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Mold Exposure and Cognitive Health During Menopausal Transitions: A Quantitative Analysis Using Secondary Data

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This is to certify that the doctoral dissertation by

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the review committee have been made.

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Walden University
2025

Abstract

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BS, University of Central Florida, 1995

Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health - Epidemiology

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Abstract

Mold exposure and menopausal transition have been independently linked to cognitive decline, yet their interaction among socioeconomically disadvantaged populations has not been well examined. This quantitative study is an exploration of how menopausal transition influences the relationship between mold exposure and cognitive health outcomes among women aged 40 to 60 living in low-income housing in the United States. Bronfenbrenner's ecological systems theory was used to guide the cross-sectional design using secondary data from the 2005–2006 National Health and Nutrition Examination Survey (NHANES; $N = 149$). Weighted logistic regression and Hayes' PROCESS (Models 2 and 4) were used to test associations and the potential moderating and mediating roles of menopausal transition (*Weighted N ≈ 7,135,536)*. The analysis showed that mold exposure remained a significant predictor of cognitive difficulty after adjusting for education, poverty-to-income ratio, and menopausal transition (OR = 1.768, 95% *CI* [1.761, 1.775], $p < .001$). Women in the postmenopausal stage were twice as likely to report cognitive difficulty as those in perimenopause (OR = 2.072, 95% *CI* [2.061, 2.083], $p < .001$). However, no significant moderating or mediating effects of menopausal transition were observed. The findings may inform public health strategies, guide housing and environmental health policies, and promote early cognitive and indoor air quality screenings and intervention efforts, particularly for women in underserved communities.

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Dedication

First, I would like to thank God, for this journey would not have been possible without Him.

I would also like to dedicate this to my supportive husband, Matt. You have held my hand and helped me through this entire process, and I am forever grateful. Your encouragement and love mean the world to me. I also want to thank my children, mother, and sister for their support and reassurance.

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Chapter 1: Introduction to the Study

Introduction

Current research on mold exposure and cognitive health during menopausal transitions is limited, especially among women aged 40 to 60 living in low-income housing. In this study, I examined whether mold exposure and menopausal transition predicted cognitive health outcomes after adjusting for socioeconomic factors, such as education and income, among midlife women living in low-income housing in the United States. I also explored whether menopausal transition served as a moderator or mediator in the relationship between mold exposure and cognitive health.

While previous research suggests potential links among these variables, the precise mechanisms remain underexplored. I sought to address this gap by examining the role of menopausal transition in the relationship between mold exposure, and cognitive health among women living in low-income housing, with consideration of socioeconomic factors, like income and education.

In this chapter, I provide background information for the study, explain its purpose, and summarize the research methods I used. I also outline potential limitations that could affect how the study may be interpreted and discuss the significance of the research within the field of public health.

Background

Cognitive health is an important health concern for aging women, due to hormonal changes, biological shifts, and environmental exposures (World Health Organization [WHO], 2024). Among these factors, mold exposure has been identified through previous research as a potential contributor to adverse cognitive health outcomes (Campbell & Weinstock, 2022; Chauhdary et al., 2021; Ehsanifar et al., 2023; Guo et al., 2021; Harding et al., 2020; Harding et al, 2019; Zheng et al., 2024). Simultaneously, hormonal changes have been associated with increased oxidative stress, neuroinflammation, and reduced neuroprotection, which contribute to cognitive impairments, including memory deficits (Allegra et al., 2023; WHO, 2024).

Existing research has linked mold exposure to adverse cognitive health outcomes, including memory deficits and neuroinflammation (Campbell & Weinstock, 2022; Chauhdary et al., 2021; Ehsanifar et al., 2023; Guo et al., 202; Harding et al., 2020; Harding et al, 2019; Zheng et al., 2024). Mold exposure has been associated with memory deficits, mood disorders, and disruptions to the gut-brain axis, which can lead to impaired cognitive performance (Campbell & Weinstock, 2022). Harding et al. (2020, 2019) found that breathing mold can trigger immune responses in the brain, leading to cognitive decline.

Socioeconomic factors, like income and education play a vital role in determining environmental exposures, like mold, and health outcomes. For example, Bovell-Ammon et al. (2021) showed that poor housing quality, including mold exposure, worsens respiratory and developmental health issues. Bovell-Ammon et al. (2021) emphasized in their research that inadequate housing conditions, including mold growth, impact low-income and minority families disproportionately.

While there is research that has examined how mold exposure effects cognitive health and the health effects of menopausal transitions, there is limited research exploring how menopausal transitions interact with mold exposure to influence cognitive health, especially among low-income women. This gap demonstrates the need to investigate the combined effects of mold exposures and menopausal transitions on cognitive health, and this study addressed this gap. In this study, I examined the combined effects of mold exposure and menopausal transitions on cognitive health, while considering socioeconomic factors like education and income. The findings from this study contributed to understanding health disparities in vulnerable populations and provided insights for targeted interventions.

Problem Statement

The specific research problem is that the exact role (moderating and/or mediating) of menopausal transitions in the relationship between mold exposure and cognitive health among women living in low-income housing is unknown. While existing research has examined mold exposure as a contributor to adverse cognitive health outcomes (Campbell & Weinstock, 2022; Chaudhary et al., 2021), as well as the effects of menopausal transition on neuroprotection and oxidative stress (Allegra et al., 2023; WHO, 2024), little research has been performed on how these factors interact. This highlights a significant gap in the literature and underscores the need for further studies on environmental and biological influences on cognitive health.

Women living in low-income housing in the United States experience higher rates of mold exposure due to the poor housing conditions, inadequate ventilation, and increased humidity levels (Bovell-Ammon et al., 2021). These environmental factors may contribute to adverse cognitive health outcomes, particularly among women undergoing menopausal transitions. The present analysis investigated whether menopausal transition directly influences cognitive health outcomes and mold exposure. The analysis also tested whether menopausal transition serves as a moderator, indicating that menopausal status alters the strength or direction of the relationship between mold exposure and cognitive health. In addition, the analysis examined whether menopausal transition operated as a mediator, hypothesizing that mold exposure influences menopausal status, which in turn affects cognitive health.

Purpose of the Study

In this quantitative study, I examined the role of menopausal transitions in the relationship between mold exposure (independent variable) and cognitive health (dependent variable) among women living in low-income housing in the United States. Specifically, I explored whether menopausal transition serves as a moderator (altering the strength or direction of the relationship) or mediator (explaining the pathway between mold exposure and cognitive health) in this relationship. Additionally, I controlled for socioeconomic factors such as education and income, as covariates to account for potential external influences.

I evaluated whether mold exposure contributed to adverse cognitive health outcomes in this population and whether menopausal transition alters or explains this relationship. I employed a quantitative, cross-sectional design using secondary data from the 2005-2006 National Health and Nutrition Examination Survey (NHANES) dataset to address this gap in the literature. Using this established dataset made it possible to explore the relationship between the variables without the need to conduct new surveys or interview this vulnerable segment of the population and it also allowed for statistical examination of the potential health risks.

The findings from this study help to clarify how mold exposure and menopausal transition influence cognitive health and shed light on how environmental and biological factors interact. The research findings may provide valuable information for developing policies and program interventions aimed at reducing health disparities among this vulnerable population segment.

Research Questions and Hypotheses

In this study, I addressed the following three overarching questions:

Research Question 1 (RQ1): Does mold exposure predict cognitive health outcomes after adjusting for menopausal transition, and socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$1): Mold exposure does not predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$1): Mold exposure does predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Research Question 2 (RQ2): Does menopausal transition moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$2): Menopausal transition does not moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$2): Menopausal transition moderates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Research Question 3 (RQ3): Does menopausal transition mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$3): Menopausal transition does not mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$3): Menopausal transition mediates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Theoretical Framework for the Study

Bronfenbrenner's ecological systems theory (EST), nested within the broader social ecological model (SEM), served as the theoretical framework guiding this study. This framework was selected because it supports an in-depth perspective and understanding of how individual health outcomes, like cognitive health, are shaped by interactions across several layers of their environmental exposures over time (Bronfenbrenner, 1974; 1994; Guy-Evans, 2024; Hayden, 2019). Therefore, the concept grounding this study was the influence of environmental and biological factors, specifically mold exposure and menopausal transition, on cognitive health in women living in low-income housing. This phenomenon reflects a multifaceted public health concern surrounding external environmental and internal physical changes.

According to Bronfenbrenner (1974; 1994), adverse development occurs through "close or proximal" environmental interactions that develop over time. This reflects changes in the individual and their social and physical surroundings (Bronfenbrenner, 1994). Within the context of this research, the EST framework was particularly applicable because the framework accounts for how proximal environmental exposures, like mold growth within the home, can negatively affect health outcomes, especially for biologically vulnerable populations like women undergoing menopausal transitions.

Bronfenbrenner's Ecological Systems Theory (1974) is categorized into five interrelated levels: the microsystem, mesosystem, exosystem, macrosystem, and chronosystem. The first level is the *microsystem,* which is the immediate environment where direct interactions occur, such as an individual's home (Brofenbrenner, 1974). This is the immediate environment where direct interactions occur, such as an individual's home (Bronfenbrenner, 1974). This study's microsystem included the residential environment and specific household exposures (e.g., mold or mildew), which may directly impact cognitive health. The next level is the *mesosystem,* which focuses on the connections between two or more microsystems (Bronfenbrenner, 1974). This research considered the relationship between the home environment and access to healthcare or educational resources, which can either mitigate or amplify the effects of mold exposure (Bronfenbrenner, 1974). (Bronfenbrenner, 1974). The *exosystem* involves environmental conditions that do not directly affect the individual but still influence their experience (Bronfenbrenner, 1974). For example, property management decisions or public housing policies that affect maintenance and environmental conditions of low-income housing may increase the risk of mold exposure without the woman's direct input (Bronfenbrenner, 1974). The *macrosystem* includes overarching societal structures, like socioeconomic inequalities, cultural beliefs, and gender norms (Bronfenbrenner, 1974). In this study, the macrosystem reflected how systemic disparities in housing access and gendered healthcare influence both the prevalence of mold exposure and the attention given to menopausal health concerns. Although the *chronosystem* level was not a primary focus in this cross-sectional study, it reflected the dimension of time, which includes changes in environmental exposures or hormonal health over the life course (Bronfenbrenner, 1974). While longitudinal data would have been needed to explore this layer, it remained a foundational part of the EST model (Bronfenbrenner, 1974).

The conceptual framework is also grounded in recent literature demonstrating that mold exposure is associated with cognitive and neurological effects (Campbell & Weinstock, 2022; Chaudhary et al., 2021) and that menopausal transition affects brain health (Allegra et al., 2023; WHO, 2024). However, research has yet to explore how these two variables interact, particularly among socioeconomically vulnerable populations. A detailed review of this literature is presented in Chapter 2.

Nature of the Study

This quantitative study employed a cross-sectional design using secondary data from the 2005-2006 National Health and Nutrition Examination Survey (NHANES) to identify patterns and associations within a defined population at a single point in time (Frankfurt-Nachmias, 2008; NCHS, 2007). The information gleaned from this study provided a valuable assessment of the relationship between environmental exposures, menopausal transition, and cognitive health. Utilizing an existing national dataset, like the 2005-2006 NHANES dataset, enabled a more robust analysis and is generalizable to the population in analyzing the relationship between environmental exposures, menopausal transition, and cognitive health because it is cost-effective, has a large sample size, and eliminates ethical concerns.

The 2005-2006 NHANES dataset was used because this survey cycle includes a self-reported measure of household mold exposure that is not consistently available in more recent NHANES datasets (NCHS, 2007). Specifically, the housing questionnaire from this dataset asks participants whether their homes had a mildew or musty odor in the past twelve (12) months (NCHS, 2007). The variable was removed from subsequent

NHANES cycles, limiting its use in more recent research. Including this indicator in the 2005-2006 cycle offered a rare opportunity to investigate self-reported mold exposure in a large, nationally representative sample (NCHS, 2007). The methodological validity of using this dataset for mold exposure studies is supported by prior research. For example, Sharpe et al. (2015) examined the variable risk for atopic disease due to indoor fungal exposure using NHANES 2005-2006 data. Furthermore, Ran et al. (2023) analyzed the association between metal exposure and allergen-specific IgE levels using the same NHANES 2005-2006 cycle.

This study focused on women aged forty to sixty years based on their menopausal transition (WHO, 2024). These age ranges allowed for an investigation of how hormonal changes might influence cognitive functioning. The NHANES dataset also included extensive demographic and health-related variables that supported the relationships between mold exposure, menopausal status proxies, and cognitive health outcomes within a single data source (NCHS, 2007).

This independent variable was mold exposure, using the NHANES question on household mildew or musty smell (NCHS, 2007). It was coded dichotomously (0 = No, 1 = Yes). The dependent variable was cognitive health status, assessed through self-reported functional limitations such as trouble concentrating or mental recall (NCHS, 2007). This variable was also treated as categorical (1 = cognitive difficulty present, 0 = not present).

Key covariates included menopausal transition indicators, education, and income. Because NHANES does not directly capture menopausal status, age was used as a proxy

(NCHS, 2007). Education level was treated as a categorical variable, and the poverty-to-income ratio (PIR) served as a continuous indicator of socioeconomic status (NCHS, 2007).

The study used logistic regression to analyze these relationships and determine whether mold exposure is a statistically significant predictor of cognitive health after adjusting for menopausal transition and socioeconomic factors. Moderation and mediation analyses were conducted using Hayes' PROCESS macro to explore whether the relationship between mold exposure and cognitive health is moderated by the menopausal stage or mediated by socioeconomic status (Hayes, 2018). This approach supported an understanding of how mold exposure may influence cognitive outcomes directly or indirectly through social and biological mechanisms (Hayes, 2018).

Through this quantitative, cross-sectional approach, the study aimed to determine whether self-reported mold exposure significantly predicts cognitive health issues in midlife women after accounting for key life-stage and socioeconomic factors.

Definitions

Bronfenbrenner's Ecological Systems Theory (EST): A theoretical model describing how individual development and health outcomes are influenced by interactions across various environmental systems. In this study, the home (*microsystem*), community (*mesosystem*), and broader socioeconomic and cultural influences (*exosystem/macrosystem*) are used to contextualize how environmental exposures and individual factors such as hormonal transitions affect cognitive health outcomes (Bronfenbrenner, 1974; Guy-Evans, 2024).

Cognitive Health: Refers to an individual's ability to think clearly, learn, and remember (CDC, 2024; CDC, 2016). In this study, cognitive health is operationalized through self-reported concentration difficulty as assessed by NHANES 2005-2006 dataset variable DPQ070, which asks whether participants experienced trouble concentrating in the past two weeks (NCHS, 2007).

Indoor Environmental Quality (IEQ): A measure of the conditions within a home or building, including air quality, presence of mold, ventilation, and moisture (EPA, 2012; EPA 2019). Poor IEQ is associated with adverse health outcomes, particularly in low-income or substandard housing (Pert, 2022; Dressel et al., 2021).

Low-Income Housing: Residences occupied by individuals or families whose income falls at or below the poverty threshold, operationalized through the Poverty Income Ratio (PIR) in NHANES, with PIR < 1.0 used to identify low-income status (NCHS, 2007; Bovell-Ammon et al., 2021).

Menopausal Transition: This is the biological process marking the end of reproductive years in women, generally divided into perimenopause, menopause, and post-menopause (WHO, 2024). Estrogen and progesterone levels change during menopausal transition (WHO, 2024; Allegra et al., 2023). These changes can influence cognitive and emotional health (WHO, 2024; Allegra et al., 2023). In this study, menopausal transition is proxied through age categories, with perimenopause at 40-44, menopause at 45-55, and post-menopause at 56+ (WHO, 2024).

Microbial Volatile Organic Compounds (MVOCs): Low molecular weight chemicals released by fungi and bacteria during their metabolic processes (Korpi et al., 1998). MVOCs are emitted into the surrounding air as metabolic by-products of microbial digestion (Korpi et al., 1998). These compounds are responsible for the characteristics of the mildew or musty odor often detected in mold-contaminated environments. (Korpi et al., 1998). Their presence can indicate that mold growth is present and has been linked to respiratory and neurological symptoms (Kori et al., 1998; EPA, 2012).

Mold Exposure: Indoor environmental exposure to mold, typically resulting from moisture or water damage, which can manifest through visible mold or olfactory signs such as mildew or musty odors (EPA, 2012; EPA, 2019; Korpi et al., 1998). The variable is captured in the NHANES 2005-2006 dataset via self-report of whether the home had a musty or mildew smell in the past 12 months (HOQ230) (NCHS, 2007). Mold exposure has been linked to respiratory issues, cognitive decline, and neurological impairments (Shiue, 2015; Harding et al., 2019); Straus, 2021).

Mycotoxins: Toxic compounds produced by some molds, like *Stachybotrys charatarum* and *Aspergillus*, that provide a competitive edge by inhibiting nearby microorganisms that are competing for the same foods (Korpi et al., 1998). These toxic compounds are common and often thrive in water-damaged environments (Korpi et al., 1998). These substances disrupt immune function and have been shown to cause neuroinflammation and cognitive impairment (Campbell & Weinstock, 2022; Chauhdary et al., 2021).

Neuroinflammation: Inflammatory responses in the brain that may result from mold exposure and can affect cognitive health. In fact, neuroinflammation has been associated with cognitive decline, memory loss, and mood disturbances (Harding et al., 2020; Ehsanifar et al., 2023).

NHANES (National Health and Nutrition Examination Survey): A nationally representative survey conducted by the CDC to assess the health and nutritional status of the United States population. The 2005-2006 cycle is used in this study due to its inclusion of key mold exposure variables that are not present in later datasets (NCHS, 2007; Sharpe et al., 2015; Ran et al., 2023).

Oxidative Stress: "An imbalance between two different types of molecules in your body: free radicals and antioxidants" (Cleveland Clinic, 2024). This imbalance, known as oxidative stress, is often implicated in cellular aging and cognitive decline (Allegra et al., 2023). Estrogen is believed to play a protective role against oxidative stress, and lower levels during menopause that may affect memory and cognitive health (Allegra et al., 2023).

Sick Building Syndrome (SBS): refers to a set of nonspecific symptoms, like headaches, fatigue, dizziness, difficulty concentrating, and irritation of the eyes, nose, or throat. Occupants of certain indoor environments report these symptoms without a diagnosable illness. These symptoms typically improve upon leaving the building, suggesting an environmental trigger (CDC, 2022; NIEHS, 2025).

Assumptions

I based this study on several key assumptions using secondary data from the 2005-2006 National Health and Nutrition Examination Survey (NCHS, 2007). First, I assumed that the NHANES data were collected following standardized protocols set forth by the Centers for Disease Control and Prevention (NCHS, 2007). I also assumed that the secondary data that I used were collected using standardized procedures consistent with established scientific and methodological approaches (NCHS, 2007). NHANES is designed to be nationally representative, and it was assumed that the dataset appropriately reflects the health status and environmental exposures during the 2005-2006 cycle (NCHS, 2007).

I assumed that participants responded truthfully and to the best of their ability, particularly regarding self-reported variables such as difficulty concentrating, presence of mildew or musty odors in the home, income, and educational attainment (NCHS, 2007). The data collected for the 2005-2006 NHANES dataset ensured confidentiality and anonymity, likely increasing the likelihood of honest responses (NCHS, 2007). Additionally, I assumed that the variable capturing whether the participant smells a mildew or musty odor within their home serves as a valid proxy for mold exposure, as previously used in prior studies (Shiue, 2015; Sharpe et al., 2015). I also assumed that age served as a reasonable proxy indicator of the menopausal transition in the absence of clinical or laboratory hormone testing, as other researchers have also relied on this variable in large-population health studies (Allegra et al., 2023).

Lastly, I assumed that the statistical methodology was appropriate to assess the relationship between mold exposure and cognitive health outcomes. Additionally, the underlying assumptions of the statistical tests used in these analyses were met and verified before the final data analysis. These assumptions varied by analytical method and are outlined below for each research question.

I used logistic regression to address Research Question 1 and assessed the assumptions to ensure the validity of the analysis. These assumptions include linearity in the logit, absence of multicollinearity, and identification of outliers (Daniel & Cross. 2019; Warner, 2013).

For Research Question 2, which involves testing for moderation using Hayes' PROCESS Model 2, it was assumed that the relationship between the independent and dependent variables was linear, the residuals were homoscedastic and normally distributed, and the errors are independent. It was also assumed that there was no multicollinearity between the moderator and predictor variables (Hayes, 2018). Additionally, it was assumed that the moderator (menopausal transition) and the independent variable (Mold exposure) interacted in a way that influenced the dependent variable (cognitive health) (Hayes, 2018; Warner, 2013).

For Research Question 3, which tests for mediation using Hayes' PROCESS Model 4, it was assumed that the variables involved in the mediation model followed a proper causal order and that the relationships among the independent variable (mold exposure), mediator (menopausal transition), and dependent variable (Cognitive health) were linear (Hayes, 2018; Warner, 2013). It was also assumed that residuals were independent and approximately normally distributed, that there was no multicollinearity among the variables, and that the variables were appropriately measured (Hayes, 2018; Warner, 2013). Furthermore, the data were assumed to be free from any outliers that could distort the mediation path and weaken the interpretability (Hayes, 2018; Warner, 2013).

Scope and Delimitations

In this study, I focused on the association between self-reported indoor mold exposure and cognitive health outcomes among women living in low-income housing as they age and transition through menopause (Ehsanifar et al., 2023; Allegra et al., 2023). Specifically, the population to be examined included women between the ages of forty and sixty who participated in the 2005-2006 National Health and Nutrition Examination Survey (NHANES) (NCHS, 2007). I selected this age range and used age as a proxy to capture the menopausal transition of women living in low-income communities. Menopausal transition has been associated with hormonal changes and increased risk of cognitive health concerns (Allegra et al., 2023; WHO, 2024). The NHANES 2005-2006 dataset was selected because it contains a unique variable that asks whether participants' homes had mildew or musty odors in the past twelve months which is a variable not consistently available in late NHANES cycles (NCHS, 2007; Shiue, 2015; Ran et al., 2023; Sharpe et al., 2015). Cognitive difficulty was measured using self-reported responses to questions on memory or concentration, using the NHANES questionnaire DPQ070 (NCHS, 2007).

The scope of this study intentionally excluded men, women outside the specified age range, or data beyond the 2005-2006 NHANES cycle (NCHS, 2007). The study was limited to women between the ages of forty to sixty years old to explore the association between mold exposure and menopausal transitions. Therefore, variables such as menopause status were estimated using age as a proxy, rather than using direct clinical or laboratory hormone levels. This delimitation was necessary due to the nature of the secondary data, though this approach was consistent with prior research (Allegra et al., 2023). Socioeconomic status was included as a covariate and measured using education and the poverty-to-income ratio (PIR) provided by NHANES (NCHS, 2007). These demographic indicators served as proxies for broader environmental and lifestyle factors (Bovell-Ammon et al., 2021).

This study also excluded clinical diagnoses of cognitive impairment, such as dementia or mild cognitive disorder. Cognitive function was assessed strictly through self-reported questionnaire responses. Furthermore, the cross-sectional design of the NHANES data limited the ability to infer causal relationships between mold exposure and cognitive health. Although NHANES provides a representative sample of the U.S. population, these findings may not be generalizable to individuals in other countries or adults outside of the sampled age range. The decision to utilize United States data and focus the literature review on studies conducted within North America was made to ensure the findings are directly applicable to public health policies and housing environments specific to the United States (von Hoffman, 2012).

Furthermore, the study also intentionally excluded examining other environmental exposures that may impact cognitive health outcomes. These additional environmental factors, like tobacco smoke, were excluded to narrow the study's focus to a manageable scope.

Narrowing the study population to women between forty and sixty years within the NHANES cycle provided a more focused insight into the potential impact of mold exposure on cognitive health outcomes during the menopausal transition. These delimitations may decrease the generalizability of the findings but enhanced the relevance and applicability to similar demographic and environmental conditions.

Limitations

Several limitations were considered when interpreting the findings of this study. A potential limitation when conducting this secondary data analysis was the lack of specificity in some datasets regarding key variables, like mold or menopausal transitions. For example, while mold exposure may be measured indirectly through housing quality indicators or self-reports, like the respondent stating they smelled a mildew or musty odor in the past twelve months, the study did not capture quantifiable data on specific species or concentration levels. These limitations may affect construct validity, as they did not capture sampling and laboratory measurements for specific mold species or exact hormone levels.

This study explored the relationship between mold exposure and cognitive health after adjusting for menopausal transitions, as well as socioeconomic factors, such as education and income, among women living in low-income housing in the United States using a nationally representative dataset. However, there were several limitations to consider. First, the variables available for analysis were limited to those collected during 2005-2006 within the NHANES dataset (NCHS, 2007). Second, the cross-sectional design based on the NHANES dataset prevented the ability to establish causal relationships. Although statistical associations were identified, conclusions about directionality or cause and effect could not be made. This limits internal validity. Lastly, as with any self-reported data, responses may be subject to recall bias or social desirability bias, which could have influenced the accuracy of certain variables within the study. The study relied on standardized, nationally validated survey items in the 2005-2006 NHANES data to mitigate these concerns. Using covariates like income and education helped control confounding variables.

Significance

While improvements have been made in understanding cognitive health in older adults, there is limited research on the environmental and socioeconomic factors that may impact cognitive health outcomes during menopausal transition. The findings from this research provided beneficial insights into how poor indoor air quality because of mold exposure may impact socioeconomically disadvantaged midlife women. This research contributes to environmental health and cognitive neuroscience by examining how mold exposure interacts with menopausal changes, which may exacerbate cognitive health outcomes in vulnerable populations. Numerous previous studies have shown that poor housing conditions related to mold and dampness are associated with various adverse health conditions, including respiratory issues, allergic reactions, and potentially cognitive effects (Du et al., 2020; Bovell-Ammon et al., 2020; Holden et al., 2022; CDC, 2022; Jacobs, 2011; Musarri & Fisk, 2007; Fisk et al., 2010). However, few studies have examined how mold exposures affect menopausal transition to influence cognitive health.

This research is significant because it examined how exposure to moldy or damp environments and socioeconomic disadvantages, such as low income and limited education, can affect women's cognitive health. This research contributed to understanding how health disparities are shaped by biology and the environment in which individuals live. This was accomplished by using age as a proxy for menopausal transition and mildew or musty smell to indicate indoor air quality in the NHANES 2005-2006 cycle.

Understanding this relationship was crucial for shaping public health policy and developing community-level interventions. The findings from this study may help inform the general population, improve housing standards, and prioritize indoor air quality assessment while targeting public health promotion efforts for women going through menopausal transitions in socially vulnerable housing communities. This research may also support and assist with gender-specific and equitably focused policies.

The findings from this study were intended to expand the knowledge showing that where individuals live influences their health outcomes, especially during menopausal transitions. This study provided a foundation for future advocacy and policy to reduce health disparities, improve living conditions, and promote cognitive wellness among women in low-income communities.

Summary

Chapter 1 included the background of this study, which focused on the relationship between mold exposure and cognitive health among midlife women experiencing menopausal transition who reside in low-income housing. I discussed the problem statement, purpose of the study, and theoretical framework in this chapter. The assumptions, scope and delimitations, and limitations were also discussed. Chapter 1 concluded with a discussion of the significance of the study. In Chapter 2, I will present a comprehensive literature review supporting this research by identifying the existing gap.

The theoretical foundation and a review of the study's methodology will also be provided in Chapter 2.

Chapter 2: Literature Review

Introduction

A complex combination of biological, environmental, and socioeconomic factors influences cognitive health in women during menopausal transition. There is a growing body of research examining how menopause affects neurocognitive functioning. However, few studies have explored how environmental exposures, like mold, may interact with hormonal changes during midlife to affect cognitive health.

Although cognitive decline and environmental exposures have been studied independently, little information is known about how these variables interact. Women living in substandard housing may be exposed to environmental hazards, like mold, and face barriers to healthcare access. The interaction between menopausal transition and mold exposure represents a critical and underexplored area of research that may contribute to health disparities in cognitive aging.

Despite the growing evidence that supports the independent links between menopause, cognitive decline, and mold exposure, there is a significant gap in research examining their intersection. Only a few studies have begun to explore how mold exposure may relate to neurological symptoms or cognitive impairment in women during the menopausal transition.

The purpose of this study was to investigate the association between mold exposure and menopausal transition on cognitive health in women between the ages of 40 and 60 who live in low-income housing, using secondary data from NHANES 2005-2006. In Chapter 1, I established the importance of public health of understanding how the intersection of environmental exposure and biological transitions contribute to cognitive decline. Chapter 2 builds upon that foundation by reviewing the literature across several categories that align with the study's variables and its conceptual model. The review of current literature helps lay the foundation for the theoretical framework, clarify the main concepts, and explore past findings tied to key variables in this study.

This chapter begins with a summary of the literature search process and continues into the conceptual and theoretical foundations that guide the research. The chapter then takes a closer look at existing research on mold exposure, menopausal transition, and cognitive decline, especially as these variables affect women living in low-income housing. Finally, this section concludes by identifying the gaps in current research and explain why this study is important and necessary.

Literature Search Strategy

In this literature review, I examined peer-reviewed research on the relationship between mold exposure, menopausal transition, and cognitive health outcomes, especially among women living in low-income housing. The review was used to identify key theoretical foundations and gaps in current knowledge and supported the direction of this study. This process consisted of multiple steps, including structured database searches, the application of clear inclusion and exclusion criteria, and systematic data extraction and organization. Each section is explained in further detail below.

Database Search

Two primary search methods were used to identify relevant journal articles for this literature review. First, electronic database searches were conducted using Walden

University's library, specifically through Academic Search Complete, CINAHL Plus with Full Text, MEDLINE with Full Text, APA PsycInfo, and ScienceDirect, among others accessible through EBSCOhost. Second, supplemental searches were conducted using Google Scholar to identify and locate peer-reviewed articles, grey literature, and dissertations.

Keywords and combinations searched included:

- *mold exposure* OR *mould exposure* OR *water damage* OR *poor indoor air quality*
- AND *low-income housing* OR *environmental health disparities*
- AND *cognitive performance* OR *brain health* OR *neurological impact* OR *cognitive decline*
- AND *gender differences* OR *menopause* OR *perimenopause* OR *post-menopause* OR *hormonal transitions*

Other search strings and terms included:

- *environmental exposures and gender*, *mold and cognitive health*, *brain health* and *housing quality*, *mold and hormonal transitions*, AND *indoor air quality* AND *cognitive decline*.

Databases searched:

- Walden University Library (public health databases)
- PubMed
- Google Scholar

Search results and refinements (2020-2026):

- *Mold exposure* OR *water damage* AND *low-income housing and cognitive performance* (374 results)
- *Mold exposure* AND *cognitive impairment* and gender AND **hormon* (862 results)
- *Mold exposure* AND *cognitive impairment* AND *gender* OR **hormon* (422 results)

Search results were screened for relevance, date range (2020-2026), and duplicates to ensure quality and alignment with the research focus.

Inclusion/Exclusion Criteria

To ensure relevancy and quality, the following inclusion criteria were applied:

- Articles must be peer-reviewed and published between 2020-2026.
- Studies must focus on populations or models relevant to cognitive and environmental health, particularly those examining women or female physiology, and address at least one of the following domains: mold exposure, cognitive health, hormonal transition, environmental inequality, or public health disparities.

Exclusion criteria:

- Articles limited to pediatric or exclusively male populations.
- Systematic reviews, meta-analysis, or conceptual papers not used for empirical analysis.
- Studies focused primarily on genetic or pharmaceutical interventions without environmental context.

Data Extraction

Articles were systematically reviewed and organized into color-coded themes reflecting key dimensions of the research landscape. These categories included environmental exposure (specifically mold – Green), hormonal and cognitive health across the menopausal transition (e.g., Pink and Yellow), theoretical or conceptual frameworks (Purple), intersectional studies combining mold exposure, menopause, and cognition (Orange), socioeconomic and housing-related factors (Blue), and research focused on datasets, methodology, or definitions (White).

Key data extracted for each article included:

- Full citation, including year of publication
- Theoretical or conceptual framework
- Study objectives, research questions, and/or hypotheses
- Description of the study population
- Methodology and intervention or study design
- Primary findings, strengths, limitations, and relevance to the research study

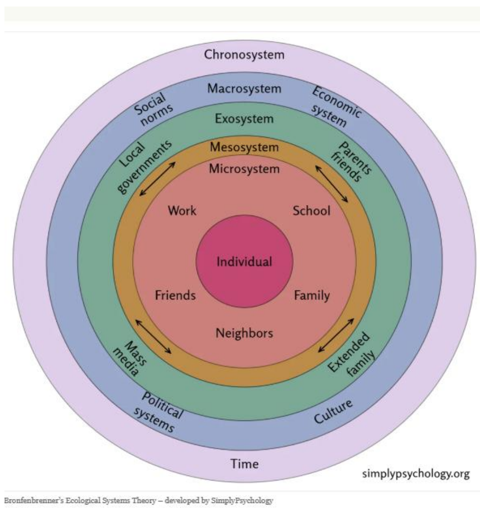
Theoretical Foundation

This study was grounded in Bronfenbrenner’s ecological systems theory (EST), which provided a comprehensive view to examine how individual health outcomes, such as cognitive health, were shaped by layered interactions across personal, social, and environmental contexts over time (Bronfenbrenner, 1979, 1994). This model was selected because it accounted for dynamic interrelationships between a person and their surrounding environments. It offered a structured approach to understanding how environmental exposures, like mold exposure, and biological factors, such as menopausal transition, interacted within broader social systems to influence cognitive health.

Bronfenbrenner's ecological systems theory (EST) provides a comprehensive framework for understanding the several layers of the environment that influence human development. These layers include the microsystem, mesosystem, exosystem, macrosystem, and chronosystem (Bronfenbrenner, 1974, 1994; Guy-Evans, 2024; Psychology Facts, 2018). Figure 1 visually represents these nested systems and how they interacted with individual development (Guy-Evans, 2024). These layers worked simultaneously to shape individual development and health. In this study, I applied EST to contextualize how women living in low-income housing during midlife may experience environmental and biological influences that could have impact their cognitive health.

Figure 1

Bronfenbrenner's Ecological System's Theory



Note. This diagram illustrates the nested environmental systems that influence an individual’s development (Guy-Evans, 2024).

The individual's home environment represents the primary setting for direct interaction at the microsystem level. For this study, the home was critical, as it included the presence of mold (operationalized through self-reported musty or mildew odors). Mold exposure at this level could have led to direct neurotoxic effects, such as inflammation, mood changes, and cognitive impairment (Harding et al., 2019; Campbell & Weinstock, 2022). These effects were particularly relevant for midlife women undergoing menopausal transition, a biological period marked by hormonal changes that may have increased susceptibility to neurological stressors (Allegra et al., 2023; WHO, 2024).

The mesosystem refers to the interrelations among multiple microsystems, such as interactions between a woman’s home and her access to healthcare, education, or support services. For example, women living in low-income housing with poor indoor air quality may have faced limited access to healthcare or educational resources that could otherwise help mitigate cognitive decline. Previous research has shown that women with less education and income are more likely to experience both environmental exposures and lack health access to healthcare resources to meet their needs (Bovell-Ammon et al., 2021; Christensen et al., 2022).

The exosystem encompasses external environments that indirectly influence the individual. In the study's context, this level included public housing policies, landlord maintenance practices, and government programs that may or may not have addressed mold remediation. While women are not directly engaged in these systems, decisions made within them may have affected the quality of their living environments and, consequently, their health outcomes. For instance, a lack of mold inspections or poor housing code enforcement may have increased exposure risks, particularly in socioeconomically disadvantaged communities (Du et al., 2020; Shiue, 2015).

The macrosystem represents broader sociocultural values, norms, and systemic inequalities. In this study, I explored how gendered health policies, income inequality, and social stigmas around aging and menopause influenced housing quality and access to health care. The intersection of systemic environmental injustice and gender disparities placed midlife women at greater risk of exposure to environmental toxins during a biologically vulnerable period (Straus, 2021; Albadrani, 2025).

Although I did not explicitly test the chronosystem in this cross-sectional study, it remained conceptually relevant. It represented the dimension of time and included life-course transitions such as menopause and long-term exposure to environmental hazards, like mold. A longitudinal design would have been needed to fully assess this component, but its presence in the theoretical framework underscored the cumulative nature of risk over time.

Bronfenbrenner's ecological systems theory (EST) supported this study's investigation into how mold exposure and menopausal transition, nested within broader social and environmental systems, influenced cognitive health outcomes in women who lived in low-income housing. I designed the research questions to examine how menopausal transition may serve as both a potential moderator and mediator between mold exposure and cognitive health. This approach illustrates the interplay between health, environment, and social contextual influences. Acknowledging the relationship between the layers represented in Bronfenbrenner's EST provided a robust foundation to understand the multi-layered determinants of cognitive health in this vulnerable population when exposed to microbial growth.

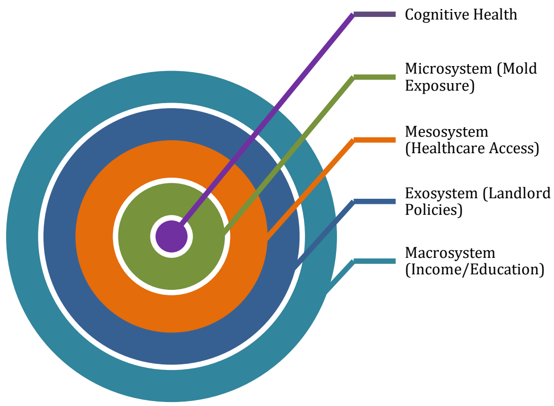
Conceptual Framework

To complement the theoretical foundation provided by Bronfenbrenner's ecological systems theory (EST), I developed a conceptual framework that visually organized the primary variables influencing midlife women's cognitive health across the theory's nested systems shown in Figure 2. At the center is cognitive health, the primary outcome of interest, shaped by environmental exposures, like mold, and life-stage changes, like menopausal transitions. Mold exposure is positioned within the microsystem, representing direct day-to-day environmental influences in the home environment. The mesosystem captures the interface between the home and external resources, like healthcare access, that might mitigate or exacerbate exposure effects.

The exosystem represents housing policy and landlord practices, which indirectly influence the living conditions of women who live in low-income housing without their direct involvement. At the outer layer, macrosystem-level factors such as income and education reflect broader social structures that shape both risk and healthcare access. Menopausal transition was examined in the analysis as either a moderator or mediator, influencing how mold exposure may have affected cognitive outcomes. While not explicitly shown in the figure, it remains conceptually tied to the chronosystem, representing biological and temporal changes over the life course. Together, this framework provides a foundation for undertsanding how environmental and social determinants converge to influence cognitive health during a vulnerable life stage.

Figure 2

Conceptual Framework of Bronfenbrenner's Ecological Systems Theory



Note. Conceptual Model Showing Mold Exposure, Menopausal Transition, and Cognitive Health Outcomes within the Ecological Systems Theory Framework.

Mold Exposure and Cognitive Health

Early Evidence Linking Mold to Systemic Health Outcomes

Mold exposure has been linked to various respiratory and systemic health issues, laying the foundation for current interest in its potential neurocognitive effects. Mudarri and Fisk (2007) estimated that 21 percent of asthma cases in the United States were attributable to indoor dampness and mold, with annual costs reaching $3.5 billion (USD). Similarly, Fisk et al. (2010) conducted a meta-analysis of 33 studies, concluding that damp indoor environments significantly increased the risk of respiratory infections and bronchitis. While these studies focused on respiratory outcomes, they provided early evidence of the widespread systemic burden of indoor mold exposure.

More recent studies have begun to explore the link between mold and brain-based health outcomes. Holden et al. (2023) found that poor indoor air quality in the closed building envelope negatively impacted respiratory health in children, a finding that parallels emerging literature suggesting that neuroinflammatory pathways may also be activated in adults. Xiao et al. (2021) further demonstrated associations between household mold exposure and increased risk for childhood asthma using national U.S. data, reinforcing the systemic effects of mold that may extend across the lifespan.

Experimental Evidence of Mold-Related Neurotoxicity

Building on this foundational work, Harding et al. (2020) investigated the effects of inhaled *Stachybotrys chartarum* spores in mice and found elevated cytokine levels in the hippocampus associated with impaired memory and increased anxiety-like behaviors. Similarly, Chauhdary et al. (2021) detailed the molecular mechanisms through which mycotoxins damage the central nervous system, including blood-brain barrier disruption, oxidative stress, and neuroinflammation. Findings from Chauhdary et al. (2021) supported the hypothesis that fungal exposure can cause neuroinflammation, compromising cognitive function. Campbell and Weinstock (2022) reported that mold exposure was associated with cognitive decline and gut-brain axis disturbances, reinforcing that fungal metabolites can impact neurological health.

Straus (2021) reviewed multiple case reports and animal studies, and concluded that mycotoxins like trichothecenes may be responsible for memory, learning, and attention impairments. These outcomes were especially severe in individuals exposed to indoor mold and water-damaged buildings. Ehsanifar et al. (2023) extended upon this research by evaluating cognitive decline in workers exposed to visible mold growth. They found that exposure to mold and water damage was associated with slower reaction times, reduced working memory, and increased self-reported mental fatigue, even after controlling for age and education. These findings were further supported by Kraft et al. (2021), who emphasized the role of mycotoxins in dysregulating the immune system and triggering neurological effects.

Zheng et al. (2024) added psychological context by documenting a significant increase in anxiety symptoms among older adults exposed to mold, particularly those with poor physical health or socioeconomic disadvantage. Though focused on anxiety, their study supported the role of mold exposure as a broader neuropsychological stressor.

Mechanisms of Neurological Impairment

Wang et al. (2024) further confirmed the biological plausibility of mold-related neurotoxicity by documenting alterations in mitochondrial function in hippocampal cells exposed to fungal toxins. These cellular disruptions are significant because mitochondrial dysfunction has been widely associated with cognitive aging and neurodegenerative diseases. Chauhdary et al. (2021) further supported these findings through a mechanistic synthesis of animal and cellular models, explaining how mycotoxins disrupt the blood-brain barrier, increase oxidative stress, and initiate neuroinflammation. Estrogen-related differences were proposed to influence the vulnerability to these exposures.

Ehsanifar et al. (2023) and Kraft et al. (2021) emphasized that mycotoxin exposure can dysregulate immune and neurological systems through oxidative stress and inflammation. Zheng et al. (2024) highlighted the psychological burden of mold in older adults, noting that anxiety symptoms worsened in those with poor physical health and lower socioeconomic status. Although their study focused on anxiety rather than cognition, it further supported mold exposure as a neuropsychological stressor. Roland (2024) also highlighted common neurological symptoms reported by individuals living in mold-affected environments, including confusion, difficulty concentrating, and “brain fog,” thereby supporting clinical concerns about mold’s cognitive effects.

Psychological and Socioeconomic Impacts of Mold Exposure

Gatto et al. (2024) conducted a state-of-the-science review on damp and mold-exposed housing, finding strong associations between exposure and mental health issues, including depression and stress. While most studies focused on psychological outcomes, many researchers have advocated for longitudinal and clinical studies to examine mold-related cognitive impacts. The National Institute of Environmental Health Sciences (NIEHS, 2025) and the Centers for Disease Control and Prevention (CDC, 2022) also noted cognitive complaints like “brain fog” and memory issues in public health materials, further supporting the relevance of mold exposure to cognitive function.

These findings across in vivo, in vitro, and occupational studies strongly support a growing consensus that mold exposure represents a significant risk factor for cognitive impairment. While environmental exposure presents both biological and psychological risks to cognitive health, another critical factor influencing midlife cognition is the menopausal transition.

Menopausal Transition and Cognitive Health

Overview of Menopausal Transition and Cognitive Symptoms

The menopausal transition is a critical life stage during which women may experience changes in cognitive performance and function (Than et al., 2023). The World Health Organization (WHO, 2024) identifies menopause as a natural biological process that typically occurrs between ages 45 and 55. Menopause is associated with symptoms like mood changes, sleep disturbances, and memory issues. These biological changes have been shown to influence mental and cognitive health outcomes, highlighting the need for this type of research.

Research has increasingly shown that this transitional phase can influence memory, attention, and higher-level functioning, mainly because of hormonal functioning, especially changes in estrogen levels, that affect neurological structures (Than et al., 2023). Than et al. (2023) conducted a longitudinal study of 15,486 women, with a mean age of 52 years, from the UK Biobank and found that both perimenopausal and postmenopausal women demonstrated poorer performance in fluid intelligence and memory compared to their premenopausal peers. Interestingly, psychomotor speed was the cognitive domain most affected by menopausal status. However, structural brain measures and menopausal hormone therapy (MHT) use did not significantly alter these associations (Than et al., 2023).

Hormonal Timing and Cognitive Outcomes

Several studies have explored the association between menopausal timing and cognitive performance. Nakanishi et al. (2025) found that earlier age at menopause was significantly associated with reduced cognitive performance and increased depressive symptoms in a large cohort of Japanese women. The findings from Nakanishi et al.'s (2025) research suggested that depressive symptoms partially mediate the relationship between menopausal timing and cognition, which shows the complex interaction between mood and memory during postmenopausal life.

Crockford et al. (2025) conducted a longitudinal cohort study of 8,168 midlife women in the United Kingdom. They concluded that a higher burden of menopausal symptoms, including hot flashes, sleep disturbances, and mood stability, predicted poorer cognitive function and increased behavioral risk markers. These associations remained statistically significant after adjusting for depressive symptoms and educational level (Crockford et al., 2025). Crockford et al.'s (2025) study suggested that the severity of menopausal symptoms may be an early warning indicator of long-term cognitive and behavioral risk, particularly in aging populations.

Biological and Molecular Mechanisms

Biological mechanisms behind these associations have been explored in depth by Sochocka et al. (2023), who identified that early and premature menopause increases the risk of cognitive impairment through processes such as beta-amyloid (β-amyloid) accumulation, mitochondrial dysfunction, and oxidative stress. Their research emphasized that early estrogen loss can disrupt memory, executive function, and attention, all of which are associated with increased vulnerability to neurodegenerative diseases such as Alzheimer's disease (Sochocka et al. 2023). These findings were complemented by Allegra et al. (2023), who reported that estrogen enhances antioxidant defenses and reduces oxidative stress in women. As estrogen declines during the menopausal transition, this protective mechanism weakens, potentially increasing neurological vulnerability (Allegra et al., 2023). While Allegra et al.'s (2023) study primarily focused on cancer, the underlying oxidative and mitochondrial pathways are relevant to cognitive decline and neurodegeneration.

Brinton et al. (2015) further advanced the understanding of neurological transitions by conceptualizing perimenopause as a "neurological transition state," which describes how estrogen withdrawal leads to a breakdown in brain energy metabolism. Brinton et al. (2015) argued that this hormonal disruption impairs synaptic function, reduces brain glucose metabolism, and serves as the precipice for future neurodegenerative disease. This bioenergetic decoupling is considered a tipping point for cognitive decline, particularly among women entering menopause (Brinton et al., 2015).

Estrogen Exposure and Brain Aging

The role of estrogen in protecting brain health is echoed in the findings of Jett et al. (2022), who reviewed the effects of both endogenous (naturally occurring) and exogenous (hormone therapy) estrogen exposures across a woman's reproductive lifespan. Their synthesis suggested that longer durations of estrogen exposure were associated with more favorable cognitive aging trajectories. At the same time, early menopause or inadequate hormone therapy was linked to an increased risk for Alzheimer's disease (Jett et al., 2022). Estrogen appears to be protective by maintaining brain metabolism, synaptic plasticity, and structural integrity, especially in areas like the hippocampus (Jett et al., 2022).

Mosconi et al. (2025) built upon the hormonal framework using neuroimaging to demonstrate that subjective memory complaints in perimenopausal and postmenopausal women were significantly associated with reduced hippocampal volume and brain glucose metabolism. These changes mirrored shifts in hormone levels, reinforcing the validity of the self-reported cognitive symptoms as early indicators of underlying neurological changes (Mosconi et al., 2025).

Synaptic Integrity and Hormonal Biomarkers

Alexander et al. (2025) examined postmortem brain tissue from a cohort of aging women and reported that synaptic integrity modified the effects of early menopause on Alzheimer's pathology. Their findings suggested that higher synaptic resilience may mitigate the adverse cognitive effects of early menopause (Alexander et al., 2025). Additionally, Nerattini et al. (2023) found that elevated gonadotropin levels, particularly follicle-stimulating hormone (FSH) and luteinizing hormone (LH), were associated with higher levels of Alzheimer's-related brain biomarkers in midlife women. These findings reinforce the need of tracking hormonal changes during menopausal transition as a potential early indicator of neurodegenerative risk (Nerattini et al., 2023).

Public Health Implications and Early Intervention

Recent syntheses of emerging research show that menopausal symptoms beyond the physiological end of menstruation may contribute to long-term adverse cognitive and neurological health outcomes. News Medical (2025) emphasized findings linking severe menopausal symptoms with mild behavioral impairments and memory difficulties. Krewson (2025) reported that early menopause, occurring before age 45, was associated with a substantially increased risk of cognitive decline later in life. Although these are journalistic summaries of peer-reviewed work, they bring attention to important public health messages that call for earlier screening and intervention.

This body of research suggests that the menopausal transition, especially when characterized by early onset or severe symptoms, represents a significant risk factor for cognitive decline. Estrogen loss, mood disturbances, and underlying neuropathological changes appear to converge during this life stage. These changes increase susceptibility to cognitive impairments and even neurodegenerative diseases. Since hormone levels and cognitive function can change simultaneously, midlife is a critical period for identifying and mitigating risk factors by implementing targeted intervention strategies. Beyond specific hormonal and environmental mechanisms, midlife represents a vulnerable period for cognitive change independent of external exposure.

Midlife Cognition

Demographic and Social Determinants of Cognitive Change

Cognitive health during midlife is a critical yet often overlooked public health concern. This period of life generally spans the ages of 40 to 60 years. This life stage can reflect changes incorporating biological transitions, environmental exposure, and cumulative life stressors affecting brain health or cognitive functioning. While much of the research on cognitive health focuses on older adults, a growing body of literature has identified midlife as a period when subtle cognitive changes may first emerge and serve as early indicators of later cognitive impairment (Paget-Blanc et al., 2025; Mosconi et al., 2025; Karlamangla et al., 2017; Greendale et al., 2021)

Evidence from longitudinal cohort studies emphasizes the variability of an individual's midlife cognitive trajectories, influenced by intrinsic factors like sex and education and extrinsic factors including lifestyle behaviors and environmental exposures. In the Cardiovascular Risk in Young Finns Study, Heiskanen et al. (2024) reported that changes in cognitive performance over a seven-year period were significantly associated with sex, age, and education level. These findings reinforce the importance of demographic and social determinants in shaping midlife cognitive health outcomes.

Lifestyle Factors and Cognitive Preservation

Similarly, Greendale et al. (2021) examined longitudinal data from women in midlife and found physical activity served as a protective factor for cognitive health. The analysis by Greendale et al. (2021) revealed that higher levels of physical activity were associated with better verbal memory and processing speed, independent of other health behaviors. These findings suggest that modifiable lifestyle factors offer opportunities for maintaining or improving cognitive health function during this transitional life stage.

Circadian Regulation and Cognitive Risk

Paget-Blanc et al. (2025) explored midlife-specific biomarkers and found that irregular rest-activity rhythms, particularly delayed timing and reduced robustness, were associated with lower cognitive performance and higher Alzheimer’s disease biomarkers in women. These findings suggest that disruptions in daily resting patterns may contribute to adverse cognitive health in midlife.

Cognitive Interventions During Midlife

Beyond observational studies, intervention research has demonstrated promise in assessing midlife and cognitive health. A systemic review and meta-analysis by Zhu et al. (2024) reported that cognitive training programs conducted during midlife improved working memory, attention, and executive function, with moderate effect sizes. These outcomes emphasize that midlife may be an optimal period for cognitive health interventions before a more pronounced decline develops.

Comorbid Interventions Affecting Midlife Cognition

In addition to lifestyle and demographic variables, comorbid health conditions prevalent in midlife may also influence cognitive health functioning. For instance, Begasse de Dhaem and Robbins (2022) reviewed research on the cognitive impact of primary and secondary headache disorders. These conditions, such as migraines and tension-type headaches, often peak during midlife (Begasse de Dhaem & Robbins, 2022). Their synthesis revealed that individuals with chronic headache disorders frequently experience impairments in attention, working memory, and executive functioning (Begasse de Dhaem & Robbins, 2022). These findings suggest that cognitive difficulties during midlife may also arise from underlying neurological conditions unrelated to aging or hormonal transitions.

Underrecognized Changes and Public Health Relevance

Despite this growing body of research, cognitive changes in midlife are frequently underrecognized or misattributed to stress, multitasking, or normal aging. Karmangla et al.'s (2017) study analyzed data from the Study of Women's Health Across the Nation

(SWAN) and reported that even in healthy midlife women, declines in processing speed and memory declines were detectable over time. Such findings suggest that subtle yet meaningful cognitive changes in midlife warrant clinical attention and may serve as early indicators for later-life cognitive health risks.

This literature included in this section will show that interactions between demographic, behavioral, and neurobiological factors may influence midlife cognitive health. While the onset of cognitive changes may be gradual, this life stage represents an important opportunity for the early risk identification and the implementation of protective strategies. Recognizing the menopausal transition as a critical period for cognitive health may guide preventative interventions and inform public health approaches and policy reform.

Intersectional Pathways: Mold Exposure, Menopause, and Cognitive Health Risks

The intersection of mold exposure and hormonal transitions represents an emerging concern in cognitive health research. Midlife women, particularly those undergoing the menopausal transition, may face unique neurocognitive risks due to the combined effects of indoor environmental toxicants, like mold, and fluctuating sex hormones. Although studies have traditionally examined mold exposure and menopause as separate domains, growing evidence supports a converging pathway that warrants further exploration.

Environmental Toxicants and Neurocognitive Risk

Mold Exposure has long been associated with various respiratory and allergic outcomes. However, its neurological implications are only beginning to gain attention.

Du et al. (2020) reviewed research on indoor mold exposure characteristics and health outcomes. They reported that molds' microbial volatile organic compounds (mVOCs) can penetrate the blood-brain barrier, potentially contributing to cognitive dysfunction. Cox-Ganser (2015) and Pert (2022) echoed this concern by identifying mold-related biotoxins such as mycotoxins and fungal fragments as plausible contributors to nonallergic systemic symptoms, including memory impairment and concentration difficulties, often observed in sick building syndrome.

Animal studies further support this biological plausibility. Guo et al. (2021) used [1H-13C]-nuclear magnetic resonance (NMR) techniques to identify altered metabolic kinetics in rats exposed to environmental toxicants in different brain regions. Their findings suggested that external exposures can differently impact neurochemical processes, with potential relevance to humans exposed to indoor mold (Guo et al., 2021). Similarly, Shiue (2015), using NHANES 2005-2006 data, found that mildew odors in housing were significantly associated with poor self-rated health, asthma, sleep disturbances, and neurological complaints among adults, many of which overlap with the symptoms experienced during menopause.

Hormonal Vulnerability and Environmental Sensitivity

The menopausal transition involves hormonal fluctuations that influence inflammatory pathways, oxidative stress, and neuroprotection. Wang et al. (2024) reported that air pollution exposure during menopause was associated with altered sex hormone levels, including estrogen and progesterone. These findings suggest that environmental exposures during this transition may exacerbate hormonal dysregulation, amplifying vulnerability to neurological consequences. Allegra et al. (2023) noted that sex-based differences in oxidative stress responses may make women more susceptible to environmental toxicants, particularly during hormonally sensitive periods.

In this context, mold exposure could act as an environmental stressor that interacts with the hormonal instability of menopause to intensify cognitive symptoms. This potential synergy is underscored by the work of Sharpe et al. (2015), who reported that exposure to indoor fungi in NHANES participants from the 2005-2006 data was associated with a variable risk of allergic and systemic responses, influenced by individual susceptibility factors such as age, sex, and immune function.

Disproportionate Impact of Low-Income and Urban Communities

Environmental injustice further compounds this risk, particularly for women living in low-income or urban housing environments. Dressel et al. (2021) showed the importance of sustained, nurse-led community partnerships to address structural inequities in environmental exposures. Similarly, Grant and Wood (2022) reported that urban residents face an increased risk of allergen exposure and related health conditions, including those caused by mold. Substandard housing conditions and limited access to preventative health care or early cognitive screening may doubly burden women undergoing menopause in these settings.

Ran et al. (2023) and Wen et al. (2022), both using NHANES 2005-2006 data, found that exposure to environmental agents like metals and fungi was significantly associated with immune sensitization and asthma. These findings show that environmental hazards can contribute to brain inflammation causing cognitive decline.

Methodological Challenges and Future Directions

Methodological challenges and limitations persist despite this emerging body of research. LaKind et al. (2012) cautioned that linking chemical exposures to chronic disease using NHANES data requires caution because of potential confounding and reverse causation. Similarly, Sun and Lipsitz (2018) emphasized the importance of advanced statistical methods and longitudinal studies to better evaluate how combined exposures affect cognitive outcomes. The literature shows that mold exposure and the menopausal transition often overlap and should not be viewed separately. Instead, these factors may interact to increase the risk of cognitive health concerns. The findings from this study may contribute to future research that examines how these variables work together using models that include environment, biology, and social factors.

Socioeconomic, Environmental, and Housing Inequities

Structural inequities in housing, environmental exposures, and neighborhood resources are key determinants of health disparities, particularly among marginalized populations. For midlife women navigating the hormonal and physiological shifts of menopause, these factors may converge to create a cumulative burden that negatively impacts cognitive health. Although environmental and biological stressors have been widely studied independently, factors like housing quality and socioeconomic status play critical roles in shaping exposure risk and overall health outcomes.

Environmental Health Disparities in Housing

People living in low-income communities are more likely to experience poor housing conditions that adversely affect their health. Jacobs (2011) reported that these communities are more exposed to indoor air quality issues like mold, inadequate ventilation, and structural damage. These issues stem from longstanding inequities in housing policies and enforcement, which can lead to chronic health problems.

Bovell-Ammon et al. (2021) emphasized the direct link between housing and health, particularly regarding federal underinvestment in affordable housing infrastructure. Their call for policy reform underscored how inadequate housing perpetuates health inequities, including those related to cognitive function, by sustaining conditions of chronic stress, environmental exposure, and neighborhood disinvestment (Bovell-Ammon et al., 2021). These structural deficits may be especially detrimental during critical periods, such as menopause, when physiological resilience is already challenged.

Additional research indicates that environmental injustices are deeply embedded in the urban housing experiences of low-income women. For example, Grant and Wood (2022) found that urban residence was strongly associated with increased allergen exposure, exacerbating risks for asthma and respiratory conditions. These adverse health outcomes share overlapping inflammatory pathways with neurological health outcomes (Grant & Wood, 2022). Dressel et al. (2021) also illustrated how sustained, community-based partnerships play a role in addressing inequitable environmental conditions that disproportionately affect underserved populations.

Historical and Structural Contexts of Housing Inequity

Housing disparities have deep historical and political roots. von Hoffman (2012) examined the development of low-income housing in the United States and described how long-standing practices like redlining, urban disinvestment, and discriminatory zoning have created lasting health risks for certain populations. Midlife women, especially those living in disadvantaged communities, are more likely to be exposed to environmental hazards, like mold, without the financial means to relocate or make repairs. These structural challenges can lead to both physical and cognitive health problems.

Intersection of Socioeconomic Status and Environmental Exposure

Recent research has examined how socioeconomic status (SES) and environmental exposures combine to affect health outcomes. Christensen et al. (2022) investigated how air pollution and neighborhood SES relate to cognitive decline and reported that people in low-SES areas experienced both higher pollution levels and increased risk for cognitive issues. These results suggest that social and environmental stressors interact together rather than operate independently. This relationship is especially important for people in midlife who may be more vulnerable when living in underserved communities.

Research using data from the NHANES 2005–2006 cycle has also shown connections between environmental toxicants and immune sensitization. Ran et al. (2023) found that exposure to certain metals was associated to higher immunoglobulin E (IgE) levels, suggesting allergic responses that may contribute to neurological inflammation. Similarly, Wen et al. (2022) reported that fungal sensitization, particularly to *Aspergillus* species, was associated with higher rates of asthma diagnoses among children in the

United States. These immune system changes may have lasting effects, including increased brain inflammation, which is a known contributor to cognitive decline.

Albadrani (2025) reported that people living in poverty faced higher mortality rates associated with indoor air pollution issues. While the study focused on mortality, it also points to other concerns, like chronic inflammation, oxidative stress, and poor access to healthcare. These interconnected issues my also influence cognitive health.

Implications for Policy and Research

To improve cognitive health in midlife, there must be a stronger focus on environmental justice, safe housing, and equitable social policies. Existing research indicates that living in poverty exacerbates the harmful effects of both environmental toxins and hormonal changes, which can raise the risk of adverse cognitive health outcomes. Future research should employ approaches incorporating neighborhood-level variables and longitudinal designs to better capture the relationship between social determinants and biological vulnerability.

Summary and Conclusions

The literature reviewed in this chapter emphasizes the increasing understanding that cognitive health in midlife is shaped by a combination of biological, environmental, and social factors. There is strong evidence that hormonal changes during menopause can negatively affect cognitive health. These changes often impact memory, attention, and decision-making, and are shaped by the duration of estrogen exposure over time.

Simultaneously, mold exposure has been increasingly associated with neurological and psychological outcomes. Although initial studies emphasized respiratory illness and immune dysregulation, new evidence indicates that mold and mycotoxins may induce oxidative stress, disrupt the blood-brain barrier, and trigger neuroinflammation, which are mechanisms directly relevant to cognitive impairment. Occupational studies, animal models and public health research have suggested that these biological processes are not only plausible but prevalent in environments that are damp, water damaged, and have poor indoor air quality.

The link between mold exposure and the menopausal transition is an important concern that remains insufficiently studied. Existing research shows that broader systemic issues exacerbate these risks. Women living in low-income or poor-quality housing are more likely to be exposed to mold and other environmental hazards. They also face more barriers getting healthcare, early diagnosis, and preventive care.

Finally, socioeconomic and environmental injustices form a critical framework for understanding how these health risks develop. Factors such as neighborhood disadvantage, decades of disinvestment in housing, and racially patterned environmental exposures compound the challenges faced by midlife women undergoing the menopausal transition. Collectively, the literature emphasizes the importance of examining mold exposure and cognitive health together.

In this chapter, I established the foundation for understanding how mold exposure, menopause, and social determinants may converge to influence cognitive outcomes in midlife women, which is a topic that remains a critical yet underexplored public health issue. While prior researchers have explored these factors independently, few studies have examined their interaction. The purpose of this study sought to address this gap by investigating associations between reported mold exposure, menopausal status, and cognitive health among women in the United States, focusing on socioeconomic disparities.

Chapter 3, I will present the methodology and research approach used to examine these relationships, including the selected data source, chosen variables, analytical framework, and procedures to address the identified gap in the literature.

Chapter 3: Research Method

Introduction

In Chapter 2, I provided a comprehensive review of the literature on mold exposure, cognitive health outcomes, and the menopausal transition, particularly among midlife women living in low-income housing. Through the review, I identified critical gaps in existing research, including limited attention to how environmental and biological risk factors interact to influence adverse cognitive health outcomes in midlife women. I also presented the theoretical and conceptual foundations of the study, grounded in Bronfenbrenner's ecological systems theory (EST). In Chapter 2, I also examined the roles of socioeconomic disparities, environmental injustice, and vulnerabilities of the studied population.

In this chapter, I describe the methodology used to explore the association between self-reported mold exposure and cognitive health outcomes among midlife women undergoing the menopausal transitions. The purpose of this study was to determine whether menopausal transitions served as a moderating or mediating variable in the relationship between mold exposure and cognitive health. Socioeconomic factors, like education and income, were included as covariates.

In this chapter, I outline the research design, target population, sampling procedures, instrumentation, operationalization of variables, data analysis plan, threats to validity, and ethical considerations. A cross-sectional, quantitative approach was employed using secondary data from the 2005-2006 National Health and Nutrition Examination Survey (NHANES). In this study, I utilized multivariate logistic regression to examine the predictive relationship between mold exposure and cognitive health. In this analysis, I conducted moderation and mediation testing using Hayes' PROCESS macro (Model 1 for moderation and Model 4 for mediation) to evaluate the influence of menopausal transition on these associations. The findings from this research may inform public health initiatives, policy developments, and housing standards that aim to reduce adverse cognitive health risks associated with mold exposure and menopausal transition in vulnerable populations.

Research Design and Rationale

In this study, I examined the role of menopausal transition in the relationship between mold exposure and cognitive health among women living in low-income housing, while considering socioeconomic factors like income and education. The independent variable was self-reported mold exposure; the dependent variable was cognitive health. The moderating and mediating variable was menopausal transition, and the covariates included income and education level. I selected a cross-sectional correlational design to explore relationships among mold exposure, menopausal transition, and cognitive health in midlife women. This design allowed me to analyze associations within a defined population at a single point in time (Frankfort-Nachmias & Nachmias, 2008). I selected the 2005-2006 NHANES dataset because it includes a unique self-reported measure of mold exposure not consistently available in later cycles (NCHS, 2007). Utilizing an existing national dataset enhanced the generalizability of findings and provided a cost-effective, ethically sound method for examining environmental health disparities.

Methodology

Target Population

In this study, I focused on midlife women between the ages of 40 and 60 residing in the United States. This age range aligns with the menopausal transition period defined by the World Health Organization (WHO, 2024). While an exact count of women in this demographic living in low-income housing could not be directly determined from NHANES, national data indicate that women in this age group frequently experience environmental and social disparities that increase their vulnerability to health outcomes such as cognitive impairment (CDC, 2023; NCHS, 2007). In this study, I examined the relationship between mold exposure and menopausal transition in relation to cognitive health, while adjusting for covariates such as socioeconomic status, including income and education.

Sampling and Sampling Procedures

I derived the sample from the 2005-2006 National Health and Nutrition Examination Survey (NHANES) cycle. This nationally representative dataset includes health, demographic, and housing data for the civilian, non-institutionalized U.S. population (NCHS, 2007). Participants included in the final analytic sample met the following inclusion criteria:

- Female
- Aged 40-60 years
- Provided responses to the mold exposure item
- Provided responses to the cognitive functioning question

- Had complete data for key covariates, including education and poverty-to-income ratio (PIR)

Women outside the 40-60 age range or with missing responses on key variables were excluded from the analysis. I applied sampling weights in all analyses to account for NHANES's complex multistage probability sampling design and to enhance the generalizability of the results to the U.S. population (CDC, 2007).

Data Sources

National Health and Nutrition Examination Survey (NHANES)

The National Health and Nutrition Examination Survey (NHANES) administered by the National Center for Health Statistics (NCHS), collects cross-sectional data through interviews and physical examinations (NCHS, 2007). NHANES employs a multistage, stratified sampling design to ensure representation across various subgroups, including minority populations and individuals living below the federal poverty line (NCHS, 2007). I selected the 2005-2006 cycle because it includes a unique self-reported item on household mold exposure: "*In the past 12 months, has your home had a mildew odor or musty smell?*" (NCHS, 2007). This variable is not available in more recent cycles. The dataset also contains demographic and health-related variables relevant to this study, including cognitive functioning, age, income, and education.

Sample Size Calculation

I conducted the sample size calculation based on the first research question: *Does mold exposure predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors among women living in low-income housing in the*

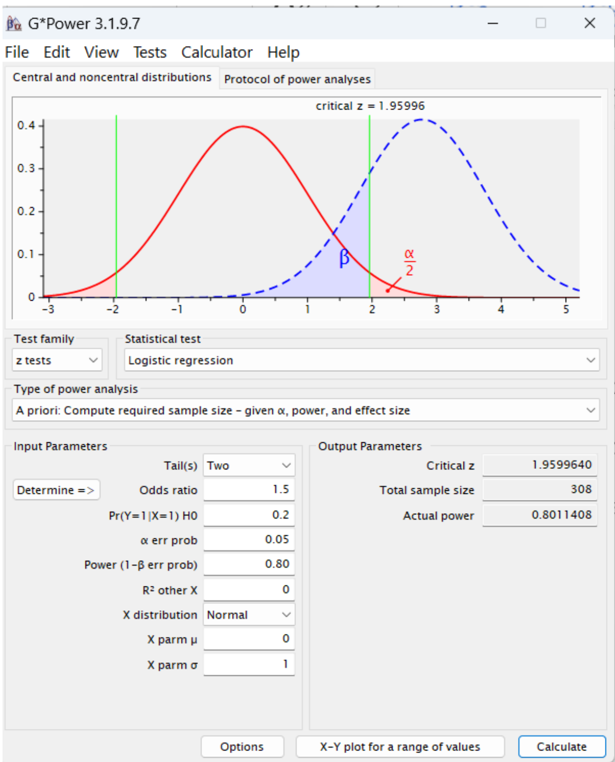
United States? I performed a preliminary power analysis using G*Power 3.1.9.7 software (Faul et al., 2009) to determine the required sample size for the primary (bivariate) relationship between mold exposure and cognitive health outcomes. Using a two-tailed test, an alpha level of .05, a power of 0.80, and an anticipated odds ratio of 1.5, I estimated a minimum required sample size was estimated at 308 participants (OR estimate based on Faul et al., 2009).

To adequately address the entire research question, I included covariates in the multivariate analysis, including menopausal transition (proxied by age), education level, and income (poverty-to-income ratio), were also included. Based on Bujang et al. (2018), logistic regression models should consist of a minimum of 10 to 15 events per covariate to maintain statistical reliability. Accordingly, I added 45 additional subjects (15 for each of the 3 covariates), which increased the sample size requirement to 353.

Including these covariates increased the recommended sample size from 308 to 353 participants to ensure the accuracy and reliability of estimates. Given the NHANES sample design and the expected prevalence of mold exposure and cognitive health issues within the targeted population, I anticipated that the dataset would exceed this threshold. Therefore, I retained all participants who met the inclusion criteria in the analysis to maximize statistical power and generalizability.

Figure 3

*G*Power 3.1.9.7 Output*



Note. G*Power 3.1.9.7 output showing the total sample size (N = 308) needed to detect an odds ratio of 1.5 with 80% power and α = 0.5 using a two-tailed logistic regression analysis.

Instrumentation and Operationalization of Constructs

I used de-identified data from the 2005-2006 National Health and Nutrition Examination Survey (NHANES), a cross-sectional survey administered by the National Center for Health Statistics and publicly available through the Centers for Disease Control and Prevention (CDC). NHANES collects comprehensive health, nutritional, and environmental data through a combination of interviews, physical examinations, and laboratory tests. For this research, I extracted specific variables from the NHANES components, including the Housing Characteristics Questionnaire, Functional Status Questionnaire, and Demographic Files.

I operationalized each variable based on NHANES survey questions that aligned with the constructs under examination. The NHANES instrument contains a wide array of items addressing environmental exposures (such as household mold), health outcomes (such as cognitive functioning), and demographic characteristics. I used self-reported data for all variables, and I recoded binary or categorical responses when necessary to align with the study's quantitative methodology.

Variables

I categorized the variables in this study into three types: independent, dependent, and covariates. These variables were derived from secondary data collected through the 2005-2006 National Health and Nutrition Examination Survey (NHANES).

- Independent Variable (Mold Exposure)

  I measured mold exposure using a self-reported item from the NHANES housing questionnaire that asked whether the home had a musty or mildew odor during the past 12 months. I coded responses as binary (1 = Yes, 0 = No).

- Dependent Variable (Cognitive Health)

  I measured cognitive health using a self-reported item from the functional status questionnaire that asked whether the respondent has difficulty with memory or experienced periods of confusion. I coded responses as binary (1 = Yes (Cognitive Difficulty), 0 = No (No Cognitive Difficulty)).

- Theoretical Variable (Menopausal Transition)

I operationalized menopausal transition using age as a proxy variable. Women aged 40 to 60 were included in the sample, with the menopausal transition estimated based on standard biological benchmarks rather than laboratory hormone levels.

I included two additional covariates to control for socioeconomic status: education and income. The poverty-to-income ratio (PIR), treated as a continuous variable, represented income levels relative to federal poverty guidelines. Gender was coded as a categorical variable with values for males and females. However, for this study, I included only female participants in the analytical sample to ensure the findings were relevant to the menopausal transition and cognitive health outcomes. This restriction supported the study's focus on women in midlife navigating biological changes and environmental exposures within low-income housing.

Table 1

Summary of Variables

Variable/Name	Data Type	Survey Question	Data Values or Units
Cognitive Health (D)	Categorical	Over the last 2 weeks, how often have you been bothered by the following problems: trouble concentrating on things, such as reading the newspaper or watching TV?	0 = No Cognitive Difficulty, "Not at all" 1 = Cognitive Difficulty: "Several days," "More than half the days," or "Nearly every day"

Variable/Name	Data Type	Survey Question	Data Values or Units
Mold Exposure (I)	Categorical	In the past 12 months, has your home had a mildew odor or musty smell?	1 = Yes 0 = No
Menopausal (C/Second I & M) Transitions	Categorical Based on Menopausal Stages	Age in years of the participant at the time of screening.	Perimenopause (40-44 years) Menopause (45-55 years) Post-Menopause (56-60 years)
Poverty-To-Income Ratio (PIR) (C)	Continuous	Poverty Income Ratio (PIR) – A ratio of family income to poverty threshold	1.0: Exactly at Poverty Line 0.5: Indicates the family income is 50% of the poverty threshold 2.0: Indicates the family income is 200% of the poverty threshold
Education Level (C)	Categorical	What is the highest grade or level of school {you have/SP has} completed or the highest degree {you have/s/he has} received?	Less than 9th grade9th -11th grade High school graduate/GED Some college or AA

Variable/Name	Data Type	Survey Question	Data Values or Units
			College graduate or above
Gender	Categorical	Gender	All Males will be excluded from the study

Note. *(I) = independent variable, (D) = dependent variable, (M) = Moderator/Mediator, (C) = Covariate

Data Analysis Plan

I conducted all statistical analyses using IBM SPSS Statistics (Version 30) and the PROCESS macro developed by Hayes (2018). I used a multivariate logistic regression model to assess whether mold exposure predicted cognitive health outcomes while adjusting for menopausal transition, education, and income. I applied descriptive statistics to summarize the sample's demographic characteristics, including age, education level, and poverty-to-income (PIR). The outcome variable, cognitive health, was coded as a binary variable and I analyzed it using odds ratio (OR) and 95% confidence intervals (CIs) to determine the strength and direction of associations.

I performed moderation analysis using Hayes' PROCESS Model 1 to assess whether age (as a proxy for the menopausal stage) moderated the relationship between mold exposure and cognitive health. I also conducted mediation analysis using PROCESS Model 4 to examine whether PIR mediated the relationship between mold exposure and cognitive health. I applied NHANES sampling weights and design variables, where appropriate, to account for the complex survey design and ensure generalizability to the U.S. population (CDC, 2007).

Research Questions and Hypotheses

I addressed three primary research questions in this study to fill the gaps identified in the literature. These questions assessed the associations between self-reported mold exposure, menopausal transition, and cognitive health outcomes while accounting for socioeconomic variables such as education and income. I paired each research question with a corresponding null and alternative hypothesis to support statistical testing.

Research Question 1 (RQ1): Does mold exposure predict cognitive health outcomes after adjusting for menopausal transition, and socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0 1$): Mold exposure does not predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1 1$): Mold exposure does predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

RQ1 Statistical Test

I used a multivariate logistic regression to assess whether mold exposure predicted binary cognitive health outcomes, adjusting for menopausal transition (proxied by age), education, and income (PIR).

Research Question 2 (RQ2): Does menopausal transition moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$2): Menopausal transition does not moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$2): Menopausal transition moderates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

RQ2 Statistical Test

I conducted a moderation analysis using Hayes' PROCESS macros Model 1 to examine whether age (as a proxy for menopausal transition) moderated the relationship between mold exposure and cognitive health, adjusting for education and income.

Research Question 3 (RQ3): Does menopausal transition mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$3): Menopausal transition does not mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$3): Menopausal transition mediates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

RQ3 Statistical Test

I conducted mediation analysis using Hayes' PROCESS macro Model 4 to determine whether age (as a proxy for menopausal transition) mediated the relationship between mold exposure and cognitive health after adjusting for education and income.

I used multivariate logistic regression, along with moderation and mediation analyses, to statistically assess the relationships among mold exposure, menopausal transition, and cognitive health outcomes. These analyses evaluated binary outcomes and modeled both direct and indirect effects while adjusting for key covariates like education and income. I selected each statistical test to address a specific research question and to test whether menopausal transition acted as a moderator or mediator in the relationship between mold exposure and cognitive health. Table 2 outlines the variables included in each model and the corresponding statistical tests.

Table 2

Statistical Tests Used in Data Analysis

Research Question	Variable	Statistical Test
RQ1	Mold Exposure (I) Cognitive Health (D) Menopausal Transition (Age) (C / Second I) Education (C) PIR (C)	Multivariate Logistic Regression
RQ2	Mold Exposure (I) Cognitive Health (D) Menopausal Transition (Age) (M) Education (C) PIR (C)PIR (C)	Moderation Analysis (PROCESS Model 2)
RQ3	Mold Exposure (I) Cognitive Health (D) Menopausal Transition (Age) (M) Education (C) PIR (C)PIR (C)	Mediation Analysis (PROCESS Model 4)

Note. *(I) = independent variable, (D) = dependent variable,
(M) = Moderator/Mediator, (C) = Covariate, PIR = Poverty-to-Income Ratio

Threats to Validity

Internal Validity

Because I relied on self-reported data from NHANES, potential threats to internal validity included measurement bias and recall error. I used age as a proxy for menopausal status because the dataset did not include direct measure of this variable, which may have introduced some misclassification. Additionally, since I employed a cross-sectional design, the analysis identified associations rather than establishing causality.

External Validity

Because of the NHANES sampling design, the findings from this study can be generalized to the broader U.S. population. However, I analyzed mold exposure data from the 2005-2006 cycle, which limited temporal generalizability. Therefore, temporal generalizability was limited. Additionally, findings were not generalizable to populations outside the 40-60 age range or men.

Construct or Statistical Conclusion Validity

I identified potential threats to construct validity because the survey questions for both mold exposure and cognitive function came from a single self-reported measure on the NHANES 2005-2006 dataset. Furthermore, using age as a proxy for menopausal transition may not have fully represented hormonal changes. Grounding the variable selections in previous literature helped mitigate potential concerns.

Statistical Conclusion Validity

I conducted a power analysis using G*Power 3.1.9.7 software (Faul et al., 2009) to ensure a sufficient sample ($N \geq 353$), following logistic regression guidelines (Bujang et al., 2018). The NHANES data implemented a complex sampling design, and I applied the appropriate sampling weights (NCHS, 2007; CDC, 2007) to improve accuracy and generalizability.

Ethical Procedures

I used secondary data from the NHANES 2005-2006 cycle, which are publicly available and de-identified. As such, I did not have direct contact with participants, and the dataset included no identifying information. The original NHANES study obtained informed consent from all participants, and their data collection complied with the U.S. Department of Health and Human Services regulations for human subject's research (NCHS, 2007). I stored all NHANES files on a password-protected computer only accessible to me. Before conducting any data analysis, I obtained approval from Walden University's Institutional Review Board (IRB) (Approval # 07-14-25-1010203).

Summary

In this chapter, I outlined the methodology for a cross-sectional, quantitative analysis of the relationship between mold exposure and cognitive health among midlife women. I used data from the 2005-2006 NHANES cycle to analyze environmental and biological factors affecting cognitive health outcomes. I applied logistic regression and Hayes' PROCESS models to test direct, moderating, and mediating effects. Through this methodological approach, I sought to provide a multifaceted understanding of how mold exposure and socioeconomic status interact with menopausal transition to influence cognitive health.

In Chapter 4, I present the results of the statistical analyses, including descriptive statistics, logistic regression findings, and the outcomes of the moderation and mediation analyses. I organized the results by research question and hypothesis to demonstrate the relationships among mold exposure, menopausal transition, socioeconomic factors, and cognitive health outcomes in midlife women.

Chapter 4: Results

Introduction

In Chapter 4, I present the statistical analysis and their results that were discussed in Chapter 3. This chapter organizes and presents the findings according to each research question and corresponding hypothesis.

The purpose of this study was to examine whether mold exposure and menopausal transition predict cognitive health outcomes after adjusting for socioeconomic factors, such as education and income, among midlife women living in low-income housing in the United States. This study also examined whether menopausal transition moderates or mediates the relationship between mold exposure and cognitive health. Environmental, biological, and social factors served as overarching themes of the study. Each of the factors served as a specific indicator for the study. Environmental factors included the reported mold exposure within the property. Biological factors consisted of menopausal transition status. Social factors included socioeconomic status, measured through education level and poverty-to-income ratio (PIR).

The research questions and hypotheses were organized according to these key indicator groupings. I conducted analyses to determine whether mold exposure was directly associated with cognitive health outcomes. I also examined whether menopausal transition moderated or mediated this relationship after adjusting for socioeconomic factors, like education and income.

The purpose of this chapter is to present the results organized by research questions and hypotheses, and include descriptive statistics, a logistic regression model, and the results of the moderation and mediation analyses. Each section provides evidence regarding the relationships among mold exposure, menopausal transition, socioeconomic factors, and cognitive health outcomes among midlife women in the United States.

This study answered the following research questions and hypotheses:

Research Question 1 (RQ1): Does mold exposure predict cognitive health outcomes after adjusting for menopausal transition, and socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis (*H_0*1): Mold exposure does not predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis (*H_1*1): Mold exposure does predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Research Question 2 (RQ2): Does menopausal transition moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis (*H_0*2): Menopausal transition does not moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$2): Menopausal transition moderates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Research Question 3 (RQ3): Does menopausal transition mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$3): Menopausal transition does not mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$3): Menopausal transition mediates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

The chapter presents descriptive statistics of the study sample, followed by results from the logistic regression, moderation, and mediation analyses. A summary of key findings concludes the chapter, preparing for interpretation and discussion in Chapter 5.

Data Collection

After receiving approval, I downloaded secondary data from the National Health and Nutrition Examination Survey (NHANES) 2005–2006 cycl from the Centers for Disease Control and Prevention (CDC) website. NHANES employs a multistage stratified probability sampling design to ensure representativeness of the civilian, noninstitutionalized U.S. population (CDC, 2007a; CDC, 2007b). For this study, the analytic sample was restricted to women between the ages of 40 and 60 who responded to the items on mold exposure, cognitive health, education, and poverty-to-income ratio (PIR).

I did not directly recruit participants because the study relied on publicly available, de-identified secondary data. Instead, I obtained information from the original 2005–2006 NHANES survey of the individuals that were recruited and participated in the study. The survey ensured representation of low-income and minority populations using oversampling strategies (CDC, 2007a; CDC, 2007b). All women who met the inclusion criteria were retained for analysis, and a weighted sample was applied (WTINT2YR) (CDC, 2007a; CDC, 2007b).

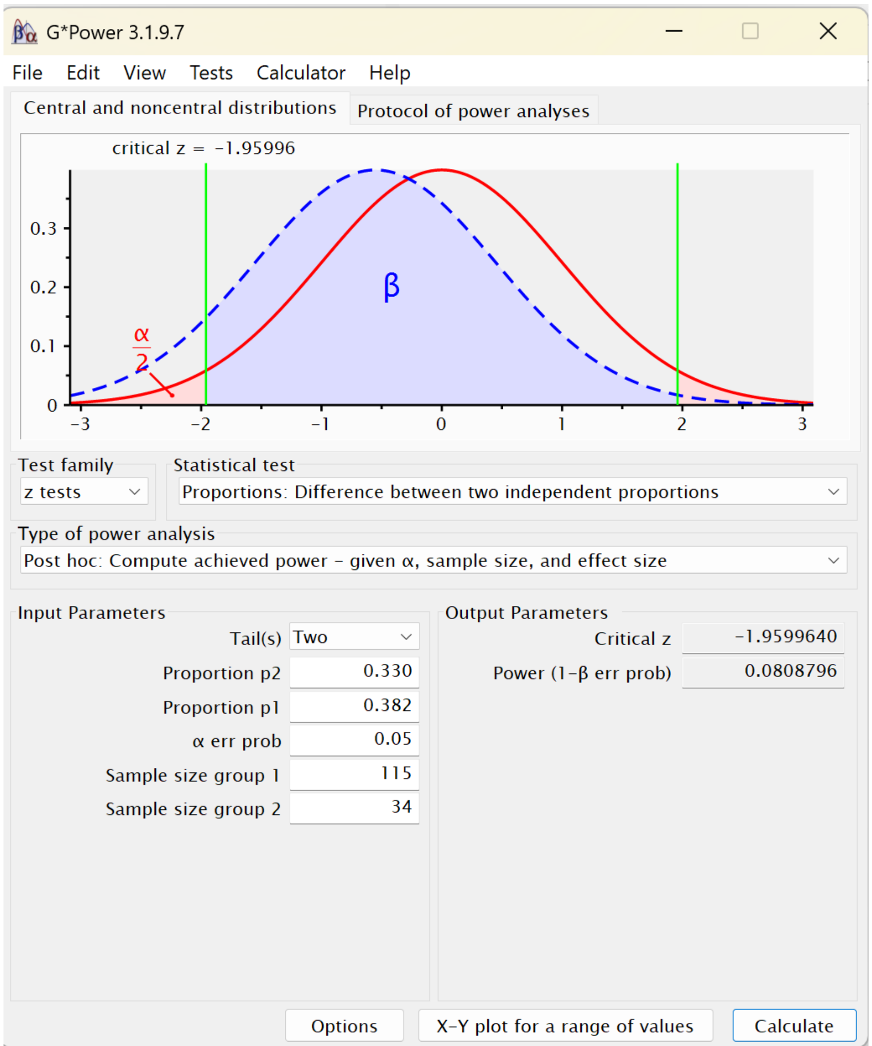
There were no deviations from the overall data collection plan described in Chapter 3. As discussed, participants who did not meet the inclusion criteria, such as those individuals outside the 40–60 age range, males, or those missing responses to key variables, were excluded from the analytic dataset. Sampling weights (WTINT2YR) were applied in all analyses to account for the complex NHANES survey design.

Two methodological considerations were noted. First, the a priori G*Power analysis indicated that a minimum of 308 participants, or 353 with the addition of covariates, was needed to achieve 80% power to detect an odds ratio of 1.5 at an alpha level of .05. However, the final unweighted analytic sample consisted of 149 women who met all inclusion criteria and provided complete responses to all study variables. To evaluate the impact of this shortfall, a post hoc sensitivity analysis was conducted in G*Power using the observed proportions of cognitive difficulty among the sampled women who were exposed to mold (38.2%), and those who were unexposed (33.0%). With the final sample size (N = 149) and an alpha level of .05, the achieved statistical power was approximately 8.1% (see Figure 4). This result indicated that the analysis was underpowered to detect small effect sizes, and this limitation is acknowledged in the limitations section of interpretation of findings in Chapter 5.

Second, during the moderation analysis, an error occurred when attempting to run PROCESS Model 2 in the most recent version of the Andrew Hayes macro (5.0). Model 2 was considered because it can be used for analyses involving more than one moderator. However, because the moderator variable for menopausal transition was coded with more than two categories, the model could not be executed. PROCESS Model 1 was used instead to address this issue. PROCESS Model 1 is widely applied for moderation analysis to produce equivalent information regarding the interaction between menopausal transition and mold exposure (Hayes, 2018; Hayes, 2022). This adjustment did not alter the conceptual alignment with research Question 2 but represented a practical change in response to a software limitation.

Figure 4

*G*Power 3.1.97 Output Showing Post Hoc Sensitivity Analysis With Proportions Of Cognitive Difficulty Among Women Who Were Exposed And Unexposed To Mold*



Note. The figure displays the post hoc sensitivity analysis conducted in G*Power 3.197. Observed proportions of cognitive difficulty were 38.2% for mold-exposed women and 33.0% for unexposed women, with a total analyzed sample size of 149. At $\alpha = .05$ (two-tailed), the statistical power achieved was approximately 8.1%

The final weighted analytic sample consisted of women aged 40–60 years, with variability in education and income levels. Education ranged from less than high school to a college graduate. PIR reflected a spectrum of socioeconomic conditions, including participants at or below the federal poverty threshold (0.5: indicates the family income is 50% of the poverty threshold) (CDC, 2007a; CDC, 2007b). Weighted descriptive statistics of the study sample are presented in the results section. These characteristics illustrate the diversity of the analytic sample and support the generalizability of findings for midlife women residing in the United States.

Using the NHANES dataset ensures that the sample was representative of the broader population of women aged 40–60 living in the United States. NHANES used a stratified sampling design which oversampled key subgroups, like low-income populations. This strengthened external validity and supported generalizability to the broader population (CDC, 2007a; CDC, 2007b). By applying NHANES sampling weights it allowed the results to represent the broader population while maintaining proportional representation of subgroups (CDC, 2007a; CDC, 2007b).

Univariate analyses were conducted to determine whether the multivariate models should include education and poverty-to-income ratio (PIR) as covariates. The weighted analyses showed that education and PIR were significantly related to cognitive health outcomes. However, only education showed a significant association in the unweighted analyses, while PIR was not statistically significant.

Because these socioeconomic factors showed meaningful associations, particularly under the weighted analyses that best represent the U.S. population, they were retained as covariates in the logistic regression, moderation, and mediation models. Including them as control variables helped ensure that the observed relationships between mold exposure, menopausal transition, and cognitive health were not explained simply by differences in education or income.

Study Results

Descriptive Statistics

I analyzed a nationally representative sub-sample of women residing in the United State**s,** drawn from the 2005–2006 cycle of the National Health and Nutrition Examination Survey (NHANES). Although NHANES includes respondents across their lifespan, this study restricted the sample to women aged 40 to 60 years (unweighted N = 149; weighted $N \approx$ 7,135,536) to approximate menopausal transition while maintaining alignment with the measurement periods for the variables in the study. All variables used in this section were obtained from the NHANES interview. The variables were analyzed using the WTINT2YR interview weight (CDC, 2007a; CDC, 2007b). Percentages shown in Table 3 are survey-weighted, and the National Population Estimate (NPE) column translates those weighted proportions into thousands of U.S. women.

Mold exposure was operationalized from the NHANES variable which asked respondents whether their home has a mildew or musty smell. Respondents could answer yes, no, or don't know. For this analysis, the variable was recoded into a dichotomous format with only yes and no responses retained. The cognitive outcome was self-reported based on the NHANES 2005-2006 variable titled trouble concentrating on things. Respondents were able to answer: not at all, several days, more than half the days, nearly every day, and don't know. For this analysis, the variable was recoded into a dichotomous format, as any difficulty or none. Because NHANES does not include a clinical menopausal status variable in this cycle, menopausal transition was proxied by age bands reflecting typical stages: perimenopause (40–44), menopause (45–55), and post-menopause (56–60). Education was dichotomized as High School or Less versus College+. College+ included respondents who attended some college, an associate's degree, a bachelor's degree, or any degree higher than a bachelor's degree. Poverty-to income ratio (PIR) was categorized into Low, Mid, and High income groups to capture socioeconomic gradients relevant to both exposure and outcome. All descriptive statistics below are weighted and accompanied by NPE to emphasize population relevance.

Table 3 shows that 1 in 5 midlife women reported a recent musty or mildew odor (20.7%), and nearly 1 in 3 reported cognitive difficulty (31.7%). The age-proxy distribution suggests the majority fall within the 45-55 band (55.6%). Educational attainment is high overall, as 65.0% fall within the College+ category. PIR indicates that almost 1 in 7 are in low-income households (family income ratio under 0.50 or 50% of the poverty threshold) (14.3%) and nearly 2 in 3 fall in the high-income category (family income ratio is 2.0 or 200% of the poverty threshold) (64.4%). These distributions provide the population context for subsequent weighted regression, moderation, and mediation analyses to answer each research question.

Table 3

Frequency Distribution of Mold Exposure, Cognitive Difficulty, Menopausal Transition, Education, and PIR Among Females Aged 40–60 in the United States (N = 149, Weighted N ≈ 7,135,536)

Variable	Category	Unweighted *N*	Weighted *N* (NPE)	Weighted % (SE)
Mold Exposure	No	115	5,655,173	79.3 (2.6)
	Yes	34	1,480,363	20.7 (2.6)
Cognitive Difficulty	No	98	4,874,946	68.3 (3.9)
	Yes	51	2,260,590	31.7 (3.9)
Menopause Transition	Perimenopause (40-44)	38	1,984,152	27.8 (4.9)
	Menopause (45-55)	83	3,967,351	55.6 (4.3)
	Post-Menopause (56-60)	28	1,184,034	16.6 (5.1)
Education Level	High School or Less	65	2,500,515	35.0 (5.2)
	College+	84	4,635,020	65.0 (5.2)
PIR Category	Low Income	31	1,017,328	14.3 (3.4)
	Mid Income	39	1,521,033	21.3 (3.6)
	High Income	79	4,597,175	64.4 (4.0)

Note. PIR = Poverty-to-Income. NPE = National Population Estimate. Categories for Menopause Stage are age-based approximations used to reflect typical stages:

perimenopause (ages 40–44), menopause (ages 45–55), and post-menopause (ages 56–60). Education was dichotomized into “High School or Less” and “College or Higher.”

Evaluation of Statistical Assumptions

Before conducting the logistic regression analyses for RQ1, RQ2, and RQ3, I took steps to ensure the assumptions for the logistic regression models were met. These assumptions included an assessment of multicollinearity and linearity in the logit for continuous predictors, as well as identifying potential outliers and influential cases (Daniel & Cross, 2019; Warner, 2013). Because all of the predictors in this study were categorical, the linearity in the logit assumption did not apply, and an evaluation did not occur.

Multicollinearity was assessed by running a linear regression with the same predictors as those included in the logistic regression models and requesting collinearity diagnostics. There was no evidence of multicollinearity, as assessed by tolerance values greater than 0.1. There were no standardized residuals greater than ±2.5 standard deviations and no leverage values greater than 0.2 (Daniel & Cross, 2019). Outliers were examined through Cook’s distance. Cook’s distance values were 0.000, suggesting no evidence of influential data points (Daniel & Cross, 2019). The same diagnostic results apply because Research Question 2 (moderation) and Research Question 3 (mediation) were tested using Hayes’ PROCESS models, which rely on logistic regression frameworks. Therefore, the statistical assumptions were sufficiently met for the logistic regression, moderation, and mediation analyses.

Statistical Findings

Results of Research Question 1

Research Question 1 (RQ1): Does mold exposure predict cognitive health outcomes after adjusting for menopausal transition, and socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis ($H_0$1): Mold exposure does not predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1$1): Mold exposure does predict cognitive health outcomes after adjusting for menopausal transition and socioeconomic factors (education and income) among women living in low-income housing in the United States.

Model Fit

A weighted logistic regression analysis was conducted to answer the first research question. The omnibus test of model coefficients indicated that the model was statistically significant $X^2(6, N = 149) = 823{,}682.53$, $p < .001$, indicating that the predictors reliably distinguished between women with and without reported cognitive difficulty. The model explained approximately 15.3% of the variance in cognitive difficulty (Nagelkerke $R^2 = .153$). The Receiver Operating Characteristic (ROC) curve analysis (see Figure 5) yielded an area under the curve (AUC) of 0.649 (95% *CI* [.555, .742]). These findings suggest poor discrimination, only slightly better than chance (Hosmer et al., 2013).

Results

The results showed that women who reported mold exposure had significantly higher odds of reporting cognitive difficulty compared with those not exposed, after adjusting for menopausal transition, education, and poverty-to-income (OR = 1.768, 95% *CI* [1.761, 1.775], $p < .001$). Menopausal transition was also significant. Compared with women in perimenopause, those in menopause had slightly elevated odds of reporting cognitive difficulty (OR = 1.083, 95% *CI* [1.079, 1.088], $p < .001$), while women in post-menopause had more than twice the odds (OR = 2.072, 95% *CI* [2.061, 2.083], $p < .001$).

Education emerged as a protective factor. Women with at least some college education or higher were less likely to report cognitive difficulty compared with those with a high school education or less (OR = 0.343, 95% *CI* [0.342, 0.345], $p < .001$). The poverty-to-income ratio was also associated with cognitive health outcomes. Women in the mid-income group, compared with women in the low-income category, had increased odds of reporting cognitive difficulty (OR = 1.368, 95% *CI* [1.361, 1.376], $p < .001$). In contrast, the women in the high-income group had significantly lower odds (OR = 0.609, 95% *CI* [0.606, 0.612], $p < .001$).

Conclusion

The findings showed that environmental exposure, socioeconomic status, and menopausal transition stage were essential predictors of self-reported cognitive health in midlife women. Additionally, these results indicated that women exposed to household mold and those in later stages of menopause were more likely to report cognitive difficulties, while higher education and higher income appeared to offer protection. Since mold exposure was a statistically significant predictor of cognitive difficulty after adjusting for menopausal transition, education, and poverty-to-income ratio (OR = 1.768, 95% *CI* [1.761, 1.775], $p < .001$), the null hypothesis for Research Question 1 was rejected. Consistent with the alternative hypothesis, women exposed to mold were more likely to report cognitive difficulty than those who were not. Table 4 presents the results of the logistic regression analysis.

Table 4

Weighted Logistic Regression Summary for the Association Between Mold Exposure and Cognitive Difficulty After Adjusting for Menopausal Transitions, Education, and PIR Among Females Aged 40–60 in the United States (Weighted N ≈ 7,135,536)

Research Question 1

Predictor Variable	OR	95% CI for OR	*p*-value
Mold Exposure (Ref = No)	1.768	[1.761, 1.775]	< .001
Menopause Transition (Ref = Peri)			
Menopause	1.083	[1.079, 1.088]	< .001
Post-Menopause	2.072	[2.061, 2.083]	< .001
Education Level (Ref = HS or Less)	0.343	[0.342, 0.345]	< .001
PIR Category (Ref = Low)			
Mid Income	1.368	[1.361, 1.376]	< .001
High Income	0.609	[0.606, 0.612]	< .001

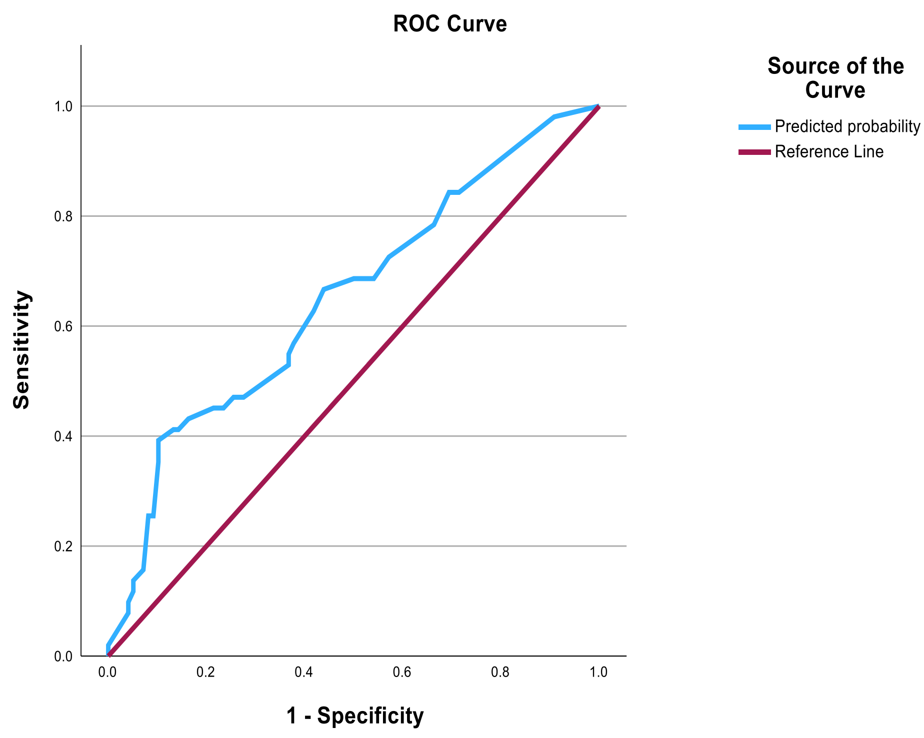
Note. Weighted sampling was conducted using NHANES 2005–2006 interview weights (WTINT2YR). All predictors were statistically significant at $p < .001$. OR = Odds Ratio. CI = Confidence Interval (95%)

Variable(s) entered: Mold Exposure (1=Yes, 0=No), Menopause Stage (1=Peri, 2=Menopause, 3=Post), Education Level (1=College+, 0=HS or Less), PIR Category (1=Low, 2=Mid, 3=High).

Reference categories: No exposure (mold exposure), Perimenopausal (menopausal transition), Low income (PIR category), and High school or Less (education level)

Figure 5

Receiver Operating Characteristic (ROC) Curve for Logistic Regression for the Association Between Mold Exposure and Cognitive Difficulty After Adjusting for Menopausal Transitions, Education, and PIR Among Females Aged 40–60 in the United States (Weighted N ≈ 7,135,536)



Note. The area under the ROC curve was 0.649, 95% CI [.555, .742], which is considered a poor discrimination, not much better than a coin toss according to Hosmer et al. (2013).

Results of Research Question 2

Research Question 2 (RQ2): Does menopausal transition moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (such as education and income) among women living in low-income housing in the United States?

Null Hypothesis (H_02): Menopausal transition does not moderate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis (H_12): Menopausal transition moderates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Model Fit

A weighted moderation logistic regression analysis was conducted to answer the second research question. The overall model was not statistically significant.

Results

The interaction between mold exposure and menopausal transition was not statistically significant after adjusting for education and poverty-to-income ratio (OR = 0.62, 95% *CI* [0.17, 2.25], $p = .4714$), indicating that menopausal transition did not moderate the relationship between mold exposure and cognitive difficulty. Figure 6 further illustrates the nonsignificant interaction. Figure 6 shows that the slopes for cognitive difficulty across menopausal stages were similar regardless of mold exposure. This information confirms the nonsignificant moderation effect.

Although the interaction was not significant, several main effects were examined. Mold exposure alone was not a significant predictor of cognitive difficulty in this model (OR = 2.96, 95% *CI* [0.20, 43.59], $p = .4300$). Menopausal transition, when considered independently, also did not reach statistical significance (OR = 1.58, 95% *CI* [0.86, 2.90], $p = .1403$). The PIR category was not significantly associated with cognitive difficulty (OR = 0.87, 95% *CI* [0.55, 1.36], $p = .5417$). However, education emerged as a significant protective factor. In contrast, women with at least some college or higher education were less likely to report cognitive difficulty compared with women with a high school education or less (OR = 0.41, 95% *CI* [0.20, 0.85], $p = .0169$).

Conclusion

The findings showed that the interaction between mold exposure and menopausal transition was not statistically significant, indicating that menopausal transition did not moderate the relationship between mold exposure and cognitive difficulty. Additionally, these findings indicated that the slopes for cognitive difficulty across menopausal stages were similar regardless of mold exposure. This information suggested that there was no meaningful effect. Since the interaction term was not statistically significant after adjusting for menopausal transition, education, and poverty-to-income ratio (OR = 0.62, 95% *CI* [0.17, 2.25], $p = .4714$), the null hypothesis for Research Question 2 was not rejected. Consistent with the null hypothesis, menopausal transition did not moderate the effect of mold exposure on cognitive health outcomes. Table 5 presents the moderation analysis results, and Figure 6 illustrates the nonsignificant interaction.

Table 5

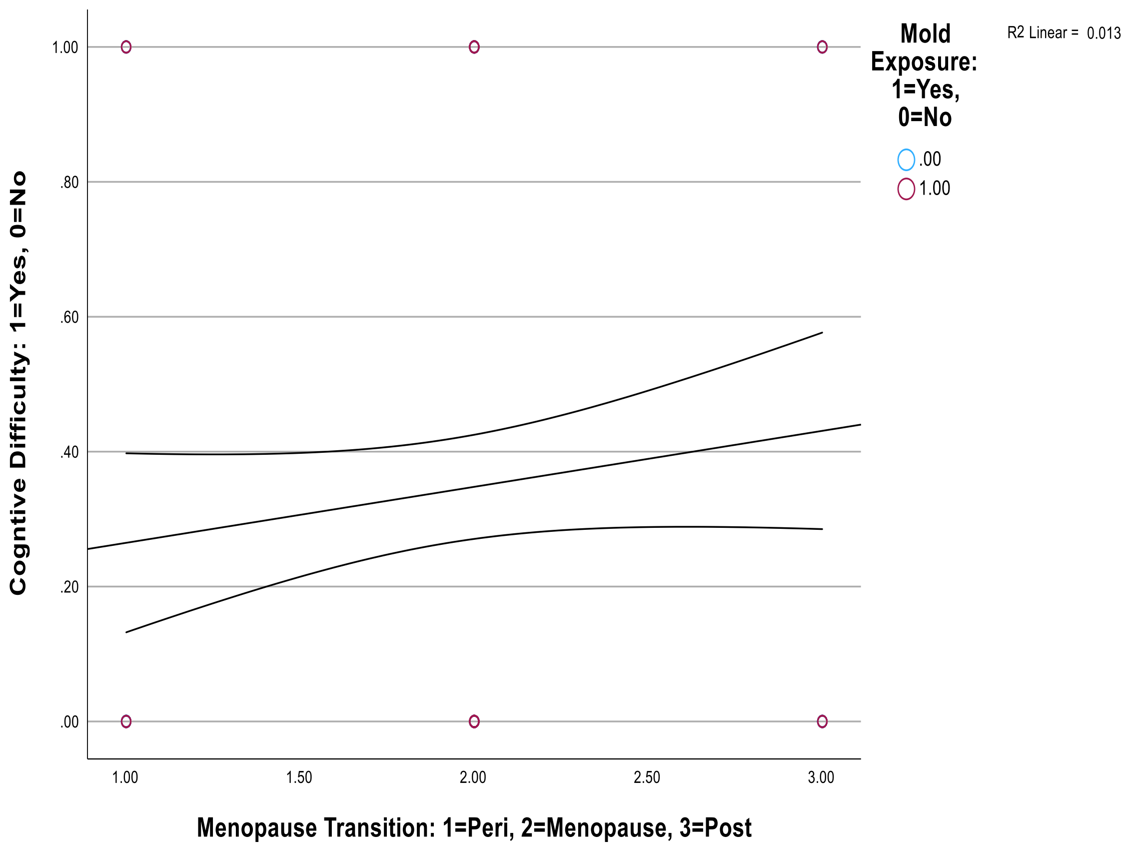
Weighted Moderation Logistic Regression Predicting Cognitive Health from Mold Exposure, Menopausal Transition, and Their Interaction, Adjusting for Education and PIR Among Women Aged 40–60 in the United States (Weighted N ≈ 7,135,536)

Research Question 2			
Predictor	OR	95% CI for OR (Lower, Upper)	*p*-value
Mold Exposure (Ref = No)	2.96	[0.20, 43.59]	.4300
Menopausal Transition (Ref = Peri)	1.58	[0.86, 2.90]	.1403
INTERACTION (Mold Exposure x Menopausal Transition)	0.62	[0.17, 2.25]	.4714
PIR Category (Ref = Low)	0.87	[0.55, 1.36]	.5417
Education Level (Ref = HS or Less)	0.41	[0.20, 0.85]	.0169

Note. Weighted sampling was conducted using NHANES 2005–2006 interview weights (WTINT2YR). The results of the weighted model were identical to the unweighted model, suggesting there was minimal influence of the weighted sampling. Statistical significance was set to $p < .05$. Coded variables: OR = Odds Ratio. CI = Confidence Interval. Int_1 = Interaction term represents MOLDEXPO x MENOTRAN.

Figure 6

Interaction Between Mold Exposure and Menopausal Transition on Cognitive Health



Note. The figure illustrates that the interaction was not significant.

Results of Research Question 3

Research Question 3 (RQ3): Does menopausal transition mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States?

Null Hypothesis (H_03): Menopausal transition does not mediate the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Alternative Hypothesis ($H_1 3$): Menopausal transition mediates the relationship between mold exposure and cognitive health after adjusting for socioeconomic factors (education and income) among women living in low-income housing in the United States.

Model Fit

A weighted mediation logistic regression analysis was conducted to answer the third research question. The overall model was not statistically significant, indicating the findings did not support a mediation effect.

Results

Mold exposure was not a statistically significant predictor of cognitive difficulty in this model after adjusting for menopausal transition, education, and poverty-to-income ratio (OR = 1.14, 95% *CI* [0.50, 2.61], $p = .7498$). Menopausal transition, analyzed as a mediator, was also not statistically significant after adjusting for education and poverty-to-income ratio (OR = 1.42, 95% *CI* [0.56, 1.37], $p = 1.945$). The PIR category was not statistically significant to cognitive difficulty (OR = 0.87, 95% *CI* [0.55, 1.36], $p = .5471$). However, education was a significant protective factor, with women who had at least some college or higher education demonstrating lower odds of reporting cognitive difficulty compared with those with a high school education or less (OR = 0.41, 95% *CI* [0.20, 0.85], $p = .0169$).

Conclusion

The findings showed that neither mold exposure nor menopausal transition was a statistically significant predictor of cognitive difficulty in the mediation model. These results indicated that menopausal transition did not mediate the relationship between mold exposure and cognitive health. Additionally, these results suggested that the indirect pathway through menopausal transition was not supported, while higher education remained a significant protective factor. Since both mold exposure (OR = 1.14, 95% *CI* [0.50, 2.61], $p = .7498$) and menopausal transition (OR = 1.42, 95% *CI* [0.56, 1.37], $p = 1.945$) were not statistically significant after adjusting for education and poverty-to-income ratio, the null hypothesis for Research Question 3 was not rejected. Consistent with the null hypothesis, menopausal transition did not mediate the effect of mold exposure on cognitive health outcomes. Table 6 presents the mediation analysis results.

Table 6

Weighted Logistic Regression Results for Mediation Model: Menopausal Transition as a Mediator Between Mold Exposure and Cognitive Health, Adjusting for Education and PIR Among Women Aged 40–60 in the United States (Weighted N ≈ 7,135,536)

Research Question 3			
Predictor	OR	95% CI for OR (Lower, Upper)	*p*-level
Mold Exposure (Ref = No)	1.14	[0.50, 2.61]	.7498
Menopausal Transition (Ref = Peri)	1.42	[0.56, 1.37]	.1945
PIR Category (Ref = Low)	0.87	[0.55, 1.36]	.5471
Education Level (Ref = HS or Less)	0.41	[0.20, 0.85]	.0169

Note. Weighted sampling was conducted using NHANES 2005–2006 interview weights (WTINT2YR). The results of the weighted model were identical to the unweighted model, suggesting there was minimal influence of the weighted sampling. Statistical significance was set to $p < .05$. Coded variables: COGHEALTH (Cognitive Difficulty: (1 = Yes, 0 = No)); MOLDEXPO (0 = No, 1 = Yes); MENOTRAN (1 = Peri, 2 = Menopause, 3 = Post); PIR_CAT (1 = Low, 2 = Mid, 3 = High); EDUCATIO (0 = HS or less, 1 = College+). OR = Odds Ratio. CI = Confidence Interval.

Summary

The statistical analyses provided insight into the relationship between mold exposure, menopausal transition, socioeconomic factors, and cognitive health among midlife women. For the first research question, weighted logistic regression analysis revealed that mold exposure, menopausal transition, education, and poverty-to-income ratio were all significant predictors of self-reported cognitive difficulty. Women exposed to mold and those in post-menopause were more likely to report cognitive difficulties. In contrast, women with higher education and higher income were less likely to report these problems. Because all predictors were statistically significant ($p < .001$), the null hypothesis for Research Question 1 was rejected.

I tested whether menopausal transition moderated the relationship between mold exposure and cognitive health. The interaction term between mold exposure and menopausal transition was not statistically significant, and neither variable was independently significant in the moderation model. Education remained a protective factor, but because the interaction between mold exposure and menopausal transition was not significant, the null hypothesis for Research Question 2 was not rejected.

The third research question examined whether menopausal transition mediated the relationship between mold exposure and cognitive health. Similar to the moderation model, the mediation analysis did not support a mediating effect of menopausal transition. Education remained as a protective factor, but because there was no mediating effect, the null hypothesis for Research Question 3 was not rejected.

The findings suggest that mold exposure, socioeconomic factors, and menopausal transition stage are relevant to cognitive health outcomes in midlife women in the United States. While mold exposure and menopausal transition were significant in the direct model, only education consistently appeared as a protective effect across all models.

These results highlight the importance of considering both environmental exposures and social determinants of health when evaluating cognitive difficulties in women during the menopausal transition.

In Chapter 5, I interpret these results based on the existing literature, acknowledge the study's limitations, and discuss recommendations for future research. In Chapter 5, I also explore the broader implications of these findings for public health, policy, and practice.

Chapter 5: Discussion, Conclusions, and Recommendations

Introduction

In this study, I examined how mold exposure may be related to cognitive health outcomes in midlife women, specifically those between the ages of 40–60 who reside in low-income housing in the United States. I focused on the role of the menopausal transition, and whether it influences this relationship. This study examined whether menopause acted as a confounding variable, moderator, or mediator in the link between mold exposure and cognitive health difficulties. Using data from the NHANES 2005-2006 cycle, this quantitative cross-sectional study analyzed a nationally representative sample of women aged 40–60 (unweighted $N = 149$; weighted $N \approx 7{,}135{,}536$). Weighted logistic regression models were applied to test three research questions. The first research question examined whether mold exposure predicted cognitive difficulty. The second research question analyzed whether menopausal transition moderated the relationship between mold exposure and cognitive health. The third research question examined whether menopausal transition mediated the relationship between mold exposure and cognitive health.

The findings showed that mold exposure, menopausal transition, and socioeconomic status were significant predictors of cognitive difficulty, but no moderating or mediating effects were observed. In this chapter, I interpret the study's findings, discuss the limitations of the study, present future recommendations and implications for social change, followed by an overall summary of the study.

Interpretation of the Findings

The study's results showed that about one in five (20.7%) of midlife women reported noticing a musty or mildew odor in their homes, and nearly one in three (31.7%) reported some level of cognitive difficulty. The most significant portion of women were between the ages of 45 and 55 (55.6%), reflecting the menopausal stage, while smaller proportions were in perimenopause (27.8%) or post-menopause (16.6%). High educational attainment was relatively high, with nearly two-thirds of women (65.0%) having at least some college education. However, household income showed variation, with 14.3% of women in the low-income category and almost two-thirds (64.4%) in the high-income group.

Environmental Factors and Cognitive Health

The weighted logistic regression model confirmed that mold exposure significantly predicted cognitive difficulty. The findings demonstrated that mold exposure significantly predicted cognitive difficulty among midlife women. Women who reported mildew or musty odors were about 1.8 times more likely to report cognitive difficulty compared with those without exposure. This aligns with prior evidence linking mold exposure to systemic and neurological effects (Campbell & Weinstock, 2022; Chauhdary et al., 2021; Shiue, 2015).

By confirming this association in a large, nationally representative dataset, these findings extend previous clinical and experimental evidence to the broader U.S. population of midlife women. As noted in Chapter 2, most previous research emphasized respiratory or allergic outcomes of mold exposure (Mudarri & Fisk, 2007; Fisk et al., 2010), while neurological impacts remain underexplored. This study helps fill that gap, supporting the plausibility that poor indoor air quality, housing quality, and mold represent environmental neurotoxic risks for women during midlife.

Socioeconomic Factors and Cognitive Health

Socioeconomic factors play a key risk factor in cognitive health. Education was a significant protective factor consistent with prior research (Heiskanen et al., 2024). Women who had some college education had lower odds of reporting cognitive difficulties compared with those who had a high school education or less. Household income also demonstrated a relationship with cognitive health outcomes. Low-income women were at a higher risk of cognitive health issues. High-income women also showed a protective effect.

These findings align with research showing socioeconomic resources as determinants of cognitive health (Christensen et al., 2022; Bovell-Ammon et al., 2021). As discussed in Chapter 2, women in low-income housing face both greater environmental exposures and fewer healthcare resources, creating greater disadvantage. The findings support this framework by demonstrating that mold exposure and socioeconomic inequities independently contribute to cognitive disparities.

Menopausal Transition and Cognitive Health

Women in post-menopause had more than twice the odds of reporting cognitive problems. This aligns with past studies that connect estrogen loss with declines in brain function (Brinton et al., 2015; Sochocka et al., 2023; Mosconi et al., 2025). In Chapter 2, studies such as Than et al. (2023) and Nakanishi et al. (2025) were reviewed and identified cognitive health issues in postmenopausal women. This information reinforces the vulnerability of this life stage.

These findings build upon this literature by documenting the impact of menopausal transition on cognitive health within the context of environmental exposures. While hormonal decline has been well established as a risk factor, few studies have considered its effect alongside mold exposure. This dual approach responds to the gap identified in Chapter 2, where environmental and biological risks were often studied independently.

Moderation and Mediation Findings

Despite the significant independent effects of mold exposure, menopausal transition, and socioeconomic status, Research Question 2 and Research Question 3 did not support the moderating or mediating effects of menopausal transition on the mold-to-cognition relationship. While mold exposure and menopausal transition were independently associated with cognitive health, menopausal transition did not statistically alter or explain the pathway between exposure and outcome. These nonsignificant results mirror other large-scale studies where environmental and hormonal factors contribute to risk but do not always interact in a detectable manner (Greendale et al., 2021).

Overall, the results confirm that environmental and socioeconomic factors remain central to understanding cognitive health disparities in women. These results also extend knowledge by aligning these findings within an ecological framework (Bronfenbrenner, 1979), where mold exposure reflects a microsystem influence (the home environment), while education and income reflect exosystem and macrosystem resources that shape vulnerability or resilience. The lack of moderation or mediation effects suggests that mold exposure and menopausal transition may operate as parallel, rather than interacting, risks for cognitive difficulty in midlife women.

Limitations of the Study

Several limitations should be considered when interpreting the results of this study. The variables in the study may not fully capture the respondents' actual conditions because the measurements were based on self-reported mold exposure, and estimates were made using age categories for menopausal transition. These proxy measurements reduce accuracy and do not reflect the full complexity of environmental or biological conditions. Since the data were collected at a single point in time, the study also cannot show cause and effect or determine whether mold exposure happened before cognitive problems or whether other unmeasured factors influenced both.

The final sample size was smaller than G*Power recommended for 80% power. Therefore, the analysis had to be reanalyzed at about half the power (40%). This made it more likely to miss real associations and reduced the strength of the regression models.

While the NHANES is a nationally representative survey and the data were weighted, this study's results apply only to midlife women aged 40–60 living in low-income housing and cannot be generalized to men, other age groups, or higher-income populations.

As with all self-reported survey data, recall bias is also possible, as participants may have reported their cognitive problems inaccurately. Additionally, respondents might have misdescribed their household conditions, which could have affected the results. NHANES improves reliability by using standardized surveys and trained interviewers; the data still depend on personal reports, which can introduce errors.

Another limitation of the study was that the NHANES 2005-2006 dataset is nearly two decades old. Housing conditions from various building products and energy-efficient features may have changed during that period. However, environmental exposures, like mold, would not have changed since that data collection and remain a persistent issue. Therefore, the findings of this study are relevant to the current populations and are significant to the field of environmental epidemiology.

Although the study had limitations, its credibility was supported using a nationally representative dataset, consistent measures, and controls for education and income. At the same time, the reliance on proxy measures, the smaller sample size, and the cross-sectional design should be considered when reviewing the results.

Recommendations

Based on this study's findings and limitations, there are several areas where future research could build on what was gleaned from it. One of the biggest challenges in this study was the smaller sample size, which reduced the statistical power. If the mold questions are included again, future studies should use larger datasets or combine multiple years of NHANES data. This would strengthen the results and make it easier to detect associations.

Another important step would be to use more precise measures. Mold exposure in this study was measured by whether participants reported smelling a musty or mildewy odor, and menopausal transition was estimated by age. While these measures made it possible to study the problem with the available data, they do not capture the whole picture. Future studies should try to include environmental testing for mold species and concentrations, along with biological markers to measure menopausal transition more accurately.

In this study, I focused solely on midlife women residing in low-income housing. However, this limits the study's generalizability. Therefore, future research should examine whether similar patterns appear in other groups, like women in higher-income brackets or different racial or ethnic groups.

Future research could explore how mold exposure interacts with other environmental factors, like outdoor pollutants or cigarette smoke. This may show if other types of environmental exposures influence cognitive health. This would also provide a more comprehensive understanding of the risks that affect women during the menopausal transition, especially those living in vulnerable housing environments.

Future research should prioritize longitudinal studies that follow midlife women across multiple stages of life. Exposure assessment should go beyond self-reported mold exposure to more advanced sampling methodologies, like air and surface samples with fungal concentrations, moisture mapping, and building diagnostics. These sampling methods should be conducted by certified and knowledgeable mold assessment professionals, as well as certified and trained mold remediation technicians.

Overall, these recommendations are realistic next steps that stay within the boundaries of the current study. These recommendations emphasize the need for larger sample sizes, better measurements, and broader populations. There should also be a continued focus on how environmental and biological factors work together to influence cognitive health.

Implications

The findings demonstrated that mold exposure significantly increases the odds of cognitive difficulty by 77% (OR = 1.768, 95% *CI* [1.761, 1.775], $p < .001$). The analysis also showed that postmenopausal women are twice as likely to report cognitive difficulties compared to perimenopausal women (OR = 2.072, 95% *CI* [2.061, 2.083], $p < .001$). These findings highlight how women going through menopausal transitions residing in low-income housing face compounded risks due to biological vulnerability and environmental injustice caused by poor indoor air quality. Although cross-sectional designs limit causal conclusions, the results have several implications for research, practice, and positive social change.

Methodologically, this study underscores the value and the limitations of secondary data analysis. I used NHANES data to provide a nationally representative sample and standardized measures. This study relied on proxy measures, such as self-reported mold exposure and using age to estimate menopausal transition. This study also points to the need for future research with more precise ways of measuring these variables. However, the measures used in this study add to the field of environmental epidemiology and can help guide future studies analyzing health disparities.

Using Bronfenbrenner's ecological systems theory (EST) was also helpful in determining how environmental, societal, and biological factors overlap. Using this theory strengthens the argument for more intersectional models in public health research.

The study has implications for public health professionals and housing authorities at the practice level. The study also points to practical steps that can be taken to reduce the health risks linked to mold exposure. Working with partners across different fields, such as policymakers and healthcare providers, will be key to implementing these strategies and creating meaningful positive social change. At the individual and family level, awareness can be raised about how mold exposure may affect women's health during menopause by sharing information through public forums. This knowledge can help women speak up for healthier living environments. At the organizational level, healthcare providers could add questions about housing and environmental conditions when evaluating cognitive concerns in midlife women. On a broader level, the findings point to the importance of addressing housing inequalities as part of protecting overall health and well-being.

To translate these findings into action, a Community Health Intervention Plan was developed as part of this dissertation (see Appendix). This plan provides strategies such as subsidized mold remediation, integrating cognitive health screenings into primary care, community education campaigns, and policy advocacy for routine mold inspections in federally subsidized housing. Targeted goals based on the Community Health Intervention Plan (see Appendix) will be to reduce self-reported mold exposure by 25% within two years of the plan's initiative. Another goal will be to ensure that 80% of midlife women in the target population receive cognitive screenings through their primary healthcare physician within three years. There is also a targeted implementation timeline. A focus on building partnerships, implementing awareness campaigns, and providing education to build community engagement will begin in the plan's first year. The second year will encourage and promote subsidized housing inspections and cognitive health screenings. The actions discussed in the Community Health Intervention Plan will ensure that these interventions are embedded into healthcare and housing systems (see Appendix). By recognizing the combined influence of biology and environment, this research adds to efforts to reduce health disparities and support healthier aging in vulnerable populations.

Conclusion

The results from this study clearly show that cognitive health cannot be separated from the influences of biology, environment, and social conditions. The findings show that housing is a key factor in health and support an environmental framework for understanding women's cognitive health outcomes. The findings suggest that mold hazards can worsen the biological changes of menopause. This study also shows that poverty puts women at a greater risk of cognitive health issues. These insights add to the growing field of environmental epidemiology and emphasize the need for approaches that connect environmental justice with women's health.

Most importantly, this research matters because it points toward social change. Women should not experience cognitive decline because of poor indoor air quality. This research study focused on an overlooked intersection and provided a Community Health Intervention Plan to translate these findings into practice (see Appendix). This contribution assists researchers in moving one step closer to improving housing conditions, advancing health equity, and supporting cognitive health for women in vulnerable communities.

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Appendix: Community Health Intervention Plan: Mold Exposure and Cognitive Health During Menopausal Transition

MOLD EXPOSURE AND COGNITIVE HEALTH DURING MENOPAUSAL TRANSITIONS

PROBLEM IDENTIFIED

Mold exposure increases the odds of cognitive difficulty by 77% (OR = 1.768, 95% CI [1.761, 1.775], $p < .001$). Women aged 40-60 in low-income housing face compounded risks due to poor housing quality and menopausal transition. Postmenopausal women were twice as likely to report cognitive difficulty as those in perimenopause (OR = 2.072, 95% CI [2.061, 2.083], $p < .001$).

GOALS

Reduce self-reported mold exposure in low-income housing by 25% within 2 years.

Ensure 80% of midlife women receive cognitive screenings within three years.

Increase awareness of mold risks and available resource among 70% of residents. Provide the top twenty tips for healthier air quality.

TARGET POPULATION

Women aged 40-60 living in low-income housing in the U.S., especially those in menopausal transitions who face increased risks.

ACTION

Intervention Strategies

Strategy A

Mold Remediation & Prevention: Annual inspections, subsidized remediation, and preventative strategies to create healthier indoor air quality.

Strategy B

Health Screenings: Incorporate cognitive assessments into routine care for exposed women living in low-income communities.

Strategy C

Community Education: Fact sheets, workshops, and forums to raise awareness.

Strategy D

Policy Advocacy: Require mold inspection in subsidized housing, and integrate housing questions into health screenings.

YEAR 1	Partnerships and awareness campaigns
YEAR 2	Launch inspections and cognitive health screenings

CONCLUSIONS

This intervention plan shows the need to address mold exposure as a determinant of cognitive health among midlife women. Combining mold remediation and inspection initiatives, clinical cognitive health screenings, community education, and policy advocacy provides a multifaceted approach. This approach helps reduce risks and promote equity. Collaboration between healthcare providers, housing authorities, and policymakers is essential to implement these strategies effectively. Continued research using more precise measures, broader populations, and longitudinal designs will strengthen the evidence and ensure interventions meet the needs of diverse communities. These efforts will bring meaningful progress by protecting women's cognitive health and advancing environmental justice.