Midlife Hypertension and Dementia: The Shadow Link

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Table of Contents

SUMMARY

The aging U.S. population faces an increasing assortment of age-associated morbidities, with dementia emerging as one of the most debilitating conditions, both financially and emotionally. Recent evidence suggests that midlife hypertension may be a significant risk factor for late-life dementia. This paper explores the hypothesis of a positive correlation between midlife hypertension and dementia, which otherwise have a complex relationship. Through a comprehensive review of epidemiological studies and proposed biological mechanisms, including vascular damage and inflammatory processes, this investigation seeks to unravel potential causal pathways linking both conditions.

Confounding variables such as lifestyle factors, genetic predispositions, and comorbid conditions are also critically examined to contextualize the association. Gaps in research, such as the need for long-term randomized trials and precision medicine approaches, highlight challenges and opportunities for further studies. Establishing hypertension as a modifiable risk factor for dementia could pave the way for innovative preventative strategies, improving both cognitive outcomes and overall quality of life. Ultimately, this research underscores the importance of early detection and intervention, aligning public health priorities with the goal of promoting a more dignified and fulfilling aging process.

1. INTRODUCTION

America is getting older.

According to Woolf and associates (Woolf et. al, 2019)[1], U.S life expectancy increased from 69.9 years to 78.9 years between 1959 and 2016. With increasing age comes an array of medical morbidities, including cardiovascular disease, malignancy, and what some would argue as the most debilitating, cognitive decline (Jaul et. al, 2017)[2].

Recent epidemiological studies suggest cognitive impairment doubles from 19% in ages 65-74 to 38% in ages 85 and above in the U.S (Hale et. al, 2020)[3]. The impact of cognitive decline, emotional distress notwithstanding, is financially burdensome, with estimated costs of dementia in the U.S to be over $500 billion in 2019 (Skaria et. al, 2022)[4]. As with any chronic medical disease, early detection and prevention is a primary goal in ongoing research. Several possible culprits have been implicated in processes leading to age-related cognitive decline, namely diabetes, stroke, and even depression (Dhakal et. al, 2020)[5].

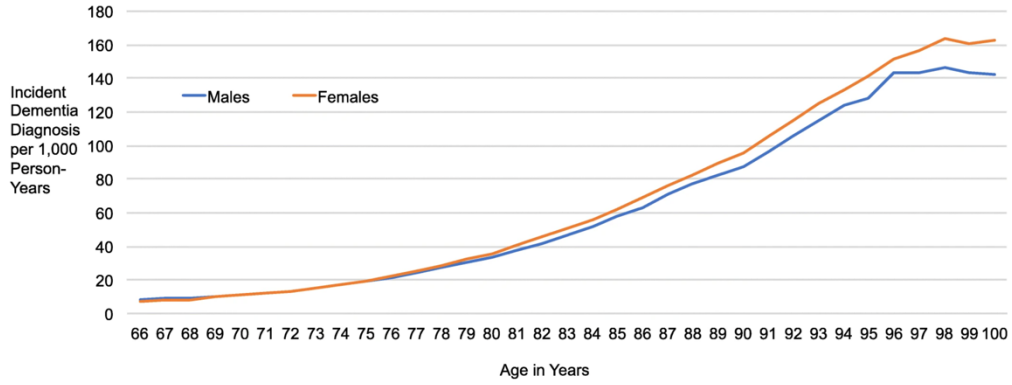

Fig. 1. Age-specific incidence rates of dementia diagnoses per 1000 person-years for males and females. Data from the Centers for Medicare and Medicaid Services (Hale et. al, 2020).

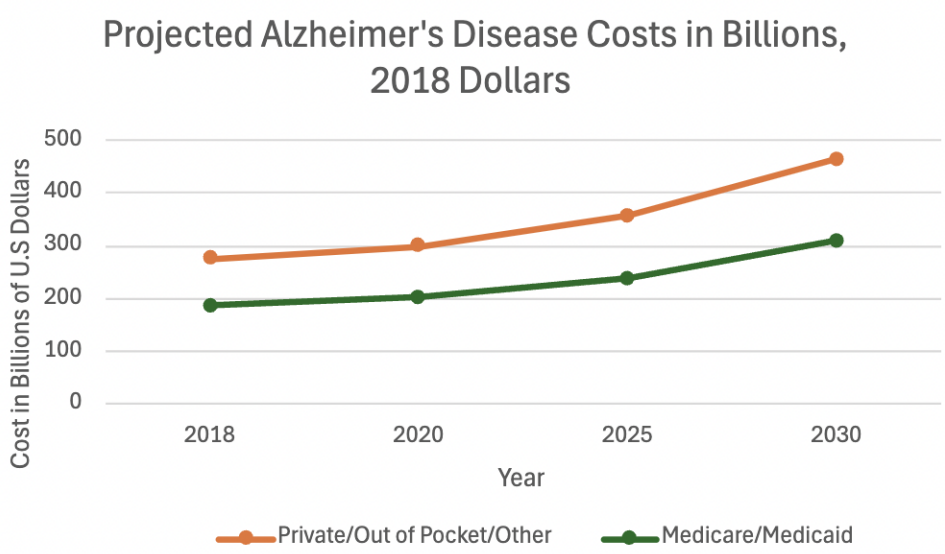

Fig. 2. Projected Alzheimer's Disease Costs in Billions of U.S Dollars (Redrawn from Skaria, 2022).

Hypertension, which happens to be diagnosed in nearly one half of all Americans above age 20, has also been proposed as a causative agent of cognitive decline, particularly dementia (Chobufo et. al, 2020)[6]. Whether there is a chronological link between hypertension and subsequent dementia is open to debate. This paper aims to investigate and contextualize the association(s) between hypertension diagnosed in midlife (typically 40-60 years old) and late-life dementia (typically diagnosed at 75 years or above) in the U.S. To achieve this end, the paper will begin by briefly defining hypertension and different stages and types of dementia, then cover various studies on the hypertension-dementia relationship, including potential confounding factors such as lifestyle and genetic risk. We hypothesize that midlife hypertension diagnoses are positively correlated with late-life dementia risk. If an association is indeed confirmed, midlife hypertension could serve as both a biomarker and therapeutic target to prevent dementia and improve patient quality of life.

2. Understanding Hypertension and Dementia

2.1 Definition of Midlife Hypertension

According to the American Academy of Family Physicians (AAFP), hypertension is defined as a blood pressure of 140-90 mmHg or higher on three separate measurements, at least one week apart (Buelt et. al, 2021)[7]. Blood pressure is measured conventionally as a systolic pressure (SP) over diastolic pressure (DP). One classification system for hypertensive diseases, commonly used in U.S clinical practice, stratifies different severities of hypertension: grade 1 or mild (140-159 SP over 90-99 DP), grade 2 or moderate (160-179 SP over 100-109 DP), and grade 3 or severe (> 180 SP over > 110 DP) (Evbayekha, 2022)[8].

2.2 Types and Stages of Dementia

Similarly, the American College of Neurology defines dementia as a decline in memory or other cognitive abilities (e.g language, problem solving, attention), severe enough to interfere with daily life (Arvanitakis, 2019)[9]. Just as with hypertension, there are different classifications for dementia, including no cognitive impairment, mild (slight difficulties in memory, language, and orientation), moderate (struggle with everyday tasks and less independence), and severe (extensive loss of basic functions), based on the Mini Mental State Examination (MMSE), a thirty-point questionnaire that quantifies dementia grade (Chapman et. al, 2016)[10]. In addition to severity, there are different types of dementia, including Alzheimer's disease, vascular dementia, Lewy Body dementia, and many others.

Despite the heterogeneity of both hypertension and dementia disease spectra, this paper will study literature that examines the correlation of any hypertensive class with any grade or type of dementia to allow simplicity of observations and analysis. For the purpose of this paper, "dementia" and "cognitive decline" will be used synonymously. When feasible, breakdown of different classifications may be necessary to explain inconsistent hypertension-dementia relationships, if present.

Although the mechanisms underlying the association between midlife hypertension and late-life dementia are extremely complex, a prevalent traditional rationale suggests hypertension induces vascular damage, disrupting blood supply to the brain and causing cognitive impairment. In our next section, we will review data from prominent clinical studies and extract more specific, proposed pathways by which hypertension leads to dementia.

3. Correlating Midlife Hypertension and Cognitive Decline

3.1 Longitudinal Studies and Clinical Evidence

The correlation between hypertension and cognitive performance has been widely studied for the past 40+ years and continues to be an expanding field of research. A seminal publication revealed that individuals from the Framingham, Massachusetts community with elevated blood pressure, particularly in midlife, were more likely to demonstrate poorer cognitive function in later life (Farmer et. al, 1990)[11]. The Framingham study is unique in being one of the first large-scale, longitudinal studies to track health outcomes over time in a homogeneous cohort (predominantly white and middle class), with fewer than 2% of participants lost to follow-up (Mahmood et. al, 2013)[12]. Studying a uniform population helped control certain socioeconomic and racial variables, minimizing confounding factors and increasing consistency of data. Despite its benefits, there are several disadvantages to the Framingham study, including its limited applicability to more diverse populations.

Not long after the Framingham Study findings, other studies followed suit and found a similar association between midlife blood pressure and dementia in other demographics. Launer and associates tracked Japanese-American men in Honolulu over one generation and found a positive correlation between midlife hypertension and late-age

dementia (Launer et. al, 2000)[13]. One provocative publication found African Americans, who were at higher risk of hypertension, had worse cognitive outcomes compared to Caucasian Americans (Levine et. al, 2019)[14]. Interestingly, when differences in blood pressure were removed between African Americans and Caucasians, the difference in risk of cognitive decline became insignificant, potentially suggesting ethnicity alone may not be a significant prognosticator for cognitive decline. Further studies evaluating ethnic variables on cognitive decline must be performed to validate Levine's findings.

Recent investigations continue to strengthen the midlife hypertension-dementia association. A study from Johns Hopkins University utilized the 6 Digit Symbol Substitution Test (DSST) as a proxy for cognitive function (Gottesman et. al, 2014)[15]. DSST assesses executive function and processing speed by requiring participants to match symbols to corresponding numbers using a reference key within 90 seconds. A significant decline in DSST scores was associated with the hypertensive group compared to the normotensive group. Complementing these findings, other researchers are leveraging advanced tools, such as neuroimaging studies, to explore novel surrogate metrics for dementia (George et. al, 2023)[16]. The following table compiles four different studies, which report mean systolic BP and corresponding dementia incidence across different groups. Overall, the clinical data presented appears compelling, particularly highlighting consistent trends across diverse methodologies. However, causality has yet to be proven.

Table 1. Compilation of results of four observational studies, demonstrating a consistent positive correlation between midlife hypertension and late-life dementia.

Author	Study	Mean Systolic BP (mmHg)	Dementia Incidence (%)
Farmer et. al (1990)	Longitudinally measured blood pressure, antihypertensive medication use, and cognitive performance: the Framingham study	>142	13.8%
		<122	8.8%
Launer et. al (2000)	Midlife blood pressure and dementia: the Honolulu-Asia aging study	168±1	14.5%
		107±5	2%
Gottesman et. al (2014)	Midlife Hypertension and 20-Year Cognitive Change: The Atherosclerosis Risk in Communities	Raw SBP not given	
		Normotensive	Delayed Word Recall Test (DWRT) = 0
		Hypertensive	DWRT = -1.124
McGrath et. al (2017)[46] *Extension of the Framingham Study examining offspring cohort from 1971-1975	Blood pressure from mid- to late life and risk of incident dementia	Raw SBP not given but Hazard Ratio (HR) for Dementia = 1.57 between Systolic Hypertension vs. Normotension	Dementia Incidence not given – see HR

3.2 Biological Mechanisms Linking Hypertension to Brain Changes

The brain is a complex, highly vascularized organ that requires constant and stable blood circulation for proper function. Hypertension, through its long-term pressure effects and mechanical trauma to blood vessels, is postulated to adversely affect blood circulation (Kim, 2023)[17]. In a similar fashion, hypertension can change the structure of cerebral blood vessels, impairing proper blood flow to the brain. Poor vascular supply leads to poor tissue

oxygenation, coined the neuroglial energy crisis, eventually causing tissue injury (Iadecola, 2016)[18].

Consequently, elevated blood pressure is hypothesized to increase leakage of the blood brain barrier, a semipermeable membrane between the intracranial blood vessels and the brain (Marimuthu, 2013)[19]. Leakage of unwanted proteins and substances is known to trigger immune inflammatory processes, causing brain buildup of beta-amyloid plaques commonly associated with Alzheimer's Disease (Wang, 2016)[20]. As a matter of fact, Shah and associates demonstrated a positive correlation between plasma B-amyloid levels and midlife hypertension, examining data from the Honolulu-Asia Aging Study (Shah et. al, 2012)[21].

Other proposed mechanisms include oxidative stress, where hypertension, through immune-mediated processes, induces free radical production, a toxin that can damage cell membrane integrity and decrease neuron function (Kowalcyzk, 2021)[22]. Through reduced blood flow to the brain, hypertension may also result in brain matter damage, characterized by accumulation of white matter lesions, potentially disrupting communication between brain regions (Li et. al, 2023)[23]. The processes by which hypertension influences cognitive function are tremendously complex, and researchers have only touched the tip of the iceberg in their findings.

3.3 Confounding Factors: Age, Lifestyle, and Genetic Predisposition

While many studies have demonstrated a strong hypertension-dementia (HD) correlation, possible confounding factors could influence the HD relationship. Several investigators argue chronological age as being the most significant independent predictor for late-life dementia (Stephan et. al, 2018)[24]. Healthcare researcher Ezra Fishman established at age 70, 0.1% were diagnosed with dementia, which more than doubled to 0.24% at age 85, and by more than ten-fold to 1.3% at age 100 (Fishman, 2017)[25]. Additionally, although midlife hypertension is strongly associated with dementia risk, some studies reveal a counterintuitive association for hypertension diagnosed in later life. A study by Power et. al suggests late-life hypertension may actually lower the risk of dementia (Power, 2013)[26]. The following table suggests that people who develop hypertension later in life (e.g 70 years old) have a smaller difference in cognition scores with their normotensive peers, compared to those who develop hypertension earlier in life, where cognitive decline worsens with longer surveillance intervals. Potential explanations include the role of hypertension in preserving adequate cerebral blood flow in aging brains that may otherwise experience hypoperfusion or vascular stiffening (de la Torre, 2012)[27]. Additionally, survivor bias, where hypertensive individuals who live into old age have unique protective factors and vascular adaptations, might mitigate the cognitive risks typically linked to elevated blood pressure earlier in life (Corrada et. al, 2017)[28]. Clearly, age complicates the relationship between blood pressure and cognition.

Table 2. Predicted Difference in Age-Adjusted Mean Cognitive Test Z Score between Hypertensives and Normotensives by Age at Onset and Duration of Time Since Hypertension Initiation.

	Time Between Hypertension Initiation and Cognitive Testing				
Age at Onset of Hypertension (Years)	5 Difference (95% Cl)	10 Difference (95% Cl)	15 Difference (95% Cl)	20 Difference (95% Cl)	25 Difference (95% Cl)
40	-	-	-.06 (-.28 to .16)	-.16 (-.33 to 0.00)	-.26 (-.42 to -.11)
45	-	.01 (-.21 to .22)	-.09 (-.24 to .05)	-.20 (-.32 to -.07)	-.30 (-.47 to -.13)
50	.07 (-.14 to .29)	-.03 (-.17 to .12)	-.13 (-.24 to -.02)	-.23 (-.38 to -.09)	-.33 (-.56 to -.11)
55	.04 (-.11 to .19)	-.06 (-.16 to .04)	-.16 (-.29 to -.03)	-.26 (-.47 to -.06)	-
60	.01 (-.10 to .12)	-.10 (-.22 to .03)	-.20 (-.40 to 0.00)	-	-
65	-.03 (-.16 to .11)	-.13 (-.33 to .07)	-	-	-
70	-.06 (-.27 to .14)	-	-	-	-

Genetic risks for dementia are another promising realm of research. The Apolipoprotein E ε4 Allele (APOE ε4), present in 20-30% of the U.S population, is a well-established risk factor for Alzheimer's Disease and Vascular Dementia (Frisoni, 1994)[29]. The APOE protein normally facilitates lipid metabolism by binding and clearing lipids, preventing beta-amyloid plaque buildup. APOE ε4 is an abnormal variant that is less efficient in lipid clearance, contributing to plaque accrual (Dunk et. al, 2022)[30]. Impaired lipid clearance may also increase cholesterol accumulation in blood vessels, causing vascular damage and resulting in hypertension (Linton et. al, 2019)[31]. In having dual effects on both hypertension and dementia, it is not unreasonable to hypothesize that APOE ε4 is the underlying risk factor behind dementia, with hypertension only as an intermediate condition, and that hypertension may not directly cause dementia. Not surprisingly, the APOE ε4 allele might serve as a biomarker and therapeutic target for both hypertension and dementia.

In addition to genetics, lifestyle factors, such as diet, might significantly confound the midlife hypertension-dementia link. For instance, high sodium intake has been shown to increase hypertension risk (Karppanen, 2006)[32], and by corollary, decrease cognitive function. Smoking has been shown to increase the risk of developing dementia by 30-50%

(Livingston et. al, 2024)[33]. Evidence suggests that toxins in cigarette smoke contribute to neuroinflammation and oxidative stress, accelerating brain aging. Sedentary lifestyles are defined by NIH as sitting, lying down, and expending little energy, such that one consumes 50% or more calories of what they metabolically process or "burn" (Owen et. al, 2010)[34]. Yan and associates revealed individuals with sedentary behavior were at 30% higher risk for dementia (Yan et. al, 2020)[35]. These adverse behaviors often exist in parallel (e.g someone who smokes is unlikely to have a healthy diet or exercise) and, combined with hypertension, likely exert a synergistic effect on dementia risks.

3.4 Comorbidities

To complicate matters, hypertension itself can lead to a wide variety of medical comorbidities that also affect cognitive function. It is widely known that hypertension can lead to chronic kidney disease (CKD). Approximately 20% of American adults with high blood pressure have CKD, which is more than three times the prevalence of CKD in adults without hypertension (Peralta et. al, 2005)[36]. Hypertension is thought to induce CKD by impairing blood flow to the kidneys and damaging kidney blood vessels, decreasing kidney function over time and ability to filter waste from the blood (Bidani et. al, 2004)[37]. When toxins, including creatinine, are not properly excreted, individuals experience a constitution of symptoms, including muscle cramps, nausea, fatigue, and changes in mental status, such as confusion and memory impairment. In turn, CKD is associated with a nearly 40% increase in odds of dementia (Zammit et. al, 2016)[47].

Hypertension can also adversely impact cardiac performance. Because of increased vascular resistance from high blood pressure, cardiac tissues require more force to generate the same output. Continued stress on cardiac tissues will eventually lead to cardiovascular failure. Inadequate cardiac output subsequently leads to poor perfusion to multiple organs. The brain is especially sensitive to fluctuations in blood circulation due to a higher requirement of oxygen and other nutrients.

Interestingly, hypertension is implicated in sleep apnea development. Sleep apnea is a condition of repeatedly interrupted respiration during sleep (Park et. al, 2011)[38]. High blood pressure can lead to increased pressure in the circulatory system, resulting in the narrowing or collapse of the airway during sleep (Bangash et. al, 2020)[39]. In fact, according to Osorio and associates, obstructive sleep apnea occurs in 40% of Alzheimer's patients (Osorio et. al, 2015)[40]. Sleep apnea-induced hypoxia likely compromises brain oxygen supply, which, over time, can cause chronic injury.

Like hypertension, diabetes is highly prevalent in the U.S. Approximately 73.6% of individuals above age 18 with hypertension in the U.S are diagnosed with diabetes, particularly type 2 (Naha, 2015)[41]. Hypertension may induce diabetes through activation of the sympathetic nervous system (i.e adrenaline) which affects insulin resistance in the pancreas. Insulin resistance subsequently may impair glucose metabolism in the brain and contribute to plaque buildup. The following table summarizes the possible factors that contribute to the pathogenesis of dementia, highlighting their interactions and cumulative impact.

Table 3. Examples of hypertension-related factors contributing to dementia pathogenesis, with variables often multi-directional.

Category	Specific Factor	Physiological Impact	Intermediate Effects	Link to Dementia
Genetic Risk Factors	**APOE ε4 allele**	Impaired lipid metabolism → Increased beta-amyloid plaques	Promotes vascular damage → Higher hypertension risk	Accelerates neurodegeneration and Alzheimer's risk
Lifestyle Factors	**High sodium intake**	Increased blood pressure	Greater risk of midlife hypertension	Associated with cognitive decline
	Smoking	Toxins contribute to neuroinflammation & oxidative stress	Accelerates brain aging	30-50% higher dementia risk
	Sedentary behavior	Reduced metabolic activity & circulation	Increased risk of hypertension	30% higher risk for dementia
Comorbid Conditions	**Chronic Kidney Disease (CKD)**	Impaired filtration → Buildup of toxins	Cognitive impairment due to systemic toxicity	40% higher dementia risk
	Cardiovascular disease	Increased vascular resistance → Cardiac strain	Poor cerebral perfusion due to low cardiac output	Reduced oxygenation contributes to neurodegeneration
	Obstructive sleep apnea (OSA)	Intermittent hypoxia → Poor brain oxygenation	Higher systemic inflammation	Found in 40% of Alzheimer's patients
	Type 2 diabetes	Insulin resistance → Impaired glucose metabolism	Increased risk of atherosclerosis & brain plaques	Plaque accumulation and cognitive decline
Hypertension as an Intermediary	**High blood pressure**	Vascular damage → Reduced cerebral blood flow	Small vessel disease in brain	Can trigger neuro-glial energy crisis
Potential Synergistic Effects	**Co-existence of multiple risk factors**	Shared inflammatory & vascular pathways	Synergistic worsening of brain function	Compounds dementia risk

The midlife hypertension-dementia association is, by no means, a simple binary relationship. As noted in an editorial, one doctor explains how mitigating dementia risk requires a holistic lifestyle approach, including physical activity, social engagement, a balanced diet, and management of chronic diseases like hypertension and diabetes (Swartz, 2024)[51]. In other words, there is a complex, multi-layered set of biological and social variables exerting both sequential and parallel effects on late-life dementia. Sequential effects in this paper refer to a chronological impact, where event A leads to event B. Parallel effects refer to two events, A and B, working in concert to lead to event C. In the real world, parallel variables can work independently or dependently of each other. Much of the scientific literature reviewed thus far attempts to control for confounding variables but given the complexities of sequential and parallel interactions, it is nearly logistically

impossible to generate bias-free data. Although peer-reviewed data should be evaluated with some skepticism, they nevertheless serve as a benchmark for future studies and understanding of the hypertension-dementia relationship.

4. Future Research and Implications

4.1 Gaps in Current Research

As presented, there have been extensive observational studies evaluating and supporting the links between midlife hypertension and dementia. There are also a plethora of factors that could confound the hypertension-dementia link. Many observational studies cited thus far have provided insights into potential intervention strategies to lower dementia risk. The gold standard for any clinical decision relies on well-designed, prospective, randomized, and controlled experiments.

Achieving "gold standard" clinical studies is difficult for the following reasons: (1) As for any clinical randomized study, sufficient follow-up time must be allowed for adequate data extraction and interpretation. The surveillance time of a midlife hypertension and late-life dementia study would necessitate 30-50 years of participant tracking, which is beyond the scope of most practical follow-up times, typically ranging from 6 months to 5 years. Researchers have used animal models to extract data more promptly, including primates and mice, whose lifespans are much shorter than humans. The major drawback of using any animal model is the inherent unreliability in measuring any dementia endpoint. For example, the Canine Cognitive Dysfunction Rating Rating Scale (CCDR) is a 50-point questionnaire used by veterinarians to assess canine mental status (Salvin et. al, 2011)[43]. The questionnaires, however, can be deeply subjective and flawed. For instance, one point of measure is to quantify how often a dog "stares blankly at the walls or floor." To gauge whether a dog is blankly staring versus curiously watching something is subject to bias and not quantifiable. Another example is evaluating whether a dog "gets lost in the house or yard," but what appears to be disorientation might actually be normal exploratory behavior or a reaction to a new environmental stimulus. Because animals cannot verbally express symptoms, assessments rely entirely on external behavior, which can easily be misinterpreted. In humans, clinicians can follow up with questions, check for understanding, or cross-reference with self-reports or imaging. A human with dementia may not be capable of verbalizing either, but presumably were capable at a previous point in their life, making verbal speech a quantifiable endpoint. Dogs and other animals therefore arguably lack a clear and objective baseline to assess cognitive changes. (2) Another challenge to a well-designed experiment is the difficulty in controlling for every confounding variable. As stated, the clinical interactions between hypertension and a variety of comorbidities and social lifestyles are too deeply embedded to logistically exclude. Therefore, it is difficult, but not impossible, to establish a clear causal pathway between any risk factor and dementia. Recursive Partitioning Analysis (RPA) is a statistical method that could help establish and rank the relative risk/significance of confounding variables. As a hypothetical example, RPA could reveal that hypertension contibutes 25% to the overall dementia risk in a particular cohort, while another comorbidity like diabetes

could contribute 20%. While RPA doesn't establish causality, it offers a valuable algorithm for quantifying the interacting factors leading to dementia.

4.2 Precision Medicine in Addressing Hypertension-Related Dementia

Gaps in current clinical research aside, perhaps current understanding of multiple variables that may affect dementia can be used to create a comprehensive patient profile to quantify individual risks, otherwise known as precision medicine. Precision medicine allows for individually tailored approaches to diagnostic and treatment strategies. Cholerton and associates propose a model that evaluate dementia risk by incorporating personalized patient history, such as protein levels (e.g. beta-amyloid, tau) and past or current medical conditions (e.g. Parkinson's) (Cholerton et. al, 2016)[44]. The following figure shows one such application of precision medicine being used to quantify dementia risk through patient APOE genotype. Additional studies are needed to establish clearly-defined models that incorporate relative risks of known variables in dementia development.

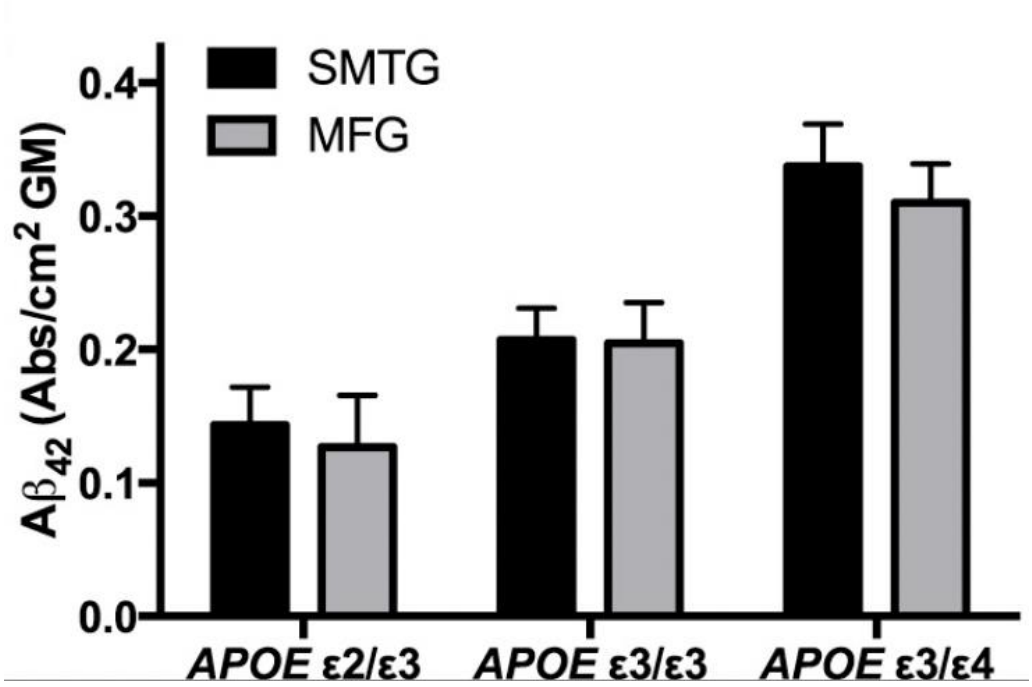

Fig. 4. Quantification of Amyloid-Beta protein (Aβ42) in superior and middle temporal gyri (SMTG) or middle frontal gyrus (MFG) in adults with genotypes APOE ε2/ε3, APOE ε3/ε3, or APOE ε3/ε4. A Precision Model approach to dementia risk assessment using the APOE gene as a biomarker (Cholerton et al, 2016).

4.3 Ethical Implications in Prevention, Treatment, and Healthcare Policy

There are ethical considerations to clinical trials that must be addressed. Even if long-term prospective, randomized studies could be practically implemented, they raise concerns about fairness and equity. To fully study whether midlife hypertension causes

late-life dementia, a control arm without hypertension must be compared to an experimental arm with medication-induced hypertension. Such an experimental design is clearly unjustifiable in current clinical standards. One possible alternative is to ask a corollary question: can anti-hypertensive medications lower dementia risk? The experimental design to answer the aforementioned question would necessitate a control arm where no treatment is given to known hypertensive patients, raising concerns of potential harm to experimental subjects and malpractice litigations. Finally, even if a clinical study confirms antihypertensive interventions lower dementia risk in hypertensive patients, society needs to grapple with healthcare disparities. How can we ensure equitable access for the uninsured or communities where medications are not readily available (e.g rural or reservation lands)? After speaking with clinicians from various specializations, including gerontology, radiology, and primary care, it became clear that socioeconomic barriers, including limited access to education and antihyperintensive therapies, can impede meaningful progress in aging care. One primary care physician, for example, described a patient: Mrs. G., a 72-year-old woman living in Fresno County with limited healthcare access. Despite repeated visits, Mrs. G struggled to consistently fill her prescription for antihypertensive medication due to limited insurance coverage. Consequently, her hypertension remained poorly controlled, increasing her risk for cardiovascular events and other health complications (Tri Do, M.D, personal interview, 18 November 2024; John Hamrick, M.D, personal interview, 18 November 2024; Sergio Mistivar, D.O, personal interview, 19 November 2024).

These challenges also have significant implications for healthcare policy. If antihypertensive medications are proven to reduce dementia risk, policymakers must prioritize patient education. Public health initiatives and awareness campaigns may need to expand their focus to include midlife as a critical window for intervention, emphasizing the importance of early hypertension screening and treatment to mitigate long-term cognitive decline. Because dementia care imposes tremendous financial burden on our healthcare system, public policy should equally be directed at increased government funds to provide patient education and treatment.

5. Conclusion

Hypertension is an extremely complex disease affecting many organs. Extensive clinical studies show a strong association between midlife hypertension and dementia risk. However, there are many possible confounding variables associated with dementia risk, including chronic kidney disease, poor cardiac performance, and genetic predispositions, as well as lifestyle and environmental factors like diet and smoking. Whether and how these variables interact with each other synergistically in cognitive decline remains to be fully determined. If a causal relationship between midlife hypertension and dementia is ever established, early intervention becomes quintessential. Just as early breast cancer detection by mammograms lower cancer-specific mortality, it stands to reason that early hypertensive diagnosis and intervention would positively impact cognitive outcomes, as well as lowering risk for other comorbidities.

Financial burdens aside, the emotional cost of caring for loved ones with dementia cannot be fully appreciated. As portrayed in *Still Alice* (Glatzer and Westmoreland, 2014)[45], a movie documenting the gradual decline of a brilliant linguistics professor due to early-onset Alzheimer's, the emotional impact of dementia on the patient and her family is nothing short of debilitating. As we gear into our next presidential administration, efforts must be lobbied to increase funding in research, awareness, and treatment of hypertension within the National Institute on Aging. In understanding the intricate relationships between midlife hypertension and dementia, we can empower individuals to age with more dignity and purpose. After all, true longevity is not about extending the quantity of life but improving the quality of life.

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