women#:~:text=Women%20are%20disproportionately%20affected%20by,with%20Alzheimer's%20disease%20are%20women.](https://www.alz.org/blog/alz/february_2016/why_does_alzheimer_s_disease_affect_more_women#:~:text=Women%20are%20disproportionately%20affected%20by,with%20Alzheimer's%20disease%20are%20women.)
58. Jose Viña and Ana Lloret, "Why Women Have More Alzheimer's Disease than Men: Gender and Mitochondrial Toxicity of Amyloid-Beta Peptide," *Journal of Alzheimer's Disease* 20, Suppl 2, S527–S533, <https://doi.org/10.3233/JAD-2010-100501>.
59. Ana García, Nieves Ramón-Bou, and Miquel Porta, "Isolated and Joint Effects of Tobacco and Alcohol Consumption on Risk of Alzheimer's Disease," *Journal of Alzheimer's Disease* 20, no. 2 (2010): 577–586, <https://doi.org/10.3233/JAD-2010-1399>.
60. Fabricio de Oliveira, "Correlations among Cognitive and Behavioral Assessments in Patients with Dementia due to Alzheimer's Disease," *Clinical Neurology and Neurosurgery* 135 (2015): 27–33, <https://doi.org/10.1016/j.clineuro.2015.05.010>.
61. Barry J. Gurland et al., "Rates of Dementia in Three Ethnoracial Groups," *International Journal of Geriatric Psychiatry* 14, no. 6 (1999): 481–493, <https://pubmed.ncbi.nlm.nih.gov/10398359/>.



62. Peggy Dilworth-Anderson et al., "Diagnosis and Assessment of Alzheimer's Disease in Diverse Populations," *Alzheimers Dementia* 4, no. 4 (2008): 305–309, <https://doi.org/10.1016/j.jalz.2008.03.001>.
63. Kumar B. Rajan et al., "Racial Differences in the Association Between Apolipoprotein E Risk Alleles and Overall and Total Cardiovascular Mortality Over 18 Years," *Journal of the American Geriatrics Society* 65, no. 11 (2017): 2425–2430, <https://doi.org/10.1111/jgs.15059>.
64. A. M. Saunders et al., "Association of Apolipoprotein E Allele Epsilon 4 with Late-Onset Familial and Sporadic Alzheimer's Disease," *Neurology* 43, no. 8 (1993): 1467–1472, <https://doi.org/10.1212/WNL.43.8.1467>.
65. Jennifer J. Manly and Richard Mayeux, "Ethnic Differences in Dementia and Alzheimer's Disease," in *Critical Perspectives on Racial and Ethnic Differences in Health in Late Life*, ed. Anderson NB, Bulatao RA, and Cohen B (National Academies Press, 2004).
66. Alan Reo, HwaJung Choi, and Marcia Valenstein, "Social Relationships and Depression: Ten-Year Follow-Up from a Nationally Representative Study," *PLoS One* 8, no. 4 (2013): <https://doi.org/10.1371/journal.pone.0062396>.
67. Ziggi Santini et al., "The Association between Social Relationships and Depression: A Systematic Review," 175, (April 2015): 53–65, <http://doi.org/10.1016/j.jad.2014.12.049>.
68. Nicholas J. Justice, "The Relationship Between Stress and Alzheimer's Disease," *Neurobiology of Stress* 8 (2018): 127–133, <http://doi.org/10.1016/j.ynstr.2018.04.002>.
69. Betzaida Tejada-Vera, Mortality From Alzheimer's Disease in the United States: Data for 2000 and 2010 (Hyattsville, MD: National Center for Health Statistics, 2013), <https://www.cdc.gov/nchs/products/databriefs/db116.htm#ref2>.
70. 2022 Alzheimer's Disease Facts and Figures, Alzheimer's Association.
71. George A. Edwards III et al., "Modifiable Risk Factors for Alzheimer's Disease."
72. Alzheimer's Association, "2009 Alzheimer's Disease Facts and Figures," *Alzheimers Dementia* 5, no. 3 (May 2009): 234–270, <http://doi.org/10.1016/j.jalz.2009.03.001>.
73. "65 and Older Population Grows Rapidly as Baby Boomers Age," United States Census Bureau, June 25, 2020, <https://www.census.gov/newsroom/press-releases/2020/65-older-population-grows.html>.
74. "Primary Care Physicians on the Front Lines of Diagnosing and Providing Alzheimer's and Dementia Care," March 11, 2020, <https://www.alz.org/news/2020/primary-care-physicians-on-the-front-lines-of-diag>.
75. Liesi E. Hebert, "Alzheimer Disease in the United States (2010–2050) Estimated Using the 2010 Census," *Neurology* 80, no. 19 (May 2013): 1778–1783, <https://doi.org/10.1212/WNL.0b013e31828726f5>.
76. "The US Population Is Aging," Urban Institute, April 3, 2015, <https://www.urban.org/policy-centers/cross-center-initiatives/program-retirement-policy/projects/data-warehouse/what-future-holds/us-population-aging#:~:text=The%20number%20of%20Americans%20ages,The%20nation%20is%20aging>.
77. Wei Xu, Changshan Wu, and Jason Fletcher, "Assessment of Changes in Place of Death of Older Adults Who Died from Dementia in the United States, 2000–2014: A Time-Series Cross-Sectional Analysis," *BMC Public Health* 20, 765 (2020), <https://doi.org/10.1186/s12889-020-08894-0>.
78. Jennifer Weuve, "Deaths in the United States Among Persons with Alzheimer's Disease (2010–2050)," *Alzheimer's & Dementia: The Journal of the Alzheimer's Association* 10, no. 2 (March 2014): e40–e46, <http://doi.org/10.1016/j.jalz.2014.01.004>.
79. 2022 Alzheimer's Disease Facts and Figures, Alzheimer's Association, 29.
80. "Data & Statistics," CDC, accessed August 15, 2022, <https://www.cdc.gov/physicalactivity/data/index.html>.
81. 2022 Alzheimer's Disease Facts and Figures, Alzheimer's Association.
82. Christopher A. Taylor et al., "Deaths from Alzheimer's Disease - United States, 1999 –2014," *MMWR: Morbidity & Mortality Weekly Report* 66, no. 20 (May 26, 2017): 521–26, <http://doi.org/10.15585/mmwr.mm6620a1>.
83. Stephen J. Ralph and Anthony J. Espinet, "Increased All-Cause Mortality by Antipsychotic

- Drugs: Updated Review and Meta-Analysis in Dementia and General Mental Health Care,” *Journal of Alzheimer's Disease Reports* 2, no. 1 (2018): 1–26, <https://doi.org/10.3233/ADR-170042>.
84. Donovan Maust et al., “Antipsychotics, Other Psychotropics, and the Risk of Death in Patients with Dementia: Number Needed to Harm,” *JAMA Psychiatry* 72, no. 5 (May 2015): 438–445, <https://doi.org/10.1001/jamapsychiatry.2014.3018>.
  85. Ronnie Daniel, “Alzheimer’s Association,” Park City Television, September 5, 2018, YouTube video, 10:13, <https://www.youtube.com/watch?v=TIcvYBdyJMI>.
  86. 2019 Alzheimer's Disease Facts and Figures (Alzheimer's Association, 2019), 321–387, <https://www.alz.org/media/documents/alzheimers-facts-and-figures-2019-r.pdf>.
  87. Current Geriatrician Shortfall (New York, NY: The American Geriatrics Society, February 2017), [https://www.americangeriatrics.org/sites/default/files/inline-files/Current-Geriatrician-Shortfall\\_0.pdf](https://www.americangeriatrics.org/sites/default/files/inline-files/Current-Geriatrician-Shortfall_0.pdf).
  88. Kirsten Barnicot et al., “Older Adult Experience of Care and Staffing on Hospital and Community Wards: A Cross-Sectional Study,” *BMC Health Services Research* 20, no. 583 (2020), <https://doi.org/10.1186/s12913-020-05433-w>.
  89. The Importance of the Optimal Nurse-To-Patient Ratio,” Wolters Kluwer, November 11, 2016, <https://www.wolterskluwer.com/en/expert-insights/the-importance-of-the-optimal-nursetopatients-ratio>.
  90. Ursula E. Bauer, “Prevention of Chronic Disease in the 21st Century: Elimination of the Leading Preventable Causes of Premature Death and Disability in the USA,” *The Lancet* 384, no. 9937 (July 2014): 45–52, <https://www.sciencedirect.com/science/article/pii/S0140673614606486#bib4>.
  91. Adrian De la Rosa et al., “Physical Exercise in the Prevention and Treatment of Alzheimer’s Disease,” *Journal of Sport and Health Science* 9, no. 5 (2020): 394–404, <https://doi-org.erl.lib.byu.edu/10.1016/j.jshs.2020.01.004>.
  92. Kirk Erickson et al., “Physical Activity, Brain Plasticity, and Alzheimer’s Disease,” *Archives of Medical Research* 43, no. 8 (2012): 615–621, <https://doi.org/10.1016/j.arcmed.2012.09.008>.
  93. Physical Activity and Health: A Report of the Surgeon General (Centers for Disease Control and Prevention, accessed February 5, 2021), <https://www.cdc.gov/nccdphp/sgr/adults.htm>.
  94. Kirk Erickson et al., “Physical Activity, Brain Plasticity, and Alzheimer’s Disease.”
  95. *Ibid.*
  96. Stephan Müller et al., “Relationship Between Physical Activity, Cognition, and Alzheimer Pathology in Autosomal Dominant Alzheimer's Disease,” *Alzheimer's & Dementia* 14, no. 11 (2018): 1427–1437, <http://doi.org/10.1016/j.jalz.2018.06.3059>.
  97. Adrian De la Rosa et al., “Physical Exercise in the Prevention and Treatment of Alzheimer’s Disease.”
  98. Kirk I. Erickson et al., “Exercise Training Increases Size of Hippocampus and Improves Memory,” *PNAS* 108, no. 7 (January 2011): 3017–3022, <https://doi.org/10.1073/pnas.1015950108>.
  99. Adrian De la Rosa et al., “Physical Exercise in the Prevention and Treatment of Alzheimer’s Disease.”
  100. Ayeisha Armstrong et al., “Chronic Stress and Alzheimer’s Disease: The Interplay Between the Hypothalamic-Pituitary-Adrenal Axis, Genetics, and Microglia,” *Biological Reviews* 96, no. 5 (October 2021): 2209–2228, <https://doi.org/10.1111/brv.12750>.
  101. “Stress a Major Health Problem in The U.S., Warns APA,” American Psychological Association, October 24, 2007, <https://www.apa.org/news/press/releases/2007/10/stress#:~:text=Twenty%2Deight%20percent%20of%20Americans,stress%20in%20the%20last%20month>.
  102. Niraj Chokshi, “Americans Are Among the Most Stressed People in the World, Poll Finds,” *The New York Times*, April 25, 2019, <https://www.nytimes.com/2019/04/25/us/americans-stressful.html>.
  103. Nicholas J. Justice, “The Relationship Between Stress and Alzheimer's Disease.”
  104. P. Björntorp, “Stress and Cardiovascular Disease,” *Acta Physiologica Scandinavica Supplementum* 640, 144–148, <https://pubmed.ncbi.nlm.nih.gov/9401628/>.
  105. Carlos C. Crestani, “Adolescent Vulnerability to Cardiovascular Consequences of Chronic

- Emotional Stress: Review and Perspectives for Future Research," *Neuroscience and Biobehavioral Reviews* 74, Pt B (March 2017): 466–475, <https://doi.org/10.1016/j.neubiorev.2016.03.027>.
106. J.A. Prenderville et al., "Adding Fuel to the Fire: The Impact of Stress on the Ageing Brain," *Trends Neuroscience* 38 (2015): 13–25, <http://doi.org/10.1016/j.jynstr.2018.04.002>.
  107. Nicholas J. Justice, "The Relationship Between Stress and Alzheimer's Disease."
  108. Rachel Sayer et al., "The Effect of Stress on the Expression of the Amyloid Precursor Protein in Rat Brain," *Neuroscience Letters* 431, no. 3 (March 2008): 197–200, <http://doi.org/10.1016/j.neulet.2007.11.032>.
  109. Nicholas J. Justice, "The Relationship Between Stress and Alzheimer's Disease."
  110. D. Baglietto-Vargas et al., "Short-Term Modern Life-Like Stress Exacerbates Abeta-Pathology and Synapse Loss in 3xTg-AD Mice," *J. Neurochem.* 134, (2015): 915–926, <https://doi.org/10.1038/s41598-019-52324-0>.
  111. H. Dong et al., "Modulation of Hippocampal Cell Proliferation, Memory, and Amyloid Plaque Deposition in APPsw (Tg2576) Mutant Mice by Isolation Stress," *Neuroscience* 127, no. 3 (2004), 601–609, <https://doi.org/10.1016/j.neuroscience.2004.05.040>.
  112. John G. Csernansky, "Plasma Cortisol and Progression of Dementia in Subjects with Alzheimer-Type Dementia," *The American Journal of Psychiatry* 163, no. 12 (December 2006): 2164–2169, <https://doi.org/10.1176/ajp.2006.163.12.2164>.
  113. Ameer Baird and William Forde Thompson, "The Impact of Music on the Self in Dementia," *Journal of Alzheimer's Disease* 61, no. 3 (2018): 827–841, <https://doi.org/10.3233/JAD-170737>.
  114. Ayeisha Armstrong et al., "Chronic Stress and Alzheimer's Disease: The Interplay Between the Hypothalamic-Pituitary-Adrenal Axis, Genetics, and Microglia."
  115. Cheng Zhang and Robert A. Rissman, "Corticotropin-Releasing Factor Receptor-1 Modulates Biomarkers of DNA Oxidation in Alzheimer's Disease Mice," *PLoS One* 12, no. 7 (2017): e0181367, <https://doi.org/10.1371/journal.pone.0181367>.
  116. Ameer Baird and William Forde Thompson, "The Impact of Music on the Self in Dementia."
  117. Christina Bryant, Henry Jackson, and David Ames, "The Prevalence of Anxiety in Older Adults: Methodological Issues and a Review of the Literature," *Journal of Affective Disorders* 109, no. 3 (2008): 233–250, <https://doi.org/10.1016/j.jad.2007.11.008>.
  118. Jack Prenderville et al., "Adding Fuel to the Fire: The Impact of Stress on the Ageing Brain," *Trends in Neurosciences* 38, no. 1 (2014): 13–25, <http://dx.doi.org/10.1016/j.tins.2014.11.001>.
  119. T. E. Seeman and E. Crimmins, "Social Environment Effects on Health and Aging: Integrating Epidemiologic and Demographic Approaches and Perspectives," *Annals of the New York Academy of Sciences* 954, (December 2001) 88–117, <https://doi.org/10.1111/j.1749-6632.2001.tb02749.x>.
  120. Ariel Frank Green, George Rebok, and Constantine G. Lyketsos, "Influence of Social Network Characteristics on Cognition and Functional Status with Aging," *International Journal of Geriatric Psychiatry* 23, no. 9 (2008): 972–978, <https://doi.org/10.1002/gps.2023>.
  121. François Béland et al., "Trajectories of Cognitive Decline and Social Relations," *The Journals of Gerontology, Series B, Psychological Sciences and Social Sciences* 60, no. 6 (2005): P320–P330, <https://doi.org/10.1093/geronb/60.6.p320>.
  122. David A. Bennett et al., "The Effect of Social Networks on the Relation Between Alzheimer's Disease Pathology and Level of Cognitive Function in Old People: A Longitudinal Cohort Study," *The Lancet. Neurology* 5, no. 5 (2006), 406–412, [https://doi.org/10.1016/S1474-4422\(06\)70417-3](https://doi.org/10.1016/S1474-4422(06)70417-3).
  123. Ibid.
  124. Valerie C. Crooks et al., "Social Network, Cognitive Function, and Dementia Incidence among Elderly Women," *American Journal of Public Health* 98, no. 7 (2008): 1221–1227, <https://doi.org/10.2105/AJPH.2007.115923>.
  125. C. Helmer et al., "Marital Status and Risk of Alzheimer's Disease: A French Population-Based Cohort Study," *Neurology* 53, no. 9 (1999): 1953–1958, <https://doi.org/10.1212/wnl.53.9.1953>.
  126. Krister Håkansson et al., "Association Between Mid-Life Marital Status and Cognitive Function in Later Life: Population Based Cohort Study," *BMJ (Clinical Research Ed.)* 339, (2009): b2462,

- <https://doi.org/10.1136/bmj.b2462>.
127. Robert S. Wilson et al., "Loneliness and Risk of Alzheimer Disease," *Archives of General Psychiatry* 64, no. 2 (2007): 234–240, <https://doi.org/10.1001/archpsyc.64.2.234>.
  128. Reijo S. Tilvis et al., "Predictors of Cognitive Decline and Mortality of Aged People Over a 10-Year Period," *The Journals of Gerontology, Series A, Biological Sciences and Medical Sciences* 59, no. 3 (2004), 268–274, <https://doi.org/10.1093/gerona/59.3.m268>.
  129. Hélène Amieva et al., "What Aspects of Social Network are Protective for Dementia? Not the Quantity but the Quality of Social Interactions is Protective Up to 15 Years Later," *Psychosomatic Medicine* 72, no. 9 (2010): 905–911, <https://doi.org/10.1097/PSY.0b013e3181f5e121>.
  130. C. Helmer et al., "Marital Status and Risk of Alzheimer's Disease: A French Population-Based Cohort Study."
  131. Arezoo Campbell, "Inflammation, Neurodegenerative Diseases, and Environmental Exposures," *Annals of the New York Academy Sciences* 1035, no. 1 (December 2004): 117–132, <https://doi.org/10.1196/annals.1332.008>.
  132. Michelle L. Block and Lilian Calderón-Garcidueñas, "Air Pollution: Mechanisms of Neuroinflammation and CNS Disease," *Trends in Neurosciences* 32, no. 9 (2009): 506–516, <https://doi.org/10.1016/j.tins.2009.05.009>.
  133. Lilian Calderón-Garcidueñas et al., "Brain Inflammation and Alzheimer's-Like Pathology in Individuals Exposed to Severe Air Pollution," *Toxicologic Pathology* 32, no. 6 (2004): 650–658, <https://doi.org/10.1080/01926230490520232>.
  134. Michelle L. Block, Luigi Zecca, and Jau-Shyong Hong, "Microglia-Mediated Neurotoxicity: Uncovering the Molecular Mechanisms," *Nature Reviews Neuroscience* 8, no. 1 (2007): 57–69, <https://doi.org/10.1038/nrn2038>.
  135. Michelle L. Block and Lilian Calderón-Garcidueñas, "Air Pollution: Mechanisms of Neuroinflammation and CNS Disease."
  136. Elisabeth Currit, "Disproportionate Exposure to Air Pollution for Low-Income Communities in the United States," *Ballard Brief*, May 2022, <https://ballardbrief.byu.edu/issue-briefs/disproportionate-exposure-to-air-pollution-for-low-income-communities-in-the-united-states>.
  137. Michelle L. Block and Lilian Calderón-Garcidueñas, "Air Pollution: Mechanisms of Neuroinflammation and CNS Disease."
  138. Lorraine Craig et al., "Air Pollution and Public Health: A Guidance Document for Risk Managers, *Journal of Toxicology and Environmental Health, Part A* 71, no. 9–10 (2008): 588–698, <https://doi.org/10.1080/15287390801997732>.
  139. Errol M. Thomson, "Air Pollution Alters Brain and Pituitary Endothelin-1 and Inducible Nitric Oxide Synthase Gene Expression," *Environmental Research* 105, no. 2 (2007): 224–233, <https://doi.org/10.1016/j.envres.2007.06.005>.
  140. Lilian Calderón-Garcidueñas et al., "Air Pollution and Brain Damage," *Toxicologic Pathology* 30, no. 3 (2002): 373–389, <https://doi.org/10.1080/01926230252929954>.
  141. Lilian Calderón-Garcidueñas et al., "Brain Inflammation and Alzheimer's-Like Pathology in Individuals Exposed to Severe Air Pollution."
  142. "Populations at Risk," American Lung Association, accessed August 17, 2022, <https://www.lung.org/research/sota/key-findings/people-at-risk>.
  143. "Air Quality - National Summary," US EPA, accessed August 17, 2022, <https://www.epa.gov/air-trends/air-quality-national-summary>.
  144. Arezoo Campbell, "Inflammation, Neurodegenerative Diseases, and Environmental Exposures."
  145. Michelle L. Block and Lilian Calderón-Garcidueñas, "Air Pollution: Mechanisms of Neuroinflammation and CNS Disease."
  146. Lilian Calderón-Garcidueñas et al., "Brain Inflammation and Alzheimer's-Like Pathology in Individuals Exposed to Severe Air Pollution."
  147. Michelle L. Block, Luigi Zecca, and Jau-Shyong Hong, "Microglia-Mediated Neurotoxicity: Uncovering the Molecular Mechanisms."
  148. Michelle L. Block and Lilian Calderón-Garcidueñas, "Air Pollution: Mechanisms of Neuroinflammation and CNS Disease."
  149. Moses Wainaina, Zhichu Chen, and Chunjiu Zhong, "Environmental Factors in the Development and Progression of Late-Onset Alzheimer's Disease," *Neuroscience Bull* 30, no. 2 (2014): 253–270, <https://doi.org/10.1007/s12264-013-1425-9>.

150. Lilian Calderón-Garcidueñas et al., "Neuroinflammation, Hyperphosphorylated Tau, Diffuse Amyloid Plaques, and Down-Regulation of the Cellular Prion Protein in Air Pollution Exposed Children and Young Adults, *Journal of Alzheimer's Disease* 28, no. 1 (2012): 93–107, <https://doi.org/10.3233/JAD-2011-110722>.
151. Moses Wainaina, Zhichu Chen, and Chunjiu Zhong, "Environmental Factors in the Development and Progression of Late-Onset Alzheimer's Disease."
152. Lilian Calderón-Garcidueñas et al., "Neuroinflammation, Hyperphosphorylated Tau, Diffuse Amyloid Plaques, and Down-Regulation of the Cellular Prion Protein in Air Pollution Exposed Children and Young Adults."
153. Reisa Sperling et al., "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."
154. Richard A, Marasco, "Current and Evolving Treatment Strategies for the Alzheimer Disease Continuum," *Emerging Therapies* 26, no. 8 (August 2020), <https://www.ajmc.com/view/current-and-evolving-treatment-strategies-for-the-alzheimer-disease-continuum>.
155. "Stages of Alzheimer's Disease," Johns Hopkins Medicine, accessed August 17, 2022, <https://www.hopkinsmedicine.org/health/conditions-and-diseases/alzheimers-disease/stages-of-alzheimer-disease>.
156. Sarah E. Tom et al., "Characterization of Dementia and Alzheimer's Disease in an Older Population: Updated Incidence and Life Expectancy With and Without Dementia," *American Journal of Public Health* 105, no. 2 (2015): 408–413, <https://doi.org/10.2105/AJPH.2014.301935>.
157. A. Burns et al., "Cause of Death in Alzheimer's Disease," *Age and Ageing* 19, no. 5 (1990): 341–344, <https://doi.org/10.1093/ageing/19.5.341>.
158. About Underlying Cause of Death, 1999-2018 (Hyattsville, MD: National Center for Health Statistics, accessed February 14, 2020), <https://wonder.cdc.gov/ucd-icd10.html>.
159. "2018 Alzheimer's Disease Facts and Figures," *Alzheimers Dement* 14, no. 3 (2018): 367–429, <https://www.alz.org/media/homeoffice/facts%20and%20figures/facts-and-figures.pdf>.
160. F. Coste et al., "An Update on Healthcare Resource Use and Economic Burden In Alzheimer's Disease In The United States (Us) From A Medicare Sample Analysis," *Value in Health* 18, no. 7 (November 2015): A754, <https://doi.org/10.1016/j.jval.2015.09.2924>.
161. Ibid.
162. "Alzheimer's Disease: Get The Facts," *UsAgainstAlzheimer's*, accessed February 1, 2021, [https://www.usagainstalzheimer.org/alzheimers-disease-get-facts?gclid=Cj0KCQiA6t6ABhDMAkRIsAONIYyy1vzDUg6O\\_YKyCCXrfrvjP\\_wj3-jYqTs5aERUvWdAyyMAb8Gg00NgaAtmHEALw\\_wcB](https://www.usagainstalzheimer.org/alzheimers-disease-get-facts?gclid=Cj0KCQiA6t6ABhDMAkRIsAONIYyy1vzDUg6O_YKyCCXrfrvjP_wj3-jYqTs5aERUvWdAyyMAb8Gg00NgaAtmHEALw_wcB).
163. Ibid.
164. Ye-Rin Lee et al., "Measuring the Economic Burden of Disease and Injury in Korea, 2015," *Journal of Korean Medical Science* 34, Suppl 1 (2019): e80, <https://doi.org/10.3346/jkms.2019.34.e80>.
165. "Definition of Economic Burden," *NCI Dictionary of Cancer Terms*, National Cancer Institute, accessed August 31, 2022, <https://www.cancer.gov/publications/dictionaries/cancer-terms/def/economic-burden>.
166. Pamela C. Spigelmyer et al., "Resistiveness to Care as Experienced by Family Caregivers Providing Care for Someone with Dementia."
167. "2016 Alzheimer's Disease Facts and Figures," *Alzheimer's and Dementia* 12, no. 4 (2016): 459–509, <https://doi.org/10.1016/j.jalz.2016.03.001>.
168. Ronnie Daniel, Alzheimer's Association BYU Presentation, May 2019.
169. Jorge Moreira da Silva, "Why You Should Care about Unpaid Care Work," *Development Matters*, March 18, 2019, <https://oecd-development-matters.org/2019/03/18/why-you-should-care-about-unpaid-care-work/#~:text=Unpaid%20care%20and%20domestic%20work%20refers%20to%20all%20non%20market,cooking%2C%20cleaning%20or%20fetching%20water>.

170. "2016 Alzheimer's Disease Facts and Figures," *Alzheimer's and Dementia* 12, no. 4 (2016): 459–509, <https://doi-org.erl.lib.byu.edu/10.1016/j.jalz.2016.03.001>.
171. Pamela C. Spigelmyer et al., "Resistiveness to Care as Experienced by Family Caregivers Providing Care for Someone with Dementia."
172. Ibid.
173. Ronnie Daniel, Alzheimer's Association BYU Presentation, May 2019.
174. Adnaan Bin Sallim et al., "Prevalence of Mental Health Disorders Among Caregivers of Patients With Alzheimer Disease," *Journal of the American Medical Directors Association* 16, no. 12 (2015): 1034–1041, <https://doi.org/10.1016/j.jamda.2015.09.007>.
175. Chenlu Gao, Nikita Y. Chapagain, and Michael K. Scullin, "Sleep Duration and Sleep Quality in Caregivers of Patients with Dementia: A Systematic Review and Meta-Analysis," *JAMA Network Open* 2, no. 8 (2019): e199891, <http://doi.org/10.1001/jamanetworkopen.2019.9891>.
176. F. Coste et al., "An Update on Healthcare Resource Use and Economic Burden In Alzheimer's Disease In The United States (Us) From A Medicare Sample Analysis."
177. "What Is Wellness?" Global Wellness Institute, accessed May 14, 2019, <https://globalwellnessinstitute.org/what-is-wellness/>.
178. Gene D. Cohen, *The Creativity and Aging Study: The Impact of Professionally Conducted Cultural Programs on Older Adults* (The George Washington University, April 30, 2006), 1–8, [https://hsrc.himmelfarb.gwu.edu/cgi/viewcontent.cgi?article=1001&context=son\\_ncafacpubs](https://hsrc.himmelfarb.gwu.edu/cgi/viewcontent.cgi?article=1001&context=son_ncafacpubs).
179. Ibid.
180. "A Lifestyle Intervention Trial to Support Brain Health and Prevent Cognitive Decline," Alzheimer's Association, accessed August 29, 2022, <https://www.alz.org/us-pointer/overview.asp>.
181. "U.S. Study to Protect Brain Health Through Lifestyle Intervention to Reduce Risk," ClinicalTrials.gov, Wake Forest University Health Sciences, accessed August 29, 2022, <https://clinicaltrials.gov/ct2/show/record/NCT03688126>.
182. U.S. Pointer (Alzheimer's Association, accessed August 29, 2022), [https://uspointer.net/US\\_POINTER\\_Brochure\\_en.pdf](https://uspointer.net/US_POINTER_Brochure_en.pdf).
183. "U.S. Study to Protect Brain Health Through Lifestyle Intervention to Reduce Risk," Wake Forest University Health Sciences.
184. U.S. Pointer, Alzheimer's Association.
185. Laura D. Baker et al., "U.S. Pointer: Lessons Learned About Delivery of a Multi-Domain Lifestyle Intervention During the COVID-19 Pandemic," *Alzheimers & Dementia* 17, no. s10 (December 2021): e055289, <https://alz-journals.onlinelibrary.wiley.com/doi/abs/10.1002/alz.055289>.
186. "RI Government, Health, Community Leaders Rally to Spark Participation in Alzheimer's Prevention Research," Butler Hospital, accessed August 29, 2022, <https://www.butler.org/memoryandaging/ri-government-health-community-leaders-rally-spark-participation-alzheimers-prevention-research>.
187. AFRO Staff, "Alzheimer's Association Addresses the Need for African-American Participation in Clinical Trials," *AFRO American Newspapers*, August 12, 2021, <https://afro.com/alzheimers-association-addresses-the-need-for-african-american-participation-in-clinical-trials/>.
188. "Alzheimer's Disease: Get The Facts," *UsAgainstAlzheimer's*, accessed December 9, 2020.
189. Christopher A. Taylor et al., "Deaths from Alzheimer's Disease - United States, 1999–2014."
190. Stephen J. Ralph and Anthony J. Espinet, "Increased All-Cause Mortality by Antipsychotic Drugs: Updated Review and Meta-Analysis in Dementia and General Mental Health Care."
191. Adrian De la Rosa et al., "Physical Exercise in the Prevention and Treatment of Alzheimer's Disease," *Journal of Sport and Health Science* 9, no. 5 (2020): 394–404, <https://doi.org/10.1016/j.jshs.2020.01.004>.
192. Kirk I. Erickson, Andrea M. Weinstein, and Oscar L. Lopez, "Physical Activity, Brain Plasticity, and Alzheimer's Disease," *Archives of Medical Research* 43, no. 8 (2012): 615–621, <https://doi.org/10.1016/j.arcmed.2012.09.008>.

193. Wei Xu, Changshan Wu, and Jason Fletcher, "Assessment of Changes in Place of Death of Older Adults Who Died from Dementia in the United States, 2000–2014: A Time-Series Cross-Sectional Analysis."
194. Jennifer Weuve, et al., "Deaths in the United States among Persons with Alzheimer's Disease (2010–2050)," *Alzheimer's & Dementia* 10, no. 2 (2014): e40–e46, <http://doi.org/10.1016/j.jalz.2014.01.004>.
195. "Alzheimer's Disease: Get The Facts," *UsAgainstAlzheimer's*, accessed February 1, 2021.