4-2024

Dementia Implications for Audiology: Cognitive Screening in Clinical Practice

Kelsey May Harris

UNIVERSITY OF NORTHERN COLORADO

Greeley, Colorado

The Graduate School

DEMENTIA IMPLICATIONS FOR AUDIOLOGY: COGNITIVE SCREENING IN CLINICAL PRACTICE

A Scholarly Project Submitted in Partial Fulfillment
of the Requirements of the Degree of
Doctor of Audiology

Kelsey May Harris

College of Natural and Health Sciences
Department of Communication Sciences and Disorders
Audiology

May 2024

This Scholarly Project by: Kelsey May Harris

Entitled: *Dementia Implications for Audiology: Cognitive Screening in Clinical Practice*

has been approved as meeting the requirement of the Degree of Doctor of Audiology in College of Natural and Health Sciences in the Department of Communication Sciences and Disorders, Program of Audiology.

Accepted by the Doctoral Scholarly Project Research Committee

_____

Deanna K. Meinke, Ph.D., Research Advisor

_____

Diane Erdbruegger, Au.D., Committee Member

_____

Jennifer Weber, Au.D., Committee Member

ABSTRACT

Harris, Kelsey. *Dementia Implications for Audiology: Cognitive Screening in Clinical Practice.* Unpublished Doctor of Audiology Doctoral Scholarly Project, University of Northern Colorado, 2024.

Dementia is a progressive, debilitating cognitive disorder recognized as one of the greatest challenges to global health in the 21st century. In recent years, dementia has gained significant importance in the international health agenda due to the increasing aging population, longer life expectancies, and the substantial socioeconomic burden. Global concern has led to increased efforts to understand, manage, and alleviate its impact. Considering the lack of curative treatments, dementia research and awareness campaigns have intensified with a focus on early detection, prevention strategies, and collaborative healthcare. Prevention has focused on identifying risk factors and modifiable aspects of lifestyle closely linked to dementia, such as hearing loss. Given the well-established and growing evidence supporting that hearing loss increases the risk of dementia, audiologists stand to play a critical role in prevention and management strategies.

The purpose of this project is to identify dementia prevention and management strategies with practical implementation recommendations for audiologists working with this high-risk population. Early identification is critical to timely, effective dementia intervention. Audiologists are among some of the first healthcare professionals that engage with patients at risk for cognitive decline highlighting the importance of their role in contributing to early detection and management of dementia. Implementing cognitive screening tools within routine clinical protocol can be a significant step toward achieving this goal. It is paramount that audiologists are

well-equipped with the right tools, knowledge, and skills to contribute to early identification and collaborative care for individuals at risk for dementia.

ACKNOWLEDGEMENTS

The successful accomplishment of this doctoral scholarly project represents the culmination of a collective effort and the support of many. I would like to express my sincerest gratitude to my research committee members, Dr. Weber and Dr. Erdbruegger for their insightful feedback, encouragement, and mentorship throughout this journey. Your commitment to my academic and professional growth has greatly influenced my passion for audiology. A special mention to my research advisor, Dr. Meinke, for your unwavering support and exceptional guidance throughout this entire research process. Your expertise, dedication, and guidance have been instrumental in shaping this research and my academic development. I am truly thankful for your mentorship.

To my family, I am profoundly grateful for your unwavering support, understanding, and encouragement. My husband, John, your patience and belief in me have sustained me during the most challenging times. Your sacrifices have not gone unnoticed, and I am immensely grateful for your selflessness. I also want to remember my beloved late mother whose boundless love, encouragement, and belief in my abilities have been a driving force in my life. Though she is no longer with us, her spirit and support continue to inspire me, and I dedicate this achievement to her memory.

This academic journey has been both memorable and rewarding due to the camaraderie, collective determination, and shared experiences of my cohort. Your support has been a source of motivation and strength, and I feel privileged to have shared this path with each one of you. To all that have helped me complete this milestone, your contributions have been immeasurable, and I am honored to have you as a part of this accomplishment.

TABLE OF CONTENTS

LIST OF ABBREVIATIONS

AD	Alzheimer's Disease
ADL	Activities of Daily Living
ARHL	Age-Related Hearing Loss
ARIA	Amyloid-Related Imaging Abnormalities
ASHA	American Speech-Language Hearing Association
BMI	Body Mass Index
CAPR	Clifton Assessment Procedures for the Elderly
CDC	Center for Disease Control and Prevention
CDR	Clinical Dementia Rating
CHAP	Chicago Health and Aging Project
CIBIC-plus	Clinician's Interview-based Impression of Change with Caregiver Input
CR	Cognitive Rehabilitation
CSF	Cerebrospinal Fluid
CST	Cognitive Stimulation Therapy
DSM-V	Diagnostic and Statistical Manual of Mental Disorder – 5th Edition
FDA	Food and Drug Administration
MAP	Memory and Aging Project
MCI	Mild Cognitive Impairment
MMSE	Mini-Mental Status Examination
MRI	Magnetic Resonance Imaging
NMDA	N-Methyl-D-Aspartate
NPI	Neuropsychiatric Inventory

PAFs	Population Attributable Fractions
PET	Positron Emissions Tomography
REM	Rapid Eye Movement
ROS	Religious Orders Study
ROT	Reality Orientation Training
RT	Reminiscence Therapy
SIB	Severe Impairment Battery
TIB	Traumatic Brain Injury
U.S.	United States
VICCCS	Vascular Impairment of Cognition Classification Consensus Study
WHO	World Health Organization

CHAPTER I

REVIEW OF THE LITERATURE

Neurocognitive Decline in Adults

For decades, researchers have been investigating evidence of an association between hearing loss and cognitive decline (Uhlmann et al., 1989). Although the mechanism linking hearing loss and cognition has yet to be confirmed, the evidence that hearing impairment increases the risk of cognitive decline and dementia has been well established (Ford et al., 2018). This developing relationship gives audiologists a unique opportunity to impact early identification and apply a multifaceted approach to care for individuals with neurocognitive decline.

Terminology

While dementia is the customary term retained in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-V), current diagnostic terminology has changed from "Dementia, Delirium, Amnestic, and other Cognitive Disorders" to the broader term of "Neurocognitive Disorders" (DSM-V; American Psychiatric Association, 2013). The general term *cognitive* relates to higher level mental processes involved with knowledge and comprehension. As opposed to the term *neurocognitive* which indicates acquired cognitive symptoms closely associated with the function of specific neural pathways, brain regions, and cortical networks in the brain (Ganguli et al., 2011).

Neurocognitive decline occurs when there is decreased mental function as compared to a previously attained level (McDonald, 2017). *Neurocognitive disorder* is an all-encompassing term that comprises delirium, mild neurocognitive disorder, and major neurocognitive disorder, commonly referred to as dementia (Sachdev et al., 2014). *Mild neurocognitive disorder*, also

referred to as mild cognitive impairment (MCI), demonstrates a decline in cognition that may cause increased effort, compensatory strategies, or accommodations without interfering with the capacity for independence in daily activities. On the contrary, impairment of cognitive function in *major neurocognitive disorder* interferes with independence in everyday activities, such as paying bills or managing medication (Simpson, 2014). As previously stated, the term dementia is commonly used analogously with major neurocognitive disorder and for the purpose and duration of this manuscript the term dementia will be utilized.

Dementia

"*Dementia"* is a chronic or progressive syndrome subsequent to an array of primary or secondary diseases or injuries that impact the brain outside of normal biological aging (C. Wang et al., 2022). These diseases and injuries cause changes in the brain which generate different types of dementia. There are over one hundred different types of dementia including Alzheimer's Disease, vascular dementia, Lewy body dementia, and mixed dementia. However, each type of dementia depends on the pathophysiological changes that underlie alterations in the brain leading to abnormal cognitive decline and characteristic symptoms (Raz et al., 2015). A primary reason for this classification and clarification of terms, is that the term dementia is often incorrectly used synonymously with Alzheimer's disease (AD) (Sachdev et al., 2014). In any instance, neurocognitive decline can occur in cognitive areas comprising of complex attention, executive function, learning and memory, language, perceptual-motor, or social cognition (DSM-V; American Psychiatric Association, 2013).

Symptoms

Dementia or MCI diagnosis requires a decline in one or more of the six key cognitive domains that generate characteristic symptoms (Cummings, 2021). Beginning with complex attention, Jha and Mukhopadhaya (2020) examine each domain and identify commonly associated symptoms. Processing speed, selective attention, and sustained attention are processes that make up complex attention. These processes affect our ability to manipulate attentional focus and may manifest as becoming easily distracted, difficulty with multiple stimuli, and inability to perform mental calculations. Executive functioning includes working memory, planning, decision making, and mental flexibility. Symptoms commonly associated with executive functioning include socially inappropriate behavior, inability to do complex projects, and difficulty organizing. Symptoms of deficits in learning and memory domain may display as requiring frequent reminders, repeating self in conversations, forgetting appointments because immediate and recent memory, free recall, recognition memory and cued recall are affected. The language domain includes expressive and receptive language deficits that affect grammar, fluency, and syntax. Some common symptoms associated with a decline in these processes may generate difficulties with word comprehension, naming, and finding a relevant word. Difficulty using stairs, recognizing objects, or navigating familiar environments are commonly seen symptoms that are altered by impairments of visual perception, praxis, and construction within the perceptual-motor domain. Finally, social cognition includes theory of mind, behavior regulation, and recognition of emotions which may present as unacceptable social behavior, disregard for safety, or insensitivity to social standards (Jha & Mukhopadhaya, 2020). Many of these symptoms or decline in cognitive functioning are similar to normal aging but differ in severity often making early identification of dementia difficult (Murman, 2015). Thus, a

multidisciplinary approach is recommended for diagnosing and managing dementia (Grand et al., 2011). A multidisciplinary team may include geriatricians, neurologists, physical therapists, nutritionists, and audiologists.

Epidemiology

Global Incidence

According to Gauthier et al. (2021) a new case of dementia is diagnosed every 3 seconds somewhere in the world. Currently over 55 million people are estimated to be living with dementia worldwide (Gauthier et al., 2021). Due to universal demographic aging and increased life expectancy, it is expected that this number will nearly triple globally, reaching 139 million by the year 2050 (Cafferata et al., 2021). A meta-analysis by Cao et al. (2020) found that dementia prevalence rates were higher in Europe and North America compared to Africa and South America. Additionally, dementia was the largest contributor to disability in developing countries, largely due to the increased proportions of older people (Cao et al., 2020). Additionally, the World Health Organization (WHO), recognizes dementia as a global public health priority. The World Health Organization authorized "The Global Action plan" in May 2017 in response to the public health dementia crisis. The "Global Action Plan on the Public Health Response to Dementia 2017-2025" aims to combat the negative impacts of dementia by increasing research, awareness, and knowledge of dementia (WHO, 2021).

United States Incidence

The Centers for Disease Control and Prevention (CDC), estimate that dementia is ranked as the 6th highest cause of death in the United States (U.S.) attributing to 121,499 deaths per year (Harrison et al., 2019). According to the Population Reference Bureau (2022) more than 7

million people are estimated to be living with dementia in the U.S. and a projected 12 million Americans will be living with dementia by 2040 (Population Reference Bureau, 2022).

Age and Sex

The global prevalence of dementia increases with age and roughly doubles every 5 years beginning after the age of 65 (Cao et al., 2020). Rajan et al. (2018) found that the annual incidence of dementia was 4 cases per 1,000 in people aged 65 – 74, 32 cases per 1,000 in people aged 75 – 84, and 76 cases per 1,000 in people aged 85 and older. The average age at clinical diagnosis for dementia is 76.2 years (Rajan et al., 2018). While the majority of dementia cases occur in the population aged 65 years or older, the prevalence of young onset dementia increased from 1.1 per 100,000 for populations aged 30 – 34 years to 77.4 per 100,000 for populations aged 60 – 64 years (Hendriks et al., 2021). A study by Nichols et al. (2022) found that women have a 1.69 higher incidence rate of dementia compared to men (Nichols et al., 2022). The results from a study by Bacigalupo et al. (2018) agreed that the prevalence of dementia increases with age and is higher in women compared to men (Bacigalupo et al., 2018).

Race and Ethnicity

A multiethnic cohort study by Lim et al. (2021), analyzed the prevalence and trends of dementia in the United States regarding racial/ethnicity variations. The results found that African Americans were found to have the highest risk of developing dementia while Asian Americans have the lowest risk. (Lim et al., 2021). Consistent with previous studies, Freedman et al. (2018) found dementia prevalence was double among Hispanic and other racial/ethnic minorities at 17% compared to 8% for non-Hispanic white adults (Freedman et al., 2018).

Common Types of Dementia

Dementia represents a complex and heterogeneous spectrum of neurodegenerative disorders, each with distinct etiologies, clinical presentations, and prognoses (Erkkinen et al., 2017). While there are over one hundred different types of dementia, among the most common are Alzheimer's Disease, vascular dementia, Lewy body dementia, and mixed dementia (Raz et al., 2015). Understanding the unique underlying pathologies, clinical features, and diagnosis criteria can aid in effective differentiation. Distinguishing subtypes of dementia is essential for timely identification, tailored treatment approaches, and improved outcomes for individuals and their families (Carlos & Josephs, 2023).

Alzheimer's Disease

Alzheimer's disease is a progressive neurodegenerative disorder that primarily affects cognitive function, memory, and behavior (Zhang et al., 2021). Marked by the gradual accumulation of abnormal protein deposits, AD poses significant challenges for identification, diagnosis, and treatment (van Oostveen & de Lange, 2021).

Epidemiology

Alzheimer's Disease is the most common dementia accounting for 60-80% of dementia cases (Thapa et al., 2020). A study by Cao et al. (2020) found that prevalence rates were highest in Europe and North America at 1,123 cases per 10,000 compared to South America at 648 cases per 10,000, Asia at 523 cases per 10,000, and Africa at 275 cases per 10,000. In the United States, almost two-thirds of dementia cases are women representing 12% of women and 9% of men 65 and older (Rajan et al., 2021). Additionally, worldwide AD is 1.9 times higher in females than in males (Cao et al., 2020). Finally, data from the Chicago Health and Aging Project (CHAP) study found adults aged 65 and older with AD are comprised of 19% of Black, 14%

Hispanic and 10% white indicating disproportionally higher rates of AD in minorities compared to whites (Rajan et al., 2021).

Pathophysiology

While it is widely accepted that AD gradually develops due to beta-amyloid and tau proteins that accumulate intracellularly as neurofibrillary tangles, the specific cause of this accumulation is still being explored (Zhang et al., 2021). A study by Hardy and Selkoe (2002) found that AD progresses several years before the development of dementia symptoms from the buildup of beta-amyloid and tau proteins. In turn, this build up, or neurofibrillary tangles, are detrimental to dendrites, neuronal cell bodies, and axonal processes which lead to a variety of cognitive changes in the brain. (Hardy & Selkoe, 2002).

Further, a study by Dean et al. (2016) aimed to examine the effect of beta-amyloid and tau protein accumulation on changes in white matter myelination in the brain. The authors analyzed lumbar punctures and magnetic resonance imaging (MRI) in 71 cognitively asymptomatic adults with genetic risk factors for AD. The lumbar puncture tests were used to differentiate participants with AD from healthy, age-matched controls and to assess AD cerebrospinal fluid (CSF) biomarker levels including beta-amyloid and tau proteins. The MRI testing for each of the participants analyzed whole-brain longitudinal and transverse relaxation rates and measured myelin water. The results found widespread age-related myelin changes especially in frontal white matter and the genu of the corpus callosum. Additionally, when comparing CSF biomarkers and MRI results, there were significant age-by-biomarker interactions suggesting beta-amyloid and tau protein levels modulate age-related changes in myelin water fraction. The results of this study further support that AD amyloid pathologies have

a significant negative influence on white matter microstructure, myelin content, and myelin water fraction (Dean et al., 2016).

Symptoms

Alzheimer's Disease is a progressive type of dementia that is manifested with memory issues, decline in abstract thinking and judgement, changes in mood, emotions, and behavior, and a variety of physical problems affecting vision and mobility (Gauthier et al., 2021). Decline in the ability to remember new information, make decisions, organize or plan are often the initial hallmark cognitive symptoms of AD (Silverberg et al., 2011). However, as the disease progresses 80% of individuals develop neuropsychiatric, behavioral, and psychological symptoms including depression, apathy, agitation, irritability, combativeness, and psychotic features (Zhao et al., 2016). Finally, seizures, dysphagia, gait disturbances, and incontinence are often seen in the latest stages of AD (Deardorff & Grossberg, 2019).

Diagnosis

Although AD can be accurately diagnosed using CSF biomarkers and amyloid positron emission tomography (PET) scans, these tests are not currently indicated for most symptomatic individuals (Johnson et al., 2013). Cerebrospinal fluid and PET objective testing is reserved for atypical, rapidly progressive, or early-onset syndromes to identify or exclude AD (Simonsen et al., 2016). Therefore, AD remains a clinical diagnosis that can be identified with high confidence through an individualized approach to history, examination, cognitive and neurological tests, and select laboratory tests (Atri, 2019). The Diagnostic and Statistical Manual - Fifth Edition of the American Psychiatric Association states that if a causative genetic mutation is not identified by genetic testing or family history, key clinical presentations of a neurocognitive disorder are required to diagnose AD. Diagnostic criteria include clear evidence of steadily progressive,

gradual decline in memory, learning, and one other cognitive domain. (DSM-V; American Psychiatric Association, 2013).

Vascular Dementia

Vascular dementia is characterized by cognitive decline resulting from impaired blood flow to the brain (Bir et al., 2021). The clinical presentation can vary widely depending on the location and severity of the vascular damage but can typically be diagnosed with accuracy (Iadecola et al., 2019). The high prevalence of cerebrovascular disease assists in making vascular dementia one of the most common types of dementia (Song et al., 2014).

Epidemiology

The second most common type of dementia is vascular dementia accounting for 15% to 20% of cases in North America and Europe with slightly higher estimates of 30% in Asia and developing countries (Wolters & Ikram, 2019). The estimated annual incidence of vascular dementia is between 2.5 – 3.8 cases per one thousand (Bir et al., 2021). The incidence of vascular dementia increases with the severity of stroke ranging from 5% at 1 year after transient ischemic attack to 34% at 1 year after severe stroke (Pendlebury & Rothwell, 2019). Additionally, the prevalence of vascular dementia was 1.8 times greater in males than in females which coincides with the tendency that males have an overall higher risk for stroke (Cao et al., 2020).

Pathophysiology

Vascular dementia is caused by blocked or reduced blood flow to the brain which deprives neurons of essential nutrients (Vijayan & Reddy, 2016). Affecting the blood vessels of the brain, cerebrovascular disease is commonly produced by, but not limited to, chronic cerebral ischemia, large or repeated strokes, diabetes, hyperlipidemia, hypertension, or transient ischemic

attacks (Song et al., 2014). Vascular dementia can further be categorized into subtypes depending on the anatomical location of tissue changes, level of involvement of extra and intracranial vessels, timeline of the initial vascular event, and the magnitude and nature of vascular pathologies (Kalaria, 2018).

Symptoms

The onset of cognitive decline from vascular dementia may range from acute to gradual, as well as present in a stepwise, fluctuating, gradually decreasing, or other complex progressions (DSM-V; American Psychiatric Association, 2013). A scientific expert panel from the Journal of the American College of Cardiology by Iadecola et al. in 2019, found less occurrence of memory decline compared to other dementias while identifying more characteristic symptoms of vascular dementia. These distinctive symptoms cause a severe disruption of instrumental activities of daily living due to impairments of executive function and processing speed. The most common vascular dementia symptoms affect mood and behavior which may present as deterioration of cognitive flexibility, judgement, decision making, hypothesis generation, initiation, and planning. Additionally, it is common in patients with vascular dementia to show delays in recall of word lists and visual content (Iadecola et al., 2019).

Diagnosis

Vascular dementia encompasses a variety of vascular contributions to cognitive impairment that can cause variability in clinical manifestations. The Vascular Impairment of Cognition Classification Consensus Study (VICCCS) in 2016 aimed to develop new guidelines for clinical diagnosis and research. This online Delphi consensus study identified four major vascular dementia subtypes including post-stroke dementia, subcortical ischemic vascular dementia, multi-infarct (cortical) dementia, and mixed dementia (Skrobot et al., 2016).

Diagnostic criteria for vascular dementia can be further outlined in the DSM-V when a mild or major neurocognitive disorder is present. Diagnostic criteria include verification of cerebrovascular disease evident from case history, physical examination, and/or neuroimaging. While the presence of memory impairment is no longer required as a diagnostic clinical feature, cognitive deficits must include decreased complex attention and frontal executive function or be followed by one or more cerebrovascular events (DSM-V; American Psychiatric Association, 2013). Further, MRI results are considered the gold standard neuroimaging technique to diagnose and differentiate vascular subtypes (Skrobot et al., 2016).

Lewy Body Dementia

Lewy body dementia is a complex neurodegenerative disorder characterized by the accumulation of abnormal protein deposits in the brain leading to cognitive decline (Milán-Tomás et al., 2021). Despite its prevalence, Lewy body dementia remains poorly understood and presents unique challenges for identification, diagnosis, and management (Chin et al., 2019).

Epidemiology

Dementia with Lewy bodies and Parkinson's disease dementia are categorized as Lewy body dementia. While likely underdiagnosed, Lewy body dementia is among the most common types of dementia, accounting for 5% of dementias with an incidence of 3.5 cases per 100,00 cases (Salinas, 2022). Further, up to 80% of individuals with Parkinson's disease develop dementia (Taylor et al., 2020). Additionally, the prevalence of Lewy body dementia is higher in males compared to females (Kane et al., 2018).

Pathophysiology

Although dementia with Lewy bodies and Parkinson's disease dementia may initially present with different symptoms, the underlying biological changes in the brain are the same (Milán-Tomás et al., 2021). Although the precise cause of Lewy body dementia is unknown, the pathological hallmark is abnormal clusters of alpha-synuclein proteins, or Lewy bodies, in the brain. These abnormal clusters develop in the neocortex disrupting neuronal activity through synaptic degeneration, neuronal death, and neurotransmitter failure (Hirczy & Salinas, 2022). The overall cognitive deficits result from a combination of alpha-synuclein, beta-amyloid, and tau protein accumulation. The alpha-synuclein proteins of Lewy body dementia ultimately effects several cerebral regions due to widespread neuronal atrophy (Jellinger, 2017).

Symptoms

Lewy body dementia presents a wide variety of cognitive, neuropsychiatric, motor, sleep, and autonomic symptoms. The variety of presentations not only differ between patients, but also can be expressed variably within a patient over time (Taylor et al., 2020). Cognitive impairments of Lewy body dementia affect attention, executive function, and visuoperceptual functions more commonly than naming and memory abilities. Additionally, an inherent characteristic of the disease is natural fluctuations of symptoms that often worsen with treatment of another symptom (Killen et al., 2015).

Diagnosis

The pathological changes in the brain due to Lewy body dementia cause characteristic symptoms that help differentiate from other dementias. The revised 2017 criteria for the clinical diagnosis of Lewy body dementia identify core clinical features that develop sporadically with no known family history in the majority of cases (Yamada et al., 2020). These fluctuating

features include cognitive symptoms affecting attention and alertness, typically well-formed and detailed visual hallucinations, rapid eye movement (REM) sleep behavior disorder, and one or more cardinal features of parkinsonism (McKeith et al., 2017).

Mixed Dementia

Mixed dementia is characterized by the coexistence of multiple underlying neurodegenerative pathologies contributing to cognitive decline (Forrest & Kovacs, 2022). Consisting of two or more types of dementia, mixed dementia is becoming increasingly recognized (Agrawal & Schneider, 2022). However, due to the convergence of diverse pathological processes, mixed dementia presents with complicated clinical presentation and diagnosis (Paraskevaidi et al., 2018).

Epidemiology

Mixed dementia refers to dementia symptoms caused by two or more coexisting pathologies (Jellinger & Attems, 2007). While previously underrecognized, emerging research supports that the percentage of mixed dementia cases in community-based studies range from more than 50% to 78% (Agrawal & Schneider, 2022). Additionally, the presence of mixed dementia increases with age and is highest in people 85 and older (Dumurgier & Tzourio, 2020).

Pathophysiology

Although AD is frequently considered the most common dementia, the underlying etiology in the vast majority of older people with dementia is an accumulation of multiple pathologies including cerebrovascular, Lewy bodies, neurofibrillary tangles, and other neurodegenerative dementia pathologies (Schneider, 2022). A review of literature by Kapasi et al. in 2017 aimed to analyze the clinical-pathological role of various brain pathologies in dementia, as well as the available clinical and biomarker data of common mixed pathologies.

Evidence from ten longitudinal, post-mortem studies supports the idea that the pathophysiological process of the majority of AD is a combined result of vascular, Lewy body, and other neurodegenerative pathologies. Included in this review is the updated data from the original 1993 Religious orders study (ROS) and the 1997 Memory and aging project (MAP) (Bennett et al., 2018). The data included 1078 consecutive deceased and autopsied clinically diagnosed subjects with cognitive decline since 1993. The results found that nearly 75% of participants diagnosed with probable AD dementia had mixed dementia with at least one additional vascular pathology. Additionally, over 60% of participants diagnosed with Lewy body dementia also had a pathologic diagnosis of AD. Finally, after including additional neurodegenerative and vascular dementia pathologies, 95% of subjects diagnosed with probable AD had mixed pathologies (Kapasi et al., 2017).

Symptoms

Despite the ambiguous clinical presentation of mixed dementia, research aims at identifying characteristic comorbid symptoms (Paraskevaidi et al., 2018). A study by Brenowitz et al. (2017) intended to evaluate whether decline in specific cognitive domains is modified by comorbid neurocognitive pathologies. Using a multivariable linear mixed-effects model, 1,603 autopsied individuals from U.S. AD Centers were evaluated in memory, attention, language, and executive function. This data obtained from the National Alzheimer's Coordinating Center compared the progression of impairment of the four cognitive domains in subjects with AD with co-occurring Lewy body disease or vascular brain injury to subjects with single occurring dementia pathologies. Results found that subjects with AD and Lewy body disease had the greatest impairment among all cognitive domains, especially executive function and attention compared to other pathology groups. Additionally, subjects with AD and vascular brain injury

were found to have a slower rate of decline in memory, attention, and language compared to AD with Lewy body disease and AD only (Brenowitz et al., 2017).

Diagnosis

While emerging research continues to support the evidence that the majority of dementia cases are due to coexisting pathologies, clear diagnostic criteria for mixed dementia are not well defined (Custodio et al., 2017). Analogous cognitive symptoms and clinical presentations combined with lack of clinically available biomarker testing create a challenge for diagnosing mixed dementia (Kapasi et al., 2017). Although there are characteristic presentations and diagnostic criteria for each of the common dementia diagnoses, clinical symptoms often overlap (Karantzoulis & Galvin, 2011). Unfortunately, there is no international consensus for mixed dementia diagnostic criteria and multiple global organizations have proposed different diagnostic standards (Custodio et al., 2017). However, the DSM-V suggests referring to the diagnostic criteria of other dementia etiologies but requires evidence from history, physical examination, or laboratory findings for a mixed dementia diagnosis (DSM-V; American Psychiatric Association, 2013).

Though AD is widely considered the most common cause of dementia, emerging research may continue to support evidence that the majority of dementias are due to multiple pathologies (Schneider, 2022). Accurate diagnosis may be difficult, however once a dementia diagnosis is reached the patient and healthcare provider can begin to discuss potential treatment and management options (C. Wang et al., 2022).

Treatment

Once probable dementia is suspected and diagnosed, the next step is to consider available treatments. The Lancet Commission 2020 report on dementia prevention, intervention and care recommends focusing treatment on physical and mental health, social care, and support (Orgeta et al., 2018). Additionally, the suggested treatment for neuropsychiatric symptom management is a combination of multicomponent interventions (Livingston et al., 2020). Unfortunately, there is currently no cure for any neurocognitive dementia (Ebrahimi et al., 2020). However, there are treatment and management options including U.S. Food and Drug Administration (FDA) approved drugs, alternative therapies, and medications to manage symptoms.

FDA Approved Drugs

The FDA has currently approved seven medications for the treatment of the most common form of dementia, AD (Chimakurthy et al., 2023). These medications are found in the drug classes of cholinesterase inhibitors, N-methyl-D-aspartate (NMDA) receptor antagonists (e.g., memantine hydrochloride), and monoclonal antibodies. While these medications have been developed to treat AD, current indications and off label applications are used to treat patients with other forms of dementia such as vascular dementia and Lewy body dementia (Cleveland Clinic, 2022).

Cholinesterase Inhibitors

Belonging to the class of Anti-Alzheimer agents, cholinesterase inhibitors play a crucial role in dementia management (Haake et al., 2020). Despite their widespread use, debates persist regarding the efficacy of these medications (Lista et al., 2023). It is essential to understand their indications, mechanisms of action, and efficacy for informed decision-making and effective treatment (Wong, 2016).

Indications and Usage. There are three prescription acetylcholinesterase inhibitor drugs, donepezil, rivastigmine, and galantamine, that are FDA approved to treat the symptoms of AD and other dementias (Haake et al., 2020). Pardo-Moreno et al. (2022) outline the drug summaries for each of these medications. Donepezil hydrochloride, commonly branded as Aricept or Adlarity is approved for the treatment of mild, moderate, and severe AD, vascular dementia, and dementia with Lewy bodies. Rivastigmine tartrate, commonly known as Exelon, is approved for the treatment of mild, moderate, and severe AD, vascular dementia, dementia with Lewy bodies, and mild to moderate Parkinson's disease dementia. Finally, galantamine hydrobromide, commonly known as Razadyne or Reminyl, is approved for treatment of mild to moderate AD, vascular dementia, and dementia with Lewy bodies. Each of these medications can be administered orally while donepezil and rivastigmine also have a topical administration option. Although there has been a wide variety of reported side effects, the most common side effects for all prescription cholinesterase inhibitor medications are nausea, vomiting, and diarrhea (Pardo-Moreno et al., 2022).

Mechanism of Action. Cholinesterase inhibitors are a class of drug known as anti-Alzheimer Agents that aim to improve symptoms by increasing neurotransmitters in the brain (Alzheimer's Association, 2023). In a review by Kamlesh Sharma (2019), one of the suggested causes of cognitive decline in patients with AD stems from the cholinergic hypothesis. The cholinergic nervous system is a mostly parasympathetic division of the autonomic nervous system that produces an important neurotransmitter, acetylcholine. Acetylcholine synthesis is essential to the function of memory, learning, digestion, blood pressure, movement, and many other functions. Conversely, acetylcholinesterase is an enzyme that inhibits the dispersal of acetylcholine by terminating neuronal transmission and activation of nearby receptors (Singh &

Jha, 2021). The cholinergic hypothesis suggests that the loss of neurotransmitters in AD and other dementias is caused by the deterioration of cholinergic neurons in the brain, or the reduction of acetylcholine synthesis. Thus, cholinesterase inhibitors intend to impede the biological activity of acetylcholinesterase consequently increasing the cholinergic levels in the brain, the function of neuronal cells, and the concentration of acetylcholine (Sharma, 2019).

Efficacy. Although cholinesterase inhibitors are approved for dementia treatment, their effectiveness has been debated (Lista et al., 2023). In fact, in 2018 France removed cholinesterase inhibitors from the list of reimbursable drugs due to lack of evidence supporting significant benefit and the perceived tendency to shift healthcare providers' focus from other interventions (Herr et al., 2021). However, a clinical review article by Glynn-Servedio and Ranola (2017) found evidence supporting the efficacy of cholinesterase inhibitors for patients with mild to moderate AD but limited data on patients with advanced stages of the disease. Compared to placebo-treated patients, patients with mild to moderate AD prescribed cholinesterase inhibitors slightly improved on a number of cognitive and functional outcome measures. The improvements were seen on measures including the Severe Impairment Battery (SIB), Mini-Mental Status Examination (MMSE), and Clinician's Interview-based Impression of Change with caregiver input (CIBIC-plus), but not others including Activities of Daily Living (ADL) scales, Neuropsychiatric Inventory (NPI), caregiver burden scales, and resource utilization scales. Though small improvements, outcome measures for cholinesterase inhibitor treatment are seen in some patients with early AD, but not all patients will benefit from this treatment. Finally, the long-term effects of this treatment are unknown since the vast majority of studies had a duration of less than one year. (Glynn-Servedio & Ranola, 2017). Due to limited

effectiveness and mixed results of cholinesterase inhibitor studies, medical treatment and research continue to look for alternative options for dementia management.

Memantine Hydrochloride

Another oral medication aimed to mitigate symptoms associated with certain types of dementia is memantine hydrochloride (Pardo-Moreno et al., 2022). This drug extends beyond standalone use and can be used in combination to alleviate cognitive deficits and enhance activities of daily living apoptosis (Zambrano et al., 2018). However, despite its distinctive mechanism of action, memantine hydrochloride faces similar efficacy challenges as cholinesterase inhibitors (Ovejero-Benito et al., 2022). Indications and Usage. Memantine hydrochloride, commonly known as Namenda, is another FDA approved oral prescription drug approved to treat symptoms of moderate to severe AD. Additionally, memantine hydrochloride is approved to be combined with donepezil, commonly known as Namzaric, to treat symptoms of moderate to severe AD and may provide a small cognitive benefit to patients with mixed vascular dementia (Pardo-Moreno et al., 2022).

Mechanism of Action. Similar to cholinesterase inhibitors, memantine hydrochloride does not affect the underlying brain changes that cause dementia. Rather, memantine hydrochloride is an NMDA antagonist that aims to improve cognition and activities of daily living by targeting glutamate, a neurotransmitter required for memory and learning (Alzheimer's Association, 2023). As described by Zambrano et al. (2018), although the physiological actions of glutamate are essential for synaptic plasticity and neuronal survival, excessive glutamate accumulation is one of the widely accepted theories for the multifactorial etiology of AD. Glutamate is the most excitatory neurotransmitter in the brain which triggers NMDA receptors. Over-activation of NMDA receptors lead to an influx of cytoplasmic calcium concentration and

reactive oxygen species which cause cell death and neurodegeneration. Accordingly, memantine hydrochloride protects against this hyperactivity by preferentially blocking NMDA receptors to modulate intracellular calcium accumulation and ultimately cellular apoptosis (Zambrano et al., 2018).

Efficacy. Although memantine hydrochloride has a different mechanism of action compared to cholinesterase inhibitors, clinical benefits are similar. Unfortunately, this medication has also shown low effectiveness in patients with AD (Ovejero-Benito et al., 2022). McShane et al. (2019) analyzed 44 trials yielding almost 10,000 participants to examine the efficacy and safety of memantine for people with dementia. The benefits of memantine were found to positively affect thinking, the ability to carry on normal ADL, and the severity of behavior and mood problems. These double-blind, parallel group, placebo-controlled, randomized trials concluded that a small clinical benefit was found in patients with moderate to severe AD, but no benefit was found in patients with mild forms of the disease. Although some clinical benefit has been observed, long-duration trials are needed to assess whether the benefits persist beyond six months and to establish whether starting memantine hydrochloride earlier would be beneficial over long-term use (McShane et al., 2019).

Monoclonal Antibodies

Monoclonal antibodies represent a groundbreaking approach in the medical management of Alzheimer's Disease related dementia (Vogt et al., 2023). These medications are aimed at altering the underlying biology of the condition rather than merely addressing its symptoms (McDade et al., 2022). Drugs in this class stand out as a significant milestone, however, efficacy of these novel therapies remain under scrutiny (Decourt et al., 2022).

Indications and Usage. Unlike the previously discussed medications, monoclonal antibodies are intended to change the underlying biology of AD and related dementias. The only two FDA approved prescription drugs currently on the market for the treatment of early AD are Aduhelm, generically known as aducanumab and Leqembi, generically known as lecanemab, (Vogt et al., 2023). Authorized under accelerated approval in 2021, Aduhelm is an injection to be administered as an intravenous infusion every four weeks (Plascencia-Villa & Perry, 2023). Similarly, Leqembi is an injection that was approved under the accelerated approval pathway in 2023 to be administered as an intravenous infusion every two weeks (Cummings et al., 2023). It is important to note that the accelerated approval pathway allows for patients with serious disease earlier access to the drug when there is an uncertain, but expected, clinical benefit (Huang et al., 2023).

Mechanism of Action. Aduhelm and Leqembi are categorized in the class of monoclonal antibody drugs and share the same mechanism of action (Cummings, 2023). Since the accumulation of amyloid beta plaques in the brain is the defining pathophysiological feature of AD, these monoclonal antibody drugs aim to modify the underlying disease (Plascencia-Villa & Perry, 2023). Both Aduhelm and Leqembi are humanized immunoglobulin gamma 1 monoclonal antibodies targeted against aggregated soluble and insoluble forms of amyloid beta plaques (McDade et al., 2022). Although not a cure for the disease, these infusion therapies reduce beta-amyloid build-up in the brain to slow cognitive and functional decline (Cai et al., 2023). While each of these disease-modifying immunotherapies aim to reduce the number of plaques in the brain, the key difference between Aduhelm and Leqembi is that they target beta-amyloids at different stages of plaque formation (Alzheimer's Association, 2023).

Efficacy. As mentioned previously regarding accelerated approval, continued approval for Aduhelm and Leqembi depends on verification of clinical benefit in Phase 3 therapeutic confirmatory trials (Huang et al., 2023). As described by Haddad et al. (2022), while Aduhelm previously completed two double blind, randomized placebo controlled 18-month trials, they were halted about half-way through in March 2019. The trials, entitled "Emerge" and "Engage," were stopped because no significant improvement of symptoms was observed. However, post hoc analysis indicated that high dose Aduhelm reduced the total size and number of beta-amyloid plaques compared to the placebo groups in both trials. However, the "Engage" study participants did not show statistically significant improvement of symptoms based on cognitive assessment rating scales. Additionally, the "Emerge" study only showed statistical significance of improved rating scores for patients with mild AD but did not meet the criteria for clinically significant improvement for patients with increased severities of the disease. Additional research is needed to confirm the clinical significance of Aduhelm but results showing reduction in beta-amyloids in early Alzheimer's provides hope for future treatment (Haddad et al., 2022).

Similar to Aduhelm, confirmatory trials are required to establish efficacy of Leqembi for continued approval. Vitek et al., (2023) described that accelerated approval for Leqembi was granted after only completing Phase 2, proof-of-concept study with an open-label extension phase of 260 days. This double-blind, randomized, placebo-controlled, "Study 201 Core" of Leqembi utilized cognitive assessment rating scales and cerebral spinal fluid core biomarkers to analyze the efficacy of the drug. The authors reported reductions in clinical decline and beta-amyloid concentrations in patients with mild AD compared to the control group at 18 months. Additionally, a Phase 3 trial, entitled the "Clarity" study, confirmed that Leqembi statistically slowed loss of cognition and function and reduced beta-amyloid plaques in the brain. These

results support the clinal efficacy of the drug but only for patients with early AD, highlighting the importance of early diagnosis. Further, Leqembi was found to have fewer adverse side effects than Aduhelm, including amyloid-related imaging abnormalities (ARIA) which is the most reported and serious adverse effect of both Leqembi and Aduhelm (Vitek et al., 2023). Defined by Roytman et al., (2023), ARIA encompasses a spectrum of MRI findings including edema and microhemorrhages in the brain. While often asymptomatic and self-resolving, rare clinically noticeable symptoms may present as headaches, confusion, dizziness, and visual disturbances (Roytman et al., 2023).

While additional research is being conducted to confirm the clinical significance in the class of monoclonal antibody drugs, current FDA approved medications for symptom management have limited effectiveness (Decourt et al., 2022). Emerging disease modifying drugs show promising potential for treatment of AD, but current strategies rely on symptom management (Vaz & Silvestre, 2020). Thus, healthcare providers must depend on alternative therapies and medications that aim to relieve symptoms and increase quality of life for patients living with dementia (Sikkes et al., 2020).

Alternative Therapies

Since there is no cure for dementia and pharmacological treatments have limited efficacy, healthcare professionals look to alternative therapies to maximize quality of life and manage symptoms for individuals living with dementia (Laver et al., 2016). Some common therapies include cognitive rehabilitation (CR), reminiscence therapy (RT), reality orientation training (ROT), and cognitive stimulation therapy (CST).

Cognitive Rehabilitation

Cognitive rehabilitation is an individualized, problem-solving therapy aimed at managing or reducing functional disability, maximizing engagement and social participation, and mitigating excess disability (Clare et al., 2019). Categorized as a restorative and compensatory therapy, CR focuses on capitalizing residual memory ability in impaired cognitive domains and optimizing unimpaired cognitive abilities (Bahar-Fuchs et al., 2013). Clare et al. (2019) found clinically significant improvements in overall quality of life, social relationships, and functional ability. These results were achieved by utilizing environmental adaptations or prompts, memory aids, procedural learning of skills, and techniques for learning or relearning relevant information (Clare et al., 2019).

Reminiscence Therapy

Reminiscence therapy involves life review with the discussion of memories and past experiences using tangible prompts like photographs, music, or familiar objects to evoke memories and stimulate conversation (Woods et al., 2018). Whereas individuals with dementia recall long-term memories more often than recent memories, RT focuses on remote memory as a tool for communication (Cotelli et al., 2012). A study by Huang et al. (2015) found that RT is effective in improving cognitive function and depressive symptoms in elderly individuals with dementia. However, although RT was associated with improved cognition, mood, and communication, effects are small and can be inconsistent (O' Philbin et al., 2018).

Reality Orientation Training

Reality orientation therapy focuses on relearning conversational skills, cooperating in social activities, and taking an interest in the world (Baines et al., 1987). Targeting the symptoms of confusion in adults with cognitive decline, ROT protocol incorporates the presentation and

repetition of orientation information to provide the patient with a better understanding of their surroundings (Carrion et al., 2013). Based on neurosensory stimulation to reorient patients temporally and spatially, ROT uses repeated and meaningful stimulation of time-place-person orientation through activities in a classroom setting or by professionals and/or caregivers on all occasions that they interact with the patient during activities of daily living (Cammisuli et al., 2016). A study by Chiu et al. (2018), utilized well-validated instruments, including the MMSE, the Clinical Dementia Rating (CDR), the Clifton Assessment Procedures for the Elderly (CAPR), and Koskela tests to find that ROT significantly improves cognitive function in adult patients aged 65 years or older with mild to moderate dementia. While significant cognitive improvement was found in ROT alone and combined with other techniques, ROT was not found to have significant improvements in behavioral or depressive symptoms for patients with dementia (Chiu et al., 2018).

Cognitive Stimulation Therapy

Utilizing a multisensory stimulation approach, CST is an evidence-based psychosocial intervention for mild-to-moderate dementia (Piras et al., 2017). Cognitive stimulation therapy was developed using a combination of effective techniques from reality orientation, reminiscence therapy, and cognitive rehabilitation (Rai et al., 2018). This therapy improves cognitive and social functioning by stimulating language, strengthening cognitive resources, maintaining social engagement, and using concepts of implicit learning (Toh et al., 2016). A study by Saragih et al., (2022) found that CST significantly improved cognitive function and alleviated depression levels among individuals with mild-to-moderate dementia. The repetitive activities of the tasks and games incorporated in CST were shown to improve connectivity in the brain, myelinate neural circuit, and generate new synapses (Saragih et al., 2022). The supporting evidence that CST

improves quality of life and cognitive benefits are key reasons that it is the most widely used psychosocial treatment for individuals with dementia (Piras et al., 2017).

Liang et al. (2020) report that there are a variety of treatments for dementia that show some improvement in symptoms and progression, but there is currently a lack of powerful disease modifying therapies that address the complexity of various dementia-causing pathologies. The limitations of these treatments highlight the importance of primary preventative strategies that target identifying and modifying factors that increase the risk of developing dementia (Liang et al., 2020).

Risk Factors and Prevention

Although prevention is preferable to treatment in all cases, it is particularly significant in the context of dementia considering current interventions do not have the capacity to significantly alter the course of the disease (Montero-Odasso et al., 2020). Dementia is considered to start decades before the presentation of clinical symptoms, thus targeting risk factors may prevent or delay disease onset (Zhang et al., 2021). In fact, according to recent estimates, approximately 40% of global dementia cases could be attributed to 12 modifiable risk factors (Lisko et al., 2021). Categorized into genetic and modifiable risk factors, early identification and prevention can help to maintain patient independence, fulfilment of social roles, and ability to work for longer periods of time (Perneczky, 2019).

Genetic Risk Factors

New technologies including neuroimaging, CSF analysis, and genomic testing, have aided in the identification of genetic biomarkers associated with dementia (Horgan et al., 2020). While apolipoprotein E type 4 allele (APOE ε4) is most commonly linked with the highest risk of dementia, as of 2021 there are more than 70 genetic regions that have been associated with

AD alone (Ranson et al., 2021). An early study by Corder et al. (1993) found that APOE ε4 was a major risk factor for late onset Alzheimer's Disease. Although this does not account for all genetic risk, it is the gene associated with the highest risk of dementia (Solomon et al., 2018). A meta-analysis of diagnosed Alzheimer's Disease by Kunkle et al. (2019), confirmed 20 previous risk loci, or a specific site within a genome, while identifying five new genome wide risk loci. Additionally, Kunkle et al. (2019) found that a family history of Alzheimer's Disease has been positively correlated with a higher risk of developing dementia. Consistent with previous research, APOE ε4 was confirmed as the most common genetic risk factor, however, additional rare variants remain to be identified (Kunkle et al., 2019). Finally, twin studies including 392 pairs of twins categorized in a five-group quantitative genetic model, also confirmed high heritability of developing dementia but also found that nongenetic risk factors play an important role in disease risk and onset (Gatz et al., 2006).

Although genetic risk factors are non-modifiable, identifying and understanding the mechanisms of these genetic biomarkers may ultimately aid in minimizing their contributions to the disease (Eid et al., 2019). Due to the limitations of current disease-altering dementia treatments, identifying genetic risk factors has not been known to alter dementia prognosis but can play a role in effective management and research purposes (Horgan et al., 2020). Interestingly, Licher et al. (2019) found that individuals at low and intermediate genetic risk with healthy lifestyle changes in regard to modifiable risk factors were at lower risk of developing dementia as compared to individuals at low and intermediate genetic risk without healthy lifestyle changes. These findings further highlight the complexity of dementia pathology and underly the importance of disease prevention in the context of modifiable risk factors and interactions with genetic factors (Lane et al., 2017).

Modifiable Risk Factors

To quantify the impact of modifiable risk factors, Population Attributable Fractions (PAFs) are used to describe the proportional contribution of a known risk factor and to estimate the proportion of cases that could be avoided if that risk factor was eliminated from the population (Welberry et al., 2023). A growing body of evidence has identified 12 potentially modifiable risk factors that are associated with a reduction in the risk of developing dementia and shown to promote healthy aging (Omura et al., 2022). The 12 modifiable risk factors were issued in the 2020 Report of The Lancet Commissions and have been ranked using PAFs. Commissions from The Lancet partner with academic institutions to explore advances in science, medicine, and global health targeting improvements in health policies and practices. *Dementia prevention, intervention, and care: 2020 report of the Lancet Commission's* interdisciplinary, international experts systematically evaluated evidence identifying these potentially modifiable risk factors which are categorized into early life, midlife, and later life risk factors (Livingston et al., 2020).

Early Life Risk Factors

Less Education. Education is the only early life risk factor with the second highest worldwide PAF of 7.5% (Mukadam et al., 2019). Higher levels of childhood education and lifelong higher educational attainment are shown to reduce the risk of dementia (Kuo et al., 2020). Recent evidence suggests that cognitive ability increases with education until the brain reaches greatest plasticity and plateaus in late adolescents (Kremen et al., 2019). Gow et al., (2012) agree that cognitive stimulation is most important in early life and that much of the apparent later cognitive effect may be attributed to people of higher cognitive function seeking out cognitively stimulating activities, occupations, and further education.

A study by Sando et al. (2008) found that education had a significant protective effect on the risk of developing AD dementia in a dose-dependent manner. The results showed that the probability of developing AD dementia was least in participants with 10 – 18 years of education, compared to participants with 8-9 years of education, and the highest risk of developing AD dementia was found in participants with 6-7 years of education (Sando et al., 2008). Additionally, epidemiological research agrees consistently finding evidence that educational level is inversely correlated with the risk of developing dementia (Litke et al., 2021).

Midlife Risk Factors

Hearing loss, traumatic brain injury (TBI), hypertension, alcohol intake, and obesity are categorized as midlife risk factors for developing dementia. Hearing loss is the number one modifiable risk factor with a PAF of 9.1% (Brett et al., 2022). The link between hearing loss and the increased probability of developing dementia will be outlined in detail in a later section.

Traumatic Brain Injury. With a global PAF of about 3%, administrative cohort and population-based studies have found evidence linking TBI with an increased risk of dementia (Brett et al., 2022). Traumatic brain injuries can include concussions, skull fractures, edema, brain injury, or bleeds occurring when an external force is transmitted to the head or body resulting in neuropathological damage and dysfunction (Mckee & Daneshvar, 2015). Categorized by severity into mild, moderate, or severe, neurological damage may occur not only at the moment of impact of TBI, known as primary injury, but also over the individual's lifetime, known as secondary injury (Gu et al., 2021). Although there is no confirmed mechanism, it has been hypothesized that TBI injury leads to the development of dementia due to degeneration of mitochondrial function, chronic neural inflammation, vascular damage, dysfunction of the blood-

brain barrier, β-amyloid pathology, tau deposition, and white matter degeneration (Gu et al., 2021).

The post-TBI acute and chronic processes that lead to neurodegeneration are still being explored, however, there is a large body of supporting evidence linking TBI, particularly with greater severity, to an increased risk of developing dementia (Brett et al., 2022). One such study by Mielke et al. (2022) used a population-based cohort with medical record abstraction confirming TBI and dementia to examine the relationship between the presence and severity of TBI and the risk of dementia. Each TBI case was randomly matched to two age, sex, and non-head injury population-based referents without TBI. The results found that the risk of dementia was significantly higher in individuals with a history of any severity TBI compared to individuals without a history of TBI (Mielke et al., 2022). Additionally, a study by Gardner et al. (2014) concurred with existing literature finding that individuals with moderate to severe TBI at 55 years or older or mild TBI at 65 years or older had a significantly increased risk of developing dementia (Gardner et al., 2014).

Hypertension. Hypertension is another midlife risk factor for dementia yielding a global PAF of about 2% (Mukadam et al., 2019). Defined by the presence of a chronic elevation of systemic blood pressure, hypertension is a progressive cardiovascular disease occurring from complex and interrelated etiologies strongly associated with functional and structural cardiac and vascular abnormalities (Giles et al., 2009). It is theorized that midlife hypertension increases the risk of dementia due to disruption of the structures and function of cerebral blood vessels leading to ischemic damage of white matter regions critical for cognitive function (Iadecola et al., 2016).

Available literature from epidemiological studies and randomized controlled trails indicates that only midlife, not late-life, hypertension is associated with an increased risk of

dementia (Singh et al., 2016). Further, a more recent study by Ou et al. (2020) also found stronger associations of dementia in midlife rather than late life. The results found that midlife hypertension was related to a 1.19- to 1.55- fold excess risk of cognitive disorders. Finally, the authors found that antihypertension medications exhibited a 21% reduction in dementia risk While encouraging, the optimal dose, duration, and type warrant further investigation (Ou et al., 2020).

Alcohol Intake. More recently, excessive alcohol intake was added to the potentially modifiable risk factors in the 2020 Report of Lancet Commission with a global PAF of about 1% (Livingston et al., 2020). Although the exact mechanism of which excessive alcohol leads to dementia has not been confirmed, a large-scale study by Topiwala et al. (2017) observed a community dwelling cohort with a follow-up at 30 years. The authors measured alcohol intake every 5 years and used MRI images and cognitive tests to investigate alcohol dependent effects on brain structure and function. The results concluded that alcohol use, even at light or moderate levels, was associated with adverse brain outcomes including differences in corpus callosum microstructure, faster lexical fluency decline, and hippocampal atrophy in a dose dependent manner (Topiwala et al., 2017).

Evidence from available literature highlights the association between excessive alcohol intake and dementia, however, understanding the evidence base can be challenging due to alcohol's strong link with cultural patterns, other sociocultural and health-related factors, and under-representation of heavy drinkers (Rehm et al., 2019). Even so, Schwarzinger et al. (2018) completed a 5-year longitudinal, retrospective study examining the association between the risk of developing dementia and excessive alcohol consumption. The results found that alcohol use disorders were associated with an increased dementia risk yielding hazard ratios of 3.34 for

women and 3.36 for men. Additionally, results indicated that alcohol use disorders were a major risk factor for developing dementia, especially early-onset dementia. The results found that 56.6% of early onset dementia cases were attributed to excessive alcohol intake and/or a documented alcohol use disorder (Schwarzinger et al., 2018).

Obesity. Considered a global epidemic and linked to an increased risk of dementia, obesity can be defined as a body mass index (BMI) of 30 and above and overweight as a BMI between 25 and 29.9 (Albanese et al., 2017). According to WHO guidelines, obesity can be measured by a waist circumference of at least 88 cm in women and more than 102 cm in men and accounts for a global PAF of about 1% (Mukadam et al., 2019). Evidence suggests that dementia risk is linked to increased BMI in midlife and is theoretically caused by early neuroinflammation effects as a result from brain insulin resistance disrupting normal metabolism in the hypothalamus and hippocampus (AL-Dalaeen & AL-Domi, 2022). A meta-analysis of longitudinal studies by Pedditizi et al. (2016) found a positive association with a risk ratio of 1.41 between midlife obesity and dementia for individuals under the age of 65 years (Pedditizi et al., 2016). Further, Veronese et al. (2017) found that weight loss of 2 kg or more in individuals with a BMI greater than 25, showed significant improvement in attention, memory, executive function, and language. While promising, supporting evidence is required to examine the long-term effects of weight loss and dementia prevention (Veronese et al., 2017).

Later Life Risk Factors

Smoking, depression, social isolation, physical inactivity, air pollution, and diabetes are categorized as later life risk factors. As the development of dementia is likely influenced by contributing factors over the life course, categorizing the major risk factors by life stages reflects the timeframe that corresponding interventions would be most effective (Hwang et al., 2023).

However, exposure accumulates slowly throughout life and interventions may take years to show benefit, suggesting that modifying risk factors at earlier stages will likely promote healthy aging and decrease the probability of developing dementia (Liu et al., 2010).

Smoking. While chronic tobacco smoking is widely known to negatively impact health, it also is associated with an increased risk of developing dementia attributing to a PAF of about 5% (Mukadam et al., 2019). Active smokers have demonstrated poorer performance in executive function, processing speed, learning, and memory as compared to non-smokers (Durazzo et al., 2014). Potential mechanisms for the harmful effects of smoking regarding dementia include oxidative stress, inflammation, and atherosclerosis resulting in deterioration of white matter microstructural integrity, cortical perfusion, glucose metabolism deficits, and global brain atrophy (Durazzo et al., 2010). A study by Choi et al. (2018) found that compared to continual smokers, long-term quitters and never-smokers had a significantly decreased risk of dementia with a hazard risk of 0.81. Furthermore, results revealed that stopping smoking even in later life stages reduced the risk of developing dementia. For men older than 65 years that stopped smoking for more than 4 years, a substantially reduced risk was observed over the subsequent 8 years (Choi et al., 2018). Notably, it is speculated that smokers have an underrated risk of dementia due to the bias of the competing risks that causes early smoking-related mortality rates (Chang et al., 2012).

Depression. With a PAF of about 4%, depression is another later life risk factor for dementia (Mukadam et al., 2019). Depression is a serious, but common, mood disorder negatively affecting feelings, thoughts, and actions leading to loss of interest or pleasure (Maske et al., 2016). Depression has been associated with an increased risk of cognitive decline, dementia, and dementia-related mortality (Zotcheva et al., 2018). The pathophysiological link

between depression and dementia has not been confirmed but common hypothesized mechanisms suggest decreased brain function is caused by neuroinflammation from increased pro-inflammatory cytokines in CFS, hypothalamus-pituitary-axis dysfunction due to decreased somatostatin-like immunoreactivity in CFS, and overactivation of the monoamine oxidase pathway that alters essential monoamine neurotransmitter levels in the brain (Hussain et al., 2020).

Evidence from available literature has shown that late-life depression is consistently associated with a twofold increased risk of dementia (Cherbuin et al., 2015). However, it has been debated whether depression is a true risk factor or an early symptom of dementia that appears before the clinical presentation of the disease (Bennett & Thomas, 2014). A large-scale population-based study by Yang et al. (2021) sought to unveil the nature of the dementia-depression association utilizing a life-course perspective. The results of the study found a statistically significant association between an increased risk of dementia and late-life depression as well as depression at any age throughout the life course yielding a 2.16 and 1.93 odds ratio, respectively. Additionally, contributions of genetic and early-life environmental factors could not account for the depression-dementia association. The results of this study support evidence from previous literature suggesting that depression is a true risk factor for dementia (Yang et al., 2021).

Social Isolation. Despite clear cultural variations in the meaning and perception of social isolation, it is an accepted risk factor for dementia with a PAF of about 4% (Livingston et al., 2020). Literature suggests that an active and socially engaged lifestyle is related to improved cognitive function in aging (Kelly et al., 2017). Independent of socio-economic and other lifestyle factors, Sommerlad et al. (2019) found that social contact has a protective effect on

dementia and more frequent contact is associated with higher cognitive reserve. It is unclear, however, if the ability to maintain more social contact may be a marker of higher cognitive reserve. Nevertheless, the 28-year follow-up study of 10,308 participants revealed that in individuals over the age of 60 greater frequency of social contact yielded a lower risk of dementia (Sommerlad et al., 2019). Further, a longitudinal cohort study of 13,984 adults 65 years or older by Saito et al. (2017) examined the association of dementia incidence with social relationship domains. The 10-year follow-up study calculated a 5-point social contact scale based on marital status, exchanging support with family members, having contact with friends, participating in community groups, and engaging in paid work. The results found that scores were linearly associated with dementia incidence and that those who scored highest were 46% less likely to develop dementia compared to those in the lowest category (Saito et al., 2017). Finally, a study by S. Wang et al. (2022) found a significant association linking high social engagement and frequent social contact with a lower risk of dementia. Likewise, loneliness was associated with a higher risk of dementia, consistent with available literature (S. Wang et al., 2022).

Physical Inactivity. Physical inactivity is a lifestyle factor that has been linked to an increased risk of dementia and has a PAF of about 2% (Mukadam et al., 2019). A large body of research suggests that physical activity may reduce the risk of dementia and that both high and low intensity leisure-time activities reduce the risk of dementia-related mortality (Zotcheva et al., 2018). Increased physical activity has been associated with greater hippocampus volume, executive function, and memory performance (Verstynen et al., 2012). The exact physiological mechanism of this relationship is not known but it is theorized that increased energy expenditure, reduction in body fat, and increased muscle strength promotes better metabolism and insulin

sensitivity, downregulation of oxidative processes, reduction in inflammatory response, and release of brain-derived neurotrophic and nerve growth factors (Castells-Sánchez et al., 2019).

While it is commonly accepted that physical activity is linked with a decreased risk of dementia, Wu et al. (2022) aimed to find the duration and amount of activity required to reduce that risk. The authors harmonized longitudinal data of 11,988 participants from 10 cohorts in 8 countries to examine the dose-response relationship between late-life physical activity and dementia incidence. The results suggested that performing 3.1 – 6.0 hours of physical activity and expending 9.1 – 18.0 of metabolic equivalent value-hours per week may reduce dementia risk (Wu et al., 2022). Finally, a study by Saint-Maurice et al. (2019) found that although long-term participation in physical activity may be important to lower mortality risk, becoming active in later adulthood may provide comparable benefits of 32% - 35% lower risk from mortality. These results provide encouraging intervention possibilities for adults between 40 – 61 years of age suggesting it is not too late to start engaging in physical activity for dementia prevention in later life stages (Saint-Maurice et al., 2019).

Air Pollution. With a global PAF of about 2%, air pollution is another recently added modifiable risk factor to the 2020 Report of Lancet Commission (Livingston et al., 2020). High nitrogen dioxide concentration, traffic exhaust fine ambient particulate matter, and residential wood burning have all been associated with increased dementia incidence (Oudin et al., 2018). Evidence from animal models suggests that airborne particulate pollutants cause an acceleration in neurodegenerative processes through amyloid precursor protein processing, Aβ deposition, and cerebrovascular and cardiovascular disease (Power et al., 2016). Further, a critical review and meta-analysis by Delgado-Saborit et al. (2021) found evidence that exposure to air pollution

was associated with reduced white and gray matter volume, smaller corpus callosum, neuroinflammation, and reduced global cognition.

Evidence from available research reveals an association between air pollution and impairments in specific cognitive domains including language, memory, attention, executive function, and visuo-spatial abilities (Delgado-Saborit et al., 2021). Overall, epidemiological literature suggests that exposure to air pollution is associated with an increased risk of developing dementia while highlighting the need for additional research to assess risk reduction and prevention measures (Peters et al., 2021).

Diabetes. The last of the 12 modifiable risk factors, with a PAF of about 1% is type 2 diabetes (Mukadam et al., 2019). Identified as the most common form and associated with dementia risk, type 2 diabetes is a condition of insulin resistance and a failure of pancreatic beta cells to secrete insulin (Selman et al., 2022). Although the etiology of cognitive dysfunction in adults with type 2 diabetes is likely multifactorial, common hypotheses suggest that chronic hyperglycemia and recurrent episodes of severe hyperglycemia cause neurodegeneration due to dysregulation of the hypothalamic-pituitary-adrenal axis, overproduction of inflammatory cytokines, increased levels of rheological factors, and cerebral microvascular disease (Strachan et al., 2011).

Evidence indicates that diabetes is a robust predictor of dementia in older adults, and individuals with type 2 diabetes are at about a 60% greater risk compared to those without diabetes (Chatterjee et al., 2015). Cognitive decline in association with diabetes has been shown to effect specific cognitive domains of episodic memory and executive function including verbal fluency, working memory, processing speed, cognitive flexibility, and cognitive control (Cholerton et al., 2016). In a prospective cohort study of 10,095 participants, Barbiellini Amidei

et al. (2021) clinically examined participants periodically over 31 years concluding that younger onset of diabetes was significantly associated with a higher risk of subsequent dementia. Further, results indicated that every 5-year earlier onset of diabetes was significantly associated with higher hazard of dementia and a faster rate of cognitive decline was associated with severe and/or longer duration of diabetes (Barbiellini Amidei et al., 2021).

"*Dementia prevention, intervention, and care: 2020 report of the Lancet Commission"* released the updated 12 risk factor life-course model of dementia ranked by PAF as hearing loss, early education, smoking, depression, social isolation, TBI, hypertension, physical inactivity, air pollution, excessive alcohol intake, obesity, and diabetes. Together these potentially reversible risk factors make up 40% of dementia risk (Livingston et al., 2020). Given that hearing loss is the leading chronic health condition among adults associated with cognitive decline and dementia, a detailed analysis is warranted to address the association, possible mechanisms, implications, and potential clinical roles, guidelines, and treatment recommendations for the field of audiology (Chern & Golub, 2019).

Hearing Loss and Cognitive Decline

Hearing loss is a rising global public health concern lacking a robust global initiative and funding to support hearing health care (Davis & Hoffman, 2019). Age-related hearing loss (ARHL) is the most common sensory deficit in the elderly leading to severe social and health issues (Huang & Tang, 2010). In addition, numerous studies have established an association with age-related hearing loss and cognitive decline, impaired function across cognitive domains, and increased risk for dementia diagnosis (Uchida et al., 2019). While the underlying mechanism for this association is not well-established, there are four theories that are most widely accepted (Bowl & Dawson, 2018). Unfortunately, not only is hearing loss commonly under-recognized,

but there are also significant barriers to treatments and interventions and a striking lack of resources and accessibility to manage hearing loss (Chadha et al., 2018). Yet research continues to seek evidence supporting preventative strategies regarding hearing loss and dementia and possible treatments to reduce risk and lessen the speed of cognitive decline (Bowl & Dawson, 2018).

Age-Related Hearing Loss

According to the *2021 World Report on Hearing* over 430 million people worldwide have disabling hearing loss and more than 1.5 billion people experience some decline in their hearing during their life course (WHO, 2021). It is estimated that this prevalence may almost double if hearing loss prevention is not prioritized (Dillard et al., 2022). By 2050, a projected 2.45 billion people will have hearing loss with over 62% in individuals older than 50 years (Haile et al., 2021). Astoundingly, on a global scale only 17% of people that need hearing aids utilize them for the treatment of their hearing loss (Orji et al., 2020). Finally, the Global Burden of Disease Study (2019) estimates that unaddressed hearing loss yields an annual global cost of $981 billion due to loss of productivity, health impacts, exclusion of the labor force, poor quality of life, costs of educational support, and other societal factors (McDaid et al., 2021).

Age-related hearing loss, or presbycusis, is a gradual, progressive, typically bilateral and symmetrical age-related sensorineural hearing loss which is most pronounced at higher frequencies (Davis et al., 2016). Sensorineural hearing loss, the most common type of permanent hearing impairment, results from damage to cochlea, auditory nerve, or central nervous system (Pacala & Yueh, 2012). The cause of ARHL is multifactorial with contributions from, but not limited to, aging, genetic factors, environmental factors, oxidative stress, and cochlear vascular changes (Cunningham & Tucci, 2017).

Characteristically ranging in severity from mild to moderate, ARHL typically affects the mid to high frequencies causing degradation of speech-understanding, especially at an increased rate or distance from the speaker (Lehmann et al., 2021). Correspondingly, speech-perception difficulties are also affected most notably in adverse listening conditions such as reverberant and noisy environments (Lee, 2015). Due to the gradual nature of ARHL, symptoms may not be noticeable or perceived as significant in the early stages, however as the severity of hearing loss increases, profound consequences emerge on verbal communication, and the social, functional, and psychological well-being of the individual (Jafari et al., 2019). In addition, ARHL has been associated with reduced quality of life, incident falls, low level of physical activity, frailty, slow gait speed, stress, social isolation, depression, and anxiety (Jayakody et al., 2018). Fortunately, with the assistance of an audiologist or hearing care professional, ARHL can be diagnosed and treated to reverse the sensory deficit (Panza et al., 2015). Although ARHL treatment may improve quality of life and other adverse interactions, there is conflicting evidence regarding the possibility that treatment may reverse or slow the rate of cognitive decline (Cherko et al., 2016). As such, due to the large body of evidence supporting the link between hearing loss and dementia risk, global initiatives focus on hearing loss as a modifiable risk factor and advocate for awareness and prevention measures (Chadha et al., 2018).

Cognitive Effects of Hearing Loss

An independent association between ARHL and dementia risk has been established in recent meta-analyses (Glick & Sharma, 2020). Moreover, longitudinal studies have demonstrated that adults with hearing loss have a higher likelihood of developing cognitive impairment and dementia and are more likely to have faster rates of decline in global cognitive function (Uchida et al., 2019). As one such study, Yuan et al. (2018) analyzed dementia risk across 11 high-quality research studies. The results found that moderate ARHL was associated with a 29% higher risk

of dementia over a follow-up period of 6 years or less and a 57% higher risk over a follow-up period of more than 6 years. Finally, the authors found a significant elevated dementia risk even in individuals with mild ARHL (Yuan et al., 2018). To further support existing literature, Loughrey et al. (2018) aimed to clarify the association of ARHL with cognitive decline, cognitive impairment, and dementia. This study evaluated 36 epidemiological studies including a total of 20,234 participants utilizing objective measures of hearing loss, cognitive function, impairment, and dementia. The results found that hearing loss had a significant association with global cognitive impairment and dementia yielding odds ratios of 2.00 and 2.42, respectively. In addition, a significant association was identified between ARHL and all domains of cognitive function including attention, delayed recall, fluency, immediate recall, processing speed, reasoning, semantic memory, visuospatial ability, and working memory (Loughrey et al., 2018). While evidence supports current literature associating hearing loss with cognitive decline and dementia, emerging evidence has gone beyond objective cognitive assessments to evaluate the physical brain changes correlated with hearing loss (Bisogno et al., 2021).

With the use of MRI imaging, hearing loss has been linked to changes in brain function and structure including reduced whole brain and temporal lobe volume (Bisogno et al., 2021). To assess whether hearing impairment was associated with reduced volumes in the auditory cortex, Lin et al. (2014) conducted a study based on a longitudinal cohort of adult participants older than 55 years over a 6.4-year period. Using MRI neuroimaging, brain volume measurements were obtained annually from individuals with a clinically significant hearing impairment and normal hearing individuals. After adjusting for multiple confounders and covariates, the results found a statistically significant decrease in whole brain and regional volumes in the right temporal lobe in individuals with a hearing impairment compared to normal hearing individuals. The

accelerated brain atrophy of the right temporal lobe regions was specifically found in the superior, middle, and inferior temporal gyri, and parahippocampus which are implicated in spoken language processing, semantic memory, and sensory integration. These results suggest that hearing impairment is independently associated with central neurodegeneration effects on brain structure and function (Lin et al., 2014).

A prospective cohort study by Armstrong et al. (2019) further supplemented evidence supporting the correlation between midlife hearing impairment and later-life reduction in temporal lobe volume. The study obtained audiometric data and MRI neuroimaging results from Baltimore Longitudinal Study of Aging participants with a mean follow-up assessment interval of 19.5 years. The results found a statistical significance in that poorer midlife hearing was associated with steeper volumetric declines in temporal structures known to affect AD pathological findings. Specifically, volumetric deterioration was seen in the right temporal gray matter, right hippocampus, and left entorhinal cortex which are associated with clinical symptoms of cognitive impairment. The findings of this study support the hypothesis that midlife hearing impairment is a risk factor for dementia in older adults via changes in brain structure (Armstrong et al., 2019). While evidence continues to support the correlation between hearing loss and changes in brain structure and function, the exact causal mechanisms for this association remain theoretical (Eckert et al., 2019).

Causal Mechanism Theories

The physiopathological mechanisms or causal relationship between hearing loss and dementia are unknown but there are several theories that aim to explain this well-established association (Slade et al., 2020). Among these four theories are auditory deprivation, social effects, cognitive load, and the common cause hypothesis (Beck et al., 2020). Currently the

complexities and interdependency of these potential causal mechanisms have yet to be determined, however clarifying the mechanistic pathway for the hearing loss – dementia association would have far-reaching implications for research, implementation, evaluation, prevention strategies, and clinical recommendations (Powell et al., 2022).

Auditory Deprivation

Adult-onset auditory deprivation is considered the systemic decrease in auditory performance overtime as associated with reduced availability of acoustic information (Arlinger et al., 1996). This reduction of auditory stimulation in adults is primarily due to untreated ARHL which often presents with difficulty understanding speech (Jafari et al., 2019). It is thought that the reduced auditory input causes a reduction in cognitive demand leading to cortical reorganization and subsequent atrophy and decline in general brain function (Cherko et al., 2016). Lack of auditory stimulation is hypothesized to diminish cortical input to the auditory brain regions causing neuroplastic changes to reallocate cortical processing to frontal brain regions resulting in cognitive decline and dementia (Beck et al., 2020).

Evidence from previous MRI and electroencephalography (EEG) studies have supported the hypothesis that auditory deprivation causes compensatory plasticity changes via cortical resource allocation from central auditory processing to frontal cortical regions (Cardon & Sharma, 2018). In one such study, Campbell and Sharma (2013) aimed to examine the cortical changes associated with hearing loss in early stages of hearing decline and speech processing. Adults with mild to moderate hearing loss and age-matched normal hearing controls completed high-density EEG measurements elicited by speech stimuli and The QuickSIN, a clinical test measuring acuity of speech perception in noise. Using obligatory, passively-elicited, cortical auditory evoked potentials (CAEP), the latency, amplitude, and source localization of the P1, N1,

and P2 components were compared between participants with hearing loss to normal hearing participants. The results found increases in latency and amplitude of the P2 CAEP, decreased activation in temporal cortex and increased activation in frontal cortical areas for participants with hearing loss compared to normal hearing controls. This indicates changes in cortical resource allocation are evident even in early stages of adult hearing loss. Additionally, behavioral performance, as indicated by QuickSIN scores, were significantly correlated with increases of P2 latency, suggesting the cortical changes observed are related to behavioral speech perception outcomes (Campbell & Sharma, 2013) Evidence supporting the theory of auditory deprivation suggests the brain's propensity to reorganize is elicited by intact sensory modalities recruiting deficient auditory cortex regions for neural processing causing neuroplastic changes and cortical atrophy that lead to cognitive decline and dementia (Cardon & Sharma, 2018).

Social Effects

Another hypothesis suggests that psychosocial factors of depression, social isolation, loneliness, and quality of life associated with ARHL precipitate neurodegeneration and cognitive decline (Babajanian & Gurgel, 2022). The concept is that of a cascade effect where hearing loss leads to a lack of communication, in turn reducing social interaction that contributes to social isolation and depression ultimately resulting in accelerated brain atrophy and cognitive decline (Beck et al., 2020). Longitudinal and experimental studies suggest an association with social isolation, depression, and reduced cognition (Cacioppo & Hawkley, 2009). Given that untreated ARHL is linked to reduced communication abilities that can severely limit social interaction or cause social disengagement, there is an increased risk of depression in individuals with hearing impairment (Panza et al., 2018). With that, depression increases the risk of cognitive decline with effects on information processing speed, executive function, working memory, visuospatial

memory, and attention (Steffens et al., 2006). Thus, cascade via social effects hypothesizes that social isolation and depression facilitate the relation between ARHL and dementia (Swain, 2020).

Evidence demonstrates that individuals with hearing loss are more likely to withdraw from society, experience social isolation, and have reduced social network (Sharma et al., 2021). For instance, The National Council on Aging (NCOA) (2000) completed a large-scale, national survey to quantify the social, psychological, and functional effects of hearing loss on older adults. The results indicated that adults with untreated hearing loss reported significantly greater feelings of sadness or depression and were less likely to participate in social activities. In addition, NCOA reported that compared to individuals with untreated hearing loss, hearing aid users experienced significant improvements in quality of life such as improved mental health, better relationships, and greater independence and security (NCOA, 2000). Current literature agrees that the psychosocial factors associated with ARHL are independently associated with loneliness and depression (Sharma et al., 2021). Further, supporting evidence suggests that loneliness and depression are associated with an increased risk of dementia and cognitive decline (Sutin et al., 2018).

In one such longitudinal prospective study, Wilson et al. (2007) sought to examine the association with loneliness and cognitive function. The clinicopathologic cohort study used a modified version of the de Jong-Gierveld Loneliness Scale to quantify loneliness and an extensive battery of cognitive measures to assess global cognition, episodic memory, semantic memory, working memory, perceptual speed, and visuospatial ability. The results found that loneliness was associated with a lower level of cognition at baseline in all domains and with a more rapid decline in cognition during follow-up. Additionally, the mean degree of loneliness

was robustly associated with cognitive decline and the development of dementia (Wilson et al., 2007). Accordingly, the theory of cascade via social effects suggests that the communication difficulties caused by hearing loss induce social isolation, loneliness, and depression that, in turn, cause an increased risk of cognitive decline and dementia (Dawes et al., 2015). Although the exact mechanism underlying the associations between ARHL, depression, and dementia risk are unknown, they are likely not mutually exclusive (Sharma et al., 2021).

Cognitive Load

Cognitive load refers to the excessive cognitive effort, or the amount of information processing required to carry out a task (Uchida et al., 2019). It has been postulated that the human brain has a limited capacity for working memory and if presented with information that exceeds this capacity, information overload occurs (Sweller, 2011). Under conditions where processing resources are limited, such as with hearing loss, the increased auditory processing cognitive load would reduce working memory capacity and tax cognitive reserve leading to an earlier expression of dementia pathology (Lin, 2013). According to the cognitive load theory, cognitive decline and subsequent dementia are caused by excess efforts to understand auditory information which divert the brain from working memory function to auditory processing (Blustein et al., 2023). For individuals with hearing loss, a higher utilization of cognitive resources is required to decipher speech signals causing reallocation from working memory and/or other cortical regions to auditory processing (Anwar et al., 2022). Essentially, hearing loss causes a constant degraded auditory signal that requires excessive auditory perceptual processing in everyday life contributing to the detriment of other cognitive processes and depletion of cognitive reserve ultimately inducing structural brain changes, neurodegeneration, and subsequent risk of dementia (Uchida et al., 2019).

The cognitive load theory has been supported by evidence from observational studies, experimental studies, and neuroimaging (Martini et al., 2014). It hypothesizes that as cognitive load is increased to decode, untangle, and comprehend auditory input, cognitive resources are shifted to auditory processing rather than other cognitive processes (Beck & Clark., 2009). This is supported by evidence indicating increased cognitive load when the auditory signal is deteriorated (Ralli et al., 2019). For instance, Zekveld et al. (2011) sought to evaluate the influence of difficult listening situations and hearing loss on the cognitive processing load. The study analyzed pupillary responses including peak amplitude, peak latency, and mean dilation. Task-related pupil responses reliably reflect the cognitive demands of tasks in multiple domains. Pupil responses were measured for adult participants with hearing loss as compared to adult participants without hearing loss during Speech Reception Threshold in stationary noise tests. Results found that pupil response systematically increased with decreasing speech intelligibility for all participants and that participants with hearing loss had a weaker decline in processing load as intelligibility increased. This supports the concept that cognitive load does increase due to a degraded speech signal and suggests that hearing loss requires consistent listening effort and thus increases cognitive load in all daily listening tasks (Zekveld et al., 2011).

Going beyond behavioral testing, Alain et al. (2018) sought to analyze the brain networks involved in supporting effortful listening under different speech manipulations. A total of 53 functional neuroimaging studies were divided based on the type of speech manipulation paradigm used including speech-in-noise (SIN), spectrally degraded speech using filtering techniques, and linguistic complexity. The results found that SIN and spectral degradation showed overlapping activation region in the left and right insula. Although there was no significant overlap in prefrontal activation across the three speech manipulation paradigms, in all

cases there was activation of other cortical regions. This suggests that differential executive networks and resources from other cortical regions are required to shift to support auditory processing when the cognitive load is increased due to a degraded speech stimulus (Alain et al., 2018). The evidence from behavioral studies supports that individuals with hearing loss experience daily listening difficulties which increase cognitive load while neuroimaging studies support that this increase in cognitive load alters resources from other cortical regions to auditory processing leading to neurodegeneration and an increased risk of developing dementia (Griffiths et al., 2020).

Common Cause Hypothesis

The common cause hypothesis is the only theory that is a co-occurring concept rather than of causal effect, implying that ARHL does not lead to or cause dementia (Beck et al., 2020). This hypothesis proposes that widespread neural degeneration is the common mechanism that underlies both age-related changes in cognition and age-related changes in sensory modalities, including hearing, vision, olfaction, and balance (Baltes & Lindenberger, 1997). This theory argues that cognition and sensation, in this case hearing, do not operate in isolation, but comprise an integrated system in which top-down and bottom-up processing are intrinsically intertwined (Kiely & Anstey, 2015). Therefore, it is proposed that hearing sensitivity and cognition may decline due to a common cause such as microvascular disease or other neurodegenerative diseases that affect the sensory organs and the central nervous system concurrently (Eckert et al., 2019). This suggests that if there is a single cause for both age-related decline in cognition and audition, then hearing loss may just be an early manifestation of dementia during its preclinical phase (Martini et al., 2014). It has been hypothesized that a common pathology can affect the cochlea, causing hearing loss, and affect ascending auditory pathways, causing

neurodegeneration and subsequent dementia (Griffiths et al., 2020). The common cause hypothesis suggests that there is a generalized effect possibly caused by inflammation and oxidative stress, vascular health, aging, genetics, frailty, and lifestyle influences such as smoking, diabetes, and excessive alcohol consumption (Rigters et al., 2017).

Overall, a clear association between ARHL and dementia has been demonstrated throughout literature despite lack of conclusive evidence as to the underlying mechanisms (Azeem et al., 2023). As there is evidence supporting all four theories, it is likely that the exact mechanisms identifying the relationships between hearing loss and dementia are complex and multifactorial (Pronk et al., 2019). Large epidemiological studies have attempted to control common causes and multiple potential pathways but have found that more than one causal mechanism is likely to be involved but has yet to be differentiated (Powell et al., 2022). With rising public health concerns, global initiatives focus on hearing loss as a modifiable risk factor and advocate for awareness and prevention measures (Chadha et al., 2018). Fortunately, with the assistance of an audiologist or hearing care professional, ARHL can be identified, diagnosed, and treated to reverse the sensory deficit (Panza et al., 2015). The population of adults with hearing loss and dementia are expected to rise, therefore audiology is likely to see an increased demand for services to support early detection, timely medical intervention, and social support (Shen et al., 2016). Thus, it is important to understand the role of audiology in hearing loss management and therapeutic strategies involving cognitive decline and dementia (Kramer & Brown, 2023).

Role of Audiologist

Audiologists have a unique opportunity to counteract hearing loss and reduce the potential for associated cognitive decline. While audiologists do not treat or manage dementia directly, they play a crucial role in improving the overall quality of life and communication

abilities of individuals affected by the condition. The first step is education, identification, and assessment of hearing function and communication difficulties to accurately diagnose hearing impairment. Once a formal assessment and diagnosis has been made, the next step is patient-centered counseling regarding treatment and communication strategies, making proper referrals, and engaging in collaborative care via a multidisciplinary approach to healthcare.

Scope of Practice

According to the American Speech-Language-Hearing Association (ASHA), "Audiologists provide patient-centered care in the prevention, identification, diagnosis, and evidence-based intervention and treatment of hearing, balance, and other related disorders for people of all ages" (ASHA, 2018). Audiologists may specialize in a variety of areas within the discipline such as pediatrics, geriatrics, educational, hearing aid dispensing, cochlear implants, research, industrial, intraoperative, vestibular and balance, forensic, military, and even animal audiology (Kramer & Brown, 2023). Although most audiologists practice in healthcare settings, there are a number of different settings in which an audiologist can practice such as the following accompanied by their corresponding estimated distribution: private practice (26%), hospital/clinic (26%), ENT/physician (25%), university (8%), government/Veteran Affairs (7%), industrial (6%), and schools (4%) (Stach & Ramachandran, 2022). However, for the purpose of this paper, the remaining sections will specifically regard audiologists working in healthcare settings with adult hearing loss patients.

Patient Education and Counseling

Patients often assume their hearing loss is a part of aging that cannot be controlled, has little impact on their lives, or know little about prevention measures (Erwin & Chen, 2023). Considering cognition is essential to communication, it falls under the responsibility of audiologists to educate about the potential devastating consequences of untreated hearing loss and the risk of dementia (Taylor & Tysoe, 2013). Patient education can lead to enhanced knowledge, quality of life, self-care, and ability to make informed decisions (Flanders, 2018). Information provided by audiologists may be overwhelming and concerning for patients, therefore the first step is taking a simplified approach to explaining the processes involved in hearing, understanding, and cognition (Watermeyer et al., 2020). By understanding these processes, the patient is better equipped to recognize the importance of early detection and treatment, as well as the risks and consequences of untreated hearing loss (Kestens et al., 2022).

Educating patients about the high-level cognitive resources involved in understanding speech provides realistic expectations of treatment outcomes as simply reversing the auditory sensory deficit does not account for the selective attention, processing speed, working memory, and cognitive flexibility required for speech understanding (Kestens et al., 2022). Finally, by counseling on the topic of hearing loss and its relationship to dementia, the communication provides a communication pathway where patients can discuss prevention, concerns, or changes about memory, cognition, and brain health (Maslow & Fortinsky, 2018). Although patients may not bring up cognition, it is likely that it is a topic that they are thinking about as about one third of older adults report a fear of developing dementia (Kessler et al., 2012). Thus, in accordance with the International Classification of Function and Disability (ICF) model, audiologists should

provide education and counseling regarding cognition and brain function in the context of hearing and communication (Gottschalk & Olson, 2020).

Assessment and Diagnosis of Hearing Loss

An assessment by an audiologist is a crucial component for any patient with suspected hearing loss (Erwin & Chen, 2023). According to the Academy of Doctors of Audiology (ADA) Practice Accreditation Standards, audiological assessment and diagnosis procedures should use systematic, evidence-based protocols to obtain a detailed case history, document patients' symptoms, and medical and pharmacological history to formulate a comprehensive test battery for differential diagnosis of the auditory and vestibular systems (ADA, 2020). The purpose of a hearing assessment is to obtain diagnostic information, assess non-auditory needs, develop a comprehensive treatment plan, and understand the patient's social needs, self-perception, motivation, communication needs, and treatment goals (Turton et al., 2020). According to ASHA, a standard audiological test battery includes case history, external ear examination, otoscopic examination, acoustic immittance procedures, air-conduction and bone-conduction pure-tone threshold measures, speech reception thresholds, and word recognition measures (American Speech-Language-Hearing Association, 2006). The results measure the function of the peripheral and central auditory nervous systems to determine the type and degree of the hearing loss which are then placed into the context of lifestyle and communication demands to determine the extent of impairment and the impacts on communication function (Stach & Ramachandran, 2022).

In addition to standard testing procedures, it is essential to examine factors outside of hearing loss that influence the client such as cognitive ability, mental health status, physical status, general health, dexterity, and visual status. These factors may influence the need for

modification in testing, treatment, additional counseling, and referrals to other professionals (Turton et al., 2020). Patients with reduced cognitive ability due to dementia present with unique challenges during an audiological assessment as they may not understand test directions, lack the ability to be conditioned to the testing protocol, may not be able to tolerate testing equipment, or may experience anxiety, agitation, or other behavioral or psychological symptoms (Hopper & Hinton, 2012). It has been suggested that modifications of general procedures established by ASHA for difficult-to-test populations be applied to patients with cognitive decline and dementia (Burkhalter et al., 2009).

Additionally, Dawes et al. (2021) suggest additional modifications to audiological assessments for individuals with dementia. Some strategies include allowing family members or caretakers to accompany the patient throughout testing to alleviate consternation, help calm and orient the patient, and to share information regarding the patient. Test strategies such as using pulsed tones instead of continuous tones may assist with alertness and orientation to the stimulus. Testing only one low, mid, and high frequency for each ear may provide an essential minimal level of audiometric information. Having the patient respond with yes rather than a button response or looking for repeatable nonverbal or activity responses can aid in obtaining reliable results. It is suggested that audiologists slow down the pace of the assessment, give frequent reminders about the task requirement, allow ample time for the patient to respond, and to speak slowly, clearly, calmly, gently, and with simple words. It is recommended that the audiologist ask the patient to repeat the instructions to be sure that the patient understands the expectation, as well as giving examples, demonstrating the tests, and giving practice runs. Finally, reduced test duration and several visits may be required in combination with other testing strategies. However, if an audiologist is unable to obtain reliable audiometric thresholds despite

modifications, objective electrophysiological tests should be considered (Dawes et al., 2021). After completing an audiological evaluation and a hearing loss diagnosis has been made, the next step is treatment considerations.

Hearing Loss Treatment

An audiologist uses the assessment and corresponding diagnosis to determine whether the patient is a candidate for amplification treatment, to evaluate the potential benefit that the patient may derive, and to recommend an appropriate system (Stach & Ramachandran, 2022). In the case of ARHL, treatment can include hearing aids, cochlear implants, assistive listening devices, aural rehabilitation, and communication strategies (Dawes et al., 2021). The primary treatment for hearing loss is the use of hearing aids which have been developed to restore the auditory signal by amplifying sounds to improve audibility (Giroud et al., 2017). According to ASHA, audiologists are responsible for selecting the electroacoustic and non-electroacoustic characteristics of the hearing aid, the physical fit, selecting the fitting rationale and advanced features, and conducting real-ear probe-mic measurements to verify prescriptive fitting targets of the hearing aid (Jorgensen, 2016). Unfortunately, regarding patients with cognitive decline and dementia, there is a lack of guidelines for best hearing aid selection and fitting practices in this population (Mamo et al., 2018).

In an attempt to assess the clinical practices of audiologists concerning cognition, Kestens et al. (2022) conducted a study highlighting the need for evidence-based guidelines regarding hearing aid fitting based on an individual's auditory-cognitive profile. The authors found that out of the 129 surveyed audiologists with a mean work experience of 8.0 years, only a minority took cognition into account when fitting hearing aids. Most respondents did not take cognition into account when fitting specific digital hearing aid features such as noise reduction

(yes: 31.8%), microphone directionality (yes: 34.1%), amplitude compression (yes: 30.2%), frequency compression (yes: 10.9%), or amplification (yes: 38.8%). However, 67.4% of audiologists reduced the number of manual programs in the hearing aid for cognitively impaired patients. Some small trends of fitting strategies were observed in respondents that indicated modification in this population. Among some methods were increasing noise reduction features, using identical directional microphone settings in each listening condition, utilizing slow-acting amplitude compression, extending acclimatization time frames, and limiting additional hearing aid programs. Although audiologists were found to take cognition into account within the case history interview and general communication strategies, a clear need for evidence-based hearing aid fitting guidelines is apparent (Kestens et al., 2022). Despite a lack conventional clinic-based hearing aid fitting guidelines, the literature suggests that even with standard approaches, hearing aid treatment is associated with reduced dementia-related behavioral symptoms and improved cognitive function (Mamo et al., 2018). The evidence supporting hearing aid use and cognitive function will be addressed in a later section of this paper. As more research emerges solidifying the association of hearing loss, hearing aid use, and cognitive function, it is evident that audiologist play a critical role in global efforts to mitigate hearing loss consequences on cognition and in early identification of cognitive decline.

CHAPTER II

APPLICATION TO THE FIELD OF AUDIOLOGY

Audiologists can play a critical role in global efforts to improve early dementia identification efforts and to mitigate consequences of hearing loss on cognition (Broome et al., 2023). Early identification of cognitive decline is crucial to the health and well-being of patients, their families, and society (Mick et al., 2015). To improve early detection, international, national, and state initiatives have focused on increasing cognitive screening efforts centering around primary care physicians (Maslow & Fortinsky, 2018). Unfortunately, there are many barriers to implementing routine, widespread cognitive screening in primary care settings (Iliffe et al., 2009). Auspiciously, audiologists can serve as potential gatekeepers positioned to optimize systematic early detection efforts (Washnik & Anjum, 2022).

Rationale for Implementation of Cognitive Screening

While audiologists do not treat or manage dementia directly, the field is in an optimal position to join collaborative efforts to increase cognitive screening (Mick et al., 2015). Considering a substantial portion of audiologists specialize in treating older adults with hearing loss, they inherently serve a population at a heightened risk of developing dementia (Beck et al., 2020). As communication experts, audiologists are some of the most equipped to differentiate common symptoms and presentations of hearing loss and dementia (Broome et al., 2023). Additionally, audiologists can ensure that potential hearing concerns are addressed prior to cognitive testing, contributing to accurate diagnostic evaluations and optimal patient care (Mick et al., 2015). Implementing cognitive screening within routine clinical protocol can be a significant step toward achieving the rising demand for early detection of cognitive decline (Washnik & Anjum, 2022).

Early Identification

Globally in 2019, dementia contributed to 33.1 million disability-adjusted life years (DALYs) making it the 6th largest cause of mortality and morbidity among older adults (Nandi et al., 2022). It is important to realize that there are various strategies to delay the progression of cognitive impairment, some patients have experienced reversion to cognitive normality, and there are reversible causes of dementia (Limpawattana & Manjavong, 2021). Thus, the detection and early diagnosis of dementia and related cognitive decline are becoming increasingly important with the ageing of our population (Yang et al., 2016). Dementia is one of the leading causes of dependence in the elderly, constituting a major economic burden for public health systems (Yang et al., 2017). Associated direct and indirect costs are considerable and not only rise from formal health care but also from the loss of productivity from both the affected individuals and their caregivers (Li et al., 2018). Early identification is critical to reduce the economic burden and allow for planning of appropriate management, early education for patients and families regarding prognosis, shared decision making for patient life planning, and improvements in patients' quality of life (Limpawattana & Manjavong, 2021).

Economic Burden

In 2019, the global economic burden of dementia was an estimated $2.8 trillion and projected to increase to $16.9 trillion by 2050 (Nandi et al., 2022). However, these estimates do not capture the broader individual aspects of the burden of dementia such as lost market and non-market productivity of patients and the value of suffering and loss of dignity (Bloom et al., 2020). Dementia care is complex and expensive involving medical care, end-of-life care, and long-term or hospice care formally from medical professionals as well as informally from family members (Langa et al., 2005). El-Hayek et al. (2019) reviewed the full scope and magnitude of

these socioeconomic expenses categorizing them into direct and indirect costs. Direct costs are goods and services in which money is explicitly exchanged. In regard to dementia, medical costs include health office visits, medications, hospital care, equipment, medical treatments, and specialized aids. Direct social and non-medical costs can consist of transportation, community and peer support, psychosocial interventions, private and publicly funded home care, special accommodations, patient income and welfare support, and household modification expenses. Whereas indirect costs can be defined as a "resource lost or invested for which no money is exchanged". These costs are derived from informal care from the unpaid inputs or the productivity losses of informal caregivers. Informal care from friends, family, or others can lead to employment challenges such as reduction or work hours, work impairment, sick leave, early retirement, or mortality (El-Hayek et al., 2019).

A comparative national cohort study by Frahm-Falkenberg et al. (2016) found that these expenses begin to increase starting as early as 10 years prior to diagnosis (Frahm-Falkenberg et al., 2016). Additionally, these associated costs are known to increase as the severity of dementia increases highlighting the importance of early identification to manage and potentially reduce the progression of dementia (Mattap et al., 2022). In fact, in 2018, an estimated $7 trillion (U.S. dollars) could have been saved by individuals that developed dementia if it had been diagnosed in the early mild stages of cognitive impairment (Alzheimer's Association, 2023). Some suggested ways to combat these growing costs include launching public health campaigns, investing in a trained workforce that is incentivized to detect and manage cognitive impairment, and leveraging healthcare professionals and technology to facilitate and improve early detection (Hilsabeck et al., 2022).

Patient-Centered Benefits

In addition to reducing the economic burden associated with dementia care costs, early detection is critical to the health and well-being of patients, their families, and society (de Vugt & Verhey, 2013). To begin with, certain causes of cognitive impairment such as depression, urinary tract infections, sleep apnea, stress and polypharmacy are reversible in some cases (Cummings et al., 2015). Moreover, in a larger percentage of cases changing behaviors, managing chronic medical conditions, and dementia-specific treatments can likely slow and sometimes prevent additional cognitive decline (Krivanek et al., 2021). Thus, the earlier cognitive impairment is detected the sooner a potential cause can be identified, or appropriate treatments can be applied to prevent further decline (Aguirre et al., 2023).

Compared to their peers, individuals with dementia have worse overall health, higher out-of-pocket healthcare expenses, shorter life expectancies, increased utilization of healthcare services, and greater likelihood of dying in hospitals (Krivanek et al., 2021). Accordingly, early identification can provide personal benefits of opportunities to plan for the future, lessen the burden on loved ones, seek education and support, and address potential safety issues such as the ability to drive and take medications as directed (Garnier-Crussard et al., 2019). Additionally, early interventions may help caregivers in anticipating and preparing for the future care role, transitions, and ability to involve the patient with the decision-making process society (de Vugt & Verhey, 2013). Finally, earlier diagnosis allows for a patient-centered, multidisciplinary healthcare approach that could improve the patient's prognosis, ensure the monitoring of mental health, slow the progression of cognitive decline, and manage symptoms and comorbidities (Ramos et al., 2021).

Early Identification Strategies in Healthcare

Since early detection of cognitive decline allows for reaping many benefits, the need for implementation strategies is evident (Yang et al., 2017). The Gerontological Society of America (GSA) recommends improving assessment of cognitive impairment by utilizing the process of KAER: "Kickstart the conversation, Assess cognitive impairment, Evaluate for dementia diagnosis, and Refer to community resources, clinical trials, or other research" (Fitzpatrick & Belza, 2021).

Additionally, Hilsabeck et al. (2022) outlined possible policy implementation strategies and their implications for early identification of cognitive decline. One strategy suggests employing education targeting the public about cognitive health and the benefits of early detection. Public health campaigns should highlight prevention measures and the difference between normal aging versus possible warning signs and symptoms to increase the likelihood of seeking professional guidance. Another suggested implementation strategy is addressing gaps in the healthcare workforce for older adults with cognitive impairment. This would include plans to develop and improve education and training initiatives across disciplines and settings, recruit and retain a dementia-capable workforce, and establish effective communication and collaboration between healthcare professionals, direct care providers, and family caregivers. Implementing these strategies could decrease duplication of services, reduce costs, minimize stress, and improve quality of care. Additionally, implementation policies should incentivize cognitive screenings for primary care physicians and other healthcare providers. Lack of time and reimbursement to conduct these screenings are some of the challenges that clinicians face. Implementing policies to increase cognitive screening efforts requires addressing insurance reimbursement benefits as well as administration and interpretation training. It has been

suggested that more healthcare education curriculums include dementia training, encourage collaboration regarding early identification, and support cognitive screenings initiatives across disciplines and settings (Hilsabeck et al., 2022). In agreeance, the Alzheimer's Association recommends implementing objective cognitive screenings to detect early decline across healthcare disciplines, not only limited to primary care physicians (Perry et al., 2018).

Cognitive Screening in Audiology

Expert consensus guidelines recognize the benefits of early identification. However, many cognitive impairments in older adults are not recognized during routine wellness and physical examinations (Lin et al., 2013). In the first time in United States history, by 2034 there will be more people over the age of 65 than children (Hilsabeck et al., 2022). The progressive rise of the elderly population will bring about an exponential increase in the incidence of age-related disease, including dementia and hearing loss (Ramos et al., 2021). It is expected that widespread demand for cognitive assessments will overwhelm existing healthcare services and infrastructures for primary care physicians (PCPs) and specialists (Sabbagh et al., 2020). In fact, only 41% of older adults with probable dementia are diagnosed suggesting the need for improved detection and communication among healthcare professionals (Amjad et al., 2018).

Most international, national, and state initiatives to increase early detection of cognitive impairment have focused on the role of PCPs regarding cognitive screening and diagnosis (Maslow & Fortinsky, 2018). Unfortunately, PCPs face many challenges and lack support to implement routine, widespread cognitive screenings (Sabbagh et al., 2020). As such, audiologists are well positioned to implement cognitive screening protocols to reduce the patient load of PCPs (Davis, 2021). The association between hearing loss and cognitive decline is well established and it is estimated that older adults with severe hearing loss are up to 5-times more

likely to develop dementia (Strutt et al., 2020). Fortunately, ARHL is highly treatable by audiologists who can serve as potential gatekeepers positioned to optimize systematic early detection efforts and collaborate with a multidisciplinary team to help manage individuals with dementia (Beck et al., 2018).

Barriers to Screening

Primary care physicians are often the first healthcare provider to be sought out by patients concerned with cognitive decline (Ramos et al., 2021). However, despite their awareness of a mandate or benefits of early detection, PCPs face challenges to cognitive screening due to limited time, lack of training, and uncertainty about what to do with a positive screening (Hilsabeck et al., 2022). In fact, it is estimated that about 50-67% of individuals 65 years or older are not recognized or diagnosed by their PCPs (Connolly et al., 2011). Surveys indicate that PCPs in the United States do not feel that they are in a position to fulfill the roles expected of them to identify cognitive decline due to lack of time (Geldmacher & Kerwin, 2013). A negative perception regarding the importance of early diagnosis, lack of training in cognitive assessment techniques, concern with high false positive and negative rates of cognitive screening tests, and lack of resources for specialist referrals and community social services are some of the reported challenges met by PCPs (Geldmacher & Kerwin, 2013).

On average, it takes between 8 to 52 months for individuals to seek medical advice after noticing behavioral changes or difficulties in daily functioning due to possible cognitive decline (Perry-Young et al., 2016). Often patients are uncertain, do not notice early symptoms of dementia, or attribute them to normal aging (Koch et al., 2010). Additionally, individuals may refuse to seek help because they are in denial of the problem, feel shameful, assume there is no treatment, or fear the consequences of taking action (de Vugt & Verhey, 2013). Consequently, if

primary care physicians wait for an expressed complaint, it could delay early identification and intervention (Perry et al., 2018).

Although some countries have recently recommended that brief cognitive screenings be administered to all older adults to identify cognitive impairments in asymptomatic individuals, the utility of routine screening is controversial suggesting clinicians reserve screening for at risk populations (Seitz et al., 2018).

Overlapping Populations

To ensure timely referrals and optimize individual outcomes of audiological interventions, audiologists should be trained to recognize cognitive impairment in older adults and join collaboration efforts for early detection (Shen et al., 2016). As a large proportion of audiologists serve the aging population, there is an increased likelihood of encountering cases of undiagnosed cognitive impairments (Nalley, 2021). Further, due to the growing evidence of the link between ARHL and dementia, individuals with ARHL are considered an at-risk population and could be used as a marker for early diagnosis (Su et al., 2017). Dementia often coexists with other disorders, particularly hearing loss, suggesting a high comorbid prevalence although the estimates vary depending on sample size and definition of hearing loss (Hubbard et al., 2018).

Globally there are over 55 million people living with dementia and 1.57 billion people that have some form of hearing loss (Gauthier et al., 2021) (McDaid et al., 2021). Given that the prevalence of hearing and cognitive impairment exponentially increases with age, it is highly likely that audiologists will experience an overlap in aging populations (Bott et al., 2020). An early study by Gold et al. (1996) found an extraordinarily high prevalence of hearing loss in adults with cognitive impairment. In a small sample of 52 patients that met criteria for probable AD, 94% of patients had a significant hearing loss (Gold et al., 1996). In a more conservative

estimate but still highly alarming, Nirmalasari et al. (2016) measured the prevalence of hearing loss in cognitively impaired individuals over 50 years old. Over a 12-month period, 133 patients with cognitive impairment from a memory clinic underwent hearing assessments. The results indicated 60% of the sample had at least a mild hearing loss in their better ear. These results highlight the disproportionately high prevalence of hearing loss in this vulnerable population (Nirmalasari et al., 2016). Considering that hearing loss and early cognitive decline are common and frequent comorbid conditions, audiologists are already in a position to screen and monitor this at-risk population in conjunction with hearing evaluations (Del Vecchio et al., 2023). It has been suggested that clinicians should be more sensitive to detecting early dementia symptoms within the first 2 years following ARHL diagnosis (Su et al., 2017).

Discriminating Symptoms

Aging is highly associated with deterioration in both cognitive functions and sensory functions, such as hearing ability (Cutajar & Tabone, 2022). As previously established, effective communication is essential to both hearing and dementia (Yue et al., 2021). As such, discriminating hearing difficulties from cognitive difficulties can prove challenging (Dawes et al., 2021). Common symptoms of declines in both hearing and cognition include difficulty following conversations, forgetfulness, and withdrawals from social gatherings (Dawes et al., 2021).

Specifically, dementia-related communication difficulties manifest as word-finding problems, restricted verbal output, repetitiousness, diminished ability to understand verbal output, and sound localization difficulty (Slaughter et al., 2014). Similarly, individuals with moderate to severe hearing loss also present with common symptoms such as confusion during conversations, repetitive questioning, inappropriate changes in conversational topics, difficulty

completing everyday tasks, and feeling of fatigue and stress (Hardy et al., 2016). Seeing as improvements in diagnostics, amplification, and aural rehabilitation continue, audiologists' focus is shifting from simply hearing to the ability to effectively communicate (Beck et al., 2018). Since there is significant overlap between symptoms of ARHL and cognitive decline, it further supports audiologists as appropriate professionals to administer cognitive screenings (Hopper & Hinton, 2012). Consequently, there is evidence that untreated hearing loss may contribute to dementia misdiagnosis due to artificially severe symptoms and implications on cognitive testing (Nirmalasari et al., 2016).

Administration Considerations

Considering the majority of questions and instructions are administered orally, performance of cognitive tests is affected by various factors and sensory impairments, especially ARHL (Ray et al., 2019). It is extraordinarily rare for the test administrator to know if the patient has subjective or diagnosed hearing loss which may impede the patient's comprehension of the verbal questions or instructions (Beck & Grisel, 2022). Specifically, there is evidence from a number of lab-based behavioral studies assessing the impact of hearing ability on cognitive test performance (Shen et al., 2016). For instance, a between-subjects study by Jorgensen et al. (2016) examined the effect of decreased audibility performance on the MMSE in individuals without cognitive impairment. A total of 125 young normal hearing participants were randomly assigned to one of five degrees of simulated hearing loss conditions. The researchers found that reduced audibility, even without the confounding of cognitive decline, significantly affected MMSE test scores resulting in greater apparent cognitive deficits as audibility decreased (Jorgensen et al., 2016).

Evidence from a study by Guerreiro and Van Gerven (2017) agreed that without taking sensory acuity into account for cognitive testing, age-related cognitive decline may be overestimated (Guerreiro & Van Gerven, 2017). It is likely that in addition to the association between hearing loss and lower cognitive function, reduced audibility in verbal testing also has a negative impact on cognitive test scores (Shen et al., 2016). Accordingly, it has been suggested to improve communication by administering tests in quiet environments, with face-to-face oral presentation, and to consider use of amplification when appropriate (Dupuis et al., 2015).

If poor performance on cognitive assessments is related to hearing dysfunction, it will likely result in false positive diagnoses, leading to unnecessary patient burden and waste of healthcare resources (Saunders et al., 2018). To combat these negative impacts, evidence suggests that restoring audibility via amplification for individuals with a hearing loss improves cognitive testing scores, often resulting in a shift of a full diagnostic category (Gaeta & John, 2021). A randomized control study by MacDonald et al. (2012) assessed how the use of commercially available hearing augmentation devices affected performance on cognitive assessment scales. A total of 192 patients with a mean age of 82.4 years were randomly selected into either the control group or the intervention group that utilized a portable personal amplification device. The MMSE was administered to each of the groups on two consecutive days. The results indicated that hearing augmentation significantly improved performance on MMSE scores compared to control group (MacDonald et al., 2012).

Similarly, a study by Gaeta et al. (2022) aimed to investigate the impact of amplification on cognitive screening performance. Participants consisted of adults ages 60 to 80 years old with bilateral mild to moderately severe sensorineural hearing loss with no history of cognitive impairment. The participants were given alternate versions of the MMSE in three conditions

varying in test order. The three conditions consisted of without hearing aids, with hearing aids, and with a personal listening device. The results found that MMSE scores significantly improved with the use of hearing aids or personal listening devices. Additionally, the majority of participants had scores that improved by at least one category of severity with the use of amplification. Demonstrating that if an individual with hearing impairment did not have access to amplification for a standard cognitive assessment there is an increased likelihood that it would result in an incorrect diagnosis. Finally, the findings highlight the similar benefits of personal listening devices that could serve as a low-cost, universal alternative to hearing aids (Gaeta et al., 2022). As such, audiologists can provide important roles in reducing potential misdiagnoses of cognitive decline by identifying hearing impairment and providing appropriate amplification before administering cognitive screening tests (Beck & Harvey., 2021).

It is well known that a high percentage of those attending dementia assessment services have hearing loss (Beck et al., 2020). In fact, Allen et al. (2003) found that 87% of those attending dementia assessment had hearing loss and of that 80% were unaware of this deficit (Allen et al., 2003). Evidence suggests that test conditions with reduced audibility, especially due to ARHL, could result in incorrectly identified cognitive impairment or misdiagnosed severity of decline (Jorgensen et al., 2016). Unfortunately, the extent to which hearing loss contributes to impaired test performance is easily underestimated and can lead to serious adverse consequences (Ray et al., 2019). Thus, awareness of and correction for individual's hearing ability is critical prior to undergoing standardized measures that contribute to a cognitive impairment diagnosis (Nirmalasari et al., 2016). The role of an audiologist can positively influence potential consequences as specialists in identifying and providing appropriate amplification for individuals with potential hearing impairment before administering cognitive screening tests (Beck &

Harvey, 2021). The growing evidence supporting improved performance on cognitive screening test scores with the use of amplification has led research to examine the potential positive effects of hearing loss treatment to counteract and reduce cognitive decline (Castiglione et al., 2016).

Implementation of Cognitive Screening

The general goal of cognitive screening is to uncover the possibility that a patient is at risk for dementia and the potential impact on speech understanding and listening ability (Beck et al., 2016). Specifically, cognitive screening can indicate the presence of MCI, identify a modest cognitive decline from previous performance without the presence of noticeable symptoms, or a severe decline that may interfere with independence (American Psychiatric Association, 2013). The clinical diagnosis for MCI or related cognitive disorders is reserved for physicians, however understanding the general cognitive domains that screening tools assess is important for audiologists (Shen et al., 2016).

Gottschalk and Olson (2020) describe the six domains that are assessed by cognitive screening tests including executive function, attention, memory, language, perceptual motor function, and social cognition. Executive function refers to the ability to self-monitor, organize, plan, reason, and problem solve. Attention allows for concentration and focus. Memory encompasses the ability to encode, store, and retrieve information. Language comprises object naming, word finding, fluency, grammar, syntax, and receptive language. Perceptual motor function allows for synchronized body movements. Social cognition allows for processing and storage of various social signals and recognition of emotions. It is important to note that these six domains do not work in isolation but dynamically interact to form cognition (Gottschalk & Olson, 2020). Currently there is no nationally recognized screening tool or standardized

screening methodology, so clinicians are tasked with choosing a screening tool that is best fit for their practice (Perry et al., 2018).

Cognitive Screening Tools

Cognitive screening is within audiologists' scope of practice, however with a variety of validated rapid psychometric screening tools there still is a lack of clearly defined guidelines (Weinstein, 2022). Audiologists can choose from a number of inexpensive and easy-to-administer cognitive screening tests but should consider specific characteristics and psychometric properties (Beck et al., 2016). According to Shulman (2000) an ideal cognitive screening test should have the following characteristics: 1) quick, brief, and easy to administer with minimal training; 2) well tolerated and accepted by patient, and easy to score; 3) relatively independent of culture, language, and education; 4) have good inter-rater and test-retest reliability; 5) high levels of sensitivity and specificity; 6) have concurrent validity as scores correlate with measures of severity and dementia rating scores; and 7) have positive predictive value (Shulman, 2000). The most widely used cognitive screening tests include the Mini Mental Status Evaluation (MMSE), the Montreal Cognitive Screening Assessment (MoCA), the Mini-Cog Test, and Cognivue (Lin, J. et al., 2013).

The Mini-Cog Test

The Mini-Cog is a brief neuropsychological screening test with an administration time of about 3-5 minutes and was designed to reduce educational, language, and cultural biases (Seitz et al., 2018). The test consists of the clock drawing test (CDT) to assess executive function and a delayed, three-word recall task to assess memory (Li et al., 2018). The CDT requires the patient to spontaneously draw a circular clock displaying a particular time and is scored with points awarded according to the components of the clock included (Beck et al., 2016). However, there

are a variety of scoring systems for the CDT which can cause the psychometric properties to vary depending on the scoring algorithm utilized (Li et al., 2018). Employing an optimal referral cutoff of 2/3, the Mini-Cog has been found to have 90% sensitivity and 71% specificity plus has been validated in multiple settings including community, primary care, and specialized clinics (Carnero-Pardo et al., 2022). Nevertheless, it has been proposed that the cognitive domains assessed by the Mini-Cog are not comprehensive enough to be fully sensitive and it has been suggested to be used in combination with other cognitive screening tests for the most effective screening (Li et al., 2018).

The Mini Mental Status Evaluation

The Mini Mental State Evaluation is an efficient test with a total administration time of 5–10 minutes and the most commonly used cognitive screening tools by both general practitioners and specialists in western countries (Judge et al., 2019). It is mainly a verbal test used to screen for orientation to place, recall, attention, comprehension, reading, writing, language, construct ability, calculation, and registration (Folstein et al., 1975). The MMSE has a total of 11 test items with a maximum score of 30 ranging in severity from no cognitive impairment with a score of 24-30, mild cognitive impairment with a score of 18-23, and severe cognitive impairment with a score of 0-17 (Tombaugh & McIntyre, 1992). Although MMSE score correlate well with functional capacity and ability to carry out instrumental activities of daily living, it is designed for individuals that are fluent in English and have at least an 8th grade education (Beck et al., 2016). Additionally, the accuracy of the test in discriminating MCI varies with different referral cutoffs ranging from 13 to 97% for sensitivity and 60-100% for specificity (Zhuang et al., 2019). Considered adequate, the MMSE had 88.3% sensitivity and 86.2% specificity for the most commonly reported cut off points (23/24 or 24/25) but has been found to

better recognize moderate cognitive impairment and be less reliable in identifying potential MCI in early stages (Beck et al., 2016).

The Montreal Cognitive Screening Assessment

Another widely used cognitive screening test, the MoCA has 13 test items and administration time is about 10-15 minutes (Gottschalk & Olson, 2020). The domains evaluated include short-term memory recall, a clock drawing test, a three-dimensional cube copy to assess visuospatial abilities, a test of executive function, orientation to time and place, memory, delayed recall, and abstraction (Nasreddine et al., 2005). The MoCA has been translated into 65 languages and dialects, has adapted versions for the blind and hearing impaired, and can be administered traditionally with a pen and paper or through digital/app versions (Washnik & Anjum, 2022). The MoCA has been found to be a better measure of cognitive function due to lack of ceiling effect and good detection of cognitive heterogeneity (Jia et al., 2021). Utilizing a referral cutoff of 23, the MoCA overall sensitivity yielded 83% and specificity 88% which reduces that rate of false positives (Carson et al., 2017). However, the MoCA is considered the lengthiest and most difficult test to administer and interpret, and it is not recommended for education levels of 12 years or less (Zhuang et al., 2019). This test remains free for clinicians, academic researchers, and teachers but requires training and certification in order to perform, score, and interpret results (Washnik & Anjum, 2022).

Cognivue®

Recently introduced and FDA-approved, Cognivue® is a physiological and psychophysical screening tool administered using a standalone computer and onscreen instructions (Rose et al., 2021). This screening tool evaluates six cognitive domains consisting of visuospatial, executive function and attention, naming and language, memory, delayed recall,

and abstraction (Raduns, 2020). Cognivue® was developed based on extensive research into the neural mechanisms of functional impairment in memory, aging, and dementia (Cahn-Hidalgo et al., 2020). It was designed to allow for easy administration and interpretation to identify cognitive decline and to track changes in cognitive function over time (Raduns, 2020). This adaptive screening tool consists of ten separately scored sub-tests within three sub-batteries including visuomotor and visual salience, perceptual processing, and memory processing (Smith et al., 2018). It is intended to be self-administered with a short, 10-minute duration which generates a one-page report with a single clinical score for easy interpretation of results (Raduns, 2020).

Unfortunately, there are limited validation studies of Cognivue®, especially studies not associated with or funded by the company (Rose et al., 2021). In one such study, Cognivue® was found to avoid misclassifications of cognitive impairment versus no impairment and demonstrate superior reliability and good psychometric validity (Cahn-Hidalgo et al., 2020). Although it was found that Cognivue® demonstrated good agreement with St. Louis University Mental Status (SLUMS) and other neuropsychological tests, it is suggested that this tool be closely reviewed until more research confirms that the test meets the standards for reliability and validity (Rose et al., 2021). Overall Cognivue® has many features of the ideal cognitive screening test given that it was designed for autonomous administration with a brief duration of about 10 minutes, to test multiple domains, and provide easy to interpret performance scores that can facilitate tracking of cognitive functions (Smith et al., 2018).

Integration into Clinical Audiology

In addition to the specific characteristics and psychometric properties of the screening tests themselves, audiologists must consider overall goals, workflow issues, and clinician style in order to choose the best screening and implementation methods for their practice (Beck et al., 2018). Integrating cognitive screening requires careful considerations regarding which patients should be screened, when and how screenings would be conducted, how to deliver results, referral procedures, and patients' perspectives of screenings performed in an audiology setting (Broome et al., 2023).

In a primary care setting, the US Preventative Task Force does not endorse universal screening for adults 65 years or older who do not exhibit signs or symptoms of cognitive impairment (Moyer, 2014). However, routine cognitive screening in audiology clinics has been suggested for patients 70 years and older given that adults with hearing loss are considered an at-risk population (Beck et al., 2018). In addition to routine screening criteria, clinicians can help identify screening candidates by including questions about memory, depression, and history of head injuries on case history forms (Remensnyder, 2012). Other strategies for identifying candidates include paying attention to any atypical communication, inappropriate affective reactions, and memory difficulties such as frequently missed appointments, confusion over simple directions, and difficulty finding words (Robinson et al., 2015). Finally, it may be helpful to communicate with family members or caretakers to address any cognition concerns and to collect information regarding memory, behavior, social isolation, and communication (Shen et al., 2016). Once a patient has been identified as a candidate for cognitive screening, clinicians should consider where screening fits into the audiological test battery and patient perception factors.

Allen et al. (2003) found that 87% of adults that attended dementia assessment sessions had hearing loss and that 80% of those evaluated were unaware of their hearing loss (Allen et al., 2003). Further, it has been found that hearing loss may directly affect cognitive assessment results which can lead to misdiagnosis (Uchida et al., 2019). As such, it has been suggested that cognitive screenings be administered after a comprehensive audiometric evaluation in which appropriate amplification, or modifications can be provided to ensure accurate results (Beck & Harvey, 2021). It is important to note that adding cognitive screening into the audiological test battery may require supplementary training and additional time and staff resources regarding addressing patient concerns and perspectives (Broome et al., 2023).

Although conducting cognitive screening within audiology services has been found to be acceptable to most patients, a survey found that patients expressed concerns regarding relevance between cognition and audition, as well as audiologists' qualifications and experience delivering and interpreting results (Broome et al., 2023). Additionally, psychological issues have been associated with the thought of cognitive screening including anxiety, irritation, embarrassment, defensiveness, and fear (Broome et al., 2023). Considering that some patients may perceive cognitive screening to be intrusive and threatening, it is important to elucidate the purpose and benefits of administration (Beck et al., 2018). Accordingly, recommending cognitive screening to patients should not solely focus on dementia identification but incorporate a discussion regarding the role of cognition in audition whereas screenings can help to rule out other issues that may cause communication difficulties (Dawes et al., 2023).

Once results have been interpreted and a failed result has been identified, it is important for the clinician to attempt to reduce the patient's anxiety or shame by reiterating that the results are not diagnostic and by facilitating the expansion of a supportive team of professionals (Beck

et al., 2018). Audiologists should have an established, multidisciplinary referral network and resources that assist the patient in understanding more about dementia and finding community support (Beck et al., 2018). Referral network should consist of, but not limited to, primary care physicians, memory care clinics, psychologists, and geriatricians (Beck et al., 2018). Additionally, referrals for formal diagnostic testing should include information about the patient's hearing abilities and needs to ensure that the influence of hearing loss or noise levels can be controlled and appropriately interpreted (Pichora-Fuller, 2015).

Many individuals in the early stages of cognitive decline are undiagnosed and audiologists may be the first to encounter and identify this population (Armero et al., 2017). While it is currently well within the scope of practice in audiology, there are multiple factors that need to be taken into account before integrating cognitive screening into a clinical setting (Shen et al., 2016). Unfortunately, there is no concordance or guidelines for implementing a screening protocol into audiology services (Washnik & Anjum, 2022). However, it is logical that this should be a part of an audiologist’s ethical responsibility and by careful consideration of the multiple associated factors, audiologists can integrate cognitive screening that best fits their practice (Gottschalk & Olson, 2020).

CHAPTER III

CRITICAL REVIEW OF THE LITERATURE

The exploration of the relationship between hearing loss and cognitive decline has traversed centuries of historical understanding and modern scientific inquiry (Vatanabe et al., 2020). While research initially focused on conventional risk factors, advancements in technology and growing awareness highlighted the relationship between hearing loss and cognitive decline (Abraham et al., 2023). Despite exponential growth in research in recent years, significant gaps persist in our understanding, necessitating multidisciplinary collaboration, innovative approaches to address research challenges, and priorities for future research.

History of the Literature

Some of the earliest historical records regarding the concept of dementia date back to around the year 2000 BC where ancient Egyptians identify that age could be accompanied by a major memory disorder (Boller & Forbes, 1998). Due to the heavy influence of the church and lack of scientific understanding during the Middle Ages, various religious or supernatural terms were used to describe cognitive decline and mental disorders (Vatanabe et al., 2020). In fact, leading to the 14th and 15th century witch hunts, the book "Hammer of the Witches" from 1948 by Heinrich Kraemer and Johann Spregner detailed how to identify and deal with 'demon-possessed' individuals which included people with dementia and other mental illnesses (Pacheco Palha & Esteves, 1997). It was not until the 19th century that dementia would be defined as a form of insanity accompanied by psychosocial incompetence and civil and legal incapacity (Albert & Mildworf, 1989). It was during this period that awareness of the difficulties in finding the diffuse cerebral causes of dementia led to the concentration of research on clinical symptoms and progression of the illness (Pacheco Palha & Esteves, 1997).

However, in 1906, Alois Alzheimer presented a case study that led to the postmortem discovery of the histopathological presence of plaques and tangles in the brain, which currently remain as the neuropathological hallmarks of Alzheimer's Disease today (Jellinger, 2006).

Risk Factors for Dementia

In the 20th century observational and epidemiological studies dominated research aiding in the identification of key risk factors for dementia (Vatanabe et al., 2020). For instance, initiated in 1948, the Framingham Heart Study is an ongoing, community-based, longitudinal, prospective cohort study aimed at identifying factors predisposing individuals to cardiovascular disease and hypertension (Satizabal et al., 2016). Complementary to the extensive accumulated cardiovascular data was the systematic assessment of cognitive function dating back to 1975 (Wolf, 2012). Now on its third generation of participants, this study has identified stroke, diabetes, hypertension, and physical inactivity as leading modifiable risk factors for cognitive decline (Satizabal et al., 2016). Moreover, established in 1990, the Rotterdam Study is a longitudinal, prospective, population-based cohort study aimed to investigate etiology, preclinical phase, prognosis, and potential intervention targets of chronic diseases among adults over the age of 54 (Oeppen & Vaupel, 2002). Regarding dementia specifically, the results identified risk factors including obesity, hypertension, diabetes, cholesterol levels, smoking, and low education (de Bruijn et al., 2015). This was one of the studies that highlighted the need to identify risk factors in order to discover the causes of diseases and potential targets for preventive interventions (Ikram et al., 2020). Longitudinal, population-based research is critical to advancing our understanding of dementia and has been successful in contributing to our understanding of dementia, including hearing loss as a risk factor.

Hearing Loss

While other risk factors were gaining early attention from researchers, among the first studies to report on the independent relationship between hearing loss cognitive decline was conducted by Uhlmann et al. (1989). The case-control study included 100 participants with dementia and 100 age, sex, and education matched non-dementia participants. The results found that the prevalence of hearing loss of 30 dB HL or greater was significantly higher in participants with dementia. Additionally, a dose-dependent relationship was observed in that greater hearing loss was associated with higher severity of cognitive decline (Uhlmann et al., 1989). Despite this evidence supporting the hypothesis that hearing impairment contributes to cognitive decline, little research had been conducted in the ensuing years to further explore this association (Lin & Albert, 2014). Progress in this research was likely hindered by lack of interaction and collaboration among specialists including otolaryngologists, audiologists, neurologists, epidemiologists, and cognitive scientists (Fortunato et al., 2016). It is also possible that because hearing loss often goes undetected, it was not as apparent as other risk factors.

It was not until the early 2010s that research regarding hearing loss and cognition began to emerge with increasing attention (Abraham et al., 2023). The increase was likely caused by the rising prevalence and negative impacts of cognitive decline, the increased global efforts to raise awareness of dementia as a growing public health priority, and the mounting demand for prevention and treatment of dementia due to the increased proportion of older individuals in the general population (Fortunato et al., 2016). Of note, a study by Lin, et al. (2011) gained significant attention in the scientific community and the media due to its robust methodology, large sample size, and long, 12-year follow-up (Powell et al., 2022). Much of the early, foundational evidence was provided by epidemiological studies but with technological

advancements have included neurobiological and neuroimaging evidence establishing the effect of hearing loss on brain structure and function (Ford et al., 2018). With an influx of supporting evidence, hearing loss was first identified as a risk factor for dementia by the Lancet Commission in 2017 and due to compelling evidence from recent perspective studies and meta-analyses, was identified as the largest risk factor in 2020 (Abraham et al., 2023). The historical gaps in research highlight the need for better interdisciplinary collaboration and improved detection of hearing loss. Nevertheless, ongoing global initiatives to raise dementia awareness and technological advancements in neurological evidence have propelled research in the field forward and continue to drive its evolution.

Gaps in the Research Literature

Hearing involves both peripheral and central auditory systems. Current scientific literature primarily considers hearing loss in the context of formalized measures of peripheral hearing ability. While pure tone audiometry is the gold standard for peripheral auditory testing, specialized tests of central auditory ability are rare in clinical assessments and large-scale epidemiological studies. The inclusion of self-reported hearing loss and lack of distinction between peripheral and central auditory ability causes heterogeneity of previous studies creating barriers to pooled evidence and causal inference regarding the role of central hearing ability within cognitive impairment and testing. Additionally, literature has theorized multiple potential mechanistic pathways linking hearing loss and dementia. However, despite large epidemiological studies that have attempted to control for contributing factors, no one theorized pathway has been clearly identified. Evidence for the treatment of hearing loss or use of hearing aids to mitigate the risk of dementia is emerging but many questions still remain. Extensive gaps in evidence for hearing loss treatment and dementia remain with considerations of efficacy,

effectiveness, and cost efficiency required to guide best practices for aural rehabilitation choices for prevention.

Research Challenges

There are many challenges to overcome when conducting research on the relationship between hearing loss and cognition which may affect the quality of current and future literature. For instance, heterogeneity within auditory science and cognitive research create obstacles for evidence synthesis. Such heterogeneity obstacles include differing means to categorizing hearing ability, defining hearing loss, utilized pure tone frequencies, variability in neurocognitive tests, utilization of cognitive screening tests, the considered cognitive domains, patient or physician report, and ways of defining cognitive change. Standardized assessment tools and protocols have not been clearly established, potentially compromising reliability and comparability. Additionally, selection effects of older adults into studies present with unique challenges. Individuals who pursue and obtain hearing loss treatment are generally subgroups consisting of individuals with higher education, higher income, and greater health seeking behaviors which are also considered protective factors for cognitive decline. Further, many studies are observational and lack the ability to account for potential confounders which weaken causal inferences. Recruitment and study participation of individuals with more severe cases of dementia are difficult to include in research studies due to their inability to follow instructions or complete tasks. Finally, ethical considerations present challenges in the form of informed consent and the well-being of participants.

Future Directions

Research identifying the association between hearing loss and dementia has made huge strides, especially in its relatively short history. However, with continued multidisciplinary collaboration, efforts for future research should focus on advanced identification of subgroups that present with a greater risk of cognitive decline and exploring options for not only intervention, but prevention. Specifically, future research should prioritize the identification the mechanistic pathway of association, management of potential bias due to sensory loss in cognitive testing, determination of hearing loss treatment possible effects of reducing or mitigating dementia risk, and examination of biomarkers or indicators of sensory loss as identifiers of pre-clinical dementia. Determination of one or multiple mechanistic pathways and whether the pathway is unique to the individual may provide the ability for a more person-centered approach to dementia prevention and intervention, as well as improved study design. This may be furthered by advancements in genetics and precision medicine. Future directions may include initiatives to expand the administration of cognitive screening tests to include allied healthcare professionals with universally standardized guidelines for screening protocols and referrals.

Summary

Dementia is a rapidly growing global health crisis that is projected to affect approximately 139 million individuals by the year 2050 (Niotis et al., 2022). This condition has far-reaching effects on individuals, caregivers, healthcare institutions, and imposes substantial global socioeconomic burdens (Li et al., 2018). Unfortunately, there is no known cure for neurodegenerative dementia, and existing medications and therapies have limited efficacy (Buccellato et al., 2023). Therefore, identifying and mitigating risk factors for dementia has

become a top priority in healthcare and research (Chowdhary et al., 2022). One such risk factor that has garnered significant attention in recent years is hearing loss, which ranks among the top modifiable risk factors associated with an elevated risk of dementia (Ying et al., 2023). Although the precise causal mechanisms underlying the link are still under investigation, mounting evidence supports the robust association between untreated hearing loss and impaired cognitive function across various domains and an increased risk of dementia diagnosis (Powell et al., 2022).

Therefore, clinical audiologists, as specialists in hearing and communication disorders, have a unique opportunity to contribute significantly to addressing the dementia crisis. Their role extends beyond improving the quality of life and communication abilities of individuals affected by dementia to also play a pivotal role in the early identification of cognitive decline (Gottschalk & Olson, 2020). By incorporating cognitive screening into their practice, audiologists can help identify potential cognitive impairments in individuals who might not have been otherwise assessed. To implement cognitive screening effectively in clinical audiology, audiologists need to be well-equipped with the necessary knowledge and skills related to dementia (Washnik & Anjum, 2022). Due to the lack of established guidelines for implementing cognitive screening tools, audiologists should carefully consider which cognitive screening tools to use and how best to implement them in their specific clinical setting. This process may involve identifying the most suitable screening instruments, developing efficient workflow procedures, and training audiologists and support staff. By integrating cognitive screening into their clinical protocols, audiologists can contribute to the early detection of cognitive decline and promote a multidisciplinary approach to providing comprehensive, holistic care to their patients.

Audiologists should continue to stay informed about the latest research and best practices in this evolving field to ensure they can make a positive impact on the lives of their patients.

REFERENCES

Abraham, A. G., Hong, C., Deal, J. A., Bettcher, B. M., Pelak, V. S., Gross, A., Jiang, K., Swenor, B., & Wittich, W. (2023). Are cognitive researchers ignoring their senses? The problem of sensory deficit in cognitive aging research. Journal of the American Geriatrics Society, 71(5), 1369-1377. https://doi.org/10.1111/jgs.18229

Academy of Doctors of Audiology. (2020). *Audiology Practice Accreditation Standards*. https://www.audiologist.org/about-us/academy-documents/accreditation-standards

Agrawal, S., & Schneider, J. A. (2022). Vascular pathology and pathogenesis of cognitive impairment and dementia in older adults. Cerebral Circulation - Cognition and Behavior, 3, 100148. https://doi.org/10.1016/j.cccb.2022.100148

Aguirre, A., Hilsabeck, R. C., O'Mahar, K., Carberry, K. E., Ayers, G., Bertelson, J., Rousseau, J. F., & Paydarfar, D. (2023). Designing an interprofessional dementia specialty clinic: Conceptualization and evaluation of a patient-centered model. Journal of Interprofessional Care, 37(2), 254-261. https://doi.org/10.1080/13561820.2022.2060194

Alain, C., Du, Y., Bernstein, L. J., Barten, T., & Banai, K. (2018). Listening under difficult conditions: An activation likelihood estimation meta-analysis. Human Brain Mapping, 39(7), 2695-2709. https://doi.org/10.1002/hbm.24031

Albanese, E., Launer, L. J., Egger, M., Prince, M. J., Giannakopoulos, P., Wolters, F. J., & Egan, K. (2017). Body mass index in midlife and dementia: Systematic review and meta-regression analysis of 589,649 men and women followed in longitudinal studies. Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring, 8(1), 165-178. https://doi.org/10.1016/j.dadm.2017.05.007

Albert, M. L., & Mildworf, B. (1989). The concept of dementia. Journal of Neurolinguistics, 4(3-4), 301-308. https://doi.org/10.1016/0911-6044(89)90022-5

AL-Dalaeen, A., & AL-Domi, H. (2022). Does obesity put your brain at risk? Diabetes & Metabolic Syndrome: Clinical Research & Reviews, 16(3), 102444. https://doi.org/10.1016/j.dsx.2022.102444

Allen, N. H., Burns, A., Newton, V., Hickson, F., Ramsden, R., Rogers, J., Butler, S., Thistlewaite, G., & Morris, J. (2003). The effects of improving hearing in dementia. *Age and Ageing*, *32*(2), 189–193. https://doi.org/10.1093/ageing/32.2.189

Alzheimer's Association. (2023). *Medications for memory, cognition and dementia-related behaviors*. Alzheimer's Disease and Dementia. Retrieved April 7, 2023, from https://www.alz.org/alzheimers-dementia/treatments/medications-for-memory

American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders. https://doi.org/10.1176/appi.books.978089042559

American Speech-Language-Hearing Association. (2006). Preferred practice patterns for the profession of audiology [Preferred Practice Patterns]. Available from www.asha.org/policy/ doi:10.1044/policy.PP2006-00274

American Speech-Language-Hearing Association. (2018). Scope of practice in audiology [Scope of Practice]. Available from www.asha.org/policy/ doi:10.1044/policy/SP2018-00353

Amjad, H., Roth, D. L., Sheehan, O. C., Lyketsos, C. G., Wolff, J. L., & Samus, Q. M. (2018). Underdiagnosis of Dementia: an Observational Study of Patterns in Diagnosis and Awareness in US Older Adults. Journal of General Internal Medicine, 33(7), 1131-1138. https://doi.org/10.1007/s11606-018-4377-y

Anwar, U., Arslan, T., Hussain, A., & Lomax, P. (2022). Next Generation Cognition-Aware Hearing Aid Devices With Microwave Sensors: Opportunities and Challenges. IEEE Access, 10, 82214-82235. https://doi.org/10.1109/access.2022.3195875

Arlinger, S., Gatehouse, S., Bentler, R. A., Byrne, D., Cox, R. M., Dirks, D. D., Humes, L., Neuman, A., Ponton, C., Robinson, K., Silman, S., Summerfield, A. Q., Turner, C. W., Tyler, R. S., & Willott, J. F. (1996). Report of the Eriksholm Workshop on Auditory Deprivation and Acclimatization. Ear and Hearing, 17(Sup 1), 87S-98S. https://doi.org/10.1097/00003446-199617031-00009

Armero, O., Crosson, S., Kasten, A., Martin, V., & Spandau, C. (2017). Cognitive Screening Model Expands Health Care Delivery. The Hearing Journal, 70(6), 12,13. https://doi.org/10.1097/01.hj.0000520664.00897.9b

Armstrong, N. M., An, Y., Doshi, J., Erus, G., Ferrucci, L., Davatzikos, C., Deal, J. A., Lin, F. R., & Resnick, S. M. (2019). Association of Midlife Hearing Impairment With Late-Life Temporal Lobe Volume Loss. JAMA Otolaryngology-Head & Neck Surgery, 145(9), 794. https://doi.org/10.1001/jamaoto.2019.1610

Atri, A. (2019). The Alzheimer's Disease Clinical Spectrum. Medical Clinics of North America, 103(2), 263-293. https://doi.org/10.1016/j.mcna.2018.10.009

Azeem, A., Julleekeea, A., Knight, B., Sohail, I., Bruyns-Haylett, M., & Sastre, M. (2023). Hearing loss and its link to cognitive impairment and dementia. Frontiers in Dementia, 2. https://doi.org/10.3389/frdem.2023.1199319

Babajanian, E. E., & Gurgel, R. K. (2022). Cognitive and behavioral effects of hearing loss. *Current Opinion in Otolaryngology & Head &Neck Surgery*, *30*(5), 339–343. https://doi.org/10.1097/moo.0000000000000825

Bacigalupo, I., Mayer, F., Lacorte, E., Di Pucchio, A., Marzolini, F., Canevelli, M., Di Fiandra, T., & Vanacore, N. (2018). A systematic review and meta-analysis on the prevalence of dementia in Europe: Estimates from the highest-quality studies adopting the DSM IV diagnostic criteria. Journal of Alzheimer's Disease, 66(4), 1471–1481. https://doi.org/10.3233/jad-180416

Bahar-Fuchs, A., Clare, L., & Woods, B. (2013). Cognitive training and cognitive rehabilitation for mild to moderate alzheimer's disease and vascular dementia. Cochrane Database of Systematic Reviews. https://doi.org/10.1002/14651858.cd003260.pub2

Baines, S., Saxby, P., & Ehlert, K. (1987). Reality orientation and reminiscence therapy. British Journal of Psychiatry, 151(2), 222–231. https://doi.org/10.1192/bjp.151.2.222

Baltes, P. B., & Lindenberger, U. (1997). Emergence of a powerful connection between sensory and cognitive functions across the adult life span: A new window to the study of cognitive aging? *Psychology and Aging*, *12*(1), 12–21. https://doi.org/10.1037/0882-7974.12.1.12

Barbiellini Amidei, C., Fayosse, A., Dumurgier, J., Machado-Fragua, M. D., Tabak, A. G., van Sloten, T., Kivimäki, M., Dugravot, A., Sabia, S., & Singh-Manoux, A. (2021). Association between age at diabetes onset and subsequent risk of dementia. JAMA, 325(16), 1640. https://doi.org/10.1001/jama.2021.4001

Beck, D. L., Bant, S., & Clarke, N. A. (2020). Hearing loss and Cognition: A Discussion for Audiologists and Hearing Healthcare Professionals. *Journal of Otolaryngology-ENT Research*, *12*(3), 72–78. https://doi.org/10.15406/joentr.2020.12.00459

Beck, D. L., & Clark, J. L. (2009). audition matters more as cognition declines: Cognition matters more as audition declines. Audiology Today, 21(2), 48.

Beck, D. L., & Grisel, J. J. (2022). Cognitive screenings in otolaryngology? The time has come. Journal of Otolaryngology ENT Research, 14(1), 56–60. https://doi.org/10.15406/joentr.14.1

Beck, D. L., & Harvey, M. (2021). Issues in cognition, audiology, and amplification. *Hearing Review*, *28*(1), 28–33.

Beck, D. L., Weinstein, B. E., & Harvey, M. (2016). Issues in cognitive screenings by audiologists. Hearing Review, 23(2), 36-42.

Beck, D. L., Weinstein, B. E., & Harvey, M. A. (2018). *Dementia screening: A role for audiologists*. The Hearing Review. https://hearingreview.com/inside-hearing/research/dementia-screening-role-audiologists

Bennett, D. A., Buchman, A. S., Boyle, P. A., Barnes, L. L., Wilson, R. S., & Schneider, J. A. (2018). Religious orders study and rush memory and aging project. Journal of Alzheimer's Disease, 64(s1). https://doi.org/10.3233/jad-179939

Bennett, S., & Thomas, A. J. (2014). Depression and dementia: Cause, consequence or coincidence? *Maturitas*, *79*(2), 184–190. https://doi.org/10.1016/j.maturitas.2014.05.009

Bir, S. C., Khan, M. W., Javalkar, V., Toledo, E. G., & Kelley, R. E. (2021). Emerging concepts in vascular dementia: A Review. Journal of Stroke and Cerebrovascular Diseases, 30(8), 105864. https://doi.org/10.1016/j.jstrokecerebrovasdis.2021.105864

Bisogno, A., Scarpa, A., Di Girolamo, S., De Luca, P., Cassandro, C., Viola, P., Ricciardiello, F., Greco, A., De Vincentiis, M., Ralli, M., & Di Stadio, A. (2021). Hearing loss and cognitive impairment: Epidemiology, common pathophysiological findings, and treatment considerations. *Life*, *11*(10), 1102. https://doi.org/10.3390/life11101102

Bloom, D. E., Khoury, A., Algur, E., & Sevilla, J. P. (2020). Valuing productive non-market activities of older adults in Europe and the US. *De Economist*, *168*(2), 153–181. https://doi.org/10.1007/s10645-020-09362-1

Blustein, J., Weinstein, B. E., & Chodosh, J. (2023). It is time to change our message about hearing loss and dementia. *Journal of the American Geriatrics Society*. https://doi.org/10.1111/jgs.18323

Boller, F., & Forbes, M. M. (1998). History of dementia and dementia in history: An overview. Journal of the Neurological Sciences, 158(2), 125–133. https://doi.org/10.1016/s0022-510x(98)00128-2

Bott, A., Meyer, C., Hickson, L., & Pachana, N. A. (2020). "it's huge, in a way." conflicting stakeholder priorities for managing hearing impairment for people living with dementia in residential aged care facilities. *Clinical Gerontologist*, *45*(4), 844–858. https://doi.org/10.1080/07317115.2020.1805537

Bowl, M. R., & Dawson, S. J. (2018). Age-related hearing loss. *Cold Spring Harbor Perspectives in Medicine*, *9*(8). https://doi.org/10.1101/cshperspect.a033217

Brenowitz, W. D., Hubbard, R. A., Keene, C. D., Hawes, S. E., Longstreth, W. T., Woltjer, R. L., & Kukull, W. A. (2017). Mixed neuropathologies and associations with domain-specific cognitive decline. *Neurology*, *89*(17), 1773–1781. https://doi.org/10.1212/wnl.0000000000004567

Brett, B. L., Gardner, R. C., Godbout, J., Dams-O'Connor, K., & Keene, C. D. (2022). Traumatic brain injury and risk of Neurodegenerative Disorder. Biological Psychiatry, 91(5), 498–507. https://doi.org/10.1016/j.biopsych.2021.05.025

Broome, E. E., Tannirandorn, P., Straus, J., Beale, P., Heffernan, E., Dening, T., & Henshaw, H. (2023). Patient perceptions of cognitive screening in Adult Audiology Services: A qualitative exploration. Frontiers in Neurology, 14. https://doi.org/10.3389/fneur.2023.1143128

Buccellato, F. R., D'Anca, M., Tartaglia, G. M., Del Fabbro, M., Scarpini, E., & Galimberti, D. (2023). Treatment of alzheimer's disease: Beyond symptomatic therapies. International Journal of Molecular Sciences, 24(18), 13900. https://doi.org/10.3390/ijms241813900

Burkhalter, C. L., Allen, R. S., Skaar, D. C., Crittenden, J., & Burgio, L. D. (2009). Examining the effectiveness of traditional audiological assessments for nursing home residents with dementia-related behaviors. *Journal of the American Academy of Audiology*, *20*(09), 529–538. https://doi.org/10.3766/jaaa.20.9.2

Cacioppo, J. T., & Hawkley, L. C. (2009). Perceived social isolation and cognition. *Trends in Cognitive Sciences*, *13*(10), 447–454. https://doi.org/10.1016/j.tics.2009.06.005

Cafferata, R. M., Hicks, B., & von Bastian, C. C. (2021). Effectiveness of cognitive stimulation for dementia: A systematic review and meta-analysis. Psychological Bulletin, 147(5), 455–476. https://doi.org/10.1037/bul0000325

Cahn-Hidalgo, D., Estes, P. W., & Benabou, R. (2020). Validity, reliability, and psychometric properties of a computerized, cognitive assessment test (Cognivue®). *World Journal of Psychiatry*, *10*(1), 1–11. https://doi.org/10.5498/wjp.v10.i1.1

Cai, W., Wu, T., & Chen, N. (2023). The amyloid-beta clearance: From molecular targets to glial and neural cells. *Biomolecules*, *13*(2), 313. https://doi.org/10.3390/biom13020313

Cammisuli, D. M., Danti, S., Bosinelli, F., & Cipriani, G. (2016). Non-pharmacological interventions for people with alzheimer's disease: A critical review of the scientific

literature from the last ten years. European Geriatric Medicine, 7(1), 57–64. https://doi.org/10.1016/j.eurger.2016.01.002

Campbell, J., & Sharma, A. (2013). Compensatory changes in cortical resource allocation in adults with hearing loss. *Frontiers in Systems Neuroscience*, *7*. https://doi.org/10.3389/fnsys.2013.00071

Cao, Q., Tan, C.-C., Xu, W., Hu, H., Cao, X.-P., Dong, Q., Tan, L., & Yu, J.-T. (2020). The prevalence of dementia: A systematic review and meta-analysis. *Journal of Alzheimer's Disease*, *73*(3), 1157–1166. https://doi.org/10.3233/jad-191092

Cardon, G., & Sharma, A. (2018). Somatosensory cross-modal reorganization in adults with age-related, early-stage hearing loss. *Frontiers in Human Neuroscience*, *12*. https://doi.org/10.3389/fnhum.2018.00172

Carlos, A. F., & Josephs, K. A. (2023). The role of Clinical Assessment in the era of biomarkers. Neurotherapeutics, 20(4), 1001–1018. https://doi.org/10.1007/s13311-023-01410-3

Carnero-Pardo, C., Rego-García, I., Barrios-López, J. M., Blanco-Madera, S., Calle-Calle, R., López-Alcalde, S., & Vílchez-Carrillo, R. M. (2022). Assessment of the diagnostic accuracy and discriminative validity of the clock drawing and mini-cog tests in detecting cognitive impairment. *Neurología (English Edition)*, *37*(1), 13–20. https://doi.org/10.1016/j.nrleng.2018.12.022

Carrion, C., Aymerich, M., Baillés, E., & López-Bermejo, A. (2013). Cognitive psychosocial intervention in dementia: A systematic review. Dementia and Geriatric Cognitive Disorders, 36(5–6), 363–375. https://doi.org/10.1159/000354365

Carson, N., Leach, L., & Murphy, K. J. (2017). A re-examination of Montreal Cognitive Assessment (MOCA) cutoff scores. *International Journal of Geriatric Psychiatry*, *33*(2), 379–388. https://doi.org/10.1002/gps.4756

Castells-Sánchez, A., Roig-Coll, F., Lamonja-Vicente, N., Altés-Magret, M., Torán-Monserrat, P., Via, M., García-Molina, A., Tormos, J. M., Heras, A., Alzamora, M. T., Forés, R., Pera, G., Dacosta-Aguayo, R., Soriano-Raya, J. J., Cáceres, C., Montero-Alía, P., Montero-Alía, J. J., Jimenez-Gonzalez, M. M., Hernández-Pérez, M., … Mataró, M. (2019). Effects and mechanisms of cognitive, aerobic exercise, and combined training on cognition, health, and brain outcomes in physically inactive older adults: The Projecte Moviment protocol. *Frontiers in Aging Neuroscience*, *11*. https://doi.org/10.3389/fnagi.2019.00216

Castiglione, A., Benatti, A., Velardita, C., Favaro, D., Padoan, E., Severi, D., Pagliaro, M., Bovo, R., Vallesi, A., Gabelli, C., & Martini, A. (2016). Aging, cognitive decline and hearing loss: Effects of Auditory Rehabilitation and training with hearing aids and cochlear implants on cognitive function and depression among older adults. *Audiology and Neurotology*, *21*(Suppl. 1), 21–28. https://doi.org/10.1159/000448350

Chadha, S., Cieza, A., & Krug, E. (2018). Global Hearing Health: Future Directions. *Bulletin of the World Health Organization*, *96*(3), 146–146. https://doi.org/10.2471/blt.18.209767

Chang, C.-C. H., Zhao, Y., Lee, C.-W., & Ganguli, M. (2012). Smoking, death, and alzheimer disease. *Alzheimer Disease & Associated Disorders*, *26*(4), 300–306. https://doi.org/10.1097/wad.0b013e3182420b6e

Chatterjee, S., Peters, S. A. E., Woodward, M., Mejia Arango, S., Batty, G. D., Beckett, N., Beiser, A., Borenstein, A. R., Crane, P. K., Haan, M., Hassing, L. B., Hayden, K. M., Kiyohara, Y., Larson, E. B., Li, C.-Y., Ninomiya, T., Ohara, T., Peters, R., Russ, T. C., …

Huxley, R. R. (2015). Type 2 diabetes as a risk factor for dementia in women compared with men: A pooled analysis of 2.3 million people comprising more than 100,000 cases of dementia. *Diabetes Care*, *39*(2), 300–307. https://doi.org/10.2337/dc15-1588

Cherbuin, N., Kim, S., & Anstey, K. J. (2015). Dementia risk estimates associated with measures of depression: A systematic review and meta-analysis. *BMJ Open*, *5*(12). https://doi.org/10.1136/bmjopen-2015-008853

Cherko, M., Hickson, L., & Bhutta, M. (2016). Auditory deprivation and health in the elderly. *Maturitas*, *88*, 52–57. https://doi.org/10.1016/j.maturitas.2016.03.008

Chern, A., & Golub, J. S. (2019). Age-related hearing loss and dementia. *Alzheimer Disease & Associated Disorders*, *33*(3), 285–290. https://doi.org/10.1097/wad.0000000000000325

Chimakurthy, A. K., Lingam, S., Pasya, S. K., & Copeland, B. J. (2023). A systematic review of dietary supplements in alzheimer's disease. *Cureus*. https://doi.org/10.7759/cureus.33982

Chin, K. S., Teodorczuk, A., & Watson, R. (2019). Dementia with lewy bodies: Challenges in the diagnosis and management. Australian & New Zealand Journal of Psychiatry, 53(4), 291–303. https://doi.org/10.1177/0004867419835029

Chiu, H.-Y., Chen, P.-Y., Chen, Y.-T., & Huang, H.-C. (2018). Reality orientation therapy benefits cognition in older people with dementia: A meta-analysis. International Journal of Nursing Studies, 86, 20–28. https://doi.org/10.1016/j.ijnurstu.2018.06.008

Choi, D., Choi, S., & Park, S. M. (2018). Effect of smoking cessation on the risk of dementia: A longitudinal study. *Annals of Clinical and Translational Neurology*, *5*(10), 1192–1199. https://doi.org/10.1002/acn3.633

Cholerton, B., Baker, L. D., Montine, T. J., & Craft, S. (2016). Type 2 diabetes, cognition, and dementia in older adults: Toward a precision health approach. *Diabetes Spectrum*, *29*(4), 210–219. https://doi.org/10.2337/ds16-0041

Chowdhary, N., Barbui, C., Anstey, K. J., Kivipelto, M., Barbera, M., Peters, R., Zheng, L., Kulmala, J., Stephen, R., Ferri, C. P., Joanette, Y., Wang, H., Comas-Herrera, A., Alessi, C., Suharya, K., Mwangi, K. J., Petersen, R. C., Motala, A. A., Mendis, S., … Dua, T. (2022). Reducing the Risk of Cognitive Decline and Dementia: WHO Recommendations. Frontiers in Neurology, 12. https://doi.org/10.3389/fneur.2021.765584

Clare, L., Kudlicka, A., Oyebode, J. R., Jones, R. W., Bayer, A., Leroi, I., Kopelman, M., James, I. A., Culverwell, A., Pool, J., Brand, A., Henderson, C., Hoare, Z., Knapp, M., & Woods, B. (2019). Individual goal-oriented cognitive rehabilitation to improve everyday functioning for people with early-stage dementia: A multicentre randomised controlled trial (The great trial). International Journal of Geriatric Psychiatry, 34(5), 709–721. https://doi.org/10.1002/gps.5076

Cleveland Clinic. (2022, March 12). *Dementia: Symptoms, types, causes, treatment & risk factors*. Cleveland Clinic. Retrieved April 2, 2023, from https://my.clevelandclinic.org/health/diseases/9170-dementia

Connolly, A., Gaehl, E., Martin, H., Morris, J., & Purandare, N. (2011). Underdiagnosis of dementia in primary care: Variations in the observed prevalence and comparisons to the expected prevalence. *Aging & Mental Health*, *15*(8), 978–984. https://doi.org/10.1080/13607863.2011.596805

Corder, E. H., Saunders, A. M., Strittmatter, W. J., Schmechel, D. E., Gaskell, P. C., Small, G. W., Roses, A. D., Haines, J. L., & Pericak-Vance, M. A. (1993). Gene Dose of

Apolipoprotein E Type 4 Allele and the Risk of Alzheimer's Disease in Late Onset Families. Science, 261(5123), 921-923. https://doi.org/10.1126/science.8346443

Cotelli, M., Manenti, R., & Zanetti, O. (2012). Reminiscence therapy in dementia: A Review. Maturitas, 72(3), 203–205. https://doi.org/10.1016/j.maturitas.2012.04.008

Cummings, J. (2021). The Role of Neuropsychiatric Symptoms in Research Diagnostic Criteria for Neurodegenerative Diseases. The American Journal of Geriatric Psychiatry, 29(4), 375-383. https://doi.org/10.1016/j.jagp.2020.07.011

Cummings, J. (2023). Anti-Amyloid Monoclonal Antibodies are Transformative Treatments that Redefine Alzheimer's Disease Therapeutics. Drugs, 83(7), 569-576. https://doi.org/10.1007/s40265-023-01858-9

Cummings, J., Apostolova, L., Rabinovici, G. D., Atri, A., Aisen, P., Greenberg, S., Hendrix, S., Selkoe, D., Weiner, M., Petersen, R. C., & Salloway, S. (2023). Lecanemab: Appropriate Use Recommendations. The Journal of Prevention of Alzheimer's Disease. https://doi.org/10.14283/jpad.2023.30

Cummings, J. L., Isaacson, R. S., Schmitt, F. A., & Velting, D. M. (2015). A practical algorithm for managing alzheimer's disease: What, when, and why? *Annals of Clinical and Translational Neurology*, *2*(3), 307–323. https://doi.org/10.1002/acn3.166

Cunningham, L. L., & Tucci, D. L. (2017). Hearing loss in adults. *New England Journal of Medicine*, *377*(25), 2465–2473. https://doi.org/10.1056/nejmra1616601

Custodio, N., Montesinos, R., Lira, D., Herrera-Pérez, E., Bardales, Y., & Valeriano-Lorenzo, L. (2017). Mixed dementia: A review of the evidence. Dementia & Neuropsychologia, 11(4), 364–370. https://doi.org/10.1590/1980-57642016dn11-040005

Cutajar, K., & Tabone, N. (2022). Hearing Loss and cognition: the perspective of healthcare professionals. Malta Journal of Health Sciences, 9(2), 7-22.

Davis, A., McMahon, C. M., Pichora-Fuller, K. M., Russ, S., Lin, F., Olusanya, B. O., Chadha, S., & Tremblay, K. L. (2016). Aging and hearing health: The life-course approach. *The Gerontologist*, *56*(Suppl 2). https://doi.org/10.1093/geront/gnw033

Davis, A. C., & Hoffman, H. J. (2019). Hearing loss: Rising prevalence and impact. *Bulletin of the World Health Organization*, *97*(10). https://doi.org/10.2471/blt.19.224683

Davis, J. (2021). Cognitive Screening in Audiology: Considerations for Nonverbal Instructions. The Hearing Journal, 74(5), 34,35. https://doi.org/10.1097/01.hj.0000752316.88088.84

Dawes, P., Emsley, R., Cruickshanks, K. J., Moore, D. R., Fortnum, H., Edmondson-Jones, M., McCormack, A., & Munro, K. J. (2015). Hearing loss and cognition: The role of hearing aids, social isolation and depression. *PLOS ONE*, *10*(3). https://doi.org/10.1371/journal.pone.0119616

Dawes, P., Littlejohn, J., Bott, A., Brennan, S., Burrow, S., Hopper, T., & Scanlan, E. (2021). Hearing assessment and rehabilitation for people living with dementia. *Ear & Hearing*, *43*(4), 1089–1102. https://doi.org/10.1097/aud.0000000000001174

Dawes, P., Robinson, G., & Leroi, I. (2023). Screening for Dementia in Audiology Clinics - Advantages, Pitfalls, and Recommendations. The Hearing Journal, 76(06), 26,27. https://doi.org/10.1097/01.hj.0000938624.32004.6d

Dean, D. C., Hurley, S. A., Kecskemeti, S. R., O'Grady, J. P., Canda, C., Davenport-Sis, N. J., Carlsson, C. M., Zetterberg, H., Blennow, K., Asthana, S., Sager, M. A., Johnson, S. C., Alexander, A. L., & Bendlin, B. B. (2016). Association of amyloid pathology with myelin

alteration in preclinical alzheimer disease. JAMA Neurology, 74(1), 41. https://doi.org/10.1001/jamaneurol.2016.3232

Deardorff, W. J., & Grossberg, G. T. (2019). Behavioral and psychological symptoms in alzheimer's dementia and vascular dementia. *Psychopharmacology of Neurologic Disease*, 5–32. https://doi.org/10.1016/b978-0-444-64012-3.00002-2

de Bruijn, R. F., Bos, M. J., Portegies, M. L., Hofman, A., Franco, O. H., Koudstaal, P. J., & Ikram, M. A. (2015). The potential for prevention of dementia across two decades: The prospective, population-based Rotterdam study. BMC Medicine, 13(1). https://doi.org/10.1186/s12916-015-0377-5

Decourt, B., Noorda, K., Noorda, K., Shi, J., & Sabbagh, M. N. (2022). Review of Advanced Drug Trials focusing on the reduction of brain beta-amyloid to prevent and treat dementia. Journal of Experimental Pharmacology, Volume 14, 331–352. https://doi.org/10.2147/jep.s265626

Delgado-Saborit, J. M., Guercio, V., Gowers, A. M., Shaddick, G., Fox, N. C., & Love, S. (2021). A critical review of the epidemiological evidence of effects of air pollution on dementia, cognitive function and cognitive decline in adult population. *Science of The Total Environment*, *757*, 143734. https://doi.org/10.1016/j.scitotenv.2020.143734

Del Vecchio, V., Tricarico, L., Pisani, A., Serra, N., D'Errico, D., De Corso, E., Rea, T., Picciotti, P. M., Laria, C., Manna, G., Franzè, A., Malesci, R., & Fetoni, A. R. (2023). Vascular factors in patients with midlife sensorineural hearing loss and the progression to mild cognitive impairment. *Medicina*, *59*(3), 481. https://doi.org/10.3390/medicina59030481

de Vugt, M. E., & Verhey, F. R. J. (2013). The impact of early dementia diagnosis and intervention on informal caregivers. *Progress in Neurobiology*, *110*, 54–62. https://doi.org/10.1016/j.pneurobio.2013.04.005

Dillard, L. K., Arunda, M. O., Lopez-Perez, L., Martinez, R. X., Jiménez, L., & Chadha, S. (2022). Prevalence and global estimates of unsafe listening practices in adolescents and young adults: A systematic review and meta-analysis. *BMJ Global Health*, *7*(11). https://doi.org/10.1136/bmjgh-2022-010501

Dumurgier, J., & Tzourio, C. (2020). Epidemiology of Neurological Diseases in older adults. Revue Neurologique, 176(9), 642–648. https://doi.org/10.1016/j.neurol.2020.01.356

Dupuis, K., Pichora-Fuller, M. K., Chasteen, A. L., Marchuk, V., Singh, G., & Smith, S. L. (2015). Effects of hearing and vision impairments on the Montreal Cognitive Assessment. *Aging, Neuropsychology, and Cognition*, *22*(4), 413–437. https://doi.org/10.1080/13825585.2014.968084

Durazzo, T. C., Mattsson, N., & Weiner, M. W. (2014). Smoking and increased alzheimer's disease risk: A review of potential mechanisms. *Alzheimer's & Dementia*, *10*(3S). https://doi.org/10.1016/j.jalz.2014.04.009

Durazzo, T. C., Meyerhoff, D. J., & Nixon, S. J. (2010). Chronic cigarette smoking: Implications for neurocognition and Brain Neurobiology. *International Journal of Environmental Research and Public Health*, *7*(10), 3760–3791. https://doi.org/10.3390/ijerph7103760

Ebrahimi, K., Jourkesh, M., Sadigh-Eteghad, S., Stannard, S. R., Earnest, C. P., Ramsbottom, R., Antonio, J., & Navin, K. H. (2020). Effects of physical activity on brain energy biomarkers in alzheimer's diseases. *Diseases*, *8*(2), 18. https://doi.org/10.3390/diseases8020018

Eckert, M. A., Vaden, K. I., & Dubno, J. R. (2019). Age-related hearing loss associations with changes in Brain Morphology. *Trends in Hearing*, *23*. https://doi.org/10.1177/2331216519857267

Eid, A., Mhatre, I., & Richardson, J. R. (2019). Gene-environment interactions in alzheimer's disease: A potential path to precision medicine. Pharmacology & Therapeutics, 199, 173–187. https://doi.org/10.1016/j.pharmthera.2019.03.005

El-Hayek, Y. H., Wiley, R. E., Khoury, C. P., Daya, R. P., Ballard, C., Evans, A. R., Karran, M., Molinuevo, J. L., Norton, M., & Atri, A. (2019). Tip of the iceberg: Assessing the global socioeconomic costs of alzheimer's disease and related Dementias and strategic implications for stakeholders. *Journal of Alzheimer's Disease*, *70*(2), 323–341. https://doi.org/10.3233/jad-190426

Erkkinen, M. G., Kim, M.-O., & Geschwind, M. D. (2017). Clinical neurology and epidemiology of the major neurodegenerative diseases. Cold Spring Harbor Perspectives in Biology, 10(4). https://doi.org/10.1101/cshperspect.a033118

Erwin, D. E., & Chen, P. (2023, March). *Hearing loss in the elderly*. StatPearls - NCBI Bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK580566/

Fitzpatrick, A., & Belza, B. (2021). A systems-wide approach for early detection and management of dementia in primary care. Innovation in Aging, 5(Supplement_1), 344–344. https://doi.org/10.1093/geroni/igab046.1335

Flanders, S. A. (2018). Effective Patient Education: Evidence and Common Sense. Medsurg Nursing, 27(1), 55-58.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "mini-mental state." *Journal of Psychiatric Research*, *12*(3), 189–198. https://doi.org/10.1016/0022-3956(75)90026-6

Ford, A. H., Hankey, G. J., Yeap, B. B., Golledge, J., Flicker, L., & Almeida, O. P. (2018). Hearing loss and the risk of dementia in later life. Maturitas, 112, 1–11. https://doi.org/10.1016/j.maturitas.2018.03.004

Forrest, S. L., & Kovacs, G. G. (2022). Current concepts of mixed pathologies in neurodegenerative diseases. Canadian Journal of Neurological Sciences, 50(3), 329–345. https://doi.org/10.1017/cjn.2022.34

Fortunato, S., Forli, F., Guglielmi, V., De Corso, E., Paludetti, G., Berrettini, S., & FetonI, A. R. (2016). A review of new insights on the association between hearing loss and cognitive decline in ageing. Acta Otorhinolaryngologica Italica, 36(3), 155–166. https://doi.org/10.14639/0392-100x-993

Frahm-Falkenberg, S., Ibsen, R., Kjellberg, J., & Jennum, P. (2016). Health, social and economic consequences of Dementias: A comparative national cohort study. European Journal of Neurology, 23(9), 1400–1407. https://doi.org/10.1111/ene.13043

Freedman, V. A., Kasper, J. D., Spillman, B. C., & Plassman, B. L. (2018). Short-term changes in the prevalence of probable dementia: An analysis of the 2011–2015 national health and aging trends study. The Journals of Gerontology: Series B, 73(suppl_1). https://doi.org/10.1093/geronb/gbx144

Gaeta, L., Azzarello, J., Baldwin, J., Ciro, C., Hudson, M. A., Johnson, C. E., & John, A. B. (2022). The impact of amplification on cognitive screening test scores. *Journal of Gerontological Nursing*, *48*(7), 31–37. https://doi.org/10.3928/00989134-20220606-05

Gaeta, L., & John, A. (2021). Hearing loss and cognitive screening. *Research in Gerontological Nursing*, *14*(4), 170–172. https://doi.org/10.3928/19404921-20210617-01

Ganguli, M., Blacker, D., Blazer, D. G., Grant, I., Jeste, D. V., Paulsen, J. S., Petersen, R. C., & Sachdev, P. S. (2011). Classification of neurocognitive disorders in DSM-5: A WORK IN PROGRESS. The American Journal of Geriatric Psychiatry, 19(3), 205–210. https://doi.org/10.1097/jgp.0b013e3182051ab4

Gardner, R. C., Burke, J. F., Nettiksimmons, J., Kaup, A., Barnes, D. E., & Yaffe, K. (2014). Dementia risk after traumatic brain injury vs Nonbrain Trauma. *JAMA Neurology*, *71*(12), 1490. https://doi.org/10.1001/jamaneurol.2014.2668

Garnier-Crussard, A., Vernaudon, J., Auguste, N., Moutet, C., Dauphinot, V., & Krolak-Salmon, P. (2019). Perception of benefits and risks of neurocognitive disorders diagnosis: A French national survey. *Journal of Alzheimer's Disease*, *67*(4), 1267–1275. https://doi.org/10.3233/jad-180403

Gatz, M., Reynolds, C. A., Fratiglioni, L., Johansson, B., Mortimer, J. A., Berg, S., Fiske, A., & Pedersen, N. L. (2006). Role of genes and environments for explaining alzheimer disease. Archives of General Psychiatry, 63(2), 168. https://doi.org/10.1001/archpsyc.63.2.168

Gauthier, S., Rosa-Neto, P., Morais, J. A., & Webster, C. (2021). (publication). World Alzheimer Report 2021 *Journey through the diagnosis of dementia*. London, England: Alzheimer's Disease International.

Geldmacher, D. S., & Kerwin, D. R. (2013). Practical diagnosis and management of dementia due to alzheimer's disease in the Primary Care Setting. *The Primary Care Companion For CNS Disorders*. https://doi.org/10.4088/pcc.12r01474

Giles, T. D., Materson, B. J., Cohn, J. N., & Kostis, J. B. (2009). Definition and classification of hypertension: An update. *The Journal of Clinical Hypertension*, *11*(11), 611–614. https://doi.org/10.1111/j.1751-7176.2009.00179.x

Giroud, N., Lemke, U., Reich, P., Matthes, K. L., & Meyer, M. (2017). The impact of hearing aids and age-related hearing loss on auditory plasticity across three months – an electrical neuroimaging study. *Hearing Research*, *353*, 162–175. https://doi.org/10.1016/j.heares.2017.06.012

Glick, H. A., & Sharma, A. (2020). Cortical neuroplasticity and cognitive function in early-stage, mild-moderate hearing loss: Evidence of neurocognitive benefit from hearing aid use. *Frontiers in Neuroscience*, *14*. https://doi.org/10.3389/fnins.2020.00093

Glynn-Servedio, B. E., & Ranola, T. S. (2017). Ache inhibitors and NMDA receptor antagonists in advanced alzheimer's disease. *The Consultant Pharmacist*, *32*(9), 511–518. https://doi.org/10.4140/tcp.n.2017.511

Gold, M., Lightfoot, L. A., & Hnath-Chisolm, T. (1996). Hearing loss in a memory disorders clinic. *Archives of Neurology*, *53*(9), 922. https://doi.org/10.1001/archneur.1996.00550090134019

Gottschalk, K., & Olson, A. (2020). Cognition and Cognitive Screeners in Audiological Management. Perspectives of the ASHA Special Interest Groups, 5(6), 1716-1721. https://doi.org/10.1044/2020_persp-20-00015

Gow, A. J., Corley, J., Starr, J. M., & Deary, I. J. (2012). Reverse causation in activity-cognitive ability associations: The Lothian Birth Cohort 1936. Psychology and Aging, 27(1), 250–255. https://doi.org/10.1037/a0024144

Grand, J. H., Caspar, S., & MacDonald, S. W. (2011). Clinical features and multidisciplinary approaches to dementia care. Journal of Multidisciplinary Healthcare, 4, 125–147. https://doi.org/10.2147/jmdh.s17773

Griffiths, T. D., Lad, M., Kumar, S., Holmes, E., McMurray, B., Maguire, E. A., Billig, A. J., & Sedley, W. (2020). How can hearing loss cause dementia? *Neuron*, *108*(3), 401–412. https://doi.org/10.1016/j.neuron.2020.08.003

Gu, D., Ou, S., & Liu, G. (2021). Traumatic brain injury and risk of dementia and alzheimer's disease: A systematic review and meta-analysis. *Neuroepidemiology*, *56*(1), 4–16. https://doi.org/10.1159/000520966

Guerreiro, M. J. S., & Van Gerven, P. W. M. (2017). Disregarding hearing loss leads to overestimation of age-related cognitive decline. *Neurobiology of Aging*, *56*, 180–189. https://doi.org/10.1016/j.neurobiolaging.2017.05.001

Haake, A., Nguyen, K., Friedman, L., Chakkamparambil, B., & Grossberg, G. T. (2020). An update on the utility and safety of cholinesterase inhibitors for the treatment of alzheimer's disease. Expert Opinion on Drug Safety, 19(2), 147–157. https://doi.org/10.1080/14740338.2020.1721456

Haddad, H. W., Malone, G. W., Comardelle, N. J., Degueure, A. E., Kaye, A. M., & Kaye, A. D. (2022). Aducanumab, a novel anti-amyloid monoclonal antibody, for the treatment of alzheimer's disease: A comprehensive review. *Health Psychology Research*, *10*(1). https://doi.org/10.52965/001c.31925

Haile, L. M., Kamenov, K., Briant, P. S., Orji, A. U., Steinmetz, J. D., Abdoli, A., Abdollahi, M., Abu-Gharbieh, E., Afshin, A., Ahmed, H., Ahmed Rashid, T., Akalu, Y., Alahdab, F., Alanezi, F. M., Alanzi, T. M., Al Hamad, H., Ali, L., Alipour, V., Al-Raddadi, R. M., … Chadha, S. (2021). Hearing loss prevalence and years lived with disability, 1990–2019: Findings from the global burden of disease study 2019. *The Lancet*, *397*(10278), 996–1009. https://doi.org/10.1016/s0140-6736(21)00516-x

Hardy, C. J., Marshall, C. R., Golden, H. L., Clark, C. N., Mummery, C. J., Griffiths, T. D., Bamiou, D.-E., & Warren, J. D. (2016). Hearing and dementia. *Journal of Neurology*, *263*(11), 2339–2354. https://doi.org/10.1007/s00415-016-8208-y

Hardy, J., & Selkoe, D. J. (2002). *The amyloid hypothesis of alzheimer's disease: Progress and problems on the road to therapeutics.* Science, 297(5580), 353–356. https://doi.org/10.1126/science.1072994

Harrison, K. L., Hunt, L. J., Ritchie, C. S., & Yaffe, K. (2019). Dying with dementia: Underrecognized and stigmatized. Journal of the American Geriatrics Society, 67(8), 1548–1551. https://doi.org/10.1111/jgs.15895

Hendriks, S., Peetoom, K., Bakker, C., van der Flier, W. M., Papma, J. M., Koopmans, R., Verhey, F. R., de Vugt, M., Köhler, S., Withall, A., Parlevliet, J. L., Uysal-Bozkir, Ö., Gibson, R. C., Neita, S. M., Nielsen, T. R., Salem, L. C., Nyberg, J., Lopes, M. A., Dominguez, J. C., … Ruano, L. (2021). Global prevalence of young-onset dementia. JAMA Neurology, 78(9), 1080. https://doi.org/10.1001/jamaneurol.2021.2161

Herr, M., Ankri, J., Diard, C., & Hiance-Delahaye, A. (2021). Removal of drugs for alzheimer's disease from the list of reimbursable drugs in France: Analysis of change in drug use, disease management and cognition using the National Alzheimer Data Bank (BNA). *Drugs & Aging*, *38*(1), 63–74. https://doi.org/10.1007/s40266-020-00817-3

Hilsabeck, R. C., Lacritz, L. H., Colvin, M. K., Espe-Pfeifer, P., Sperling, S. A., Arnett, P. A., & Perry, W. (2022). Cognition is a critical vital sign for older adults: The need for early detection of cognitive impairment is now. *Policy Insights from the Behavioral and Brain Sciences*, *9*(2), 188–195. https://doi.org/10.1177/23727322221110261

Hirczy, S., & Salinas, M. (2022). Clinical presentation, diagnosis, and pathogenesis of dementia with Lewy Bodies. *Psychiatric Annals*, *52*(10), 398–403. https://doi.org/10.3928/00485713-20220907-01

Hopper, T., & Hinton, P. (2012). Hearing Loss among Individuals with Dementia: Barriers and Facilitators to Care. Canadian Journal of Speech-Language Pathology & Audiology, 36(4).

Horgan, D., Nobili, F., Teunissen, C., Grimmer, T., Mitrecic, D., Ris, L., Pirtosek, Z., Bernini, C., Federico, A., Blackburn, D., Logroscino, G., & Scarmeas, N. (2020). Biomarker testing: Piercing the fog of alzheimer's and related dementia. Biomedicine Hub, 5(3), 1–22. https://doi.org/10.1159/000511233

Huang, H.-C., Chen, Y.-T., Chen, P.-Y., Huey-Lan Hu, S., Liu, F., Kuo, Y.-L., & Chiu, H.-Y. (2015). Reminiscence therapy improves cognitive functions and reduces depressive symptoms in elderly people with dementia: A meta-analysis of randomized controlled trials. Journal of the American Medical Directors Association, 16(12), 1087–1094. https://doi.org/10.1016/j.jamda.2015.07.010

Huang, L.-K., Kuan, Y.-C., Lin, H.-W., & Hu, C.-J. (2023). Clinical trials of new drugs for alzheimer disease: A 2020–2023 update. Journal of Biomedical Science, 30(1). https://doi.org/10.1186/s12929-023-00976-6

Huang, Q., & Tang, J. (2010). Age-related hearing loss or presbycusis. *European Archives of Oto-Rhino-Laryngology*, *267*(8), 1179–1191. https://doi.org/10.1007/s00405-010-1270-7

Hubbard, H. I., Mamo, S. K., & Hopper, T. (2018). Dementia and Hearing Loss: Interrelationships and Treatment Considerations. Seminars in speech and language, 39(3), 197–210. https://doi.org/10.1055/s-0038-1660779

Hussain, M., Kumar, P., Khan, S., Gordon, D. K., & Khan, S. (2020). Similarities between depression and Neurodegenerative Diseases: Pathophysiology, challenges in diagnosis and treatment options. *Cureus*. https://doi.org/10.7759/cureus.11613

Hwang, P. H., Ang, T. F., De Anda-Duran, I., Liu, X., Liu, Y., Gurnani, A., Mez, J., Auerbach, S., Joshi, P., Yuan, J., Devine, S., Au, R., & Liu, C. (2023). Examination of potentially modifiable dementia risk factors across the adult life course: The Framingham Heart Study. *Alzheimer's & Dementia*. https://doi.org/10.1002/alz.12940

Iadecola, C., Duering, M., Hachinski, V., Joutel, A., Pendlebury, S. T., Schneider, J. A., & Dichgans, M. (2019). Vascular cognitive impairment and dementia. *Journal of the American College of Cardiology*, *73*(25), 3326–3344. https://doi.org/10.1016/j.jacc.2019.04.034

Iadecola, C., Yaffe, K., Biller, J., Bratzke, L. C., Faraci, F. M., Gorelick, P. B., Gulati, M., Kamel, H., Knopman, D. S., Launer, L. J., Saczynski, J. S., Seshadri, S., & Zeki Al Hazzouri, A. (2016). Impact of hypertension on cognitive function: A scientific statement from the American Heart Association. *Hypertension*, *68*(6). https://doi.org/10.1161/hyp.0000000000000053

Ikram, M. A., Brusselle, G., Ghanbari, M., Goedegebure, A., Ikram, M. K., Kavousi, M., Kieboom, B. C., Klaver, C. C., de Knegt, R. J., Luik, A. I., Nijsten, T. E., Peeters, R. P., van Rooij, F. J., Stricker, B. H., Uitterlinden, A. G., Vernooij, M. W., & Voortman, T. (2020). Objectives, design and main findings until 2020 from the Rotterdam Study. European Journal of Epidemiology, 35(5), 483–517. https://doi.org/10.1007/s10654-020-00640-5

Iliffe, S., Robinson, L., Brayne, C., Goodman, C., Rait, G., Manthorpe, J., & Ashley, P. (2009). Primary care and dementia: 1. diagnosis, screening and Disclosure. International Journal of Geriatric Psychiatry, 24(9), 895–901. https://doi.org/10.1002/gps.2204

Jafari, Z., Kolb, B. E., & Mohajerani, M. H. (2019). Age-related hearing loss and tinnitus, dementia risk, and auditory amplification outcomes. *Ageing Research Reviews*, *56*, 100963. https://doi.org/10.1016/j.arr.2019.100963

Jayakody, D. M. P., Almeida, O. P., Speelman, C. P., Bennett, R. J., Moyle, T. C., Yiannos, J. M., & Friedland, P. L. (2018). Association between speech and high-frequency hearing loss and depression, anxiety and stress in older adults. *Maturitas*, *110*, 86–91. https://doi.org/10.1016/j.maturitas.2018.02.002

Jellinger, K. A. (2006). Alzheimer 100 – highlights in the history of alzheimer research. Journal of Neural Transmission, 113(11), 1603–1623. https://doi.org/10.1007/s00702-006-0578-3

Jellinger, K. A. (2017). Dementia with lewy bodies and parkinson's disease-dementia: Current concepts and controversies. *Journal of Neural Transmission*, *125*(4), 615–650. https://doi.org/10.1007/s00702-017-1821-9

Jellinger, K. A., & Attems, J. (2007). Neuropathological evaluation of mixed dementia. Journal of the Neurological Sciences, 257(1-2), 80-87. https://doi.org/10.1016/j.jns.2007.01.045

Jha, A., & Mukhopadhaya, K. (2020). Memory, cognitive impairment and dementia. Alzheimer's Disease, 1–20. https://doi.org/10.1007/978-3-030-56739-2_1

Jia, X., Wang, Z., Huang, F., Su, C., Du, W., Jiang, H., Wang, H., Wang, J., Wang, F., Su, W., Xiao, H., Wang, Y., & Zhang, B. (2021). A comparison of the mini-mental state examination (MMSE) with the Montreal Cognitive Assessment (MOCA) for mild

cognitive impairment screening in Chinese middle-aged and older population: A cross-sectional study. *BMC Psychiatry*, *21*(1). https://doi.org/10.1186/s12888-021-03495-6

Johnson, K. A., Minoshima, S., Bohnen, N. I., Donohoe, K. J., Foster, N. L., Herscovitch, P., Karlawish, J. H., Rowe, C. C., Carrillo, M. C., Hartley, D. M., Hedrick, S., Pappas, V., & Thies, W. H. (2013). Appropriate use criteria for amyloid PET: A report of the amyloid imaging task force, the Society of Nuclear Medicine and Molecular Imaging, and the Alzheimer's Association. Alzheimer's & Dementia, 9(1). https://doi.org/10.1016/j.jalz.2013.01.002

Jorgensen, L. E. (2016). Verification and validation of hearing aids: Opportunity not an obstacle. *Journal of Otology*, *11*(2), 57–62. https://doi.org/10.1016/j.joto.2016.05.001

Jorgensen, L. E., Palmer, C. V., Pratt, S., Erickson, K. I., & Moncrieff, D. (2016). The effect of decreased audibility on MMSE performance: A measure commonly used for diagnosing dementia. Journal of the American Academy of Audiology, 27(04), 311–323. https://doi.org/10.3766/jaaa.15006

Judge, D., Roberts, J., Khandker, R. K., Ambegaonkar, B., & Black, C. M. (2019). Physician practice patterns associated with diagnostic evaluation of patients with suspected mild cognitive impairment and alzheimer's disease. *International Journal of Alzheimer's Disease*, *2019*, 1–8. https://doi.org/10.1155/2019/4942562

Kalaria, R. N. (2018). The pathology and pathophysiology of vascular dementia. *Neuropharmacology*, *134*, 226–239. https://doi.org/10.1016/j.neuropharm.2017.12.030

Kane, J. P., Surendranathan, A., Bentley, A., Barker, S. A., Taylor, J.-P., Thomas, A. J., Allan, L. M., McNally, R. J., James, P. W., McKeith, I. G., Burn, D. J., & O'Brien, J. T. (2018).

Clinical prevalence of lewy body dementia. Alzheimer's Research & Therapy, 10(1). https://doi.org/10.1186/s13195-018-0350-6

Kapasi, A., DeCarli, C., & Schneider, J. A. (2017). Impact of multiple pathologies on the threshold for clinically overt dementia. *Acta Neuropathologica*, *134*(2), 171–186. https://doi.org/10.1007/s00401-017-1717-7

Karantzoulis, S., & Galvin, J. E. (2011). Distinguishing alzheimer's disease from other major forms of dementia. Expert Review of Neurotherapeutics, 11(11), 1579–1591. https://doi.org/10.1586/ern.11.155

Kelly, M. E., Duff, H., Kelly, S., McHugh Power, J. E., Brennan, S., Lawlor, B. A., & Loughrey, D. G. (2017). The impact of social activities, social networks, social support and social relationships on the cognitive functioning of Healthy Older Adults: A systematic review. *Systematic Reviews*, *6*(1). https://doi.org/10.1186/s13643-017-0632-2

Kessler, E.-M., Bowen, C. E., Baer, M., Froelich, L., & Wahl, H.-W. (2012). Dementia worry: A psychological examination of an unexplored phenomenon. *European Journal of Ageing*, *9*(4), 275–284. https://doi.org/10.1007/s10433-012-0242-8

Kestens, K., Degeest, S., & Keppler, H. (2022). The views and experience of audiologists working in Flemish hearing aid centers concerning cognition within Audiological Practice. *American Journal of Audiology*, *31*(2), 338–347. https://doi.org/10.1044/2022_aja-21-00186

Kiely, K. M., & Anstey, K. J. (2015). Common cause theory in aging. *Encyclopedia of Geropsychology*, 1–11. https://doi.org/10.1007/978-981-287-080-3_118-1

Killen, A., Flynn, D., De Brún, A., O'Brien, N., O'Brien, J., Thomas, A. J., McKeith, I., & Taylor, J.-P. (2015). Support and information needs following a diagnosis of dementia with

Lewy Bodies. International Psychogeriatrics, 28(3), 495–501. https://doi.org/10.1017/s1041610215001362

Koch, T., Iliffe, S., & EVIDEM-ED project. (2010). Rapid appraisal of barriers to the diagnosis and management of patients with dementia in primary care: a systematic review. BMC family practice, 11, 1-8

Kramer, S. J., & Brown, D. K. (2023). *Audiology: Science to practice*. Plural Publishing, Inc.

Kremen, W. S., Beck, A., Elman, J. A., Gustavson, D. E., Reynolds, C. A., Tu, X. M., Sanderson-Cimino, M. E., Panizzon, M. S., Vuoksimaa, E., Toomey, R., Fennema-Notestine, C., Hagler, D. J., Fang, B., Dale, A. M., Lyons, M. J., & Franz, C. E. (2019). Influence of young adult cognitive ability and additional education on later-life cognition. *Proceedings of the National Academy of Sciences*, *116*(6), 2021–2026. https://doi.org/10.1073/pnas.1811537116

Krivanek, T. J., Gale, S. A., McFeeley, B. M., Nicastri, C. M., & Daffner, K. R. (2021). Promoting successful cognitive aging: A ten-year update. *Journal of Alzheimer's Disease*, *81*(3), 871–920. https://doi.org/10.3233/jad-201462

Kunkle, B. W., Grenier-Boley, B., Sims, R., Bis, J. C., Damotte, V., Naj, A. C., Boland, A., Vronskaya, M., van der Lee, S. J., Amlie-Wolf, A., Bellenguez, C., Frizatti, A., Chouraki, V., Martin, E. R., Sleegers, K., Badarinarayan, N., Jakobsdottir, J., Hamilton-Nelson, K. L., Moreno-Grau, S., … Pericak-Vance, M. A. (2019). Genetic meta-analysis of diagnosed alzheimer's disease identifies new risk loci and implicates AB, Tau, immunity and lipid processing. Nature Genetics, 51(3), 414–430. https://doi.org/10.1038/s41588-019-0358-2

Kuo, C.-Y., Stachiv, I., & Nikolai, T. (2020). Association of Late Life Depression, (non-) modifiable risk and protective factors with dementia and alzheimer's disease: Literature

review on current evidences, preventive interventions and possible future trends in prevention and treatment of dementia. *International Journal of Environmental Research and Public Health*, *17*(20), 7475. https://doi.org/10.3390/ijerph17207475

Lane, C. A., Hardy, J., & Schott, J. M. (2017). Alzheimer's disease. *European Journal of Neurology*, *25*(1), 59–70. https://doi.org/10.1111/ene.13439

Langa, K. M., Plassman, B. L., Wallace, R. B., Herzog, A. R., Heeringa, S. G., Ofstedal, M. B., Burke, J. R., Fisher, G. G., Fultz, N. H., Hurd, M. D., Potter, G. G., Rodgers, W. L., Steffens, D. C., Weir, D. R., & Willis, R. J. (2005). The aging, demographics, and memory study: Study Design and Methods. *Neuroepidemiology*, *25*(4), 181–191. https://doi.org/10.1159/000087448

Laver, K., Dyer, S., Whitehead, C., Clemson, L., & Crotty, M. (2016). Interventions to delay functional decline in people with dementia: A systematic review of Systematic Reviews. BMJ Open, 6(4). https://doi.org/10.1136/bmjopen-2015-010767

Lee, J. Y. (2015). Aging and speech understanding. *Journal of Audiology and Otology*, *19*(1), 7–13. https://doi.org/10.7874/jao.2015.19.1.7

Lehmann, J., Christen, N., Barilan, Y. M., & Gannot, I. (2021). Age-related hearing loss, speech understanding and Cognitive Technologies. *International Journal of Speech Technology*, *24*(2), 509–516. https://doi.org/10.1007/s10772-021-09817-z

Li, X., Dai, J., Zhao, S., Liu, W., & Li, H. (2018). Comparison of the value of mini-cog and MMSE screening in the rapid identification of Chinese outpatients with mild cognitive impairment. *Medicine*, *97*(22). https://doi.org/10.1097/md.0000000000010966

Liang, J., Lu, L., Li, J., Qu, X., Li, J., Qian, S., Wang, Y., Jia, R., Wang, C., & Xu, Y. (2020). Contributions of modifiable risk factors to dementia incidence: A bayesian network

analysis. Journal of the American Medical Directors Association, 21(11). https://doi.org/10.1016/j.jamda.2020.04.006

Licher, S., Ahmad, S., Karamujić-Čomić, H., Voortman, T., Leening, M. J., Ikram, M. A., & Ikram, M. K. (2019). Genetic predisposition, modifiable-risk-factor profile and long-term dementia risk in the general population. Nature Medicine, 25(9), 1364–1369. https://doi.org/10.1038/s41591-019-0547-7

Lim, U., Wang, S., Park, S. Y., Bogumil, D., Wu, A. H., Cheng, I., Haiman, C. A., Le Marchand, L., Wilkens, L. R., White, L., & Setiawan, V. W. (2021). Risk of alzheimer's disease and related dementia by sex and Race/ethnicity: The Multiethnic Cohort Study. *Alzheimer's & Dementia*, *18*(9), 1625–1634. https://doi.org/10.1002/alz.12528

Limpawattana, P., & Manjavong, M. (2021). The mini-cog, clock drawing test, and three-item recall test: Rapid cognitive screening tools with comparable performance in detecting mild NCD in older patients. *Geriatrics*, *6*(3), 91. https://doi.org/10.3390/geriatrics6030091

Lin, F. R. (2013). Hearing Loss and Aging. Women and Health, 1449-1462. https://doi.org/10.1016/b978-0-12-384978-6.00098-4

Lin, F. R., & Albert, M. (2014). Hearing loss and dementia - who is listening? Aging & Mental Health, 18(6), 671-673. https://doi.org/10.1080/13607863.2014.915924

Lin, F. R., Ferrucci, L., An, Y., Goh, J. O., Doshi, J., Metter, E. J., Davatzikos, C., Kraut, M. A., & Resnick, S. M. (2014). Association of Hearing Impairment with brain volume changes in older adults. *NeuroImage*, *90*, 84–92. https://doi.org/10.1016/j.neuroimage.2013.12.059

Lin, F. R., Thorpe, R., Gordon-Salant, S., & Ferrucci, L. (2011). Hearing loss prevalence and risk factors among older adults in the United States. *The Journals of Gerontology Series A:*

Biological Sciences and Medical Sciences, *66A*(5), 582–590. https://doi.org/10.1093/gerona/glr002

Lin, J. S., O'Connor, E., Rossom, R. C., Perdue, L. A., & Eckstrom, E. (2013). Screening for cognitive impairment in older adults: A systematic review for the U.S. Preventive Services Task Force. *Annals of Internal Medicine*. https://doi.org/10.7326/0003-4819-159-9-201311050-00730

Lisko, I., Kulmala, J., Annetorp, M., Ngandu, T., Mangialasche, F., & Kivipelto, M. (2021). How can dementia and disability be prevented in older adults: Where are we today and where are we going? Journal of Internal Medicine, 289(6), 807–830. https://doi.org/10.1111/joim.13227

Lista, S., Vergallo, A., Teipel, S. J., Lemercier, P., Giorgi, F. S., Gabelle, A., Garaci, F., Mercuri, N. B., Babiloni, C., Gaire, B. P., Koronyo, Y., Koronyo-Hamaoui, M., Hampel, H., & Nisticò, R. (2023). Determinants of approved acetylcholinesterase inhibitor response outcomes in alzheimer's disease: Relevance for precision medicine in Neurodegenerative Diseases. Ageing Research Reviews, 84, 101819. https://doi.org/10.1016/j.arr.2022.101819

Litke, R., Garcharna, L. C., Jiwani, S., & Neugroschl, J. (2021). Modifiable risk factors in alzheimer disease and related Dementias: A Review. *Clinical Therapeutics*, *43*(6), 953–965. https://doi.org/10.1016/j.clinthera.2021.05.006

Liu, S., Jones, R. N., & Glymour, M. M. (2010). Implications of lifecourse epidemiology for research on determinants of adult disease. *Public Health Reviews*, *32*(2), 489–511. https://doi.org/10.1007/bf03391613

Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., Brayne, C., Burns, A., Cohen-Mansfield, J., Cooper, C., Costafreda, S. G., Dias, A., Fox, N., Gitlin, L.

N., Howard, R., Kales, H. C., Kivimäki, M., Larson, E. B., Ogunniyi, A., … Mukadam, N. (2020). Dementia prevention, intervention, and care: 2020 report of The Lancet Commission. *The Lancet*, *396*(10248), 413–446. https://doi.org/10.1016/s0140-6736(20)30367-6

Loughrey, D. G., Kelly, M. E., Kelley, G. A., Brennan, S., & Lawlor, B. A. (2018). Association of age-related hearing loss with cognitive function, cognitive impairment, and dementia. *JAMA Otolaryngology–Head & Neck Surgery*, *144*(2), 115. https://doi.org/10.1001/jamaoto.2017.2513

MacDonald, A. A., Joyson, A., Lee, R., Seymour, D. G., & Soiza, R. L. (2012). The effect of hearing augmentation on cognitive assessment scales at admission to hospital. *The American Journal of Geriatric Psychiatry*, *20*(4), 355–361. https://doi.org/10.1097/jgp.0b013e3182107e88

Mamo, S. K., Reed, N. S., Price, C., Occhipinti, D., Pletnikova, A., Lin, F. R., & Oh, E. S. (2018). Hearing loss treatment in older adults with cognitive impairment: A systematic review. *Journal of Speech, Language, and Hearing Research*, *61*(10), 2589–2603. https://doi.org/10.1044/2018_jslhr-h-18-0077

Martini, A., Castiglione, A., Bovo, R., Vallesi, A., & Gabelli, C. (2014). Aging, cognitive load, dementia and hearing loss. *Audiology and Neurotology*, *19*(Suppl. 1), 2–5. https://doi.org/10.1159/000371593

Maske, U. E., Buttery, A. K., Beesdo-Baum, K., Riedel-Heller, S., Hapke, U., & Busch, M. A. (2016). Prevalence and correlates of DSM-IV-TR major depressive disorder, self-reported diagnosed depression and current depressive symptoms among adults in Germany. *Journal of Affective Disorders*, *190*, 167–177. https://doi.org/10.1016/j.jad.2015.10.006

Maslow, K., & Fortinsky, R. H. (2018). Nonphysician care providers can help to increase detection of cognitive impairment and encourage diagnostic evaluation for dementia in community and Residential Care Settings. *The Gerontologist*, *58*(suppl_1). https://doi.org/10.1093/geront/gnx171

Mattap, S. M., Mohan, D., McGrattan, A. M., Allotey, P., Stephan, B. C., Reidpath, D. D., Siervo, M., Robinson, L., & Chaiyakunapruk, N. (2022). The economic burden of dementia in low- and middle-income countries (LMICS): A systematic review. *BMJ Global Health*, *7*(4). https://doi.org/10.1136/bmjgh-2021-007409

McDade, E., Cummings, J. L., Dhadda, S., Swanson, C. J., Reyderman, L., Kanekiyo, M., Koyama, A., Irizarry, M., Kramer, L. D., & Bateman, R. J. (2022). Lecanemab in patients with early alzheimer's disease: Detailed results on biomarker, cognitive, and clinical effects from the randomized and open-label extension of the phase 2 proof-of-concept study. Alzheimer's Research & Therapy, 14(1). https://doi.org/10.1186/s13195-022-01124-2

McDaid, D., Park, A.-L., & Chadha, S. (2021). Estimating the global costs of hearing loss. *International Journal of Audiology*, *60*(3), 162–170. https://doi.org/10.1080/14992027.2021.1883197

McDonald, W. M. (2017). Overview of neurocognitive disorders. FOCUS, 15(1), 4–12. https://doi.org/10.1176/appi.focus.20160030

Mckee, A. C., & Daneshvar, D. H. (2015). The neuropathology of Traumatic Brain Injury. *Handbook of Clinical Neurology*, 45–66. https://doi.org/10.1016/b978-0-444-52892-6.00004-0

McKeith, I. G., Boeve, B. F., Dickson, D. W., Halliday, G., Taylor, J.-P., Weintraub, D., Aarsland, D., Galvin, J., Attems, J., Ballard, C. G., Bayston, A., Beach, T. G., Blanc, F., Bohnen, N., Bonanni, L., Bras, J., Brundin, P., Burn, D., Chen-Plotkin, A., … Kosaka, K. (2017). Diagnosis and management of dementia with Lewy Bodies. *Neurology*, *89*(1), 88–100. https://doi.org/10.1212/wnl.0000000000004058

McShane, R., Westby, M. J., Roberts, E., Minakaran, N., Schneider, L., Farrimond, L. E., Maayan, N., Ware, J., & Debarros, J. (2019). Memantine for dementia. *Cochrane Database of Systematic Reviews*. https://doi.org/10.1002/14651858.cd003154.pub6

Mick, P., Reed, M., & Pichora-Fuller, M. (2015). Hearing, cognition, and Healthy Aging: Social and public health implications of the links between age-related declines in hearing and cognition. Seminars in Hearing, 36(03), 122–139. https://doi.org/10.1055/s-0035-1555116

Mielke, M. M., Ransom, J. E., Mandrekar, J., Turcano, P., Savica, R., & Brown, A. W. (2022). Traumatic brain injury and risk of alzheimer's disease and related dementias in the population. *Journal of Alzheimer's Disease*, *88*(3), 1049–1059. https://doi.org/10.3233/jad-220159

Milán-Tomás, Á., Fernández-Matarrubia, M., & Rodríguez-Oroz, M. C. (2021). Lewy Body Dementias: A Coin with Two Sides? Behavioral Sciences, 11(7), 94. https://doi.org/10.3390/bs11070094

Montero-Odasso, M., Ismail, Z., & Livingston, G. (2020). One third of dementia cases can be prevented within the next 25 years by tackling risk factors. the case "for" and "against." Alzheimer's Research & Therapy, 12(1). https://doi.org/10.1186/s13195-020-00646-x

Moyer, V. A. (2014). Screening for cognitive impairment in older adults: U.S. Preventive Services Task Force Recommendation Statement. Annals of Internal Medicine, 160(11), 791. https://doi.org/10.7326/m14-0496

Mukadam, N., Sommerlad, A., Huntley, J., & Livingston, G. (2019). Population attributable fractions for risk factors for dementia in low-income and middle-income countries: An analysis using cross-sectional survey data. The Lancet Global Health, 7(5). https://doi.org/10.1016/s2214-109x(19)30074-9

Murman, D. (2015). The impact of age on cognition. Seminars in Hearing, 36(03), 111–121. https://doi.org/10.1055/s-0035-1555115

Nalley, C. (2021). Practice benefits of integrating cognitive screening services. The Hearing Journal, 74(4). https://doi.org/10.1097/01.hj.0000743692.41362.c1

Nandi, A., Counts, N., Chen, S., Seligman, B., Tortorice, D., Vigo, D., & Bloom, D. E. (2022). Global and regional projections of the economic burden of alzheimer's disease and related Dementias from 2019 to 2050: A value of Statistical Life Approach. *eClinicalMedicine*, *51*, 101580. https://doi.org/10.1016/j.eclinm.2022.101580

Nasreddine, Z. S., Phillips, N. A., Bédirian, V. R., Charbonneau, S., Whitehead, V., Collin, I., Cummings, J. L., & Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: A Brief Screening Tool For Mild Cognitive Impairment: MOCA: A BRIEF SCREENING TOOL FOR MCI. Journal of the American Geriatrics Society, 53(4), 695-699. https://doi.org/10.1111/j.1532-5415.2005.53221.x

National Council on the Aging. (2000). The consequences of untreated hearing loss in older persons. Society of Otorhinolaryngology and Head-Neck Nursing. 18(1):12-6.

Nichols, E., Steinmetz, J. D., Vollset, S. E., Fukutaki, K., Chalek, J., Abd-Allah, F., Abdoli, A., Abualhasan, A., Abu-Gharbieh, E., Akram, T. T., Al Hamad, H., Alahdab, F., Alanezi, F. M., Alipour, V., Almustanyir, S., Amu, H., Ansari, I., Arabloo, J., Ashraf, T., … Vos, T. (2022). Estimation of the global prevalence of dementia in 2019 and forecasted prevalence in 2050: An analysis for the global burden of disease study 2019. The Lancet Public Health, 7(2). https://doi.org/10.1016/s2468-2667(21)00249-8

Niotis, K., Akiyoshi, K., Carlton, C., & Isaacson, R. (2022). Dementia prevention in clinical practice. Seminars in Neurology, 42(05), 525–548. https://doi.org/10.1055/s-0042-1759580

Nirmalasari, O., Mamo, S. K., Nieman, C. L., Simpson, A., Zimmerman, J., Nowrangi, M. A., Lin, F. R., & Oh, E. S. (2016). Age-related hearing loss in older adults with cognitive impairment. *International Psychogeriatrics*, *29*(1), 115–121. https://doi.org/10.1017/s1041610216001459

Oeppen, J., & Vaupel, J. W. (2002). Broken limits to life expectancy. Science, 296(5570), 1029–1031. https://doi.org/10.1126/science.1069675

Omura, J. D., McGuire, L. C., Patel, R., Baumgart, M., Lamb, R., Jeffers, E. M., Olivari, B. S., Croft, J. B., Thomas, C. W., & Hacker, K. (2022). Modifiable risk factors for alzheimer disease and related dementias among adults aged ≥45 years — United States, 2019. MMWR. Morbidity and Mortality Weekly Report, 71(20), 680–685. https://doi.org/10.15585/mmwr.mm7120a2

O' Philbin, L., Woods, B., Farrell, E. M., Spector, A. E., & Orrell, M. (2018). Reminiscence therapy for dementia: An abridged Cochrane systematic review of the evidence from randomized controlled trials. Expert Review of Neurotherapeutics, 18(9), 715–727. https://doi.org/10.1080/14737175.2018.1509709

Orgeta, V., Mukadam, N., Sommerlad, A., & Livingston, G. (2018). The Lancet Commission on dementia prevention, Intervention, and care: A call for action. Irish Journal of Psychological Medicine, 36(2), 85–88. https://doi.org/10.1017/ipm.2018.4

Orji, A., Kamenov, K., Dirac, M., Davis, A., Chadha, S., & Vos, T. (2020). Global and regional needs, unmet needs and access to hearing aids. *International Journal of Audiology*, *59*(3), 166–172. https://doi.org/10.1080/14992027.2020.1721577

Ou, Y.-N., Tan, C.-C., Shen, X.-N., Xu, W., Hou, X.-H., Dong, Q., Tan, L., & Yu, J.-T. (2020). Blood pressure and risks of cognitive impairment and dementia. *Hypertension*, *76*(1), 217–225. https://doi.org/10.1161/hypertensionaha.120.14993

Oudin, A., Segersson, D., Adolfsson, R., & Forsberg, B. (2018). Association between air pollution from residential wood burning and dementia incidence in a longitudinal study in northern Sweden. *PLOS ONE*, *13*(6). https://doi.org/10.1371/journal.pone.0198283

Ovejero-Benito, M. C., Ochoa, D., Enrique-Benedito, T., del Peso-Casado, M., Zubiaur, P., Navares, M., Román, M., & Abad-Santos, F. (2022). Pharmacogenetics of Donepezil and memantine in healthy subjects. *Journal of Personalized Medicine*, *12*(5), 788. https://doi.org/10.3390/jpm12050788

Pacala, J. T., & Yueh, B. (2012). Hearing deficits in the older patient. *JAMA*, *307*(11), 1185. https://doi.org/10.1001/jama.2012.305

Pacheco Palha, A., & Esteves, M. F. (1997). The origin of dementia praecox. Schizophrenia Research, 28(2–3), 99–103. https://doi.org/10.1016/s0920-9964(97)00117-5

Panza, F., Lozupone, M., Sardone, R., Battista, P., Piccininni, M., Dibello, V., La Montagna, M., Stallone, R., Venezia, P., Liguori, A., Giannelli, G., Bellomo, A., Greco, A., Daniele, A., Seripa, D., Quaranta, N., & Logroscino, G. (2018). Sensorial frailty: Age-related hearing

loss and the risk of cognitive impairment and dementia in later life. *Therapeutic Advances in Chronic Disease*, *10*, 204062231881100. https://doi.org/10.1177/2040622318811000

Panza, F., Solfrizzi, V., & Logroscino, G. (2015). Age-related hearing impairment—a risk factor and frailty marker for dementia and ad. *Nature Reviews Neurology*, *11*(3), 166–175. https://doi.org/10.1038/nrneurol.2015.12

Paraskevaidi, M., Martin-Hirsch, P. L., & Martin, F. L. (2018). Progress and challenges in the diagnosis of dementia: A critical review. ACS Chemical Neuroscience, 9(3), 446–461. https://doi.org/10.1021/acschemneuro.8b00007

Pardo-Moreno, T., González-Acedo, A., Rivas-Domínguez, A., García-Morales, V., García-Cozar, F. J., Ramos-Rodríguez, J. J., & Melguizo-Rodríguez, L. (2022). Therapeutic approach to alzheimer's disease: Current treatments and new perspectives. Pharmaceutics, 14(6), 1117. https://doi.org/10.3390/pharmaceutics14061117

Pedditizi, E., Peters, R., & Beckett, N. (2016). The risk of overweight/obesity in mid-life and late life for the development of dementia: A systematic review and meta-analysis of longitudinal studies. *Age and Ageing*, *45*(1), 14–21. https://doi.org/10.1093/ageing/afv151

Pendlebury, S. T., & Rothwell, P. M. (2019). Incidence and prevalence of dementia associated with transient ischaemic attack and stroke: Analysis of the population-based Oxford Vascular Study. The Lancet Neurology, 18(3), 248–258. https://doi.org/10.1016/s1474-4422(18)30442-3

Perneczky, R. (2019). Dementia treatment versus prevention. Dialogues in Clinical Neuroscience, 21(1), 43–51. https://doi.org/10.31887/dcns.2019.21.1/rperneczky

Perry, W., Lacritz, L., Roebuck-Spencer, T., Silver, C., Denney, R. L., Meyers, J., McConnel, C. E., Pliskin, N., Adler, D., Alban, C., Bondi, M., Braun, M., Cagigas, X., Daven, M.,

Drozdick, L., Foster, N. L., Hwang, U., Ivey, L., Iverson, G., … Golden, T. (2018). Population Health Solutions for assessing cognitive impairment in geriatric patients. *Innovation in Aging*, *2*(2). https://doi.org/10.1093/geroni/igy025

Perry-Young, L., Owen, G., Kelly, S., & Owens, C. (2016). How people come to recognise a problem and seek medical help for a person showing early signs of dementia: A systematic review and meta-ethnography. *Dementia*, *17*(1), 34–60. https://doi.org/10.1177/1471301215626889

Peters, R., Mudway, I., Booth, A., Peters, J., & Anstey, K. J. (2021). Putting fine particulate matter and dementia in the wider context of noncommunicable disease: Where are we now and what should we do next: A systematic review. *Neuroepidemiology*, *55*(4), 253–265. https://doi.org/10.1159/000515394

Pichora-Fuller, M. K. (2015). Cognitive decline and hearing health care for older adults. American Journal of Audiology, 24(2), 108–111. https://doi.org/10.1044/2015_aja-14-0076

Piras, F., Carbone, E., Faggian, S., Salvalaio, E., Gardini, S., & Borella, E. (2017). Efficacy of cognitive stimulation therapy for older adults with vascular dementia. Dementia & Neuropsychologia, 11(4), 434–441. https://doi.org/10.1590/1980-57642016dn11-040014

Plascencia-Villa, G., & Perry, G. (2023). Lessons from antiamyloid-β immunotherapies in Alzheimer's disease. Handbook of Clinical Neurology, 267-291. https://doi.org/10.1016/b978-0-323-85555-6.00019-9

Population Reference Bureau. (2022). *Fact sheet: U.S. dementia trends*. PRB Resource Library. Retrieved February 22, 2023, from https://www.prb.org/resources/fact-sheet-u-s-dementia-trends

Powell, D. S., Oh, E. S., Reed, N. S., Lin, F. R., & Deal, J. A. (2022). Hearing loss and cognition: What we know and where we need to go. *Frontiers in Aging Neuroscience*, *13*. https://doi.org/10.3389/fnagi.2021.769405

Power, M. C., Adar, S. D., Yanosky, J. D., & Weuve, J. (2016). Exposure to air pollution as a potential contributor to cognitive function, cognitive decline, brain imaging, and dementia: A systematic review of Epidemiologic Research. *NeuroToxicology*, *56*, 235–253. https://doi.org/10.1016/j.neuro.2016.06.004

Pronk, M., Lissenberg-Witte, B. I., van der Aa, H. P., Comijs, H. C., Smits, C., Lemke, U., Zekveld, A. A., & Kramer, S. E. (2019). Longitudinal relationships between decline in speech-in-noise recognition ability and cognitive functioning: The Longitudinal Aging Study amsterdam. *Journal of Speech, Language, and Hearing Research*, *62*(4S), 1167–1187. https://doi.org/10.1044/2018_jslhr-h-ascc7-18-0120

Raduns, A. (2020). Assessing cognitive impairment with a computerized screening tool. *Nursing*, *50*(11), 67–68. https://doi.org/10.1097/01.nurse.0000718368.57145.5e

Rai, H., Yates, L., & Orrell, M. (2018). Cognitive stimulation therapy for dementia. *Clinics in Geriatric Medicine*, *34*(4), 653–665. https://doi.org/10.1016/j.cger.2018.06.010

Rajan, K. B., Weuve, J., Barnes, L. L., McAninch, E. A., Wilson, R. S., & Evans, D. A. (2021). Population estimate of people with clinical alzheimer's disease and mild cognitive impairment in the United States (2020–2060). Alzheimer's & Dementia, 17(12), 1966–1975. https://doi.org/10.1002/alz.12362

Rajan, K. B., Weuve, J., Barnes, L. L., Wilson, R. S., & Evans, D. A. (2018). Prevalence and incidence of clinically diagnosed alzheimer's disease dementia from 1994 to 2012 in a

population study. Alzheimer's & Dementia, 15(1), 1–7. https://doi.org/10.1016/j.jalz.2018.07.216

Ralli, M., Gilardi, A., Stadio, A. D., Severini, C., Greco, A., de Vincentiis, M., & Salzano, F. (2019). Hearing loss and Alzheimer?s disease: A Review. The International Tinnitus Journal, 23(2). https://doi.org/10.5935/0946-5448.20190014

Ramos, H., Pardo, J., Sánchez, R., Puchades, E., Pérez-Tur, J., Navarro, A., & Moreno, L. (2021). Pharmacist-physician interprofessional collaboration to promote early detection of cognitive impairment: Increasing diagnosis rate. *Frontiers in Pharmacology*, *12*. https://doi.org/10.3389/fphar.2021.579489

Ranson, J. M., Rittman, T., Hayat, S., Brayne, C., Jessen, F., Blennow, K., van Duijn, C., Barkhof, F., Tang, E., Mummery, C. J., Stephan, B. C., Altomare, D., Frisoni, G. B., Ribaldi, F., Molinuevo, J. L., Scheltens, P., Llewellyn, D. J., Abramowicz, M., Altomare, D., … Visser, L. (2021). Modifiable risk factors for dementia and dementia risk profiling. A user manual for Brain Health Services—part 2 of 6. Alzheimer's Research & Therapy, 13(1). https://doi.org/10.1186/s13195-021-00895-4

Ray, M., Dening, T., & Crosbie, B. (2019). Dementia and hearing loss: A narrative review. *Maturitas*, *128*, 64–69. https://doi.org/10.1016/j.maturitas.2019.08.001

Raz, L., Knoefel, J., & Bhaskar, K. (2015). The neuropathology and cerebrovascular mechanisms of dementia. Journal of Cerebral Blood Flow & Metabolism, 36(1), 172–186. https://doi.org/10.1038/jcbfm.2015.164

Rehm, J., Hasan, O. S., Black, S. E., Shield, K. D., & Schwarzinger, M. (2019). Alcohol use and dementia: A systematic scoping review. *Alzheimer's Research & Therapy*, *11*(1). https://doi.org/10.1186/s13195-018-0453-0

Remensnyder, L. S. (2012). Audiologists as gatekeepers and it's not just for hearing loss. *Audiology Today*, *24*(4), 24–31.

Rigters, S. C., Bos, D., Metselaar, M., Roshchupkin, G. V., Baatenburg de Jong, R. J., Ikram, M. A., Vernooij, M. W., & Goedegebure, A. (2017). Hearing impairment is associated with smaller brain volume in aging. *Frontiers in Aging Neuroscience*, *9*. https://doi.org/10.3389/fnagi.2017.00002

Robinson, L., Tang, E., & Taylor, J.-P. (2015). Dementia: Timely diagnosis and early intervention. BMJ, 350(jun15 14). https://doi.org/10.1136/bmj.h3029

Rose, A. F., Gilbertson, A. F., Cottrell, C., & Tampi, R. R. (2021). Cognitive screening for adult psychiatric outpatients: Comparison of the Cognivue®® to the Montreal Cognitive Assessment. *World Journal of Psychiatry*, *11*(7), 265–270. https://doi.org/10.5498/wjp.v11.i7.265

Roytman, M., Mashriqi, F., Al-Tawil, K., Schulz, P. E., Zaharchuk, G., Benzinger, T. L. S., & Franceschi, A. M. (2023). Amyloid-Related Imaging Abnormalities: An Update. American Journal of Roentgenology, 220(4), 562-574. https://doi.org/10.2214/ajr.22.28461

Sabbagh, M. N., Boada, M., Borson, S., Chilukuri, M., Doraiswamy, P. M., Dubois, B., Ingram, J., Iwata, A., Porsteinsson, A. P., Possin, K. L., Rabinovici, G. D., Vellas, B., Chao, S., Vergallo, A., & Hampel, H. (2020). Rationale for early diagnosis of mild cognitive impairment (MCI) supported by emerging Digital Technologies. *The Journal of Prevention of Alzheimer's Disease*, 1–7. https://doi.org/10.14283/jpad.2020.19

Sachdev, P. S., Blacker, D., Blazer, D. G., Ganguli, M., Jeste, D. V., Paulsen, J. S., & Petersen, R. C. (2014). Classifying neurocognitive disorders: The DSM-5 approach. Nature Reviews Neurology, 10(11), 634–642. https://doi.org/10.1038/nrneurol.2014.181

Saint-Maurice, P. F., Coughlan, D., Kelly, S. P., Keadle, S. K., Cook, M. B., Carlson, S. A., Fulton, J. E., & Matthews, C. E. (2019). Association of leisure-time physical activity across the adult life course with all-cause and cause-specific mortality. *JAMA Network Open*, *2*(3). https://doi.org/10.1001/jamanetworkopen.2019.0355

Saito, T., Murata, C., Saito, M., Takeda, T., & Kondo, K. (2017). Influence of social relationship domains and their combinations on incident dementia: A prospective cohort study. *Journal of Epidemiology and Community Health*, *72*(1), 7–12. https://doi.org/10.1136/jech-2017-209811

Salinas, M. (2022). Dementia with lewy bodies. *Psychiatric Annals*, *52*(10), 396–396. https://doi.org/10.3928/00485713-20220810-07

Sando, S. B., Melquist, S., Cannon, A., Hutton, M., Sletvold, O., Saltvedt, I., White, L. R., Lydersen, S., & Aasly, J. (2008). Risk-reducing effect of education in alzheimer's disease. *International Journal of Geriatric Psychiatry*, *23*(11), 1156–1162. https://doi.org/10.1002/gps.2043

Saragih, I. D., Tonapa, S. I., Saragih, I. S., & Lee, B.-O. (2022). Effects of cognitive stimulation therapy for people with dementia: A systematic review and meta-analysis of randomized controlled studies. International Journal of Nursing Studies, 128. https://doi.org/10.1016/j.ijnurstu.2022.104181

Satizabal, C. L., Beiser, A. S., Chouraki, V., Chêne, G., Dufouil, C., & Seshadri, S. (2016). Incidence of dementia over three decades in the Framingham Heart Study. New England Journal of Medicine, 374(6), 523–532. https://doi.org/10.1056/nejmoa1504327

Saunders, G. H., Odgear, I., Cosgrove, A., & Frederick, M. T. (2018). Impact of hearing loss and amplification on performance on a cognitive screening test. *Journal of the American Academy of Audiology*, *29*(07), 648–655. https://doi.org/10.3766/jaaa.17044

Schneider, J. A. (2022). Neuropathology of Dementia Disorders. *CONTINUUM: Lifelong Learning in Neurology*, *28*(3), 834–851. https://doi.org/10.1212/con.0000000000001137

Schwarzinger, M., Pollock, B. G., Hasan, O. S., Dufouil, C., Rehm, J., Baillot, S., Guibert, Q., Planchet, F., & Luchini, S. (2018). Contribution of alcohol use disorders to the burden of dementia in France 2008–13: A nationwide retrospective cohort study. *The Lancet Public Health*, *3*(3). https://doi.org/10.1016/s2468-2667(18)30022-7

Seitz, D. P., Chan, C. C., Newton, H. T., Gill, S. S., Herrmann, N., Smailagic, N., Nikolaou, V., & Fage, B. A. (2018). Mini-cog for the diagnosis of alzheimer's disease dementia and other dementias within a primary care setting. Cochrane Database of Systematic Reviews. https://doi.org/10.1002/14651858.cd011415.pub2

Selman, A., Burns, S., Reddy, A. P., Culberson, J., & Reddy, P. H. (2022). The role of obesity and diabetes in dementia. *International Journal of Molecular Sciences*, *23*(16), 9267. https://doi.org/10.3390/ijms23169267

Sharma, K. (2019). Cholinesterase inhibitors as alzheimer's therapeutics (review). *Molecular Medicine Reports*. https://doi.org/10.3892/mmr.2019.10374

Sharma, R. K., Chern, A., & Golub, J. S. (2021). Age-related hearing loss and the development of cognitive impairment and late-life depression: A scoping overview. *Seminars in Hearing*, *42*(01), 010–025. https://doi.org/10.1055/s-0041-1725997

Shen, J., Anderson, M. C., Arehart, K. H., & Souza, P. E. (2016). Using cognitive screening tests in Audiology. American Journal of Audiology, 25(4), 319–331. https://doi.org/10.1044/2016_aja-16-0032

Shulman, K. I. (2000). Clock-drawing: Is it the Ideal Cognitive Screening Test? *International Journal of Geriatric Psychiatry*, *15*(6), 548–561. https://doi.org/10.1002/1099-1166(200006)15:6<548::aid-gps242>3.0.co;2-u

Sikkes, S. A. M., Tang, Y., Jutten, R. J., Wesselman, L. M. P., Turkstra, L. S., Brodaty, H., Clare, L., Cassidy-Eagle, E., Cox, K. L., Chételat, G., Dautricourt, S., Dhana, K., Dodge, H., Dröes, R., Hampstead, B. M., Holland, T., Lampit, A., Laver, K., Lutz, A., … Bahar-Fuchs, A. (2020). Toward a theory-based specification of non-pharmacological treatments in aging and dementia: Focused reviews and methodological recommendations. Alzheimer's & Dementia, 17(2), 255–270. https://doi.org/10.1002/alz.12188

Silverberg, N. B., Ryan, L. M., Carrillo, M. C., Sperling, R., Petersen, R. C., Posner, H. B., Snyder, P. J., Hilsabeck, R., Gallagher, M., Raber, J., Rizzo, A., Possin, K., King, J., Kaye, J., Ott, B. R., Albert, M. S., Wagster, M. V., Schinka, J. A., Cullum, C. M., … Ferman, T. J. (2011). Assessment of cognition in early dementia. Alzheimer's & Dementia, 7(3). https://doi.org/10.1016/j.jalz.2011.05.001

Simonsen, A. H., Herukka, S.-K., Andreasen, N., Baldeiras, I., Bjerke, M., Blennow, K., Engelborghs, S., Frisoni, G. B., Gabryelewicz, T., Galluzzi, S., Handels, R., Kramberger, M. G., Kulczyńska, A., Molinuevo, J. L., Mroczko, B., Nordberg, A., Oliveira, C. R., Otto, M., Rinne, J. O., … Waldemar, G. (2016). Recommendations for CSF AD biomarkers in the diagnostic evaluation of Dementia. Alzheimer's & Dementia, 13(3), 274–284. https://doi.org/10.1016/j.jalz.2016.09.008

Simpson, J. R. (2014). DSM-5 and neurocognitive disorders. American Academy of Psychiatry and the Law, 42(2), 159–164.

Singh, A., & Jha, A. (2021). Cholinesterase inhibitors used for the management of alzheimer's disease: A Review. *Journal of Pharmaceutical Research International*, 121–128. https://doi.org/10.9734/jpri/2021/v33i60a34463

Singh, M., Raj, D., Raina, S. K., Gandhi, M. K., & Chander, V. (2016). Using multistate observational studies to determine role of hypertension and diabetes as risk factors for dementia. *Journal of Neurosciences in Rural Practice*, *07*(S 01). https://doi.org/10.4103/0976-3147.196462

Skrobot, O. A., O'Brien, J., Black, S., Chen, C., DeCarli, C., Erkinjuntti, T., Ford, G. A., Kalaria, R. N., Pantoni, L., Pasquier, F., Roman, G. C., Wallin, A., Sachdev, P., Skoog, I., Taragano, F. E., Kril, J., Cavalieri, M., Jellinger, K. A., Kovacs, G. G., … Kehoe, P. G. (2016). The vascular impairment of Cognition Classification Consensus Study. *Alzheimer's & Dementia*, *13*(6), 624–633. https://doi.org/10.1016/j.jalz.2016.10.007

Slade, K., Plack, C. J., & Nuttall, H. E. (2020). The effects of age-related hearing loss on the brain and cognitive function. *Trends in Neurosciences*, *43*(10), 810–821. https://doi.org/10.1016/j.tins.2020.07.005

Slaughter, S. E., Hopper, T., Ickert, C., & Erin, D. F. (2014). Identification of hearing loss among residents with dementia: Perceptions of Health Care Aides. *Geriatric Nursing*, *35*(6), 434–440. https://doi.org/10.1016/j.gerinurse.2014.07.001

Smith, A. D., Duffy, C., & Goodman, A. D. (2018). Novel computer-based testing shows multi-domain cognitive dysfunction in patients with multiple sclerosis. *Multiple Sclerosis*

Journal - Experimental, Translational and Clinical, *4*(2), 205521731876745. https://doi.org/10.1177/2055217318767458

Solomon, A., Turunen, H., Ngandu, T., Peltonen, M., Levälahti, E., Helisalmi, S., Antikainen, R., Bäckman, L., Hänninen, T., Jula, A., Laatikainen, T., Lehtisalo, J., Lindström, J., Paajanen, T., Pajala, S., Stigsdotter-Neely, A., Strandberg, T., Tuomilehto, J., Soininen, H., & Kivipelto, M. (2018). Effect of the Apolipoprotein E Genotype on Cognitive Change During a Multidomain Lifestyle Intervention: A Subgroup Analysis of a Randomized Clinical Trial. JAMA Neurology, 75(4), 462. https://doi.org/10.1001/jamaneurol.2017.4365

Sommerlad, A., Sabia, S., Singh-Manoux, A., Lewis, G., & Livingston, G. (2019). Association of social contact with dementia and cognition: 28-year follow-up of the Whitehall II Cohort Study. *PLOS Medicine*, *16*(8). https://doi.org/10.1371/journal.pmed.1002862

Song, J., Lee, W. T., Park, K. A., & Lee, J. E. (2014). Association between risk factors for vascular dementia and adiponectin. *BioMed Research International*, *2014*, 1–13. https://doi.org/10.1155/2014/261672

Stach, B. A., & Ramachandran, V. (2022). *Clinical audiology: An introduction*. Plural Publishing.

Steffens, D. C., Otey, E., Alexopoulos, G. S., Butters, M. A., Cuthbert, B., Ganguli, M., Geda, Y. E., Hendrie, H. C., Krishnan, R. R., Kumar, A., Lopez, O. L., Lyketsos, C. G., Mast, B. T., Morris, J. C., Norton, M. C., Peavy, G. M., Petersen, R. C., Reynolds, C. F., Salloway, S., … Yesavage, J. (2006). Perspectives on depression, mild cognitive impairment, and cognitive decline. *Archives of General Psychiatry*, *63*(2), 130. https://doi.org/10.1001/archpsyc.63.2.130

Strachan, M. W., Reynolds, R. M., Marioni, R. E., & Price, J. F. (2011). Cognitive function, dementia and type 2 diabetes mellitus in the elderly. *Nature Reviews Endocrinology*, *7*(2), 108–114. https://doi.org/10.1038/nrendo.2010.228

Strutt, P. A., Barnier, A. J., Savage, G., Picard, G., Kochan, N. A., Sachdev, P., Draper, B., & Brodaty, H. (2020). Hearing loss, cognition, and risk of neurocognitive disorder: Evidence from a longitudinal cohort study of older adult Australians. *Aging, Neuropsychology, and Cognition*, *29*(1), 121–138. https://doi.org/10.1080/13825585.2020.1857328

Su, P., Hsu, C.-C., Lin, H.-C., Huang, W.-S., Yang, T.-L., Hsu, W.-T., Lin, C.-L., Hsu, C.-Y., Chang, K.-H., & Hsu, Y.-C. (2017). Age-related hearing loss and dementia: A 10-year National Population-based study. *European Archives of Oto-Rhino-Laryngology*, *274*(5), 2327–2334. https://doi.org/10.1007/s00405-017-4471-5

Sutin, A. R., Stephan, Y., Luchetti, M., & Terracciano, A. (2018). Loneliness and risk of dementia. *The Journals of Gerontology: Series B*, *75*(7), 1414–1422. https://doi.org/10.1093/geronb/gby112

Swain, S. K. (2020). Age related hearing loss and cognitive impairment - A current perspective. *International Journal of Research in Medical Sciences*, *9*(1), 317. https://doi.org/10.18203/2320-6012.ijrms20205863

Sweller, J. (2011). Cognitive load theory. *Psychology of Learning and Motivation*, 37–76. https://doi.org/10.1016/b978-0-12-387691-1.00002-8

Taylor, B., & Tysoe, B. (2013). Interventional audiology: partnering with physicians to deliver integrative and preventive hearing care. Hearing Review, 20(12), 16-22.

Taylor, J.-P., McKeith, I. G., Burn, D. J., Boeve, B. F., Weintraub, D., Bamford, C., Allan, L. M., Thomas, A. J., & O'Brien, J. T. (2020). New evidence on the management of lewy

body dementia. The Lancet Neurology, 19(2), 157–169. https://doi.org/10.1016/s1474-4422(19)30153-x

Thapa, S., Singh, P., Jain, D. K., Bharill, N., Gupta, A., & Prasad, M. (2020). Data-driven approach based on feature selection technique for early diagnosis of alzheimer's disease. 2020 International Joint Conference on Neural Networks (IJCNN). https://doi.org/10.1109/ijcnn48605.2020.9207359

Toh, H. M., Ghazali, S. E., & Subramaniam, P. (2016). The acceptability and usefulness of cognitive stimulation therapy for older adults with dementia: A narrative review. International Journal of Alzheimer's Disease, 2016, 1–11. https://doi.org/10.1155/2016/5131570

Tombaugh, T. N., & McIntyre, N. J. (1992). The mini-mental state examination: A comprehensive review. *Journal of the American Geriatrics Society*, *40*(9), 922–935. https://doi.org/10.1111/j.1532-5415.1992.tb01992.x

Topiwala, A., Allan, C. L., Valkanova, V., Zsoldos, E., Filippini, N., Sexton, C., Mahmood, A., Fooks, P., Singh-Manoux, A., Mackay, C. E., Kivimäki, M., & Ebmeier, K. P. (2017). Moderate alcohol consumption as risk factor for adverse brain outcomes and cognitive decline: Longitudinal Cohort Study. BMJ. https://doi.org/10.1136/bmj.j2353

Turton, L., Souza, P., Thibodeau, L., Hickson, L., Gifford, R., Bird, J., Stropahl, M., Gailey, L., Fulton, B., Scarinci, N., Ekberg, K., & Timmer, B. (2020). Guidelines for best practice in the audiological management of adults with severe and profound hearing loss. *Seminars in Hearing*, *41*(03), 141–246. https://doi.org/10.1055/s-0040-1714744

Uchida, Y., Sugiura, S., Nishita, Y., Saji, N., Sone, M., & Ueda, H. (2019). Age-related hearing loss and cognitive decline — the potential mechanisms linking the two. *Auris Nasus Larynx*, *46*(1), 1–9. https://doi.org/10.1016/j.anl.2018.08.010

Uhlmann, R. F., Larson, E. B., Rees, T. S., Koepsell, T. D., & Duckert, L. G. (1989). Relationship of hearing impairment to dementia and cognitive dysfunction in older adults. JAMA, 261(13), 1916–1919

van Oostveen, W. M., & de Lange, E. C. (2021). Imaging techniques in alzheimer's disease: A review of applications in early diagnosis and longitudinal monitoring. International Journal of Molecular Sciences, 22(4), 2110. https://doi.org/10.3390/ijms22042110

Vatanabe, I. P., Manzine, P. R., & Cominetti, M. R. (2020). Historic concepts of dementia and alzheimer's disease: From Ancient Times to the present. Revue Neurologique, 176(3), 140–147. https://doi.org/10.1016/j.neurol.2019.03.004

Vaz, M., & Silvestre, S. (2020). Alzheimer's disease: Recent treatment strategies. European Journal of Pharmacology, 887, 173554. https://doi.org/10.1016/j.ejphar.2020.173554

Veronese, N., Facchini, S., Stubbs, B., Luchini, C., Solmi, M., Manzato, E., Sergi, G., Maggi, S., Cosco, T., & Fontana, L. (2017). Weight loss is associated with improvements in cognitive function among overweight and obese people: A systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, *72*, 87–94 https://doi.org/10.1016/j.neubiorev.2016.11.017

Verstynen, T. D., Lynch, B., Miller, D. L., Voss, M. W., Prakash, R. S., Chaddock, L., Basak, C., Szabo, A., Olson, E. A., Wojcicki, T. R., Fanning, J., Gothe, N. P., McAuley, E., Kramer, A. F., & Erickson, K. I. (2012). Caudate nucleus volume mediates the link between

cardiorespiratory fitness and cognitive flexibility in older adults. *Journal of Aging Research*, *2012*, 1–11. https://doi.org/10.1155/2012/939285

Vijayan, M., & Reddy, P. H. (2016). Stroke, vascular dementia, and alzheimer's disease: Molecular links. Journal of Alzheimer's Disease, 54(2), 427–443. https://doi.org/10.3233/jad-160527

Vitek, G. E., Decourt, B., & Sabbagh, M. N. (2023). Lecanemab (BAN2401): An anti–beta-amyloid monoclonal antibody for the treatment of alzheimer disease. *Expert Opinion on Investigational Drugs*, *32*(2), 89–94. https://doi.org/10.1080/13543784.2023.2178414

Vogt, A.-C. S., Jennings, G. T., Mohsen, M. O., Vogel, M., & Bachmann, M. F. (2023). Alzheimer's disease: A brief history of immunotherapies targeting amyloid β. *International Journal of Molecular Sciences*, *24*(4), 3895. https://doi.org/10.3390/ijms24043895

Wang, C., Song, P., & Niu, Y. (2022). The management of Dementia Worldwide: A review on policy practices, clinical guidelines, end-of-life care, and challenge along with Aging Population. BioScience Trends, 16(2), 119–129. https://doi.org/10.5582/bst.2022.01042

Wang, S., Molassiotis, A., Guo, C., Leung, I. S., & Leung, A. Y. (2022). Association between social integration and risk of dementia: A systematic review and meta-analysis of longitudinal studies. *Journal of the American Geriatrics Society*, *71*(2), 632–645. https://doi.org/10.1111/jgs.18094

Washnik, N., & Anjum, J. (2022). Integrating mild cognitive impairment screening tools in Audiologic Care: A Review. *Perspectives of the ASHA Special Interest Groups*, *7*(5), 1415–1431. https://doi.org/10.1044/2022_persp-22-00062

Watermeyer, J., Kanji, A., & Brom, L. (2020). “what’s going on with my ears?”: Some reflections on managing uncertainty in the audiology consultation. *American Journal of Audiology*, *29*(3), 1–9. https://doi.org/10.1044/2020_aja-19-00116

Weinstein, B. E. (2022). Fundamentals of screening for mild cognitive impairment and/or dementia. Hearing Review. 30(1).

Welberry, H. J., Tisdell, C. C., Huque, M. H., & Jorm, L. R. (2023). Have we been underestimating modifiable dementia risk? an alternative approach for calculating the combined population attributable fraction (PAF) for modifiable dementia risk factors. American Journal of Epidemiology. https://doi.org/10.1093/aje/kwad138

Wilson, R. S., Krueger, K. R., Arnold, S. E., Schneider, J. A., Kelly, J. F., Barnes, L. L., Tang, Y., & Bennett, D. A. (2007). Loneliness and risk of alzheimer disease. *Archives of General Psychiatry*, *64*(2), 234. https://doi.org/10.1001/archpsyc.64.2.234

Wolf, P. A. (2012). Contributions of the Framingham Heart Study to stroke and dementia epidemiologic research at 60 years. Archives of Neurology, 69(5), 567. https://doi.org/10.1001/archneurol.2011.977

Wolters, F. J., & Ikram, M. A. (2019). Epidemiology of Vascular Dementia. Arteriosclerosis, Thrombosis, and Vascular Biology, 39(8), 1542–1549. https://doi.org/10.1161/atvbaha.119.311908

Wong, C. W. (2016). Pharmacotherapy for dementia: A practical approach to the use of cholinesterase inhibitors and memantine. Drugs & Aging, 33(7), 451–460. https://doi.org/10.1007/s40266-016-0372-3

Woods, B., O'Philbin, L., Farrell, E. M., Spector, A. E., & Orrell, M. (2018). Reminiscence therapy for dementia. *Cochrane Database of Systematic Reviews*, *2018*(3). https://doi.org/10.1002/14651858.cd001120.pub3

World Health Organization. (2021). World report on hearing. World Health Organization.

Wu, W., Ding, D., Zhao, Q., Xiao, Z., Luo, J., Ganguli, M., Hughes, T. F., Jacobsen, E., Haan, M. N., van Dang, K., Lima-Costa, M. F., Blay, S. L., de Castro-Costa, E., Ng, T. P., Gwee, X., Gao, Q., Gureje, O., Ojagbemi, A., Bello, T., … Sachdev, P. S. (2022). Dose-response relationship between late-life physical activity and incident dementia: A pooled analysis of 10 cohort studies of memory in an international consortium. *Alzheimer's & Dementia*, *19*(1), 107–122. https://doi.org/10.1002/alz.12628

Yamada, M., Komatsu, J., Nakamura, K., Sakai, K., Samuraki-Yokohama, M., Nakajima, K., & Yoshita, M. (2020). Diagnostic criteria for dementia with Lewy bodies: Updates and Future Directions. Journal of Movement Disorders, 13(1), 1–10. https://doi.org/10.14802/jmd.19052

Yang, L., Yan, J., Jin, X., Jin, Y., Yu, W., Xu, S., & Wu, H. (2016). Screening for dementia in older adults: Comparison of mini-mental state examination, Mini-Cog, clock drawing test and AD8. *PLOS ONE*, *11*(12). https://doi.org/10.1371/journal.pone.0168949

Yang, L., Yan, J., Jin, X., Jin, Y., Yu, W., Xu, S., Wu, H., Xu, Y., & Liu, C. (2017). Estimation of diagnostic performance of dementia screening tests: Mini-mental state examination, Mini-Cog, clock drawing test and Ascertain Dementia 8 questionnaire. *Aging & Mental Health*, *22*(8), 948–952. https://doi.org/10.1080/13607863.2017.1320701

Yang, W., Li, X., Pan, K., Yang, R., Song, R., Qi, X., Pedersen, N. L., & Xu, W. (2021). Association of life-course depression with the risk of dementia in late life: A nationwide Twin Study. *Alzheimer's & Dementia*, *17*(8), 1383–1390. https://doi.org/10.1002/alz.12303

Ying, G., Zhao, G., Xu, X., Su, S., & Xie, X. (2023). Association of age-related hearing loss with cognitive impairment and dementia: An umbrella review. Frontiers in Aging Neuroscience, 15. https://doi.org/10.3389/fnagi.2023.1241224

Yuan, J., Sun, Y., Sang, S., Pham, J. H., & Kong, W.-J. (2018). The risk of cognitive impairment associated with hearing function in older adults: A pooled analysis of data from eleven studies. *Scientific Reports*, *8*(1). https://doi.org/10.1038/s41598-018-20496-w

Yue, T., Chen, Y., Zheng, Q., Xu, Z., Wang, W., & Ni, G. (2021). Screening tools and assessment methods of cognitive decline associated with age-related hearing loss: A Review. *Frontiers in Aging Neuroscience*, *13*. https://doi.org/10.3389/fnagi.2021.677090

Zambrano, P., Suwalsky, M., Jemiola-Rzeminska, M., & Strzalka, K. (2018). Studies on the interaction of NMDA receptor antagonist memantine with cell membranes: A mini-review. *Chemico-Biological Interactions*, *283*, 47–50. https://doi.org/10.1016/j.cbi.2018.01.022

Zekveld, A. A., Kramer, S. E., & Festen, J. M. (2011). Cognitive load during speech perception in noise: The influence of age, hearing loss, and cognition on the pupil response. *Ear & Hearing*, *32*(4), 498–510. https://doi.org/10.1097/aud.0b013e31820512bb

Zhang, X.-X., Tian, Y., Wang, Z.-T., Ma, Y.-H., Tan, L., & Yu, J.-T. (2021). *The epidemiology of alzheimer's disease modifiable risk factors and prevention.* The Journal of Prevention of Alzheimer's Disease, 1–9. https://doi.org/10.14283/jpad.2021.15

Zhao, Q.-F., Tan, L., Wang, H.-F., Jiang, T., Tan, M.-S., Tan, L., Xu, W., Li, J.-Q., Wang, J., Lai, T.-J., & Yu, J.-T. (2016). The prevalence of neuropsychiatric symptoms in Alzheimer's disease: Systematic review and meta-analysis. Journal of Affective Disorders, 190, 264-271. https://doi.org/10.1016/j.jad.2015.09.069

Zhuang, L., Yang, Y., & Gao, J. (2019). Cognitive assessment tools for mild cognitive impairment screening. *Journal of Neurology*, *268*(5), 1615–1622. https://doi.org/10.1007/s00415-019-09506-7

Zotcheva, E., Bergh, S., Selbæk, G., Krokstad, S., Håberg, A. K., Strand, B. H., & Ernstsen, L. (2018). Midlife physical activity, psychological distress, and dementia risk: The hunt study. *Journal of Alzheimer's Disease*, *66*(2), 825–833. https://doi.org/10.3233/jad-180768