Beyond "The Change": How Menopause Impacts Health and Employment

Lucia Torres Frasele*

Abstract

Despite the vast medical literature on the health effects of menopause, its economic implications remain unexplored. During women's reproductive years, hormones like estrogen are protective of health. Menopause, associated with a gradual yet pronounced decrease in estrogen, induces a distinct change in women's health trajectory that may have an impact on employment. This paper uses detailed data on the reported natural age of menopause for women in the Health and Retirement Study (HRS) to estimate the impact of menopause on health and employment. To address issues in identification that arise when analyzing menopause, such as confounding factors and measurement error, this paper uses the genetic predisposition for the timing of menopause, specifically the associated polygenic risk score (PGS), as an instrument for the reported age of natural menopause. There are three principal findings. First, consistent with the medical literature, crossing the menopause threshold there is an economically substantive and statistically significant acceleration in health conditions. The decline in health is more than triple that for pre-menopause years. Second, menopause is associated with a substantial reduction in the likelihood of working for pay by just under 2 percentage points every year after menopause, which accumulates to an 18 percentage point reduction in ten years. Combining these estimates, the associated IV estimate of the impact of health on employment indicates that the diagnosis of an additional medical condition reduces the likelihood of working for pay by between 49 to 77 percentage points depending on the exact specification. This is a substantial effect given that 78% of women work for pay prior to menopause. The key take-away is that essentially an additional diagnosis of a medical condition results in exit from employment for middle age to older women.

Keywords: women's health, menopause, aging, employment

JEL classification: I12, J14, J16, J21, J22

*Department of Economics at Syracuse University, e-mail: ltorresf@syr.edu

Acknowledgements: I would like to thank my advisor, Gary V. Engelhardt, for his invaluable guidance, support, and encouragement throughout this research. His insights and advice have been instrumental in shaping this work. Any errors or omissions are my own responsibility.

1 Introduction

Menarche and menopause mark the beginning and end of reproductive age for women. While labor, health, and population economists have allocated a great deal of effort to the understanding of fertility on the health and labor supply of teenage, young adult, and middle-aged women (e.g., Goldin and Katz, 2002; Hotz and Miller, 1988; Hotz et al., 1999; Mincer and Polachek, 1974), there has been remarkably little (if any) research on the impact of fertility cessation. In fact, on a broader societal level, menopause was not openly discussed for a long period and was sometimes referred to as "The Change," a euphemism that often carried negative connotations, which portrayed women becoming irritable or difficult. The lack of economic research is striking, given the widely known medical role of menopause in raising the risk of cardiovascular disease, osteoporosis and arthritis (Aloia et al., 1985; Namavari et al., 2024; Roman-Bias et al., 2009; Samargandy et al., 2020, 2022; van der Schouw et al., 1996; Visniauskas et al., 2023, among others), all of which may plausibly affect middle aged and older women's capacity to work and participate in the labor market.

Medically speaking, menarche refers to the first menstrual period, initiating a monthly cycle of fertile days and menstrual bleeding that can last for several decades; whereas menopause refers to the end of this cycle, a point in time when a woman has her final menstrual period (FMP) resulting from the loss of ovarian function (Soules et al., 2001). The range of normal timing for natural menopause is between ages 45 and 55 (Utian, 1999). In the physiological stage leading up menopause, referred to as menopausal transition, ovarian function starts to decline, leading to decreased production of hormones necessary for reproduction, most notably estrogen. During this stage, menstrual periods become irregular. This stage is often associated with mood swings, joint pain, fatigue, and hot flashes (Santoro, 2016). During reproductive years, these hormones are protective of women's health, including cardiovascular and musculoskeletal health (Bay-Jensen et al., 2013; Iorga et al., 2017), so that menopause induces a distinct change in women's health trajectory that may

have an impact on labor supply.

This paper uses detailed data on the reported natural age of menopause, health, and labor-market activity for women in the Health and Retirement Study (HRS) to estimate the impact of natural menopause on health and employment. The HRS is a nationally representative longitudinal study that started in 1992, surveying individuals 50 and older every two years, as well as their partners or spouses (regardless of age). Originally, the HRS included individuals born in 1941 and earlier, but has been adding younger cohorts of individuals in their 50s regularly every six years. Although menopause is an important milestone, the HRS only began collecting menopause-related data in the 2008 wave. Specifically, the HRS asks women the age at which they experienced their FMP, which represents the age at natural menopause.

There are three empirical challenges in determining the causal impact of menopause. First, any effect on health and labor supply may be confounded by other factors, including age-related changes in health or labor market attachment that are independent of menopause. This could be age-related progression of diseases such as cardiovascular, or arthritis, that naturally arise in the early 50s. The long arm of lifestyle choices like poor diet, smoking, or alcohol consumption also present as comorbidities as women age. Changes in family composition and support (e.g. older parents in need for care, or having an "empty nest" after children move out) as well as more general unobserved heterogeneity in health and labor supply may also confound estimates.

Second, there may be measurement error in self-reported health. Given that menopause age in the HRS is reported retrospectively, there is potential for recall bias, where older women might be recalling an event that occurred a decade or more before, whereas younger women may be recalling a much more recent event, or have yet to complete their menopausal transition. In addition, as women report health that justifies their labor force participation, confirmation bias can arise (Bound, 1991; Dwyer and Mitchell, 1999).

Third, not all women go through natural menopause. Some experience secondary or induced menopause prior to the advent of what otherwise would have been natural menopause. This happens from iatrogenic ovarian failure, either due to surgical removal of both ovaries (often due to serious conditions such as ovarian cancer or endometriosis), or cancer treatments like chemotherapy or radiotherapy (Rodriguez and Shoupe, 2015; Utian, 1999). The surgical removal of the uterus (hysterectomy), although not technically considered as secondary menopause, with or without the removal of ovaries, stops menstrual bleeding prematurely and is associated with a progressive decrease in ovarian function leading to an earlier menopause (Farquhar et al., 2005; Siddle et al., 1987). Additionally, procedures like endometrial ablation, or treatments like hormonal contraception, or hormonal replacement therapy can stop menstrual bleeding without affecting ovarian function, masking menopause-related bleeding patterns and symptoms (Davis et al., 2015). Consequently, the sample of women who experience and are able to distinguish natural menopause is not random, possibly inducing sample-selection bias. Ultimately, any or all of these concerns would render standard estimates biased and inconsistent.

To circumvent these concerns and recover (asymptotically) unbiased and consistent health and employment estimates, the analysis exploits respondents' genetic data gathered by the HRS. Between 2006 and 2012, the HRS collected DNA from respondents through saliva samples, achieving a more than 80% success rate. Since then, the HRS used genome-wide association studies (GWAS) to construct polygenic risk scores (PGS) for 73 phenotypes, such as smoking, diabetes, depressive symptoms, and others (Ware et al., 2024). Each PGS is derived from a single, validated GWAS, which finds the association between genetic variants and the phenotype. The PGS is a score from the weighted average of those genetic variants, based on how strong the association is. These scores represent an individual's genetic risk for each phenotype, so that the higher the score, the greater the genetic predisposition for that trait or condition. The age of menopause score reflects how late a woman is genetically predisposed to experience menopause, with a higher score indicating a later age. This score is

used as an instrument to construct a predicted age of natural menopause that is determined solely by genetics and plausibly independent of confounders and measurement error. In addition, since age of menarche is a strong predictor for early hysterectomy (Wilson and Mishra, 2016), the menarche PGS is used as an exclusion restriction to account for any selection bias. Importantly, even though the HRS provides researchers scores calculated separately for non-Hispanic individuals of European and African descent, and Hispanics (Ware et al., 2024), the underlying GWAS used to calculate these PGS are actually based on populations of European ancestry only (i.e. the genetic weights from the European ancestry group were applied to the genetic profiles of individuals of African descent and Hispanics). Given that genetic scores derived from European studies may be biased and less reliable when applied to other populations (Martin et al., 2017), this analysis focuses exclusively on women from European ancestry.

The empirical analysis begins with the relationship between menopause and health, or the first-stage. It focuses on women with data available within a ten-year span before and after the genetically predicted age of menopause, who did not undergo a hysterectomy before natural menopause. The final sample includes 3,320 women drawn from all the HRS cohorts except the CODA and AHEAD. The analysis looks at how predicted menopause timing affects health by using the total number of reported health conditions as the initial measure of health. This is constructed as follows. First, the HRS asks if doctors have ever diagnosed respondents with eight medical conditions: high blood pressure, diabetes, cancer (excluding skin cancer), chronic lung diseases, heart conditions, strokes, psychiatric issues, and arthritis. Then, these conditions are summed so that the focal outcome ranges from zero to eight. The first-stage estimates are consistent with the findings of the medical literature: menopause leads to an immediate and lasting impact on health. Prior to menopause, the total number of conditions is rising very slowly; post-menopause, there is a statistically significant acceleration of health conditions, more than tripling pre-menopausal trends.

Then the analysis turns to the relationship between menopause and labor supply, or the

reduced-form. The focal measure of employment is work for pay. Menopause is associated with a reduction in the likelihood of working for pay by just under 2 percentage points every year after menopause, which accumulates to a 18 percentage point reduction in ten years. The effects of employment are concentrated entirely on full-time work, with no changes in the mix between full-time and part-time employment or in unemployment. Overall, these results highlight a clear shift in labor supply patterns that aligns with the timing of menopause, suggesting that menopause induces a significant health shock that changes how women approach work.

Given the strength of the first-stage relationship, the first-stage and the reduced-form can be combined into instrumental variables (IV) estimates of the impact of health on labor supply. The identifying assumption is that conditional on genetic predisposition for other phenotypes and other controls, purely genetic timing of natural menopause is conditionally exogenous and affects labor supply only through health. The IV estimates indicate that the diagnosis of an additional medical condition reduces the likelihood of working for pay by 49 to 77 percentage points depending on the exact specification. This is a substantial effect, given that 78% of women in the sample work for pay prior to menopause.

The medical literature has found specific pathways between menopause and health conditions, most notably cardiovascular conditions, bone density loss (osteoporosis) and joint deterioration (arthritis). The CDC has identified these conditions as the leading causes of disabilities among adults in the U.S (Centers for Disease Control and Prevention, 2020). This pattern is evident in the HRS sample of pre-menopause data used, where the prevalence of cardiovascular conditions and arthritis are 6.8% and 25.4%, respectively. The first-stage analysis is consistent with the medical literature: the post-menopause impacts are especially strong for arthritis, with its prevalence growth rate nearly tripling. Consequently, the analysis then turns to labor supply estimates for these specific conditions. The IV estimates show a significant decrease in employment. Essentially, if a woman gets one of these conditions, they stop working for pay.

Notably, pre-menopause Body Mass Index (BMI) and smoking status play a significant role in post-menopausal health. Research suggests that women with higher BMI experience a less pronounced drop in estrogen levels around menopause, which may help mitigate some health impacts (Park et al., 2017). In contrast, women who ever smoked pre-menopause experience a more severe drop of estrogen (Randolph et al., 2011). When analyzing the sample by BMI, the strongest effects are observed in individuals with BMI below 30. Similarly, when splitting the sample by smoking status pre-menopause, the effects are greater and statistically significant among smokers. The analysis contains additional robustness checks and extensions that confirm known associations in the medical literature.

While it is difficult to place this paper's estimates in context with previous studies, given that menopause has gone unstudied in the economics literature, this analysis is more generally related to two strands of the literature on the health and labor supply of women. Earlier literature reviews, such as Currie and Madrian (1999), concluded that health problems can influence workforce participation, primarily focusing on general health issues. While some studies have looked at women's health and work outcomes (e.g., Ettner, 1997; Loprest et al., 1995), they mostly have focused on general physical or mental health, ignoring issues like menopause that are crucial for understanding why older women stay in or leave the workforce. The analysis also ties into broader research on the determinants of women's labor supply (Blundell and Macurdy, 1999; Killingsworth and Heckman, 1986). One specific medical condition that has been analyzed in the literature is arthritis, where Mitchell and Burkhauser (1990) found that arthritis affects the labor force participation of women, with women aged 45 to 64 (including those in the normal menopause age range) suffering the greatest impact, reducing their participation by half. However, Mitchell and Burkhauser (1990) did not examine the effect of menopause per se.

Overall, the results and IV methodology of this paper are novel and open a pathway for the study of a large number of related research questions on women's health and aging that are discussed in the conclusion. The remainder of this paper will proceed as follows.

Section 2 gives background about menopause and health implications; Section 3 describes the data and sample construction. Section 4 discusses the identification strategy. Section 5 analyzes the impact of menopause on health and presents the first-stage results, and Section 6 presents the reduced-form results on labor supply. Section 7 discusses the IV results. Additional analyses are presented in Section 8.

2 Menopause in Women's Health

Women's health is intrinsically linked to the stages of the reproductive years, marked by two significant milestones: menarche and menopause.[1] Menarche, the first menstrual period, signifies the onset of a woman's reproductive potential, initiating a monthly cycle that can continue for approximately three decades, barring interruptions from pregnancy, breastfeeding, or other factors (Barbo, 2002; Lacroix et al., 2023). Hormonal changes begin at menarche and stabilize during peak fertile years. The reproductive years conclude with menopause, the moment when the final menstrual period (FMP) occurs due to the loss of ovarian function. This is determined retrospectively after a woman has gone twelve months without any menstrual bleeding (WHO, 1996).

Several hormones play a crucial role in regulating the stages of the female reproductive cycle. Estrogen is essential for menstrual cycle regulation and fertility. It exists in four forms: estrone (E1), predominant after menopause; estradiol (E2), the most important and prevalent in premenopausal women; estriol (E3), produced by the placenta; and estetrol (E4), generated by the fetal liver during pregnancy (Coelingh Bennink, 2004; Visser and Coelingh Bennink, 2009). Ovarian follicles are fluid-filled sacs containing unfertilized eggs. When follicles reach a certain stage of development, they are ready for ovulation; these are called antral follicles. Luteinizing Hormone (LH) and Follicle-Stimulating Hormone (FSH)

[1] In this study, the term "women" refers to individuals assigned female at birth (AFAB). The HRS data discussed in section 3 and used in the empirical analysis categorizes gender (male or female) based on the interviewer's assessment rather than respondent self-identification. The pronouns used here for women will be they/them.

orchestrate ovarian function by triggering ovulation and supporting ovarian follicle development.[2] Inhibin B, a hormone produced by developing follicles, inhibits FSH production, so a decrease of this hormone will raise FSH levels and reduce fertility (Rosewell and Curry, 2018). Anti-Müllerian hormone (AMH) is a protein made by healthy ovarian follicles that helps regulate follicle growth (Mossa and Ireland, 2018). All these hormones, as well as the count of antral follicles, serve as key biomarkers of ovarian reserve and reproductive stages from menarche to menopause.

To standardize the reproductive aging timeline, the Staging of Reproductive Aging Workshop (STRAW) established a system in 2001, later revised in 2012 (Harlow et al., 2012; Soules et al., 2001). This system divides a woman's natural reproductive aging into stages, based on menstrual regularity and hormone levels, from menarche to menopause, regardless of age, demographic characteristics or lifestyle factors. These stages are shown in Table 1, and go from Stage -5 to Stage +2. First, the timeline organizes the reproductive lifespan into three broad phases: reproductive, menopausal transition, and post-menopause. The reproductive phase begins with menarche (typically between ages 10 and 16 (Lacroix et al., 2023)) and includes the early, peak, and late reproductive years. In the early years (Stage -5), menstrual cycles can be irregular as the body adjusts to hormonal changes. During the peak years (Stage -4), cycles typically stabilize, while the late reproductive years (Stage -3) are marked by subtle changes in hormone levels, although not enough to be seen in bleeding irregularities.

The menopausal transition includes two stages: early (Stage -2), where cycles begin to lengthen in a regular pattern by more than seven days, and late (Stage -1), the last stage with menstrual bleeding, where intervals of amenorrhea are longer than 60 days. According to Santoro and Randolph (2011), women may experience health challenges as they transition. The transition starts with the first irregular menstrual period, and it ends with the FMP, often lasting several years. This is characterized by increasingly erratic hormonal fluctuations

[2]High levels of FSH are associated with infertility.

Table 1: STRAW Staging

PHASE	**Reproductive**				**Menopausal Transition**		**Meno-pause**	**Postmenopause**			
Stage:	Early (-5)	Peak (-4)	Late (-3)		Early (-2)	Late (-1)	(0)	Early (+1)			Late (+2)
Substage			-3b	-3a	Perimenopause			+1a	+1b	+1c	
Duration	Variable				Variable	1-3 years		2 years (1+1)		3-6 years	Remaining lifespan
PRINCIPAL CRITERIA											
Menstrual cycle:	Variable to Regular	Regular	Regular	Subtle changes	Cycle length change (+7 days)	Interval of amenorrhea (+60 days)	FMP				
SUPPORTIVE CRITERIA											
Blood serum biomarkers:											
FSH			Low	Variable	Variable, elevated	Elevates to >25 IU/l		Variable, elevated		Stabilizes	
AMH			Low	Low	Low	Low		Low		Very low	
Inhibin B				Low	Low	Low		Low		Very low	
Ovarian reserve biomarkers:											
Antral Follicle Count				Low	Low	Low		Very low		Very low	

Notes. This table represents the system developed in the Staging of Reproductive Aging Workshop (STRAW). This table is adapted from Harlow et al. (2012). During reproductive years, FSH usually ranges between 4.7 to 21.5 IU/l (International Units per liter)

as ovarian function starts to decline. Elevated FSH levels is the main signal of the start of the transition, and is predictive of vasomotor symptoms like hot flashes and night sweats (Randolph et al., 2005). Additionally, increased FSH before the FMP is associated with rising blood pressure and the risk of hypertension, which may also be influenced by weight gain and changes in body fat distribution (Samargandy et al., 2022). The decline in estrogen during this period also affects the female genital tract early in the transition (Santoro, 2016), with symptoms such as vaginal dryness, burning, irritation, the lack of lubrication during sexual intercourse and urinary tract infections, forming part of the condition known as genitourinary syndrome of menopause (GSM) (Portman and Gass, 2014). While these symptoms may emerge during the transition, they are more prevalent among postmenopausal women. This span can bring other physical and psychological symptoms, including sleep disturbances and mood changes.[3] There is an associated rise in diabetes risk during the early transition, around seven years before FMP, although estradiol levels closer to menopause seem less impactful (Park et al., 2017). Low levels of AMH can predict the FMP within the next 12 months (Finkelstein et al., 2020).[4] The median age for entering the menopause transition is 47, and for menopause itself, 51 (Santoro, 2016).

In this STRAW staging system, menopause is considered Stage 0, happening at the FMP. However, twelve months have to pass until menopause is recognized and a woman is officially in the first post-menopause phase (Stage +1), so that the first part of this stage, +1a goes undetected prospectively.[5] Within Stage +1, Substage +1b, symptoms like hot

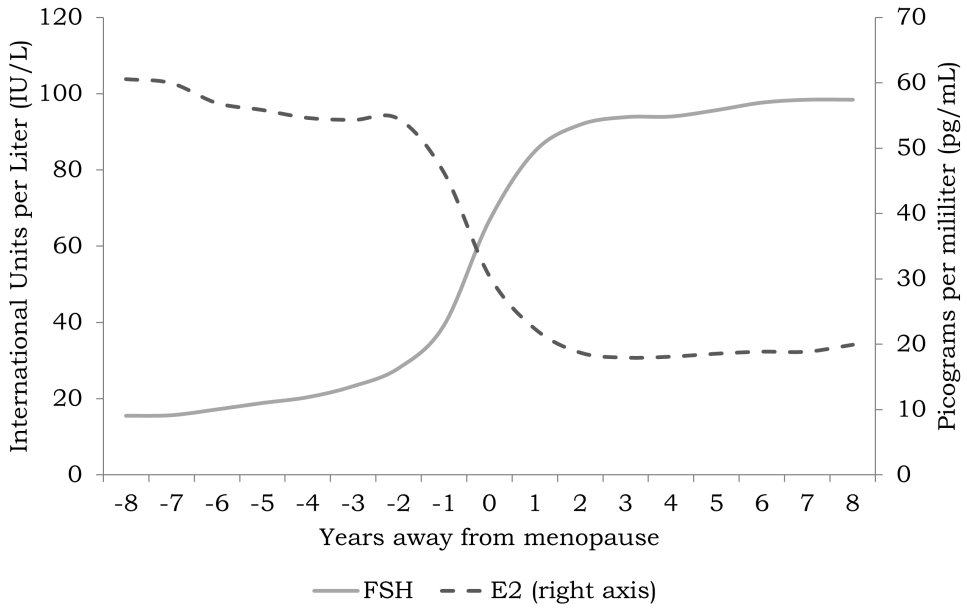
[3]Hormone replacement therapy is used to manage the menopause-related symptoms. However, its use is associated with an increased risk of breast cancer, depending on the type and duration of therapy (Colditz, 1998; Colditz et al., 1987).

[4]AMH is measured in nanograms per milliliter ng/mL and with a level <10ng/mL there is a 51% to 79% likelihood that a woman in the menopause transition will get the FMP within the next 12 months (Finkelstein et al., 2020).

[5]Perimenopause, although colloquially identified as synonymous with menopausal transition, technically includes the 12 months following the FMP. Similarly, the term "menopause" is frequently used informally to refer to the transition, creating some confusion. Even within medical literature, there is sometimes inconsistency in defining menopause as either the FMP itself or the point 12 months after the FMP when menopause is officially recognized. Despite these ambiguities, terms like perimenopause and menopause are primarily used in clinical settings with patients, while research in the last couple of decades has consistently centered on the FMP as the key reference point.

flashes still occur, as hormone levels are still fluctuating. By the end of this stage, Substage +1c, hormone levels stabilize and vasomotor symptoms ease. Finally, Stage +2, or late post-menopause, represents the final stage, lasting from hormonal stabilization through the remainder of a woman's life. A summary of the associated blood serum and ovarian reserve biomarkers for each stage are also in the table.

Figure 1: Average levels of FSH and Estradiol by years away from menopause (Estradiol units on right axis)



Notes. Adapted from Randolph et al. (2011). This figure shows the population mean levels of follicle-stimulating hormone (FSH) and estradiol (E2) from eight years before to eight years after the final menstrual period (FMP). FSH is measured in international units per liter (IU/L), ranging from 15 IU/L to 98 IU/L, and estradiol is measured in picograms per milliliter (pg/mL), ranging from 60 pg/mL to 19 pg/mL.

Figure 1 illustrates the shifts around menopause for selected hormones. The late menopausal transition, occurring between years -2 and 0, marks the onset of the most significant hormonal changes, characterized by, on average, rising serum FSH levels, and declining estradiol levels (Hale et al., 2007).[6] After the FMP, estradiol continues to decline, whereas FSH continues to rise, stabilizing approximately two years after menopause (Randolph et al., 2011). A key takeaway from this figure and Table 1 is that menopause is not characterized

[6]FSH level of >40 IU/L is a biomarker for late menopausal transition (Randolph et al., 2006)

by an abrupt change in women's physiology; instead, the biological changes unfold gradually over several years.

These hormonal changes are linked to higher cardiovascular disease risks, including coronary artery disease and stroke, as estradiol's protective effects diminish (Iorga et al., 2017; van der Schouw et al., 1996; Visniauskas et al., 2023).[7] High blood pressure (HBP) and hypertension risk increase around menopause, with post-menopausal trajectories shaped by pre-menopausal blood pressure levels: remaining stable if previously normal, accelerating if low, and decelerating if high (Samargandy et al., 2022). Additionally, central arterial stiffness, closely linked to hypertension and cardiovascular conditions, rises significantly between one year before to one year after FMP (Samargandy et al., 2020).

Menopause also raises the risk of osteoporosis and arthritis, again due to declining estradiol (Aloia et al., 1985; Bay-Jensen et al., 2013). Osteoarthritis is a degenerative disease causing joint inflammation and cartilage deterioration, while rheumatoid and psoriatic arthritis are autoimmune conditions leading to inflammation, pain, and stiffness. These conditions primarily affect postmenopausal women, with early menopause further raising the risk of rheumatoid arthritis (Namavari et al., 2024; Roman-Bias et al., 2009).

As noted in the introduction, one analytical complication is that not all women go through natural menopause.[8] A particularly important concern is hysterectomy. It stops menstrual bleeding prematurely and is associated with a progressive decrease in ovarian function leading to an earlier menopause, even when ovaries remain intact (Farquhar et al., 2005; Siddle et al., 1987).[9] In fact, hysterectomy is the most common major non-obstetric surgery, with a prevalence of 22.1% among women between 45 and 64 (Gorina et al., 2024).

[7] Lower levels of estradiol after hysterectomy, with or without the removal of the ovaries, also increases the risk for cardiovascular diseases (Falkeborn et al., 2000).

[8] Unfortunately, the HRS does not ask about procedures like oophorectomy, endometrial ablation, or the use of hormonal birth control or hormone replacement therapy.

[9] Hysterectomies can be classified into three types: total (removal of the uterus and cervix), subtotal (removal of the upper part of the uterus), or radical (removal of the uterus, cervix, and part of the vagina, usually due to cancer), and they can be conducted via abdominal, vaginal, or laparoscopic methods.

More than 50% of hysterectomies are performed alongside the removal of both ovaries (bilateral oophorectomy). This combination is less common among women in fertile years, between 15 to 44, where only 37% of hysterectomies include oophorectomy. The age group with the highest percentage of women undergoing both procedures is 50 to 54 years, with a 78% incidence (Parker, 2010; Whiteman et al., 2008). In the HRS sample of data used and described in detail in the next section, 44% of women for whom there are genetic data underwent hysterectomies, and 79% of those women had it before menopause. Consequently, any analysis of menopause on health and labor supply must utilize a data source that also asks about hysterectomies.

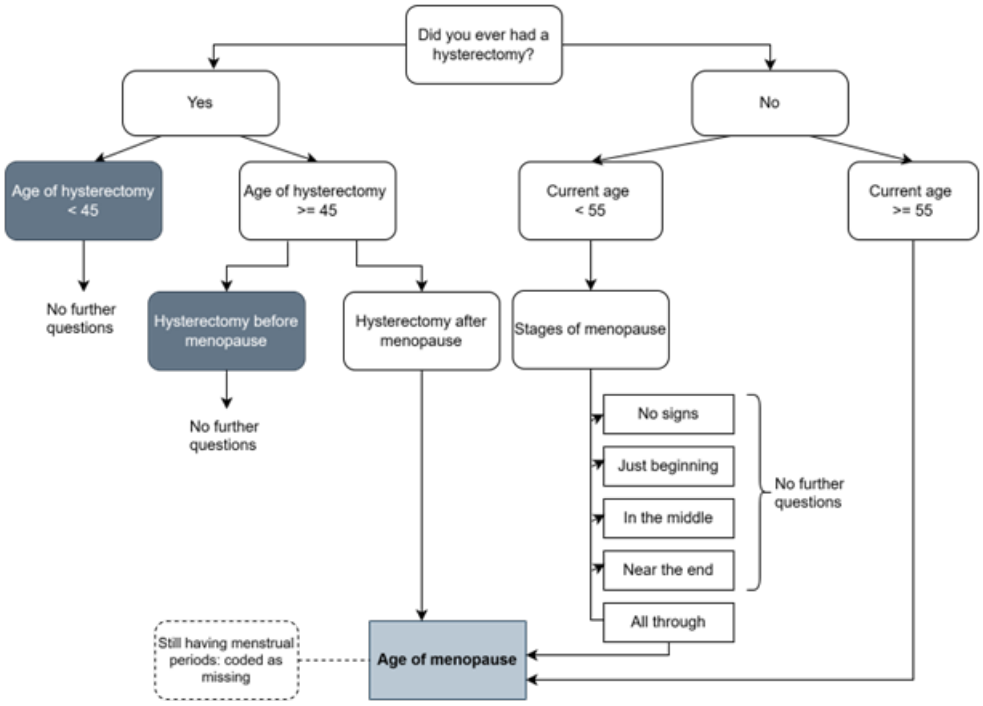
3 Sample Construction and Data Analysis

This paper uses data from the HRS, which contains both questions on health conditions and menopause. It begins by asking respondents if a doctor has ever diagnosed them with the following eight key conditions: (1) high blood pressure or hypertension, (2) diabetes or high blood sugar, (3) cancer or malignant tumor (excluding minor skin cancer), (4) chronic lung diseases (e.g., chronic bronquitis, emphysema), (5) heart problems (like heart attack, coronary heart disease, angina, congestive heart failure, among others), (6) strokes, (7) psychiatric issues (including emotional or nervous problems), and (8) arthritis or rheumatism.[10] If affirmative, then they are asked detailed follow-up questions. The main health variable in the analysis below is the sum of all these conditions, ranging from zero to eight.

The HRS started asking about menopause and related questions in the 2008 wave. As depicted in Figure 2, the menopause subsection starts by first asking about hysterectomy: if the woman had one and the age at which it occurred. If the hysterectomy happened before age 45, no further menopause-related questions are asked. If the hysterectomy occurred after or equal to age 45, the survey asks whether it was performed after the FMP, meaning after

[10]Doctors are referred as specialists, general practitioners, or osteopaths, excluding chiropractors and nurses.

Figure 2: HRS Menopause Subsection Flowchart



Notes. Flowchart depicting the menopause and hysterectomy questions in the health section of the HRS. This figure illustrates the sequence of questions related to menopause and hysterectomy.

going through menopause. For a woman aged 55 or older or a woman who had a hysterectomy after their FMP, the survey inquires about the age at which they went through menopause. If the woman is 55 or older and has not finished going through menopause and is still having menstrual periods, they do not need to answer and the answer is coded as missing. Women under 55 years who did not have a hysterectomy get asked about the stage of menopause, ranging from "without a sign" to "all through," with the specific age of menopause asked if they replied as such. In subsequent waves after a woman first answers this subsection, if the woman mentioned they finished with menopause or had a hysterectomy, this subsection was not asked again.[11]

[11]The HRS asked questions about the stages of menopause only between 2008 and 2020. In 2020, menopause was redefined as occurring 12 months after the FMP. Despite this change, the question about menopause age still referred to the age of the FMP. Starting in 2022, the question was updated to ask only the age at which women finished menopause, using the updated definition.

However, due to occasional survey errors, some women were asked the menopause subsection multiple times, resulting in varying responses. To address this, this paper develops a system to reconcile any conflicting responses across waves for the menopause and hysterectomy data. First, it cleans the age of menopause variable from reporting and measurement errors.[12] Second, it calculates the difference between the age of menopause and current age.[13] Third, it selects the age of menopause based on that difference; if the difference between current and reported menopause age is within a 5-year window, it selects the highest menopause age reported in that window. This is done in order to account for potential change in the FMP in more recent waves. If the difference exceeds 5 years, it relies on the initial reported age.[14] Finally, if a hysterectomy was reported several times, the initial reported age of hysterectomy is retained. If they reported having a hysterectomy before menopause, but provided the age of menopause, the menopause information is kept.

Labor force participation, demographic, and health variables in the analysis sample are from the RAND HRS dataset. The menopause and hysterectomy data are from the health section of the survey. The genetic data used to construct the PGS were collected by HRS during the core interview between waves 2006 and 2012. The PGS are extracted from the sensitive health data from HRS (Health and Retirement Study, 2024a,b; Ware et al., 2024).[15] The analysis is restricted to women of European ancestry for which the HRS provides PGS data. All HRS cohorts are included except CODA and AHEAD, as these cohorts were surveyed primarily after retirement age, which is beyond the scope of this study. Additionally, the sample is limited to data ten years before and after menopause, and excludes women who underwent hysterectomies before menopause.

[12]Responses indicating ongoing menstrual periods or unknown menopause ages will be considered as missing data. Reported menopause ages above 90 years old are considered likely typos due to the high unlikeliness of getting menopause at that age and be treated as missing values as well.

[13]Excluding any observation where the difference is less than negative one. Negative one values are kept because it assumes it is possible to have a misreported current age versus the age of menopause.

[14]This is done unless the age reported is under 30, in which case the highest reported age is used, because this was likely a typo, not many cases.

[15]The 73 phenotypes are listed in Table A.2 in the Appendix.

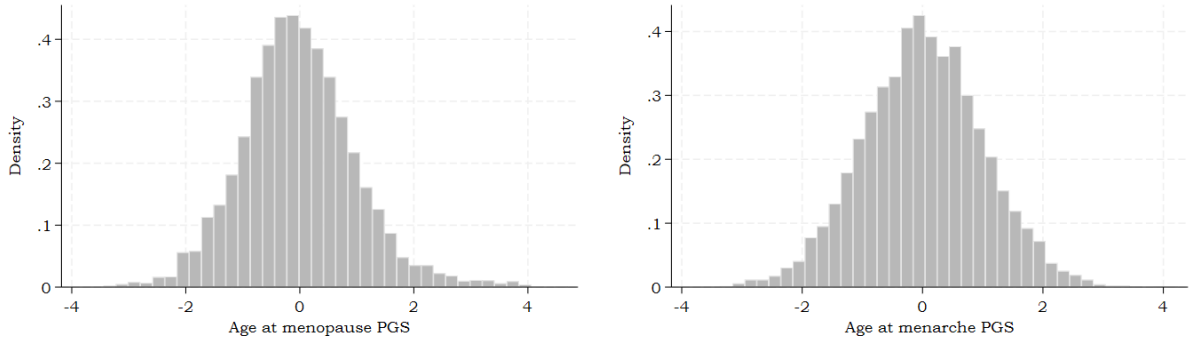
Table 2: Sample Summary Statistics

	All	Menopause	
		Before	After
	(1)	(2)	(3)
Panel A: Demographics			
Age	54.87	48.27	56.38
	(4.56)	(3.36)	(3.27)
HS dropout (%)	7.71	6.75	7.93
High school (%)	35.78	33.77	36.24
Some college (%)	26.85	28.03	26.58
College or more (%)	29.66	31.45	29.25
Married (%)	74.75	82.55	72.96
Divorced (%)	14.80	9.99	15.90
Never married (%)	3.94	3.62	4.02
Panel B: Labor Force Participation			
Working for pay (%)	69.84	77.95	67.98
Full time (%)	50.15	57.72	48.41
Part time (%)	19.69	20.23	19.57
Unemployed (%)	2.59	3.27	2.44
Not in labor force (%)	32.20	21.03	34.76
Panel C: Health			
Total health conditions	1.10	0.81	1.17
	(1.15)	(1.02)	(1.17)
High blood pressure (%)	28.13	19.10	30.21
Diabetes (%)	7.75	5.17	8.34
Cancer (%)	6.56	4.19	7.11
Lung disease (%)	4.93	4.40	5.05
Cardiovascular (%)	8.34	6.75	8.71
Psychiatric conditions (%)	16.20	15.55	16.35
Arthritis (%)	37.82	25.36	40.68
Panel D: PGS			
Age at menopause - European	0.168		
	(1.031)		
Age at menarche - European	-0.005		
	(0.992)		
Person-year observations	15,238	2,843	12,395
Observations	3,320		

Notes. Column (1) shows the mean and standard deviation for selected variables for the sample of 15,238 person-year observations and 3,320 women of European ancestry from the HRS. Columns (2)–(3) show the same for before and after predicted menopause. Predicted menopause is defined in equation 4 in the next section. The final sample for the analysis is restricted to data available ten years before and after predicted menopause, excluding women who reported having a hysterectomy before menopause. The average predicted age of menopause in the final sample is 51 years, ranging from 43 to 57 years.

Column (1) of Table 2 gives the summary statistics for selected variables for the full sample. There are 15,238 person-year observations on 3,320 women. The average age is 55,

Figure 3: Histograms for selected PGS



(a) PGS for Age at Menopause

(b) PGS for Age at Menarche

Notes. Histograms of the polygenic risk scores (PGS) for the age of menopause and the age of menarche. The PGS for both outcomes were derived from data obtained from the HRS. The left histogram displays the distribution of the PGS for age of menopause, while the right histogram shows the distribution for age of menarche.

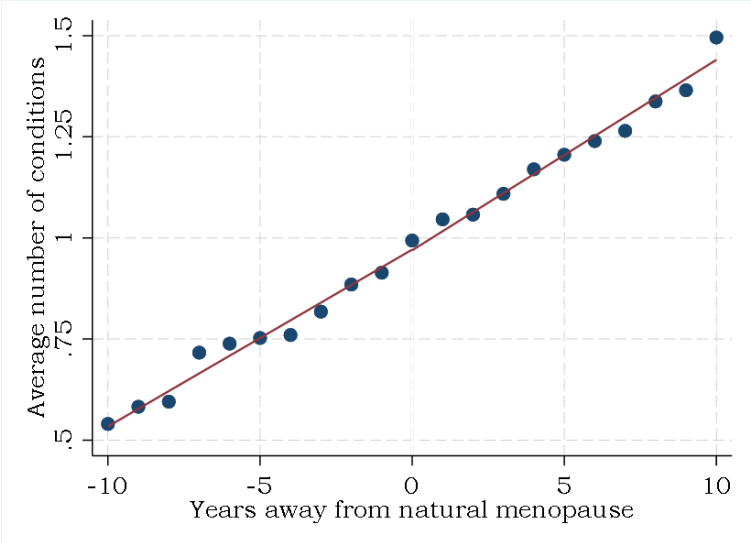
married and with high-school diploma. Most of them work for pay, in a full-time capacity. The highest prevalence of health conditions are seen in high blood pressure and arthritis. Columns (2) and (3) show similar statistics for the subsamples of person-year observations before and after menopause, respectively. Panel A of Figure 3 shows the histograms for PGS of menopause and Panel B of Figure 3 shows PGS for menarche, for women of European ancestry.

4 Identification strategy

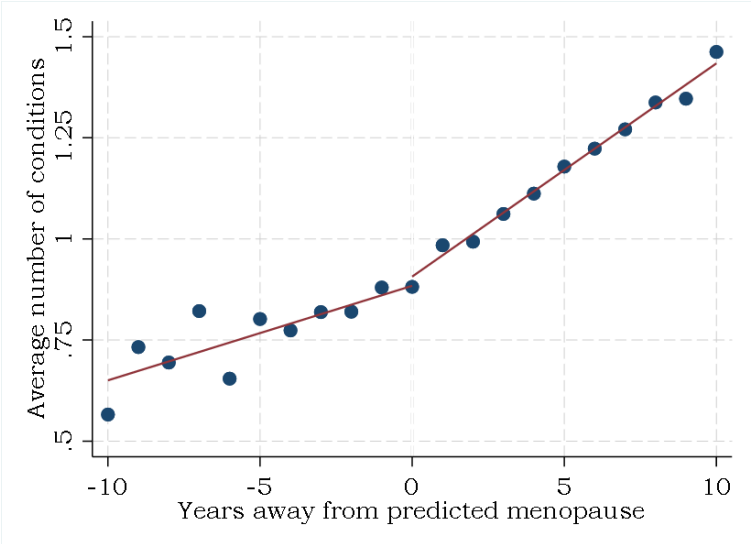
Panel (a) in Figure 4 illustrates the relationship between the number of medical conditions and years away from natural menopause for the sample of women without hysterectomy. This is a bin scatter plot, where the horizontal axis represents years away from natural menopause, using the FMP as the zero anchor point. The vertical axis represents number of conditions averaged by each year. As a frame of reference, this figure overlays fitted lines showing the relationship separately for before and after menopause. The lines indicate that, on average, women experience a steady increase in the number of medical conditions each

year as they approach menopause, where this rate of increase appears to remain stable even after menopause. It would seem, therefore, that there is no impact of menopause on health.

Figure 4: Number of health conditions and menopause



(a) Natural

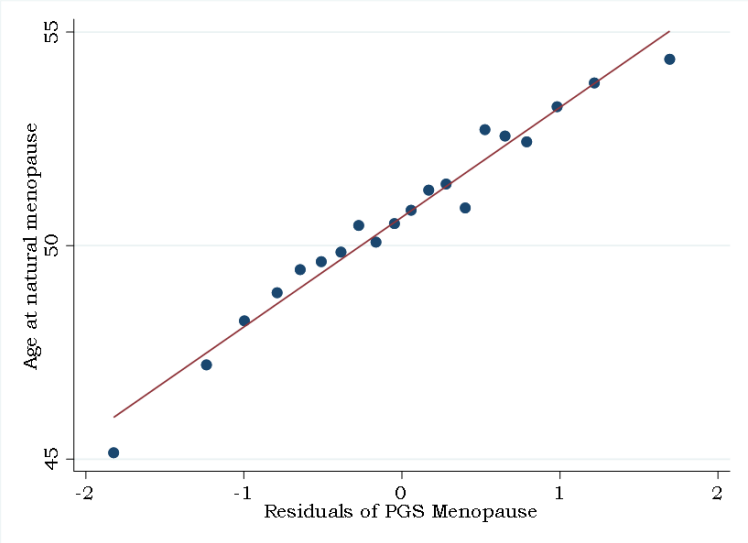


(b) Predicted

Notes. Two subfigures showing the relationship between the number of medical conditions and years away from menopause. The top panel presents a bin scatter plot for women without a hysterectomy, with years away from natural menopause on the horizontal axis (FMP=0) and average medical conditions on the vertical axis. The bottom panel shows the same relationship, but for predicted menopause age based on the estimation of equation 4.

However, as detailed in the introduction, there are three empirical challenges in determining whether the relationship in Figure 4a can be interpreted as the causal impact of menopause: confounding factors, such as other age-related changes in health that naturally affect middle aged and older women; measurement error in reported age of natural menopause; and sample-selection bias from hysterectomies. Ultimately, any or all of these concerns would render standard estimates biased and inconsistent, although the sign of the bias is indeterminate.

Figure 5: Relationship between age of menopause and PGS for menopause, conditional on other PGS



Notes. Bin scatter plot illustrating the relationship between age of menopause and the predicted residuals from regressing the PGS for menopause on other PGS, $\hat{u}_i^o$.

To circumvent these, the analysis uses a novel IV approach, whereby the PGS for age of menopause is used as an instrument for reported age at natural menopause.[16] In order to be a valid instrument, the PGS for menopause must satisfy the exclusion restriction. One concern is that some of the genes associated with menopause in the GWAS (Day et al., 2015) also have positive weights in the PGS for other phenotypes. Therefore, the first step in the

[16]Ideally, if data on blood serum levels of various hormones were available, these could be instrumented using the PGS to mitigate measurement error associated with self-reported data. Unfortunately, the HRS does not provide such values. Consequently, the effects estimated in this analysis are anchored to the FMP.

IV approach is to isolate that part of the PGS of age at menopause that is independent of other PGS.

Let P^o denote the PGS for age at menopause and P^e denote that for menarche. The independent part of P^o is obtained through the following auxiliary regression:

$$P_i^o = \alpha_0 + \alpha_1 \mathbf{P}_{-oi} + u_i^o, \tag{1}$$

where $\mathbf{P}_{-oi}$ is the vector of 73 PGSs other than that for menopause. Appendix Table A.3 shows the estimates for equation (1).[17] The R^2 from this regression is 0.36, which implies that 64% of the variation in the PGS of menopause is independent of the PGS for other phenotypes. Similarly, for P^e:

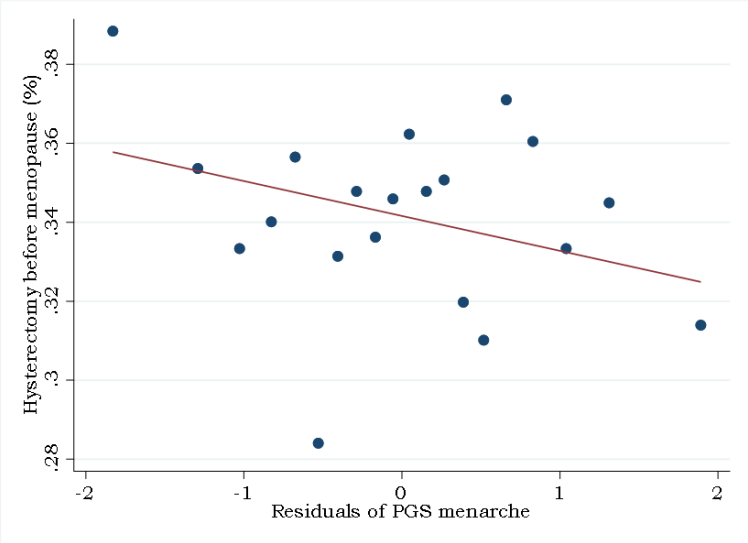
$$P_i^e = \theta_0 + \theta_1 \mathbf{P}_{-ei} + u_i^e, \tag{2}$$

where $\mathbf{P}_{-ei}$ is the vector of 72 PGSs other than that for menarche. The R^2 from this regression is 0.20, implying 80% of the variation in the PGS of menarche is independent of other phenotypes. Overall, the majority of the variation in the menopause and menarche PGS is independent of other phenotypes.

Figure 5 graphs the age of natural menopause versus the estimated residuals from equation (1), $\hat{u}_i^o$. A strong positive association is observed between these variables, consistent with findings from Zhao et al. (2021), which showed that, even when controlling for factors such as BMI, smoking status, education, and contraceptive use, the PGS for menopause for white women of European ancestry, remain significantly correlated with the natural age of menopause. Similarly, Figure 6 is a bin scatter plot of the hysterectomy probability and predicted residuals from equation (2), $\hat{u}_i^e$. This figure is consistent with the medical litera-

[17]The HRS recommends including the first five Principal Component Analysis (PCA) eigenvectors as covariates when regressing on PGS. However, incorporating these eigenvectors does not lead to statistically significant changes in the estimates (Ware et al., 2024).

Figure 6: Relationship between hysterectomy and PGS for Menarche, conditional on other PGS



Notes. Bin scatter plot illustrating the relationship between the probability of getting a hysterectomy before menopause and the predicted residuals from regressing the PGS for menarche on other PGS, $\hat{u}_i^e$.

ture, where higher age of menarche is considered a strong predictor for lower risk of early hysterectomy (Wilson and Mishra, 2016).

In the second step, to correct for selection due to hysterectomy, parameters of the following equations are estimated (Heckman, 1979):

$$H_i = \mu_0 + \mu_1 \hat{u}_i^e + \mu_2 \hat{u}_i^o + \eta_i, \tag{3}$$

$$A_i = \gamma_0 + \gamma_1 \hat{u}_i^o + \zeta_i, \quad (\text{if } H_i = 0) \tag{4}$$

where $H_i = 1$ if a woman had a hysterectomy before menopause, and 0 otherwise, A_i represents each woman's natural age of menopause, and $\hat{u}_i^o$ and $\hat{u}_i^e$ are residuals from (1) and (2), respectively. Therefore, (4) is the outcome equation and (3) is the selection equation.

The estimation results are shown in Table 3. Panel A shows that for the outcome equation, and panel B shows that for the selection equation. There are two take-aways.

First, consistent with Figure 5, the menopause PGS is a strong predictor of age at menopause. Based on the standard errors in parentheses, the null hypothesis that the PGS has no impact on age of menopause $\gamma_1 = 0$ versus the one-sided alternative can be rejected at well below the 1% significance level. Therefore, the menopause PGS, P^o, is a relevant instrument. Second, the menarche PGS, P^e, is a strong exclusion restriction in the selection equation. The estimation correlation of the errors in (3) and (4) is $\hat{\rho} = 0.08$, consistent with the medical literature (Wilson and Mishra, 2016), although statistically different at only the 12% level.

Table 3: Heckman selection estimation results for age at natural menopause, standard errors in parentheses

Dependent variable:	Age of natural menopause
Panel A: Main equation	
$\hat{u}_i^o$	2.570
	(0.090)
Constant	50.571
	(0.099)
Panel B: Selection equation	
H_i	-3.196
	(0.060)
$\hat{u}_i^o$	0.005
	(0.028)
$\hat{u}_i^e$	0.065
	(0.026)
Constant	1.243
	(0.025)
ρ	0.083
	(0.053)
Observations	6,894

Notes. This table reports the Maximum Likelihood Estimation of the Heckman model regression for age of reported natural menopause. The selection equation evaluates the probability of having had a hysterectomy before menopause using the predicted residuals from equation 2, $\hat{u}_i^e$, as the exclusion restriction. The sample for this estimation includes all European women for whom there is PGS information. Standard errors in parentheses.

Finally, the estimates from (4) are used to make a predicted years away from menopause:

$$\hat{M}_{it} = Age_{it} - \hat{A}_i, \tag{5}$$

where Age_{it} is each woman's age in each survey wave. Importantly, the variation in $\hat{M}_{it}$, is, by construction, due to independent variation in genetic predisposition.

5 Impact of menopause on health

Figure 4b, shows the relationship between number of medical conditions (h) and predicted menopause ($\hat{M}$). Compared to Panel (a) in the figure, there is now a clear shift in health trajectory following menopause. Women's health deteriorates faster after relative to before menopause.

Given that the relationship between health conditions and predicted menopause appears linear both before, and especially after, to determine the statistical strength of this relationship the graphical analysis now moves to a regression-based framework using the following linear specification:

$$h_{it} = \kappa_0 + \kappa_1 D_{it} + \kappa_2 \hat{M}_{it} + \kappa_3 \hat{M}_{it} \cdot D_{it} + \kappa_4 \mathbf{X}_i + \epsilon_{it}, \tag{6}$$

where h_{it} represents the health status of individual i in time t, D_{it} is an indicator function that takes the value of 1 after predicted menopause ($D_{it} = \mathbb{1}(\hat{M}_{it} \geq 0)$) and $\mathbf{X}_i$ is the vector of controls.[18] In (6), the coefficient κ_1 reflects the impact in level at menopause, κ_2 the pre-menopause trend, and κ_3, the focal parameter, captures the change in trend after menopause. The focal hypothesis is that there no change in trend ($\kappa_3 = 0$) versus an increase in trend ($\kappa_3 > 0$).[19]

Table 4 presents the results from estimating (6) by OLS with health status measured by the total number of medical conditions. The focal estimates are shown in the third row. In

[18] $\hat{M}_{it} = 0$ represents the moment at predicted menopause

[19] The estimate of κ_3 is identified by pure genetic variation in time away from menopause. A key identification assumption is that age does not have an independent effect on health except through years away predicted menopause, $\hat{M}_{it}$. This is the standard assumption in the medical literature where the effect of menopause in women is relative to their FMP. Specifically, this was illustrated in Figure 1, where health fluctuates with hormones and not the calendar age at menopause.

Table 4: Effect of menopause on total number of health conditions, standard errors in parentheses and p-values in square brackets

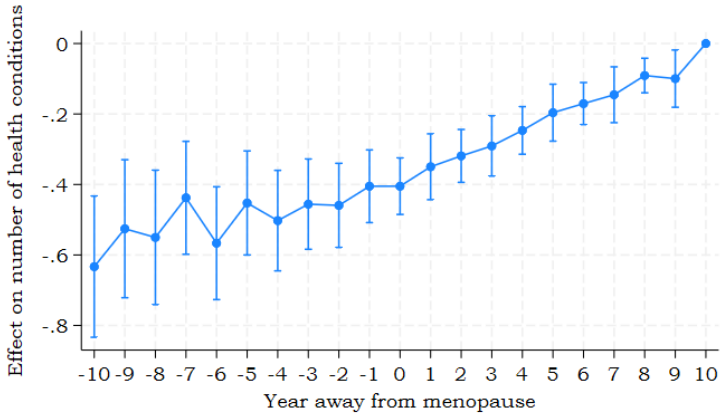
Dependent variable:	Total number of health conditions		
	(1)	(2)	(3)
$\hat{\kappa}_1$: Impact at menopause	0.039	0.035	-0.000
	(0.029)	(0.028)	(0.038)
$\hat{\kappa}_2$: Trend before menopause	0.015	0.011	0.015
	(0.008)	(0.007)	(0.008)
$\hat{\kappa}_3$: Post-menopause trend change	0.035	0.023	0.019
	(0.008)	(0.008)	(0.009)
	[0.000008]	[0.0015]	[0.0143]
Person-year observations	15,238	15,238	13,786
Controls	no	yes	yes

Notes. Columns (1) and (2) show the OLS regression for equation 6 for the full sample. Column (3) shows the same for the sample excluding observations in the immediate vicinity of predicted menopause (years -1 to 1). For all columns, the dependent variable is the total number of health conditions and the independent variables are D_{it}, measuring the impact at menopause, $\hat{M}_{it}$ measuring the trend before menopause and the interaction term $D_{it} \cdot \hat{M}_{it}$, that measures the post-menopause trend change. Column (1) shows the regression without any controls. Column (2) and (3) include dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the one-tailed test $H_0 : \kappa_3 = 0$ vs. $H_a : \kappa_3 > 0$, in square brackets.

Column (1), $\hat{\kappa}_3 = 0.035$ is estimated without controls. It indicates an additional 0.035 conditions for each additional year after predicted menopause. Hence, 10 years after menopause, a woman would have 0.35 additional conditions. The pre-menopause mean number of conditions is 0.81 (from Column (2) of Table 2), so that this estimate is economically modest in size. With a standard error of 0.029 (clustered at the individual level), this effect is statistically different from zero at the 0.002% level of significance as shown by the p-value in square brackets. Column (2) adds controls for survey waves, education level, marital status, and census division. Column (3) presents estimates excluding observations in the immediate vicinity of predicted menopause (-1 to 1), which, as shown in Figure 1, correspond to the years when estradiol (FSH) is falling (rising), with identification relying on the transition and post-menopausal periods. The results are similar.[20]

[20] As an additional robustness check, equation 6 was estimated using both components in Equation 5, Age_{it} and $\hat{A}_i$, instead of $\hat{M}_{it}$, to assess whether age directly affects the results. The results are not statistically different.

Figure 7: Coefficients for the estimation of non linear first-stage with 95% confidence intervals



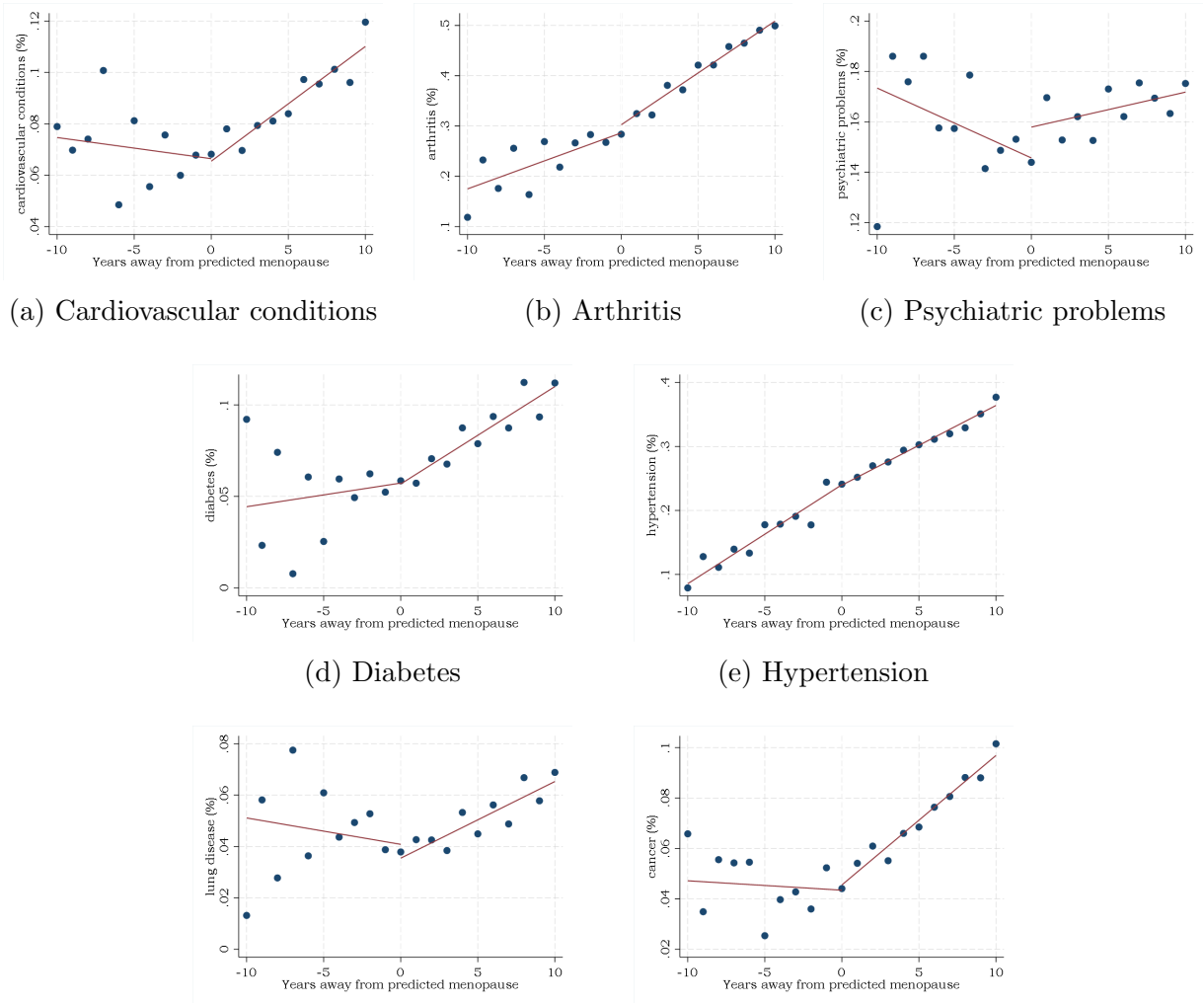
Notes. Robustness check relaxing the linearity assumption in equation 6 by replacing D_{it}, $\hat{M}_{it}$, and their interaction terms with dummy variables for each single year away from predicted menopause. The figure presents the estimates and 95% confidence intervals for each of these dummies for the non-linear first-stage estimation of the total number of health conditions, controlling for survey waves, demographic variables and census division.

Finally, as a robustness check, the linearity assumption in equation 6 is relaxed. This is done by replacing D_{it}, $\hat{M}_{it}$, and their interaction terms with dummy variables for each single year away from predicted menopause. The estimates for each year away from menopause for the total number of health conditions are presented in Figure 7. Consistent with the hormonal patterns in Figure 1, the health profile is relatively flat until roughly year -2, then begins to rise, aligning with the linear specification results.

The medical literature has identified some health conditions that can worsen after menopause, such as cardiovascular and arthritis. Figure 8 shows the relationship between predicted menopause and each of the medical conditions that comprise the focal variable.[21] Table 5 presents the OLS estimate of κ_3 (the change in trend) separately for each health condition, with p-values adjusted for multiple comparisons. The conditions that are most responsive to menopause are arthritis, cancer and lung disease. Despite the prevailing view

[21] Heart problems and stroke are condensed to cardiovascular conditions.

Figure 8: Selected health conditions and predicted menopause



(a) Cardiovascular conditions (b) Arthritis (c) Psychiatric problems

(d) Diabetes (e) Hypertension

(f) Lung disease (g) Cancer

Notes. Seven subfigures (panels a to g) showing bin scatter plots of the relationship between years away from predicted menopause and the prevalence of various health conditions. Each panel represents a different condition: (a) cardiovascular disease, (b) arthritis, (c) psychiatric problems, (d) diabetes, (e) hypertension, (f) lung disease, and (g) cancer. The horizontal axis represents years away from predicted menopause, while the vertical axis shows the prevalence of each condition.

in the medical literature that cardiovascular risk rises, the estimates for that condition are equivocal.[22] The remaining results align with the medical research suggesting menopause has no impact on diabetes and high blood pressure.

[22]Breast cancer is the most prevalent cancer among women, with longer exposure to estradiol increasing the likelihood of developing this cancer. A study by Chavez-MacGregor et al. (2005) indicates that having more menstrual cycles throughout life raises breast cancer risk, consistent with earlier research linking long-term exposure to ovarian hormones with increased breast cancer risk.

Table 5: Effect of menopause on trends of the prevalence of selected health conditions, standard error in parentheses and p-values in square brackets

		$\hat{\kappa}_3$: Post-menopause trend change		
	(1)	(2)	(3)	(4)
Dependent variable	Pre-menopause mean of dep. variable			
Arthritis	0.254	0.0136	0.0113	0.0095
		(0.0039)	(0.0039)	(0.0044)
		[0.0002]	[0.0017]	[0.0152]
		{0.0030}	{0.0120}	{0.0879}
Cardiovascular	0.068	0.0047	0.0030	0.0034
		(0.0022)	(0.0022)	(0.0026)
		[0.0164]	[0.0857]	[0.0900]
		{0.0699}	{0.2927}	{0.3337}
Cancer	0.038	0.0053	0.0048	0.0042
		(0.0019)	(0.0019)	(0.0023)
		[0.0033]	[0.0068]	[0.0342]
		{0.0150}	{0.0410}	{0.1598}
Psychiatric problems	0.155	0.0067	0.0037	0.0039
		(0.0032)	(0.0030)	(0.0034)
		[0.0180]	[0.1073]	[0.1243]
		{0.0699}	{0.2927}	{0.3337}
Diabetes	0.052	0.0027	0.0014	0.0015
		(0.0019)	(0.0019)	(0.0022)
		[0.0735]	[0.2352]	[0.2525]
		{0.1419}	{0.4096}	{0.4216}
Lung disease	0.044	0.0045	0.0034	0.0026
		(0.0017)	(0.0017)	(0.0019)
		[0.0042]	[0.0235]	[.0844]
		{0.0150}	{0.1109}	{0.3337}
High blood pressure	0.191	-0.0021	-0.0043	-0.0053
		(0.0034)	(0.0034)	(0.0038)
		[0.5315]	[0.2043]	[0.1698]
		{0.4995}	{0.5365}	{0.6004}
Person-year observations		15,238	15,238	13,786
Controls		no	yes	yes

Notes. Column (1) show the mean of each health condition pre-menopause, indicating the prevalence of the condition. Columns (2) and (3) show the OLS regression for equation 6 for the full sample, for selected health conditions. Column (4) shows the same for the sample excluding observations in the immediate vicinity of predicted menopause (years -1 to 1). This table presents only the coefficients for the interaction term $D \cdot \hat{M}_{it}$, that measures the post-menopause trend change. Column (2) shows the regression without any controls. Column (3) and (4) include dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the one-tailed test $H_0 : \kappa_3 = 0$ vs. $H_a : \kappa_3 > 0$ (except for high blood pressure that is two-tailed test), in square brackets; p-values using the Romano-Wolf multiple hypothesis correction (Clarke et al., 2020), in curly brackets.

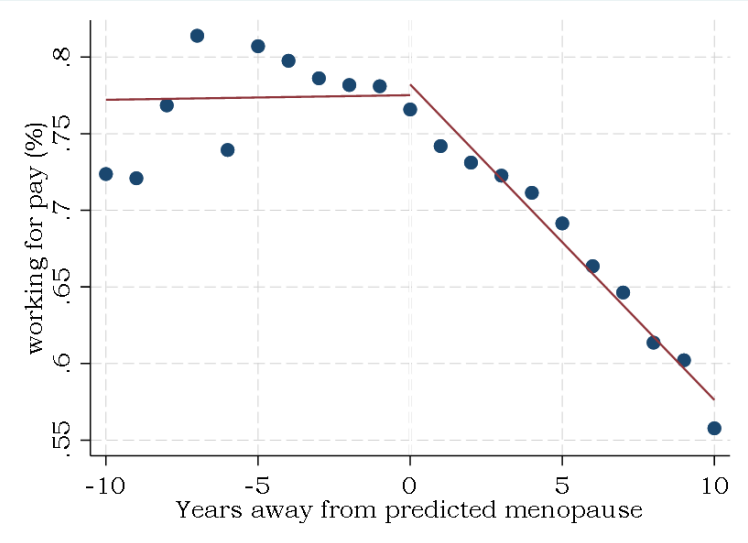
6 The impact of menopause on employment

Next, the analysis turns to the relationship between menopause and employment, or the reduced-form. Here, the focal measure of employment is "working for pay." Figure 9 presents a bin scatter plot of the probability of working for pay by each year away from predicted menopause. There is a distinct change in the trajectory of employment following menopause, altering women's engagement in the labor market that mimics the change in health illustrated in Figure 4b. Table 6 presents estimates from the following reduced-form model,

$$y_{it} = \beta_0 + \beta_1 D_{it} + \beta_2 \hat{M}_{it} + \beta_3 \hat{M}_{it} \cdot D_{it} + \beta_4 \mathbf{X}_i + \upsilon_{it}, \tag{7}$$

where y_{it} is employment, with the same right-hand side variables as in the first-stage equation (6). The focal parameter is β_3, which captures the difference in employment trend from before and after the menopause timing. The organization of the table is isomorphic to that in Table 4.

Figure 9: Working for pay and predicted menopause



Notes. Bin scatter plot illustrating the probability of working for pay by years away from predicted menopause. The horizontal axis represents years away from predicted menopause, and the vertical axis shows the probability of employment, averaged for each year.

Table 6: Effects of menopause on trends of the probability of working for pay, standard errors in parentheses and p-values in square brackets

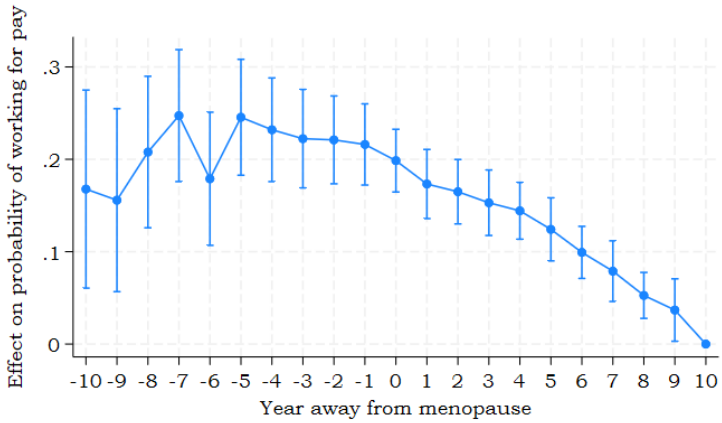
Dependent variable:	Working for pay		
	(1)	(2)	(3)
$\hat{\beta}_1$: Impact at menopause	0.0016	-0.0013	-0.0091
	(0.0138)	(0.0136)	(0.0189)
$\hat{\beta}_2$: Trend before menopause	0.0004	-0.0002	0.0019
	(0.0035)	(0.0034)	(0.0040)
$\hat{\beta}_3$: Post-menopause trend change	-0.0189	-0.0177	-0.0205
	(0.0038)	(0.0038)	(0.0044)
	[0.0000007]	[0.000003]	[0.000003]
Person-year observations	15,238	15,238	13,786
Controls	no	yes	yes

Notes. Columns (1) and (2) show the OLS regression for equation 7 for the full sample. Column (3) shows the same for the sample excluding observations in the immediate vicinity of predicted menopause (years -1 to 1). For all columns, the dependent variable is the total number of health conditions and the independent variables are D, measuring the impact at menopause, $\hat{M}_{it}$ measuring the trend before menopause and the interaction term $D \cdot \hat{M}_{it}$, that measures the post-menopause trend change. Column (1) shows the regression without any controls. Column (2) and (3) includes dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the two-tailed test in square brackets.

The focal estimates are shown in the third row. $\hat{\beta}_3 = -0.0189$ in Column (1) and was estimated without controls. It indicates a reduction of 1.89 percentage points in the probability of working for pay for each additional year after predicted menopause. Hence, 10 years after menopause, a woman would have reduced their employment by 18.9 percentage points. The pre-menopause probability of working for pay is 78% (from Column (2) of Table 2), so that this estimate represents a 24.2% decrease in the probability of working for pay after 10 years. With a standard error of 0.0038 (clustered at the individual level) this effect is statistically different from zero at well below the 1% level of significance as shown by the p-value in square brackets. Column (2) adds controls for survey waves, education level, marital status, and census division. Column (3) excludes years -1 to 1. The results are not economically or statistically different. Column (4) relaxes the linearity assumption. The estimates for each year away from menopause for the probability of working for pay are

presented in Figure 10, echoing the linear specification findings.

Figure 10: Coefficients for the estimation of non linear reduced-form with 95% confidence intervals



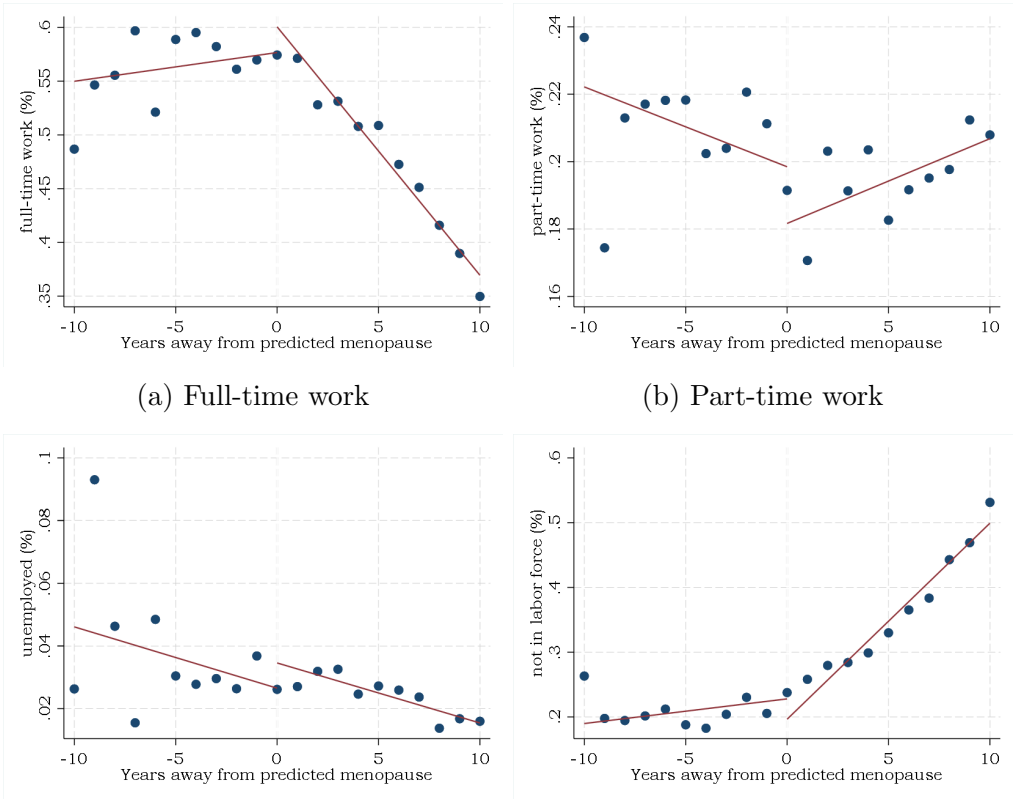
Notes. Robustness check relaxing the linearity assumption in equation 7 by replacing D_{it}, $\hat{M}_{it}$, and their interaction terms with dummy variables for each single year away from predicted menopause. The figure presents the estimates and 95% confidence intervals for each of these dummies for the non-linear reduced-form estimation of the probability of working for pay, controlling for survey waves, demographic variables and census division.

Figure 11 illustrates the relationship between alternative measures of employment, labor force participation, and predicted menopause. Table 7 presents the estimates of β_3 for each measure. The effects of employment are concentrated entirely on full-time work, with no changes in the mix between full-time and part-time employment or in unemployment. However, menopause is associated with a reduction in labor force participation, as shown in the bottom row.

7 The effect of health on employment: IV approach

Overall, menopause is associated with a reduction in health and employment. Given the strength of the first-stage relationship, these first-stage and the reduced-form results can be combined into instrumental variables (IV) estimates of the impact of health on employment,

Figure 11: Labor force participation outcomes and predicted menopause



(a) Full-time work

(b) Part-time work

(c) Unemployed

(d) Not in labor force

Notes. Four subfigures showing bin scatter plots of labor force outcomes by years away from predicted menopause. The panels illustrate the probability of: (a) working full time, (b) working part time, (c) being unemployed, and (d) not being in the labor force. The horizontal axis represents years away from predicted menopause, and the vertical axis shows the average probability for each outcome.

the model for which is

$$y_{it} = \delta_0 + \delta_1 h_i + \delta_2 D_{it} + \delta_3 \hat{M}_{it} + \delta_4 \mathbf{X}_i + \omega_{it}. \tag{8}$$

In (8), δ_1 is the effect of an additional health condition on the probability of employment. The identifying assumption is that conditional on genetic predisposition for other phenotypes and other controls, $\mathbf{X}_i$, the purely genetic timing of natural menopause is conditionally exogenous and affects employment only through health.

The first row of Table 8 reports the OLS estimates for the parameters in (8). Column

Table 7: Effects of menopause on trends of the probability of selected labor force participation outcomes, standard errors in parentheses and p-values in square brackets

		$\hat{\beta}_3$: Post-menopause trend change		
	(1)	(2)	(3)	(4)
Dependent variable	Pre-menopause mean of dep. variable			
Full-time work	0.577	-0.0230	-0.0230	-0.0219
		(0.0044)	(0.0043)	(0.0050)
		[0.0000002]	[0.0000001]	[0.00001]
		{0.0010}	{0.0010}	{0.0010}
Part-time work	0.202	0.0040	0.0053	0.0014
		(0.0035)	(0.0035)	(0.0041)
		[0.2539]	[0.1266]	[0.7252]
		{0.4456}	{0.2577}	{0.8751}
Unemployed	0.033	0.0010	0.0011	0.0010
		(0.0016)	(0.0017)	(0.0021)
		[0.5560]	[0.4992]	[0.6319]
		{0.5894}	{0.5225}	{0.8751}
Not in labor force	0.210	0.0219	0.0204	0.0233
		(0.0037)	(0.0036)	(0.0041)
		[0.000000003]	[0.00000002]	[0.00000002]
		{0.0010}	{0.0010}	{0.0010}
Person-year observations		15,238	15,238	13,786
Controls		no	yes	yes

Notes. Column (1) show the mean of each health condition pre-menopause, indicating the prevalence of the condition. Columns (2) and (3) show the OLS regression for equation 7 for the full sample, for the probability of several outcomes of labor force participation. Column (4) shows the same for the sample excluding observations in the immediate vicinity of predicted menopause (years -1 to 1). This table presents only the coefficients for the interaction term $D \cdot \hat{M}_{it}$, that measures the post-menopause trend change. Column (2) shows the regression without any controls. Columns (3) and (4) include dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the two-tailed test in square brackets; p-values using the Romano-Wolf multiple hypothesis correction (Clarke et al., 2020), in curly brackets.

(1) shows results without controls. $\hat{\delta}_1 = -0.0717$, which indicates that an additional health condition reduces employment by 7.2 percentage points. Given a pre-menopause employment rate of 78%, this corresponds to a 9% decline in the likelihood of employment. Row 3 presents the IV estimate of δ_1, using the years away after predicted menopause, captured

Table 8: Average marginal effects of health on the probability of working for pay, standard errors in parentheses

Dependent variable :	Working for pay			
	(1)	(2)	(3)	(4)
OLS	-0.0717	-0.0702	-0.0708	-0.0700
	(0.0060)	(0.0059)	(0.0059)	(0.0059)
Probit	-0.0671	-0.0661	-0.0670	-0.0660
	(0.0054)	(0.0054)	(0.0055)	(0.0054)
IV	-0.5416	-0.7746	-1.0521	-0.4919
	(0.1506)	(0.2857)	(0.4930)	(0.0603)
IV Probit	-0.5211	-0.7514	-1.0423	-0.4955
	(0.1562)	(0.2892)	(0.5003)	(0.0605)
Person-year observations	15,238	15,238	13,786	15,238
Controls	no	yes	yes	yes

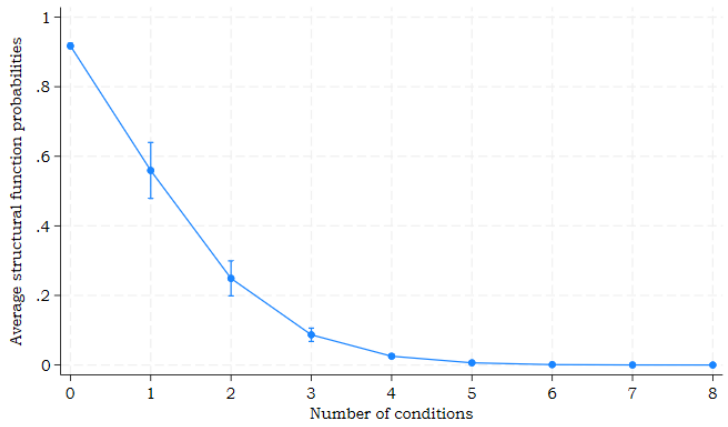
Notes. Columns (1) to (3) show, in the first row, $\hat{\delta}_1$ from the OLS regression of working for pay on the total number of health conditions, controlling for years away from predicted menopause ($\hat{M}_{it}$), D_{it} and additional controls ($\mathbf{X}_i$) for columns (2) and (3). Column (3) shows each estimation excluding observations in the immediate vicinity of predicted menopause (years -1 to 1), including controls. Column (4) shows the regression for the full sample, including controls, but relaxing the linearity assumption by adding dummy variables for each year away from predicted menopause to the estimation. The second row shows the Probit regression results of the same specifications for each column. Row 3 shows the IV regression of working for pay on health, instrumenting with the interaction of $D \cdot \hat{M}_{it}$ for columns (1) to (3), for column (4) the instruments are the dummies for years away from predicted menopause ($\hat{M}_{it}$) 1 to 10. Row 4 shows the same as row 3 for IV Probit specification. Columns (2) to (4) include as controls dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses.

,

by the interaction $\hat{M}_{it} \cdot D_{it}$, as the instrument for health, h_{it}. The IV results in Column (2), with controls, suggest that an additional medical condition reduces employment by 77 percentage points. A comparison of the OLS to IV estimates suggests that OLS is biased upward through a combination of confounders, measurement errors, etc. The p-value for the Hausman test of the equality of the OLS and the IV estimates is $p = 0.0079$. Column (4) relaxes the linearity assumption, and the IV estimate of δ_1 from equation (8) is calculated using a dummy for each year after predicted menopause as instruments for health. The result, $\hat{\delta}_1 = -0.49$, suggests that an additional medical condition reduces employment by 49 percentage points, with a standard error of 0.0603 (clustered at the individual level),

which is statistically significant at the 1% level. Although this IV point estimate is smaller, given the precision of the estimation, Column (4) is not statistically different than the linear estimate in Column (2). Overall, the impact of an additional medical condition is to reduce the likelihood of employment by between 49 and 77 percentage points.

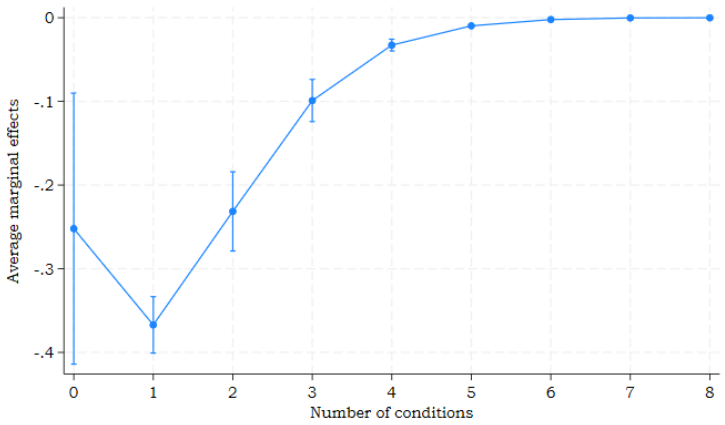
Figure 12: Predicted probability of working by number of conditions with 95% confidence intervals



Notes. This figure shows the predicted probability of working for pay by number of conditions. The predicted probabilities in this figure are calculated after estimating equation 8 with all controls with IV Probit

There are a number of additional ways to gauge the economic significance of the IV estimates. The first is to compare the IV estimate with the overall sample mean of employment prior to menopause. From Table 2 (Column 2) this was 78%. By this metric, one additional health condition almost effectively results in a woman exiting employment. A second is to calculate the average employment probability predicted by the IV estimate for each value of h (the number of health conditions) over the full sample. This is shown in Figure 12. For instance, a woman with no medical conditions (a value of zero on the horizontal axis) has a predicted probability of working for pay of 92%, this probability declines as they get more conditions. Women who transition from being healthy to having four or more health conditions effectively leave the labor force. A third way is represented in Figure 13, which illustrates how the average marginal effects change depending on her health status

Figure 13: Average marginal effect of health on employment
by number of pre-existing conditions with 95% confidence intervals



Notes. This figure shows the average marginal effect of health on working for pay after menopause by number of conditions. The average marginal effects in this figure are calculated after estimating equation 8 with all controls with IV Probit.

pre-menopause (i.e., the number of pre-existing medical conditions). For women with no pre-existing health conditions (a value of zero on the horizontal axis) being diagnosed with one reduces the probability of working for pay by 25 percentage points. For women with one existing condition, an additional diagnosis decreases this probability by 36 percentage points. However, as the baseline number of health conditions increases, the impact of an additional condition on labor force participation diminishes. This pattern suggests that for women who transition through menopause with multiple health conditions, an extra condition has a much smaller marginal effect on their likelihood of working.

Finally, Table 9 shows the average marginal effects of health by labor force participation sub-status. Mirroring the reduced-form results in Table 7, the main pathways are reduction in full-time employment and reduction in labor force participation. Table 10 breaks the results into specific health conditions. Again, most effects are loaded on arthritis.

Table 9: Average marginal effects of health on employment status from IV estimation, standard errors in parentheses and p-values in square brackets

	Average marginal effects of health			
Dependent variable	(1)	(2)	(3)	(4)
Full-time work	-0.6561	-1.0074	-1.1258	-0.5652
	(0.1841)	(0.3666)	(0.5345)	(0.0688)
	[0.0004]	[0.0060]	[0.0352]	[2.2e-16]
	{0.0040}	{0.0480}	{0.1129}	{0.0010}
Part-time work	0.1150	0.2331	0.0740	0.0735
	(0.1051)	(0.1719)	(0.2121)	(0.0383)
	[0.2738]	[0.1752]	[0.7272]	[0.0551]
	{0.4456}	{0.2278}	{0.8142}	{0.0629}
Unemployed	0.0276	0.0492	0.0513	-0.0437
	(0.0471)	(0.0746)	(0.1088)	(0.0147)
	[0.5574]	[0.5097]	[0.6375]	[0.0029]
	{0.5844}	{0.5235}	{0.8142}	{0.0100}
Not in labor force	0.6250	0.8919	1.1959	0.6904
	(0.1626)	(0.3146)	(0.5471)	(0.0759)
	[0.0001]	[0.0046]	[0.0288]	[9.1e-20]
	{0.0040}	{0.0480}	{0.1129}	{0.0010}
Person-year observations	15,238	15,238	13,786	15,238
Controls	no	yes	yes	yes

Notes. Columns (1) to (3) shows the average marginal effects of health on selected labor force participation variables from the IV estimation of equation 8 instrumenting with the interaction of $D \cdot \hat{M}_{it}$, which measure the magnitude of the change in trends of health conditions after menopause. Column (3) shows the IV estimation excluding observations in the immediate vicinity of predicted menopause (years -1 to 1), including controls. Column (4) shows the regression for the full sample, including controls, but relaxing the linearity assumption by estimating dummy variables for each year away from predicted menopause, the instruments are the dummies for years away from predicted menopause ($\hat{M}_{it}$) 1 to 10. Column (1) shows the IV regression for all the sample without any controls. Columns (2) to (4) include as controls dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the two-tailed test in square brackets; p-values using the Romano-Wolf multiple hypothesis correction (Clarke et al., 2020), in curly brackets.

8 Heterogeneity analysis

A series of additional heterogeneity analyses are performed by splitting the sample along key physiological dimensions: body mass index (BMI), smoking status, and age at menopause (before and after age 50). In particular, the medical literature has documented a number

Table 10: Average marginal effects of specific health conditions on employment status from IV estimation, standard errors in parentheses and p-values in square brackets

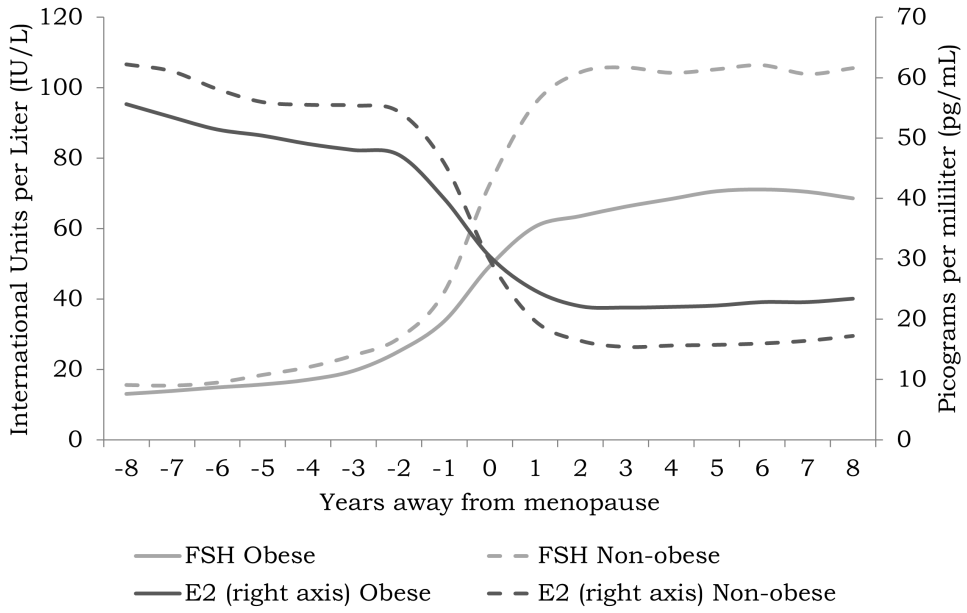
Dependent variable :	Working for pay			
	(1)	(2)	(3)	(4)
Arthritis	-1.3941	-1.5684	-2.1515	-0.8624
	(0.4715)	(0.6169)	(1.0605)	(0.1017)
	[0.0031]	[0.0110]	[0.0425]	[2.3e-17]
	{0.1000}	{0.1700}	{0.3000}	{0.0500}
Cardiovascular conditions	-4.0415	-5.8330	-5.9904	-6.4411
	(2.0466)	(4.4139)	(4.6403)	(2.5748)
	[0.0483]	[0.1863]	[0.1967]	[0.0124]
	{0.1400}	{0.2400}	{0.3700}	{0.5300}
Cancer	-3.6008	-3.7213	-4.9369	-4.0092
	(1.5117)	(1.6911)	(2.8943)	(0.8030)
	[0.0172]	[0.0278]	[0.0881]	[0.0000006]
	{0.1300}	{0.2400}	{0.3700}	{0.2600}
Person-year observations	15,238	15,238	13,786	15,238
Controls	no	yes	yes	yes

Notes. Columns (1) and (2) shows the average marginal effects of selected health conditions on working for pay. This comes from an IV regression where the dependent variable is working for pay and endogenous variable variates to each condition, instrumented with the change in trends of that condition after menopause. Column (1) shows the regression without any controls. Column (2) includes dummy variables for each survey wave, education levels (high school dropout, high school, some college, and college or more), marital status (married, divorced or separated, widowed, and never married) and census divisions. Standard errors clustered at the individual level, in parentheses; p-values for the two-tailed test in square brackets; p-values using the Westfall-Young multiple hypothesis correction (Westfall and Young, 1993), in curly brackets.

of differential effects of menopause on health for different subgroups of the population. For example, hormonal levels related to menopause vary by obesity (Park et al., 2017). Figure 14 shows that while estrogen levels are slightly lower for obese women (BMI greater than 30) before menopause, the decline in estrogen starting two years before the FMP is less pronounced compared to non-obese women. Hormones also vary by smoking status (Randolph et al., 2011). Figure 15 shows that estradiol levels are higher for smokers during the menopause transition, but after menopause, the level mirrors that of non-smokers, meaning a deeper drop. These menopausal hormonal changes negatively impact respiratory health and decrease lung function after menopause for women who ever smoke pre-menopause (Camp-

bell et al., 2018). Finally, health effects also vary by age of menopause. Long term exposure to estrogen and other hormones, which occurs for women experiencing late-age menopause, can increase the risk for certain conditions, such as breast cancer (Chavez-MacGregor et al., 2005).

Figure 14: Average levels of FSH and Estradiol by obesity and years away from menopause (Estradiol units on right axis)

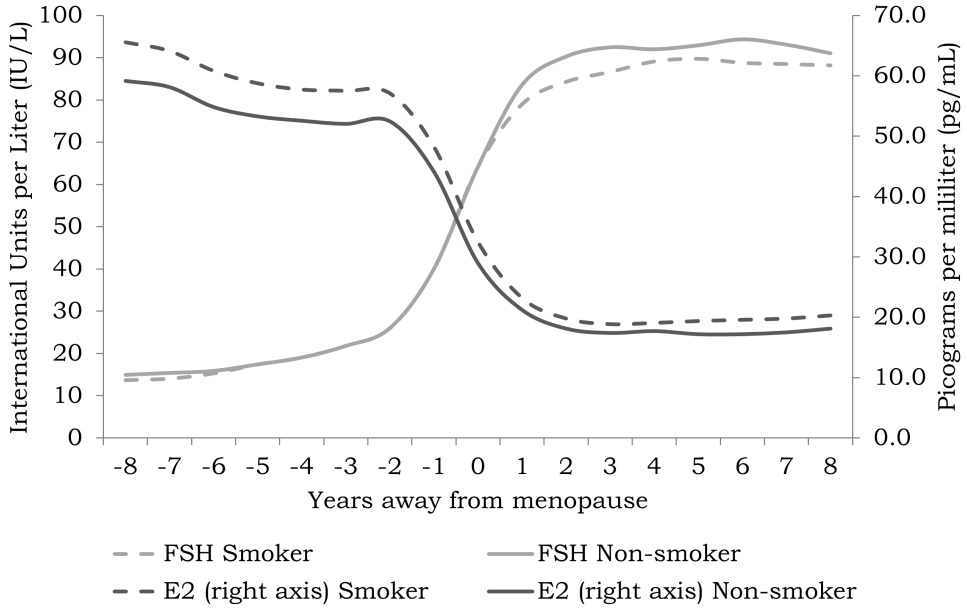


Notes. Adapted from Randolph et al. (2011). This figure shows the population mean levels of follicle-stimulating hormone (FSH) and estradiol (E2) from eight years before to eight years after the final menstrual period (FMP), separated by obesity status. Obesity is defined as a body mass index (BMI) greater than 30. FSH is measured in international units per liter (IU/L), and estradiol is measured in picograms per milliliter (pg/mL).

In light of this, Table 11 shows the first-stage, reduced-form, and IV estimates for these sample splits. The first-stage estimates in Column (2) comport with known associations in the medical literature: menopause has bigger effects for women with low BMI, smokers, and women who experience late menopause. The reduced-form estimates show differential reductions in employment for the same sub groups, but given the standard errors, the estimated effects are not precise enough to make strong distinctions on labor supply behavior.[23]

[23]For completeness, Column (4) shows the IV estimates, but they, like the reduced-form, are not precise

Figure 15: Average levels of FSH and Estradiol by smoking status and years away from menopause (Estradiol units on right axis)



Notes. Adapted from Randolph et al. (2011). This figure shows the population mean levels of follicle-stimulating hormone (FSH) and estradiol (E2) from eight years before to eight years after the final menstrual period (FMP), separated by pre-menopause smoking status. FSH is measured in international units per liter (IU/L), and estradiol is measured in picograms per milliliter (pg/mL).

9 Conclusion

Despite the vast medical literature on the health effects of menopause, its economic implications remain unexplored. During women's reproductive years, hormones like estrogen, are protective of health. Menopause, associated with a gradual yet pronounced decrease in estrogen, induces a distinct change in women's health trajectory. This paper uses detailed data on the reported natural age of menopause for women in the Health and Retirement Study (HRS) and a novel IV identification strategy to estimate the impact of menopause on health and employment. The resulting causal health estimates are largely consistent with known associations in the medical literature. Furthermore, employment declines in lock-step with these changes in health after menopause, indicating a strong link between women's health

enough to make clear distinctions across subgroups.

Table 11: Robustness checks and extensions,
standard error in parentheses and p-values in square brackets

	(1) N	(2) First-stage	(3) Reduced-form	(4) I.V.
Panel A: Sample by BMI level				
Low BMI (< 30)	12,702	0.036	-0.015	-0.417
		(0.008)	(0.004)	(0.143)
		[6.2e-06]	[0.001]	[0.003]
High BMI (≥ 30)	2,536	0.013	-0.024	-1.815
		(0.016)	(0.008)	(2.158)
		[0.402]	[0.002]	[0.400]
Panel B: Sample by Smoking status				
Non-smoker	11,119	0.013	-0.010	-0.771
		(0.010)	(0.005)	(0.691)
		[0.193]	[0.033]	[0.265]
Smoker	4,119	0.034	-0.028	-0.810
		(0.012)	(0.006)	(0.293)
		[0.003]	[2.3e-06]	[0.006]
Panel C: Sample by age				
Early menopause (≤ 50)	4,852	0.008	0.002	0.205
		(0.014)	(0.007)	(1.017)
		[0.575]	[0.823]	[0.840]
Late menopause (> 50)	10,386	0.030	-0.028	-0.922
		(0.009)	(0.004)	(0.285)
		[0.001]	[2.6e-10]	[0.001]

Notes. Column (1) presents the number of observations for each panel estimation. Columns (2) to (4) display the first-stage, reduced-form, and IV estimates, respectively, for each panel. Panel A divides the sample by pre-menopause BMI, categorizing low BMI as below 30 and high BMI as 30 or above. Panel B splits the sample by pre-menopause smoking status, distinguishing between non-smokers and smokers. Panel C divides the sample by education level, with "high education" referring to some years of college, a college degree or more, and "low education" referring to high school or less. Panel D divides the sample based on predicted menopause age, categorizing women with a predicted menopause age of 50 or younger as experiencing early menopause, and those with a predicted menopause age later than 50 as experiencing late menopause. Standard errors clustered at the individual level, in parentheses; p-values in square brackets.

and capacity to work in middle and older ages. The key take-away is that an additional diagnosis of a medical condition essentially results in exit from employment for middle age to older women.

In principle, the analysis could be extended directly in a number of ways. First, due to the manner in which the HRS constructed the PGS, the study focuses exclusively on women of European ancestry. This generates difficulty in extrapolating the results to women of African and Hispanic descent. Further research by race and ethnicity is clearly warranted given the

importance of these populations in public health. Second, since the results are driven by hormonal changes, access to more detailed biological data would be ideal. Blood serum levels of key hormones such as FSH and estradiol could provide valuable insights and could potentially be instrumented using the PGS. The SWAN study offers this type of data along with genetic profiles, making it a promising resource for future research. Additionally, SWAN surveys women prior to the menopausal transition, offering a unique opportunity to analyze hormonal dynamics throughout this critical period. Third, incorporating clinical biomarkers alongside self-reported data can offer a more robust understanding of how menopause impacts health. For instance, the HRS includes valuable biomarker data collected during three rounds between 2006 and 2016. Key measures include total and HDL cholesterol, which indicate lipid levels; glycosylated hemoglobin (HbA1c), a marker of glycemic control; C-reactive protein (CRP), a general indicator of systemic inflammation; and cystatin C, a measure of kidney function. Additionally, in 2016, the HRS conducted the Venous Blood Study, which supplemented the core study with detailed blood sample analyses. This study provided data for various tests, including the comprehensive metabolic panel and nutrient levels. This data along with the previously collected DNA, enable the calculation of epigenetic clocks. These biomarkers can potentially enhance the precision of analyses exploring the relationship between menopause and health outcomes.

In addition, it would be helpful to better understand the mechanisms by which changes in health result in changes in women's approach to work. For example, a key finding was that much effect occurs through arthritis. Hence, it would be interesting to see the extent to which arthritis complications are related to specific job or occupational requirements, such as lifting, crouching, prolonged standing, etc.; similarly, whether there are any effects of menopause on productivity and wages. To the extent that health influences productivity, this should manifest in wages during both the menopausal transition and post-menopause. In a separate analysis, these effects were examined and the findings were equivocal, likely due to the age composition of the HRS sample (individuals 50 and older) and limitations in

statistical power. However, this is an important avenue for future research.

More broadly, this paper demonstrates that menopause is a significant negative health shock for middle aged and older women. Hence, it would be natural to expect spillovers into other domains. For example, first, women affected by menopause-related health issues may transition to Social Security Disability Insurance (SSDI) or claim early retirement benefits rather than leaving the labor force entirely. Future research should explore how menopause-related health conditions influence claims for SSDI or Social Security claims. Second, given the well-known interaction between spouses in work and retirement decisions, it would be valuable to explore how spousal labor supply responds to an adverse shock like menopause. This includes examining the timing of retirement decisions and how menopause may shape joint retirement planning. Third, there is an extensive literature in sociology and demography that highlights the caregiving roles of middle-aged women, often balancing responsibilities for both aging parents and children. Given that this paper finds that menopause has a negative impact on health, exploring how this impacts intergenerational caregiving and support could provide important insights into these dynamics. Fourth, menopause itself could have a direct effect on household formation and marriage/divorce at older ages. Associated physical challenges, like vaginal dryness and reduced libido, coupled with psychological impacts during the menopausal transition, can strain intimacy and dilute the strength of relationships. Lastly, the age at which menopause occurs may influence longevity and mortality outcomes, potentially contributing to differences in mortality rates between men and women. Research on this link could deepen our understanding of gender disparities in health and lifespan.

References

Aloia, J. F., Cohn, S. H., Vaswani, A., Yeh, J. K., Yuen, K., and Ellis, K. (1985). Risk factors for postmenopausal osteoporosis. *The American Journal of Medicine*, 78(1):95–100.

Barbo, D. M. (2002). Reproductive health over the life phases: An overview. *Clinical Obstetrics and Gynecology*, 45(4):00024.

Bay-Jensen, A. C., Slagboom, E., Chen-An, P., Alexandersen, P., Qvist, P., Christiansen, C., Meulenbelt, I., and Karsdal, M. A. (2013). Role of hormones in cartilage and joint metabolism: understanding an unhealthy metabolic phenotype in osteoarthritis. *Menopause*, 20(5):578–586. Supported by Non-U.S. Government funding.

Blundell, R. and Macurdy, T. (1999). Chapter 27 - labor supply: A review of alternative approaches. volume 3 of *Handbook of Labor Economics*, pages 1559–1695. Elsevier.

Bound, J. (1991). Self-reported versus objective measures of health in retirement models. *The Journal of Human Resources*, 26(1):106–138.

Campbell, B., Davis, S., Abramson, M., Mishra, G., Handelsman, D., Perret, J., and Dharmage, S. (2018). Menopause, lung function and obstructive lung disease outcomes: a systematic review. *Climacteric*, 21(1):3–12.

Centers for Disease Control and Prevention (2020). Disability and health related conditions. Accessed: 2024-11-01.

Chavez-MacGregor, M., Elias, S. G., Onland-Moret, N. C., van der Schouw, Y. T., Van Gils, C. H., Monninkhof, E., Grobbee, D. E., and Peeters, P. H. (2005). Postmenopausal Breast Cancer Risk and Cumulative Number of Menstrual Cycles. *Cancer Epidemiology, Biomarkers Prevention*, 14(4):799–804.

Clarke, D., Romano, J. P., and Wolf, M. (2020). The romano–wolf multiple-hypothesis correction in stata. *The Stata Journal*, 20(4):812–843.

Coelingh Bennink, H. J. (2004). Are all estrogens the same? *Maturitas*, 47(4):269–275. Proceedings of The 3rd European Consensus Development Conference on Sex Sterioids and Cardiovascular Diseases.

Colditz, G. A. (1998). Relationship Between Estrogen Levels, Use of Hormone Replacement Therapy, and Breast Cancer. *JNCI: Journal of the National Cancer Institute*, 90(11):814–823.

Colditz, G. A., Willett, W. C., Stampfer, M. J., Rosner, B., Speizer, F. E., and Hennekens, C. H. (1987). Menopause and the risk of coronary heart disease in women. *New England Journal of Medicine*, 316(18):1105–1110.

Currie, J. and Madrian, B. C. (1999). Chapter 50 health, health insurance and the labor market. volume 3 of *Handbook of Labor Economics*, pages 3309–3416. Elsevier.

Davis, S. R., Lambrinoudaki, I., Lumsden, M., Mishra, G. D., Pal, L., Rees, M., Santoro, N., and Simoncini, T. (2015). Menopause. *Nature Reviews Disease Primers*, 1(1):15004.

Day, F. R., Ruth, K. S., Thompson, D. J., et al. (2015). Large-scale genomic analyses link reproductive aging to hypothalamic signaling, breast cancer susceptibility and brca1-mediated dna repair. *Nature Genetics*, 47(11):1294–1303.

Dwyer, D. S. and Mitchell, O. S. (1999). Health problems as determinants of retirement: Are self-rated measures endogenous? *Journal of Health Economics*, 18(2):173–193.

Ettner, S. L. (1997). Is working good for you? evidence on the endogeneity of mental and physical health to female employment. Unpublished paper, Harvard School of Public Health.

Falkeborn, M., Schairer, C., Naessén, T., and Persson, I. (2000). Risk of myocardial infarction after oophorectomy and hysterectomy. *Journal of Clinical Epidemiology*, 53(8):832–837.

Farquhar, C. M., Sadler, L., Harvey, S. A., and Stewart, A. W. (2005). The association of hysterectomy and menopause: a prospective cohort study. *BJOG: An International Journal of Obstetrics & Gynaecology*, 112(7):956–962.

Finkelstein, J. S., Lee, H., Karlamangla, A., Neer, R. M., Sluss, P. M., Burnett-Bowie, S.-A. M., Darakananda, K., Donahoe, P. K., Harlow, S. D., Prizand, S. H., Joffe, H., Kumar, A., Martin, D. E., McConnell, D., Merrilat, S., Morrison, A., Pastore, L. M., Randolph, J. F., Greendale, G. A., and Santoro, N. (2020). Antimullerian hormone and impending menopause in late reproductive age: The study of women's health across the nation. *The Journal of Clinical Endocrinology and Metabolism*, 105(4):e1862–e1871.

Goldin, C. and Katz, L. F. (2002). The power of the pill: Oral contraceptives and women's career and marriage decisions. *Journal of Political Economy*, 110(4):730–770.

Gorina, Y., Elgaddal, N., and Weeks, J. D. (2024). Hysterectomy among women age 18 and older: United states, 2021.

Hale, G. E., Zhao, X., Hughes, C. L., Burger, H. G., Robertson, D. M., and Fraser, I. S. (2007). Endocrine features of menstrual cycles in middle and late reproductive age and the menopausal transition classified according to the staging of reproductive aging workshop (straw) staging system. *The Journal of Clinical Endocrinology and Metabolism*, 92(8):3060–3067.

Harlow, S. D., Gass, M., Hall, J. E., Lobo, R., Maki, P., Rebar, R. W., Sherman, S., Sluss, P. M., de Villiers, T. J., and STRAW 10 Collaborative Group (2012). Executive summary of the stages of reproductive aging workshop + 10: addressing the unfinished agenda of staging reproductive aging. *Menopause (New York, N.Y.)*, 19(4):387–395.

Health and Retirement Study (2024a). HRS Core Interview Survey waves 2008-2020. Public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740). Ann Arbor, MI.

Health and Retirement Study (2024b). RAND HRS Longitudinal File 2020. Public use dataset. Produced and distributed by the University of Michigan with funding from the National Institute on Aging (grant number NIA U01AG009740 and NIA R01AG073289). Ann Arbor, MI.

Heckman, J. J. (1979). Sample selection bias as a specification error. *Econometrica*, 47(1):153–161.

Hotz, V. J. and Miller, R. A. (1988). An empirical analysis of life cycle fertility and female labor supply. *Econometrica*, 56(1):91–118.

Hotz, V. J., Sanders, S. G., and McElroy, S. W. (1999). Teenage childbearing and its life cycle consequences: Exploiting a natural experiment. NBER Working Papers 7397, National Bureau of Economic Research, Inc.

Iorga, A., Cunningham, C. M., Moazeni, S., Ruffenach, G., Umar, S., and Eghbali, M. (2017). The protective role of estrogen and estrogen receptors in cardiovascular disease and the controversial use of estrogen therapy. *Biology of Sex Differences*, 8(1):33.

Killingsworth, M. R. and Heckman, J. J. (1986). Chapter 2 female labor supply: A survey. volume 1 of *Handbook of Labor Economics*, pages 103–204. Elsevier.

Lacroix, A. E., Gondal, H., Shumway, K. R., and Langaker, M. D. (2023). *Physiology, Menarche*. StatPearls Publishing, Treasure Island (FL).

Loprest, P., Rupp, K., and Sandell, S. H. (1995). Gender, disabilities, and employment in the health and retirement study. *Journal of Human Resources*, pages S293–S318.

Martin, A. R., Gignoux, C. R., Walters, R. K., Wojcik, G. L., Neale, B. M., Gravel, S., Daly, M. J., Bustamante, C. D., and Kenny, E. E. (2017). Human demographic history impacts genetic risk prediction across diverse populations. *American Journal of Human Genetics*, 100(4):635–649. Published online 2017 Mar 30.

Mincer, J. and Polachek, S. (1974). Family investments in human capital: Earnings of women. *Journal of political Economy*, 82(2, Part 2):S76–S108.

Mitchell, J. M. and Burkhauser, R. V. (1990). Disentangling the effect of arthritis on earnings: a simultaneous estimate of wage rates and hours worked. *Applied Economics*, 22(10):1291–1309.

Mossa, F. and Ireland, J. J. (2018). Anti-müllerian hormone (amh). In Skinner, M. K., editor, *Encyclopedia of Reproduction (Second Edition)*, pages 222–226. Academic Press, Oxford, second edition edition.

Namavari, N., Jokar, M., Ghodsian, A., Jahromi, H. K., and Rahmanian, V. (2024). Menopausal state and rheumatoid arthritis: a systematic review and meta-analysis. *BMC Rheumatology*, 8(1):48.

Park, S. K., Harlow, S. D., Zheng, H., Karvonen-Gutierrez, C., Thurston, R. C., Ruppert, K., Janssen, I., and Randolph Jr, J. F. (2017). Association between changes in oestradiol and follicle-stimulating hormone levels during the menopausal transition and risk of diabetes. *Diabetic Medicine*, 34(4):531–538.

Parker, W. H. (2010). Bilateral oophorectomy versus ovarian conservation: effects on long-term women's health. *Journal of Minimally Invasive Gynecology*, 17(2):161–166.

Perry, J. R. B., Day, F. R., Elks, C. E., Sulem, P., et al. (2014). Parent-of-origin-specific allelic associations among 106 genomic loci for age at menarche. *Nature*, 514(7520):92–97.

Portman, D. J. and Gass, M. L. (2014). Genitourinary syndrome of menopause: new terminology for vulvovaginal atrophy from the international society for the study of women's sexual health and the north american menopause society. *Climacteric*, 17(5):557–563. PMID: 25153131.

Randolph, John F., J., Crawford, S., Dennerstein, L., Cain, K., Harlow, S. D., Little, R., Mitchell, E. S., Nan, B., Taffe, J., and Yosef, M. (2006). The Value of Follicle-Stimulating Hormone Concentration and Clinical Findings as Markers of the Late Menopausal Transition. *The Journal of Clinical Endocrinology Metabolism*, 91(8):3034–3040.

Randolph, John F., J., Sowers, M., Bondarenko, I., Gold, E. B., Greendale, G. A., Bromberger, J. T., Brockwell, S. E., and Matthews, K. A. (2005). The Relationship of Longitudinal Change in Reproductive Hormones and Vasomotor Symptoms during the Menopausal Transition. *The Journal of Clinical Endocrinology Metabolism*, 90(11):6106–6112.

Randolph, J. F. J., Zheng, H., Sowers, M. R., Crandall, C., Crawford, S., Gold, E. B., and Vuga, M. (2011). Change in follicle-stimulating hormone and estradiol across the menopausal transition: effect of age at the final menstrual period. *The Journal of Clinical Endocrinology and Metabolism*, 96(3):746–754.

Rodriguez, M. and Shoupe, D. (2015). Surgical menopause. *Endocrinology and Metabolism Clinics of North America*, 44(3):531–542. Postmenopausal Endocrinology.

Roman-Bias, J. A., Castaneda, S., Largo, R., and Herrero-Beumont, G. (2009). Osteoarthritis associated with estrogen deficiency. *Arthritis research therapy*, 11(5):241–241.

Rosewell, K. L. and Curry, T. E. (2018). Reproductive senescence in the female. In Skinner, M. K., editor, *Encyclopedia of Reproduction (Second Edition)*, pages 250–254. Academic Press, Oxford, second edition edition.

Samargandy, S., Matthews, K. A., Brooks, M. M., Barinas-Mitchell, E., Magnani, J. W., Janssen, I., Hollenberg, S. M., and El Khoudary, S. R. (2020). Arterial stiffness accelerates within 1 year of the final menstrual period: The swan heart study. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 40(4):1001–1008.

Samargandy, S., Matthews, K. A., Brooks, M. M., Barinas-Mitchell, E., Magnani, J. W., Thurston, R. C., and Khoudary, S. R. E. (2022). Trajectories of blood pressure in midlife women: Does menopause matter? *Circulation Research*, 130(3):312–322.

Santoro, N. (2016). Perimenopause: From research to practice. *Journal of Women's Health*, 25(4):332–339. PMID: 26653408.

Santoro, N. and Randolph, J. F. (2011). Reproductive hormones and the menopause transition. *Obstetrics and Gynecology Clinics of North America*, 38(3):455–466. Perimenopause.

Siddle, N., Sarrel, P., and Whitehead, M. (1987). The effect of hysterectomy on the age

at ovarian failure: identification of a subgroup of women with premature loss of ovarian function and literature review. *Fertility and Sterility*, 47(1):94–100.

Soules, M. R., Sherman, S., Parrott, E., Rebar, R., Santoro, N., Utian, W., and Woods, N. (2001). Executive summary: Stages of reproductive aging workshop (straw). *Climacteric*, 4(4):267–272.

Utian, W. H. (1999). The international menopause menopause-related terminology definitions. *Climacteric*, 2(4):284–286. PMID: 11915855.

van der Schouw, Y., van der Graaf, Y., Steyerberg, E., Eijkemans, M., and Banga, J. (1996). Age at menopause as a risk factor for cardiovascular mortality. *The Lancet*, 347(9003):714–718.

Visniauskas, B., Kilanowski-Doroh, I., Ogola, B. O., McNally, A. B., Horton, A. C., Imulinde Sugi, A., and Lindsey, S. H. (2023). Estrogen-mediated mechanisms in hypertension and other cardiovascular diseases. *Journal of Human Hypertension*, 37(8):609–618.

Visser, M. and Coelingh Bennink, H. J. (2009). Clinical applications for estetrol. *The Journal of Steroid Biochemistry and Molecular Biology*, 114(1):85–89. Proceedings of the 18th International Symposium of The Journal of Steroid Biochemistry and Molecular Biology.

Ware, E., Hornish, U., Nolte, M., and Faul, J. (2024). HRS Polygenic Scores (2006-2012 Genetic Data). Sensitive health data, Health and Retirement Study. Ann Harbor, Michigan.

Westfall, P. and Young, S. (1993). *Resampling-Based Multiple Testing: Examples and Methods for p-Value Adjustment*. Wiley Series in Probability and Statistics. Wiley.

Whiteman, M. K., Hillis, S. D., Jamieson, D. J., Morrow, B., Podgornik, M. N., Brett, K. M., and Marchbanks, P. A. (2008). Inpatient hysterectomy surveillance in the united states, 2000-2004. *American Journal of Obstetrics and Gynecology*, 198(1):34.e1–34.e7.

WHO (1996). Research on the menopause in the 1990s: Report of a WHO Scientific Group. Technical report.

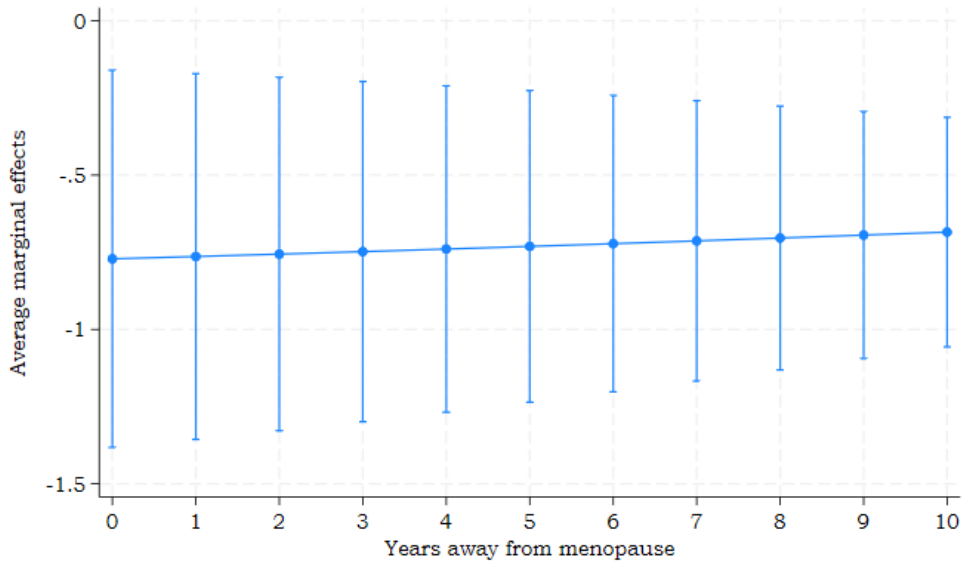
Wilson, L. F. and Mishra, G. D. (2016). Age at menarche, level of education, parity and the risk of hysterectomy: A systematic review and meta-analyses of population-based observational studies. *PloS ONE*, 11(3):e0151398.

Zhao, W., Smith, J. A., Bielak, L. F., Ruiz-Narvaez, E. A., Yu, M., Hood, M. M., Peyser, P. A., Kardia, S. L. R., and Harlow, S. D. (2021). Associations between polygenic risk score for age at menarche and menopause, reproductive timing, and serum hormone levels in multiple race/ethnic groups. *Menopause*, 28(7):819–828.

Appendix

Figures

Figure A.1: Average marginal effects by years away from menopause with 95% confidence intervals



Notes. This figure reflects the average marginal effect of health on working for pay after menopause by years away from menopause. This is constructed from the IV Probit model from Column (2) specification in Table 8.

Tables

Table A.1: STRAW Staging

Stage	**Criteria**
Stage -5	Starts with the age of menarche, during this period, menstrual cycle can be variable because the woman body is adjusting to the change in hormones.
Stage -4	Peak of the reproductive years, where the menstrual cycle becomes stable.
Stage -3	This period is characterized for regular to subtle changes at the end. This period can be divided into 2 substages: -3b and -3a
Substage -3b	Low levels of AMH and FSH.
Substage -3a	Subtle changes in flow length. Hormone levels start to variate. AMH and Inhibin B low. Antral follicle count low.
Stage -2	Start of the menopausal transition. Length of the menstrual cycle starts to lengthen by more than 7 days, although in a regular way. FSH variable and elevated. AMH, Inhibin B and antral follicle count low.
Stage -1	Last period when a woman experiences menstrual discharge. Interval of amenorrhea exceeds 60 days. FSH exceeds 25 IU/L. AMH, Inhibin B and antral follicle count low.
Stage +1	12 months after the lack of a menstrual period (amenorrhea), a person can be certain that they have achieved menopause and pass to this stage. This stage comprises three sub-stages.
Substage +1a	Woman is unaware of being in this substage until 12 months have pass. Now antral follicle count is very low.
Substage +1b	FSH variable and elevated. Vasomotor symptoms are the most likely.
Substage +1c	This is where hormone levels to stabilize, vasomotor symptoms start to dissipate.
Stage +2	Late post-menopause stage will happen after hormone level have stabilize and will last until the end of a woman lifespan.

Table A.2: List of PGS

Phenotype	GWAS	Notes
Age at menarche PGS	Perry et al. (2014)	
Age at menopause PGS	Day et al. (2015)	
General cognition PGS	Davies (CHARGE, 2015)	
Body Mass Index (BMI) PGS	Locke (GIANT, 2015)	
Height PGS	Wood (GIANT, 2014)	
Schizophrenia PGS	Ripke (PGC, 2014)	
Educational attainment PGS (2)	Okbay (SSGAC, 2016)	
Ever smoker PGS	Furberg (TAG, 2010)	
Alzheimer's disease PGS (1)	Lambert (IGAP, 2013)	Without APOE status variants
Alzheimer's disease PGS (2)	Lambert (IGAP, 2013)	With APOE status variants
Waist circumference PGS	Shungin (GIANT, 2015)	
Waist-to-hip ratio PGS	Shungin (GIANT, 2015)	
Neuroticism PGS	Okbay (SSGAC, 2016)	
Subjective well-being PGS	Okbay (SSGAC, 2016)	
Depressive symptoms PGS	Okbay (SSGAC, 2016)	
Coronary artery disease PGS	Schunkert (CARDIoGRAM, 2011)	
Myocardial infarction PGS	Nikpay (CARDIoGRAMplusC4D, 2015)	
Plasma cortisol PGS	Bolton (CORNET, 2014)	
Type 2 diabetes PGS	Morris (DIAGRAM, 2012)	
ADHD PGS (1)	Neale (PGC, 2010)	Attention Deficit Hyperactivity Disorder
ADHD PGS (2)	Demontis (PGC, 2017)	Attention Deficit Hyperactivity Disorder
Mental health cross disorder	Smoller (PGC, 2013)	
Major depressive disorder PGS (1)	Ripke (PGC, 2013)	
Number of cigarettes per day PGS	Furberg (TAG, 2010)	
Extraversion PGS	van den Berg (GPC, 2016)	
Autism PGS	Anney (PGC, 2017)	
Longevity PGS	Broer (CHARGE, 2015)	
Antisocial behavior PGS	Tielbeek (BROAD, 2017)	
Education attainment PGS (3)	Lee (SSGAC, 2018)	
Obsessive compulsive disorder PGS	(IOCDF-GC & OCGAS, 2017)	
Age at first birth PGS	Barban (Sociogenome, 2016)	Combined, female and male GWAS
Number of children ever born PGS	Barban (Sociogenome, 2016)	Female and male GWAS
Major depressive disorder PGS (2)	Wray (PGC, 2018)	

Contd. List of PGS

Phenotype	**GWAS**	**Notes**
Post traumatic stress disorder PGS	Duncan (PGC, 2018)	African, European & Combined GWAS
High density lipoprotein (HDL) PGS	Willer (GLGC, 2013)	
Low density lipoprotein (LDL) PGS	Willer (GLGC, 2013)	
Total cholesterol PGS	Willer (GLGC, 2013)	
Anxiety PGS	Otowa (ANGST, 2016)	Factor score and Case control
Blood urea nitrogen PGS	Wuttke (CKDGen, 2019)	European and transancestry GWAS
Chronic kidney disease PGS	Wuttke (CKDGen, 2019)	European and transancestry GWAS
Diastolic blood pressure PGS	Liang (COGENT, 2017)	
Systolic blood pressure PGS	Liang (COGENT, 2017)	
Body mass index PGS (2)	Yengo (GIANT, 2018)	
Height PGS (2)	Yengo (GIANT, 2018)	
Age at smoking initiation PGS	Liu (GSCAN, 2019)	
Cigarettes per day PGS	Liu (GSCAN, 2019)	
Drinks per week PGS	Liu (GSCAN, 2019)	
Smoking cessation PGS	Liu (GSCAN, 2019)	
Smoking initiation PGS	Liu (GSCAN, 2019)	
Hypertension PGS	Liang (COGENT, 2017)	
Cannabis use PGS	Pasman (ICCUKB, 2019)	
Alzheimer's disease PGS (3)	Kunkle (IGAP, 2019)	pT=1 with APOE/TOMM40 region
Alzheimer's disease PGS (4)	Kunkle (IGAP, 2019)	pT=1 without APOE/TOMM40 region
Alzheimer's disease PGS (5)	Kunkle (IGAP, 2019)	pT=0.01 with APOE/TOMM40 region
Alzheimer's disease PGS (6)	Kunkle (IGAP, 2019)	pT=0.01 without APOE/TOMM40 region
Alcohol dependence PGS	Walters (PGC, 2018)	
Pulse pressure PGS	Liang (COGENT, 2017)	
eGFR PGS	Wuttke (CKDGen, 2019)	European and Transancestry GWAS
Educational attainment PGS (3)	Lee (SSGAC, 2018)	
HbA1c PGS	Wheeler (MAGIC, 2017)	European and African GWAS
General cognition PGS (2)	Davies (CHARGE, 2018)	
Bipolar disorder PGS	Sklar (PGC, 2011)	

Table A.3: Regressions of PGS of Menopause and Menarche on other PGS, standard errors in parentheses and p-values in square brackets

	PGS Menopause	PGS Menarche
	(1)	(2)
General cognition PGS	0.074	-0.053
	(0.011)	(0.013)
	[0.000]	[0.000]
Body Mass Index (BMI) PGS	-0.025	-0.171
	(0.024)	(0.026)
	[0.301]	[0.000]
Height PGS	-0.430	0.171
	(0.028)	(0.032)
	[0.000]	[0.000]
Schizophrenia PGS	-0.062	-0.035
	(0.020)	(0.022)
	[0.002]	[0.108]
Educational attainment PGS (2)	0.021	0.014
	(0.019)	(0.021)
	[0.273]	[0.503]
Ever smoker PGS	-0.072	0.017
	(0.011)	(0.012)
	[0.000]	[0.166]
Alzheimer's disease PGS (1)	-0.497	1.545
	(1.337)	(1.490)
	[0.710]	[0.300]
Alzheimer's disease PGS (2)	0.467	-1.497
	(1.338)	(1.491)
	[0.727]	[0.315]
Waist circumference PGS	0.071	-0.006
	(0.024)	(0.026)
	[0.003]	[0.824]
Waist-to-hip ratio PGS	0.027	0.033
	(0.014)	(0.015)
	[0.049]	[0.029]
Neuroticism PGS	0.003	-0.010
	(0.014)	(0.016)
	[0.855]	[0.513]
Subjective well-being PGS	-0.014	0.010
	(0.011)	(0.012)
	[0.198]	[0.412]
Depressive symptoms PGS	-0.005	-0.021
	(0.013)	(0.014)
	[0.691]	[0.134]
Coronary artery disease PGS	0.014	-0.002
	(0.011)	(0.012)
	[0.215]	[0.856]
Myocardial infarction PGS	-0.015	-0.012
	(0.011)	(0.012)
	[0.174]	[0.316]
Plasma cortisol PGS	0.030	0.022
	(0.010)	(0.011)
	[0.002]	[0.050]
Type 2 diabetes PGS	0.053	-0.003
	(0.013)	(0.015)
	[0.000]	[0.838]
Attention Deficit Hyperactivity	-0.036	0.007
Disorder (ADHD) PGS (1)	(0.012)	(0.013)
	[0.002]	[0.587]
Attention Deficit Hyperactivity	-0.039	-0.025
Disorder (ADHD) PGS (2)	(0.011)	(0.012)
	[0.000]	[0.040]

Contd. Regressions of PGS of Menopause and Menarche on other PGS, standard errors in parentheses and p-values in square brackets

	PGS Menopause (1)	PGS Menarche (2)
Mental health cross disorder	0.158	-0.021
	(0.019)	(0.021)
	[0.000]	[0.310]
Major depressive disorder PGS (1)	-0.036	0.013
	(0.011)	(0.013)
	[0.002]	[0.302]
Number of cigarettes per day PGS	-0.040	-0.007
	(0.011)	(0.012)
	[0.000]	[0.568]
Extraversion PGS	-0.019	-0.001
	(0.011)	(0.012)
	[0.076]	[0.899]
Autism PGS	-0.042	-0.019
	(0.016)	(0.017)
	[0.007]	[0.281]
Longevity PGS	0.056	-0.006
	(0.012)	(0.013)
	[0.000]	[0.668]
Antisocial behavior PGS	-0.033	0.024
	(0.010)	(0.011)
	[0.001]	[0.035]
Education attainment PGS (3)	0.061	-0.032
	(0.031)	(0.035)
	[0.052]	[0.365]
Obsessive compulsive disorder PGS	-0.012	-0.004
	(0.010)	(0.011)
	[0.216]	[0.708]
Age at first birth PGS	-0.088	-0.309
combined GWAS	(0.091)	(0.102)
	[0.334]	[0.002]
Age at first birth PGS,	0.110	0.306
female GWAS	(0.075)	(0.084)
	[0.146]	[0.000]
Age at first birth PGS,	0.057	0.161
male GWAS	(0.037)	(0.041)
	[0.121]	[0.000]
Number of children ever born PGS,	-0.079	0.074
combined GWAS	(0.107)	(0.119)
	[0.460]	[0.535]
Number of children ever born PGS,	0.040	-0.020
female GWAS	(0.085)	(0.094)
	[0.636]	[0.835]
Number of children ever born PGS,	0.058	-0.023
male GWAS	(0.054)	(0.060)
	[0.285]	[0.700]
Major depressive disorder PGS (2)	0.015	-0.038
	(0.013)	(0.015)
	[0.251]	[0.009]
Post traumatic stress disorder PGS,	0.007	-0.010
African GWAS	(0.012)	(0.013)
	[0.522]	[0.449]
Post traumatic stress disorder PGS,	-0.027	0.005
European GWAS	(0.015)	(0.017)
	[0.083]	[0.792]
Post traumatic stress disorder PGS,	-0.029	-0.012
combined GWAS	(0.015)	(0.016)
	[0.045]	[0.459]

Contd. Regressions of PGS of Menopause and Menarche on other PGS, standard errors in parentheses and p-values in square brackets

	PGS Menopause (1)	PGS Menarche (2)
High density lipoprotein (HDL) PGS	-0.002	0.016
	(0.013)	(0.014)
	[0.857]	[0.258]
Low density lipoprotein (LDL) PGS	-0.122	-0.018
	(0.026)	(0.029)
	[0.000]	[0.535]
Total cholesterol PGS	0.117	0.010
	(0.028)	(0.031)
	[0.000]	[0.741]
Anxiety factor score PGS	0.053	0.019
	(0.014)	(0.015)
	[0.000]	[0.206]
Anxiety case control PGS	-0.045	0.022
	(0.013)	(0.015)
	[0.001]	[0.138]
Blood urea nitrogen PGS,	-0.007	-0.197
European GWAS	(0.032)	(0.035)
	[0.832]	[0.000]
Blood urea nitrogen PGS,	0.026	0.152
transancestry GWAS	(0.031)	(0.035)
	[0.395]	[0.000]
Chronic kidney disease PGS	0.163	-0.075
	(0.042)	(0.047)
	[0.000]	[0.114]
Chronic kidney disease PGS,	-0.101	0.008
transancestry GWAS	(0.041)	(0.046)
	[0.015]	[0.865]
Diastolic blood pressure PGS	0.023	-0.009
	(0.028)	(0.031)
	[0.412]	[0.774]
Body mass index PGS (2)	-0.000	-0.071
	(0.018)	(0.020)
	[0.981]	[0.000]
Height PGS (2)	0.173	0.010
	(0.018)	(0.020)
	[0.000]	[0.601]
Age at smoking initiation PGS	0.040	0.104
	(0.012)	(0.014)
	[0.001]	[0.000]
Cigarettes per day PGS	-0.016	0.016
	(0.011)	(0.012)
	[0.138]	[0.187]
Drinks per week PGS	-0.001	-0.004
	(0.010)	(0.012)
	[0.917]	[0.708]
Smoking cessation PGS	0.044	-0.034
	(0.012)	(0.013)
	[0.000]	[0.009]
Smoking initiation PGS	0.050	0.021
	(0.012)	(0.014)
	[0.000]	[0.125]
Hypertension PGS	-0.010	-0.027
	(0.012)	(0.014)
	[0.401]	[0.049]
Cannabis use PGS	0.046	-0.005
	(0.011)	(0.012)
	[0.000]	[0.697]

Contd. Regressions of PGS of Menopause and Menarche on other PGS, standard errors in parentheses and p-values in square brackets

	PGS Menopause	PGS Menarche
	(1)	(2)
Alzheimer's disease PGS (3)	6.599	7.542
	(6.625)	(7.382)
	[0.319]	[0.307]
Alzheimer's disease PGS (4)	-1.319	-1.598
	(1.305)	(1.454)
	[0.312]	[0.272]
Alzheimer's disease PGS (5)	-6.546	-7.580
	(6.629)	(7.387)
	[0.323]	[0.305]
Alzheimer's disease PGS (6)	1.400	1.614
	(1.376)	(1.534)
	[0.309]	[0.293]
Alcohol dependence PGS	-0.018	-0.008
	(0.010)	(0.012)
	[0.087]	[0.514]
Pulse pressure PGS	0.048	-0.006
	(0.033)	(0.037)
	[0.141]	[0.868]
Systolic blood pressure PGS	-0.031	0.039
	(0.046)	(0.051)
	[0.502]	[0.448]
eGFR PGS,	0.191	-0.252
European GWAS	(0.058)	(0.064)
	[0.001]	[0.000]
eGFR PGS,	-0.141	0.157
Transancestry GWAS	(0.059)	(0.065)
	[0.016]	[0.016]
Educational attainment PGS (3)	-0.010	-0.090
	(0.029)	(0.032)
	[0.718]	[0.005]
HbA1c PGS,	-0.019	-0.009
African GWAS	(0.010)	(0.011)
	[0.064]	[0.419]
HbA1c PGS,	-0.002	-0.011
European GWAS	(0.011)	(0.013)
	[0.846]	[0.387]
General cognition PGS (2)	-0.046	0.007
	(0.014)	(0.015)
	[0.001]	[0.633]
Bipolar disorder PGS	-0.056	0.031
	(0.012)	(0.014)
	[0.000]	[0.025]
Age at menarche PGS	0.013	
	(0.011)	
	[0.232]	
Age at menopause PGS		0.016
		(0.013)
		[0.232]
Constant	0.056	0.041
	(0.020)	(0.022)
	[0.004]	[0.062]
Observations	6,894	6,894
R^2	0.364	0.196

Notes. This table reports OLS estimates of regressions of the dependent variables on the 72 PGS for other phenotypes. Standard errors in parentheses; p-values in square brackets.