The Evolutionary Ecology of Menopause

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I, Megan Arnot, confirm that the work presented in this thesis is my own. Where the information has been derived from other sources, I confirm this has been indicated in the thesis. The work presented in Chapter 3 was previously published in *Ecology and Evolution* as *Current ecology, not ancestral dispersal patterns, influences menopause symptoms severity* by Yuping Yang, me, and Ruth Mace. This study was conceived by all of the authors, I carried out the statistical analysis and wrote the paper, Yuping Yang assisted with the data collection and contributed to the paper revisions, and Ruth Mace supervised the project and contributed to paper revisions. The work presented in Chapter 4 was previously published in *Royal Society Open Science* as *Sexual Frequency is associated with age of natural menopause: results from the Study of Women's Health Across the Nation*, and co-authored with Ruth Mace. All authors conceived of the project. Ruth Mace supervised the project, I completed the statistical analysis and wrote the manuscript, and we both contributed to revisions and manuscript edits. The work presented in Chapter 5 is published in *PLOS One* under the title *The relationship between support, stress, and menopause symptoms*. This was co-authored with two of my supervisors, Emily Emmott and Ruth Mace. I conceived of the idea, wrote the manuscript, and completed statistical analysis. Emily Emmott and Ruth Mace supervised the project, and contributed to the editing of the manuscript. The work presented in Chapter 6 is published in *Scientific Reports* and co-authored by Ruth Mace.

Abstract

Despite appearing to be maladaptive, the human menopause and prolonged post-reproductive lifespan are thought to have been shaped in our evolutionary history by natural selection. As a result, there has been a great deal of research looking at the inclusive fitness benefits of a post-reproductive lifespan. However, there are still many things we do not know about menopause, such as whether current variation in menopause timing is the result of evolutionary trade-offs, whether menopause symptoms require an evolutionary explanation, and how post-reproductive care functions in a sample of women from the United Kingdom. This thesis focuses on trying to fill these identified gaps in the literature using data from the United Kingdom, United States, and China. I find no evidence for menopause symptoms being facultative, nor that menopause timing varies in the way predicted by current evolutionary models. However, I do find that a later menopause is predicted by an increased likelihood of pregnancy, suggesting an energetic trade-off. Further, I show that menopause symptoms predictably vary relative to one's ecology, with a more stressful environment predicting worse symptoms. When looking at caring behaviour, I found evidence in favour of it being facultative relative to fecundity status, with pre-menopausal women caring more for their parents, while post-menopausal women spent more time caring for their grandchildren. Finally, I present evidence for an earlier menopause predicting a greater number of grandchildren, suggesting that women are able to offset the costs of being post-reproductive by increasing indirect fitness. Results from this thesis suggest that many aspects of the menopausal transition are plastic, and often vary in a way predicted by evolutionary theory. Through understanding these trends, it may allow those who experience menopause more autonomy over the transition. Further, my research on fecundity status and caring behaviours demonstrates the behavioural implications of energetic trade-offs.

Impact statement

Menopause is an under-researched topic, however, it has gained some interest from evolutionary anthropologists in recent years. Despite this, it is not clear whether current trends we see in variation in menopause timing and menopause symptoms can be explained from an evolutionary framework. In this thesis, I apply principles derived from human behavioural ecology to understand if aspects of the menopausal transition today can be understood as the result of evolutionary trade-offs. As well as being of academic interest, understanding the influences of menopause timing and symptoms has clear clinical implications: through understanding the demographic correlates of such, it will assist in informing practitioners with how to treat those experiencing menopause; and by understanding how ecological factors impact the menopausal transition, it may allow individuals to have more control over this period of their life. Research disseminating from this thesis has already had demonstrable impact: the published version of Chapter 3 has been downloaded over 30,000 times, and results from Chapters 3, 4 and 5 have been publicised in popular media, including national television and radio.

Table of Contents

List of Tables

List of Figures

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CHAPTER ONE

Evolutionary theory and menopause

1.1 Introduction

Natural selection is a process which favours traits that best increase the survival and reproductive success of organisms relative to their local ecology. As a result, many of the traits we see throughout the natural world have clear evolutionary benefits; or at least are neutral in terms of their effect on fitness, meaning they neither enhance nor hinder the individual's survival and/or reproductive success. However, occasionally we see phenotypes that appear to be detrimental to an organism's fitness. Known as maladaptive traits or evolutionary puzzles, these are harder to understand from an evolutionary framework – as why would natural selection have selected for a phenotype that hinders survival or reproductive success? Humans, in particular, show a suite of both behavioural and physical traits that appear to hinder fitness; examples include behaviours such as the tendency to over-consume fatty foods (Neel, 1962; Hales & Barker, 2001; Speakman, 2013), the prolonged use of ineffective (e.g. homeopathic) medicine (Tanaka et al., 2009), engagement in harmful bodily practices (Koziel et al., 2010; Howard & Gibson, 2017; Wander, 2017), and the negative correlation between wealth and fertility (Borgerhoff Mulder, 1998).

In addition to these behavioural practices, human females also display a physiological feature that does not immediately make sense from an evolutionary framework: complete reproductive cessation at midlife. Known as menopause, this event usually occurs at around the age of 50 (Laisk et al., 2019), meaning that – assuming the maximum human lifespan is around 122 years (Walker & Herndon, 2008) –women could potentially spend over half their lives post-reproductive. This is puzzling, as why would selection have favoured a phenotype that massively limits reproductive success by rendering a woman infertile for a large proportion of her life? In addition to this, no other primates experience menopause coupled with an extended post-reproductive lifespan outside of captivity (Walker & Herndon, 2008; Alberts et al., 2013), with only humans and a handful of cetaceans displaying this trait (Ellis et al., 2018a; Ellis et al., 2018b). While some argue that the human menopause does not require an evolutionary explanation, and that it is simply a by-product of

factors such as the extended human lifespan (Pavard et al., 2008), antagonistic pleiotropy (Wood et al., 2000), or phylogenetic constraints (Huber & Fieder, 2018); others believe that the trait has been shaped by evolution, and therefore that it has hidden fitness benefits. These hidden benefits include protection against maternal mortality (Williams, 1957), positive survival outcomes associated with having a post-reproductive grandmother (Hawkes et al., 1998), and avoiding the potential reproductive conflict that can emerge from multigenerational breeding (Cant & Johnstone, 2008).

There is currently good empirical support for menopause and a post-reproductive lifespan having evolutionary origins (reviewed later in section 1.3.3.3); however, there are still many things unclear in the literature. Is menopause an evolved or maintained trait? Is age of menopause a facultative today? Do menopause symptoms require an evolutionary explanation? If menopause was selected for, is it still adaptive today? In this thesis – through looking at the demographic correlates of aspects of the menopausal transition – I aim to start answering some of these questions. By understanding the different social, cultural, and ecological factors that associate with aspects of the menopausal transition, this research will contribute to our evolutionary understanding of menopause and the post-reproductive lifespan.

In the rest of this chapter, I expand on the theoretical framework that will be used to inform the hypotheses tested throughout this thesis. I then briefly explain the proximate understanding of why menopause happens before introducing the prevailing non-adaptive and adaptive explanations of menopause. I finish by elaborating on the aims and outline of this thesis.

1.2 Theoretical overview

This thesis will utilise theories and concepts from behavioural ecology (Krebs & Davies, 1993) to understand the variation in age of natural menopause and the behaviours often associated with fertility status. Behavioural ecology arose in the 1970s from the field of ethology, and was known initially as 'sociobiology', with its aim to use biology and evolution to understand animal behaviour. The application of sociobiology to humans was first addressed in E. O. Wilson's (1975) book of the same name, and, from here, the field of human behavioural ecology emerged, which draws upon principles from anthropology, evolutionary biology, and psychology. It is now considered to be a cross-disciplinary subject, with research disseminating from

the field having implications for understanding human physiology, diseases, and public health (Nesse & Stearns, 2008; Smith et al., 2011; Stearns, 2012; Wells et al., 2017; Arnot et al., 2020). In sum, behavioural ecologists believe that natural selection is effective in selecting phenotypes that maximise fitness relative to the local ecology. This school of thought is derived from evolutionary biology, which typically relies on measuring genetic processes; however, the empirical testing of hypotheses within behavioural ecology typically relies on measuring phenotypes, which means there is the implicit assumption that phenotypic patterns are valid proxies of heritable patterns. This issue is known as the phenotypic gambit (Grafen, 1984), and while there are continuing debates about the validity of this approach when looking at the selection of certain traits, there is a growing body of evidence supporting the idea that phenotypic analysis is a useful approximation of genetic architecture (Cheverud, 1988; Roff, 1995; Reale & Festa-Bianchet, 2000).

1.2.1 Evolutionary theory and human behavioural ecology

1.2.1.1 Questions and answers

In biology, when looking at traits, we generally want to know why they are the way they are. However, asking why something happens is not necessarily straightforward. For example, if I were to ask "why does a peacock have a colourful tail?" one scientist might tell you that the spacing of the tail feather barbules results in a unique scattering of light waves that produces the iridescent colouration we see (Burgess et al., 2006); while another might tell you that peacocks have colourful tails because of sexual selection and peahen mate choice (Zahavi, 1975). Here, both scientists are correct, but they address different levels of explanation. First identified by Mayr (1961) and then developed into a conceptual framework by Tinbergen (1963), biological phenomena can be explained at a proximate level as a means of trying to understand the ontogeny of the trait and the underlying physiological mechanism; but they can also be understood from an evolutionary – or ultimate – perspective, in which we try and understand the trait in the context of its phylogenetic history and adaptive function. As such, Tinbergen concluded there are four categories of question which fit into two levels of explanation:

1) Ultimate explanations, which attempt to explain *why* an organism presents certain traits:
    i. What is the function of the trait?

ii. What is the phylogenetic history of the trait?

2) Proximate explanations which attempt to explain *how* the organism's traits work:

iii. How did the trait develop ontogenically?

iv. How does the trait work mechanistically?

When asking the question "why do humans have menopause?" for scientists concerned with proximate explanations, the answer to this question would be something like: because a finite reserve of follicles are produced during gestation, and due to ovulation and atresia we run out of eggs around the age of 50 (see section 1.3.1). This answer is of course correct, but it is not necessarily what evolutionary thinkers are interested in. Rather, evolutionary scientists are more concerned with when and why the trait emerged. However, proximate knowledge is still required for a holistic understanding, as completely ignoring them may result in nonsense hypotheses (Tadinac, 2020). As such, efforts to integrate proximate and ultimate understandings of menopause have been made (Laisk et al., 2019; Fraser et al., 2020).

1.2.1.2 Variation

Evolution by natural selection predicts that the differential survival and reproduction of organisms relative to their local ecology has resulted in the diversity of life we see today. Here, organisms with traits better suited to their environment will have a greater chance of surviving and reproducing, meaning that their genes are more likely to be passed on to subsequent generations, and their phenotypes will be selected for and become more prevalent within that particular ecology. As the global environment is variable there is not one optimal phenotype, so the variation in phenotypes across species we see today is the result of the different ecological pressures under which they all evolved. A simple example of this is adaptations to extreme temperatures: many animals that live in cold environments – such as whales and seals – have thick layers of fat that serve the adaptive function of heat retention (Parry, 1949). Under cold conditions, this phenotype is adaptive; however, if animals in hotter environments were to have excess fat, it could result in them over-heating, and thus be detrimental to survival. This demonstrates the importance of considering the local ecology when trying to understand phenotypic variation.

As well as the environment producing phenotypic variation, behavioural and cultural practices have also been observed to create selective pressures themselves in humans. For example, amongst the Bajau people of Southeast Asia, breath-hold

diving is required to maintain their subsistence lifestyle. Described as marine hunter-gatherers, this group has been living on houseboats for over 1000 years (Sather, 1997), and are renowned for their ability to dive to depths of over 70m (Schagatay, 2014). Comparative genomic research has found that, in the Bajau, natural selection has favoured genetic variants that result in an increased spleen size, and therefore a larger reservoir of oxygenated red blood cells that allows them to dive for longer periods (Ilardo et al., 2018). Therefore, a cultural practice itself has caused a selective pressure that resulted in changes at the genetic level. Similarly, the development of agriculture is thought to have resulted in the prolonged production of lactase into adulthood (Holden & Mace, 1997; Swallow, 2003; Itan et al., 2009; Ranciaro et al., 2014), and a genetic mutation that protects against kuru (a disease caused by an infectious protein in brain tissue) has been found in the Fore people of Papua New Guinea who traditionally engaged in the cultural practice of mortuary cannibalism (Mead et al., 2009).

With physiology being adaptive relative to the local environment, behavioural ecologists also predict that behaviour will optimally vary according to local ecology (Mace, 2014; Barsbai et al., 2021). Therefore, it is thought that natural selection has allowed us to weigh up the costs and benefits of different strategies and adopt the one that results in the greatest fitness. Evidence has been found for many behaviours to vary in such a way, including foraging (Macarthur & Pianka, 1966; Hill et al., 1987), marriage patterns (Fortunato, 2015), and parental investment (Trivers, 1972; Lawson & Mace, 2011). Human behavioural ecologists therefore perceive many aspects of our physiology to be plastic, with plasticity here referring to the range of potential phenotypes (both behavioural and physical) that can result from a single genotype, through environmental interactions that can occur both during development and throughout the life course (Pigliucci et al., 2006).

1.2.1.3 Life history theory

Human behavioural ecologists often interpret and analyse phenotypic variation and plasticity from a life history framework, which is a theory that was initially developed to explain diversity between species in the animal kingdom (Charnov, 1991; Roff, 1992; Stearns, 1992; Charnov, 1993). An organism's life history refers to the age-specific schedules of growth, fecundity, and mortality, in addition to the traits connected to these schedules (e.g. growth and development), and those that result

from them (e.g. age of sexual maturity, maximum lifespan) (Stearns, 1992). Central to life history theory is the 'Principle of Allocation' (Cody, 1966), which states that resources – such as time and energy – are finite, and cannot be simultaneously invested in multiple tasks. As a result of this, all organisms will face trade-offs regarding how to optimally allocate these resources. Phenotypes with the optimal allocation strategy will be the fittest, and therefore should be selected for, with the optimal strategies not being objective but contingent on environmental conditions (Stearns & Koella, 1986; Charnov, 1991; Stearns, 1992; Charnov, 1993).

The first fundamental trade-off is between growth and reproduction. In line with the Principle of Allocation, any energy allotted to current growth means forgoing current reproduction, and vice versa. Thus, a degree of 'bet-hedging' is required here, as future reproduction is contingent on the organism having a future in which it can reproduce. It would therefore not be optimal for an organism to invest in future reproduction if it has a high risk of predation and therefore a risk of early mortality. This trade-off is used to explain the stature of human pygmies, where it is thought their small stature evolved as a result of selection for early reproduction due to high mortality rates (Migliano et al., 2007). The second key trade-off is between offspring number and offspring quality (Lack, 1954). Also known as a quantity-quality trade-off, this refers to the idea that parents must 'choose' between their own fertility and the success of their offspring. Parents have limited resources available for investment, and – all else being equal – each additional offspring will reduce the average level of investment across the progeny (Kaplan & Gangestad, 2005; Lawson & Mace, 2009; Lawson & Borgerhoff Mulder, 2016; Lynch, 2016). The theory underlying this trade-off is rooted in research conducted by ornithologist David Lack, who stated that the clutch size of birds had "evolved through natural selection to correspond with the largest number of young for which the parents can on the average find enough food" (Lack, 1954:22). To show this, Lack (1954) manipulated the offspring number of birds by either adding or removing eggs, and found that unmanipulated clutches produced the most surviving young, suggesting that the birds were adaptively optimising the number of offspring they have in a way that increase their fitness.

Whether an organism invests energy into growth or reproduction, or offspring quantity or quality, is thought to be dependent upon their risk of extrinsic mortality (death from an external force, such as predation) and resource availability

(Stearns, 1992). When the environment is harsher (e.g. scarce resources, higher mortality), it is predicted that it would be adaptive to preferentially allocate resources towards reproduction and offspring quantity to ensure genetic propagation. On the other hand, in more forgiving ecologies, organisms are able to age more slowly, allowing them to allocate more resources to growth related activities and later life reproduction (Charnov, 1991; Charnov, 1993). For instance, individuals in low mortality environments may be better off delaying reproduction and investing in their own survival and growth, or investing in any existing offspring they have (Stearns & Kawecki, 1994). This is observed in humans, where those living in low mortality environments often delay their age at first birth (Quinlan, 2007), while investing more in offspring quantity has been shown to be optimal in environments with high child mortality (Wilson & Daly, 1997; Belsky et al., 2010). The timing of life history traits in response to local conditions is thought to be indicative of the life history strategy of the species or individual. Life history strategies have been modelled to lie on a fast-slow continuum, whereby 'fast-strategists' preferentially allocate energy towards tasks related to reproduction and offspring quantity, while slow strategists are more concerned with early life growth and late life reproduction, and therefore offspring quality (Promislow & Harvey, 1990). Life history traits thought to covary with a faster strategy include a smaller body size, earlier reproduction, larger litters, and a shorter lifespan, whereas slow strategists would present the converse characteristics. Life history strategies are relative; so, while a gorilla (for example) is a slow strategist compared to a mouse, relative to a whale it has a faster life history strategy.

Life history theory was initially developed to explain the variation in life history traits *between* species; however, it has since been expanded to understand the physiological and behavioural differences we see within species. It is therefore thought that the effect of ecological conditions on life history traits can not only be applied to the evolution of species-specific life histories, but also the variance within these strategies that occur within the species-typical range (Stearns & Koella, 1986). Some research has found that, in humans, harsher, more unpredictable environments associate with traits thought to be indicative of a faster life history, such as an earlier menarche and an earlier age of first birth in females (Wilson & Daly, 1997; Ellis et al., 1999; Nettle et al., 2010; Nettle, 2011; Uggla & Mace, 2016), indicating energy being

preferentially invested into reproduction and offspring quantity in response to ecological uncertainty.

As well as current ecological cues associating with life history traits, conditions present in early life have also been thought to determine one's 'pace of life'. Here, it is thought that childhood can act as a blueprint of what can be expected during adulthood (Csatho & Birkas, 2018; Kavanagh & Kahl, 2018). As such, there is a large body of research showing that early-life adversity (e.g. poverty, parent-offspring conflict) associates with the development of traits typically associated with a fast life history strategy later in the life course, such as accelerated puberty, earlier reproduction, and faster physiological maturation (Chisholm et al., 2005; Nettle et al., 2010; Young et al., 2019; McDermott et al., 2021).

More recently, the life history framework has been used to model variation behavioural and psychological traits (Ellis et al., 2009). Here, it is presumed that different life history strategies can be identified by looking at behavioural and psychological traits that indicate how much the actor considers the future. For instance, an unpredictable environment has been associated with a tendency to focus on short-term goals and engage in risky behaviour, which has led some to characterise these behaviours (and others associated with future discounting) as fast life history traits (Ellis et al., 2009; Griskevicius et al., 2011; Ellis et al., 2012; Chen & Chang, 2016; Young et al., 2019). It has also been proposed that it is possible to measure 'the speed' of one's life history strategy using psychometric tools, the most popular of which being the Arizona Life History Battery (and its shorter version, the 'mini-K') which includes questions related to family relationships, altruism, religiosity, and self-control (Figueredo et al., 2004; Figueredo et al., 2007). A slow life history according to this scale is characterised by high levels of conscientiousness, religiosity, prosociality, emotional stability, agreeableness, and a secure attachment style. Despite its popularity, it does not include any questions about traditional life history traits (e.g. age at first birth). Efforts to validate the scale by correlating it with biological life history traits have not been fruitful, with some studies finding that a slow life history as predicted by the psychometric scale associates with biometrically fast life history traits (Copping et al., 2014; Woodley of Menie et al., 2017; Mathes, 2018; Mededovic, 2020).

As the application of life history theory to aspects of human physiology and behaviour has grown, as have the criticisms of both the use of the framework and

the framework itself (Nettle & Frankenhuis, 2019; Nettle & Frankenhuis, 2020; Sear, 2020a). Life history theory was initially developed within the field of biology to understand variation in phenotypes that result from trade-offs. However, life history theory as applied to humans has had a strong focus on the fast-slow continuum, and behaviours as a proxy of 'life history strategy'. The continuum is borne from work in evolutionary biology, which showed that *species* can be organised based on their life history traits, with fast strategists prioritising reproduction, and slow strategists investing more energy into growth and later life reproduction (Promislow & Harvey, 1990). This continuum was then expanded to be applied to variation in human life history traits (both traditional and non-traditional), with the idea that some humans are fast strategists and others slow strategists. However, there is not currently a formal model precisely outlining what we would predict to find if life history strategies did vary predictably across a within-species continuum. Further, there is currently little empirical evidence that life history traits do covary within humans in the way one would expect according to the fast-slow continuum. In a sequence analysis of data from the United States (US), it was shown that life history traits did not cluster together in the expected way, and that individuals did not follow coherent life history strategies, so could therefore not be labelled as fast or slow strategists (Sheppard & Van Winkle, 2020). Further, using data from the United Kingdom (UK) it was shown that age at menarche did not consistently predict the timing of other life history traits, suggesting that pubertal timing is not an accurate predictor of 'life history strategy' (Lawn et al., 2020). Early life adversity also does not always associate with life history traits in adulthood in the expected way, with there being some evidence that risk of mortality affecting juveniles actually strengthens selection for late-life survival, which contradicts the predictions that harsh conditions during childhood would 'speed up' one's life history and consequentially shorten their lifespan (Moorad et al., 2019).

It may be that some of the confusion within the human life history literature derives from the fact that the verbal models were constructed in a WEIRD (Western, Educated, Industrialised, Rich, Democratic; Henrich et al., 2010) setting without considering the effect of culture on many life history traits. For instance, childhood adversity is thought to speed up one's pace of life, but many events often thought of as stressful (e.g. number of house moves, paternal absence) are not generalisable cross-culturally or temporally. Where research into life history strategies has been

carried out in non-WEIRD societies, some results from WEIRD studies have been replicated. For instance, a study using data from 22 small-scale societies showed that populations with a high juvenile mortality rate also had an earlier average age of puberty, menarche and first reproduction (Walker et al., 2006), supporting the notion that risk of mortality accelerates one's life history strategy. However, throughout the literature there are exceptions. A cross-cultural analyses found no evidence for father-absence being predictive of accelerated puberty (Sear et al., 2019), demonstrating that this 'stressor' is likely unique to WEIRD societies; and research from Brazil found that early life adversity predicted a delayed, as opposed to an accelerated, menarche (Wells et al., 2019). However, it may be that the ecological conditions modelled to affect life history traits have become too broad. Differences in the timing of life history traits was initially attributed to the risk of extrinsic mortality; however, few human studies actually include this measure in their models. Though some research has shown that a greater risk of extrinsic mortality accelerates reproductive schedules and increases criminality (Uggla & Mace, 2015; Uggla & Mace, 2016), most research using human data uses vague proxies of extrinsic mortality like ecological harshness, adverse childhood conditions, and socioeconomic status (e.g. Nettle et al., 2010). As previously alluded to, what is considered 'harsh' is both culturally and temporally specific, meaning that many predictions and results cannot be generalised.

Further, it may perhaps be that the initial predictions pertaining to extrinsic mortality are not correct at all (Moorad et al., 2019; André & Rousset, 2020). Though the idea that extrinsic mortality should shape the evolution of life history traits has been used by evolutionary biologists, behavioural ecologists, and evolutionary psychologists to generate hypotheses, it has recently been proposed that different life histories have not emerged as a result of differing risk of extrinsic mortality, but rather as the result of *density-dependent competition* (André & Rousset, 2020). Here, it is thought that extrinsic mortality indirectly affects life history evolution through altering the population density within the environment, which then impacts the intensity of competition for resources. When extrinsic mortality is lower, the intensity of competition increases because more individuals are maintained in the environment, meaning that traits that are adaptive under intense competition (e.g. higher levels of offspring investment) will be favoured when extrinsic mortality is low. Similarly, when there are high levels of extrinsic mortality, resource competition

is relaxed, and so traits that are beneficial when there is a lower population density will be favoured. Therefore, a greater understanding of the effect of population density on competition in humans may be required to generate predictions pertaining to life history traits and strategies in the future.

1.3 Menopause

Though there is currently a great deal of debate about many aspects of how life history theory is applied to humans, it is generally agreed that humans have odd life history schedules compared to other mammals (Key, 2000). Humans are often considered to be slow strategists: we grow slowly, have prolonged childhoods, single births, and long lifespans. However, we also present many traits characteristic of a fast life history strategy, including having multiple offspring at once, early weening, and short interbirth intervals (Mace, 2000). In addition to this, humans also display a life history trait that is unique amongst primates: early reproductive senescence in females coupled with post-fecund survival.

The termination of female fecundity is known as the 'menopause', which can be defined biomedically as the irreversible cessation of menstrual function. This event is identified retrospectively after 12 months of amenorrhea in the absence of external influences such as pregnancy or hormonal medication (Kirchengast & Ruhli, 2013). The number of follicles (cells that surround immature eggs) is established in utero at around 5 months gestation, and is therefore finite at about 7 million (Wallace & Kelsey, 2010). By the time a woman reaches puberty, this number will have declined to ~400,000 (Baker, 1963), with menopause occurring once the ovarian reserve has declined to below 1000 (Faddy et al., 1992). Globally, average age of natural menopause varies between 44.6 to 54.5 depending on geographic region, with the average age of menopause in the UK being 50 (Laisk et al., 2019). Prior to reproductive senescence, peri-menopause occurs during which menstrual periods will begin to become irregular and estrogen levels will drop (however, it is still possible to become pregnant). While there is a large amount of cross-cultural variation, for many the peri-menopause is also accompanied by a range of unpleasant psychological (e.g. mood swings, depression, anxiety), physical (e.g. vaginal dryness, reduced sex drive), and vasomotor symptoms (e.g. night sweats, hot flashes) (Burger et al., 2007), which many choose to manage with medication such as hormone replacement therapy. These symptoms are also often reported to extend past the final menstrual period,

and well into post-menopause (Avis et al., 2015; Avis et al., 2018). The symptom patterning has resulted in menopause being seen as a process or transition, rather than a standalone event (Sievert, 2006).

Despite menopause being an important and unique aspect of the human life history, it is rarely discussed where menopause fits within the life history framework. As previously reviewed, life history theory has been used by researchers to model the timing of various life history traits (e.g. age at first birth, menarche), how they cluster with one another, and also used to inform speculations about what the timing of various life history traits means for one's life history strategy. However, even though menopause is a key life history trait, and its timing appears to be plastic in response to ecological factors (reviewed in section 1.3.2), it is seldom modelled or analysed in the same way other traits that affect fitness are. Predictions about the timing of various life history traits are often generated using principles derived from life history theory, with researchers generally looking at how specific ecological factors (e.g. harshness, extrinsic mortality) relate to life history traits. However, menopause is rarely included in such research, and it is also not clear what the predictions are in regards to how ecological factors predicted to affect the development of various life history traits would impact menopause timing. It seems there has been a strong focus in life history research on modelling the timing of traits that influence the start of reproduction, with traits that directly affect the end of reproduction being neglected.

To work out where menopause fits within a life history framework, we firstly need to understand how the timing of the trait relates to different trade-offs. For example, in terms of the trade-off between growth and reproduction, could it be that if one invests more into early life growth then they experience menopause later? Or in relation to the trade-off between offspring quantity and quality, might it be that somehow the energetics relating to the reproductive system are responsive to how many children one has, and so ones a woman has 'enough' children, energy is switched from maintaining the ovulatory system and direct reproduction to increasing fitness through investing in relatives? These thoughts are speculative, of course, but could be a consideration for future research.

Though little research has been conducted directly linking menopause timing and life history theory, some researchers have looked at how menopause timing associates with early life conditions. As previously stated, advocates of the idea of a fast-slow life history continuum in humans have suggested that one's childhood

environment can set their 'pace of life', with a harsher environment putting the individual on more of a 'life fast, die young' trajectory (Belsky et al., 2010). There is quite a large body of research into how prenatal and early life conditions relate to menopause timing; however, as this research has been largely carried out by epidemiologists the results are not discussed in the context of trade-offs or life history theory, and much of the research has been carried out using data from WEIRD countries. Results have shown that women who were exposed to famine during early life (Elias et al., 2003), not breastfed (Hardy & Kuh, 2002; Mishra et al., 2007), experienced parental divorce (Hardy & Kuh, 2005; Mishra et al., 2007), born into a lower socioeconomic position (Lawlor et al., 2003; Hardy & Kuh, 2005), and exposed to cigarette smoke prenatally (Strohsnitter et al., 2008) are at risk of an earlier menopause. As it seems a harsher childhood environment associates with an earlier menopause, it could lead us to conclude that an earlier menopause is a 'fast life history trait'; but how this conclusion fits in with the timing of other life history traits could be brought into question. Firstly, having higher fertility is generally considered to be characteristic of a fast life history, yet having more children is predictive of a later menopause (which based on research from early life conditions would be considered a slow life history trait) (Mishra et al., 2017). Similarly, timing of menarche is often taken to be indicative of one's life history strategy, yet there is no consistent relationship between menopause timing and age at menarche across studies (Nagel et al., 2005; Mishra et al., 2017; Bjelland et al., 2018; Sheppard & Van Winkle, 2020), so it does not seem that people who 'start earlier' finish earlier, and vice versa. Secondly, the effects of early life conditions on menopause timing do not appear to be universal (for conflicting results on the effects of socioeconomic position and famine see: Lumey & Stein, 1997; Hardy & Kuh, 2002), and are likely culturally specific, with the majority of studies being carried out using data from Western Europe, the US, and Australia. Finally, just because we might observe menopause to covary sometimes with other life history traits, it does not mean we have a solid model predicting why menopause would behave in a certain way. The lack of such a model within evolutionary anthropology is a huge shortfall, as at present, there is nothing guiding our predictions for what would be the optimal age of menopause given one's local ecology and current number of offspring. Though, it might be that menopause timing has no relation to fitness, and therefore an evolutionary model is not required. As I outline shortly, there are many factors

observed to influence timing, and so it could be that the age at which women enter menopause is simply the result of these factors interacting with one another with no regard for fitness. However, we do not know if this is the case or not, as it has not been researched. Hence, for a more complete picture of where menopause fits within the human life history, more attention should be paid to understanding current variation in the trait from a fitness maximising perspective.

As well as it not being clear where menopause fits within a life history framework, it is also not clear why we evolved the trait in the first place. Humans regard it to be a usual part of the life course, yet it is not a trait one would predict to evolve as a result of natural selection, as it does not make sense for selection to favour a trait that reduces the ability of the organism to directly reproduce. Further complicating the presence of the trait is the fact that humans are the only primate species to experience menopause coupled with a post-reproductive lifespan. In captivity, other primate species are occasionally observed to experience menopause followed by a short period of post-reproductive life due to their lifespans being artificially elongated; but in the wild, the reproductive systems of females senesce at the same rate as the rest of their body (Walker & Herndon, 2008; Atsalis & Videan, 2009). Not only is the trait rare in primates, but elsewhere in the mammalian class only a few species of cetacean have been observed to exhibit the trait (Johnstone & Cant, 2010; Foster et al., 2012; Brent et al., 2015; Croft et al., 2017; Photopoulou et al., 2017; Ellis et al., 2018a; Ellis et al., 2018b). Our phylogenetic distance from these species suggests that we evolved it convergently, but whether it was in response to the same selective pressures is unknown.

The apparent detrimental effect of menopause on fitness coupled with its rareness amongst mammals means it is a trait of interest for evolutionary anthropologists. Prior to addressing evolutionary hypotheses concerning menopause, the proximate understanding of menopause will first be outlined alongside the epidemiological literature looking at variation in menopause timing and symptoms reporting.

1.3.1 Physiology

As stated, a woman is generally identified as having experienced menopause retrospectively after 12 months with no periods (in the absence of other influences, such as hormonal birth control). This amenorrhea is caused by a loss of ovarian

follicles, which are aggregations of cells that contain oocytes, or immature eggs. Follicles are also the primary site of ovarian steroid production: during the preovulatory, or follicular, phase of the menstrual cycle, the follicles produce estradiol; with progesterone (and also small amounts of estradiol) then being produced during the postovulatory, or luteal, phase. The estradiol and the progesterone secreted by the follicle cause the hypothalamus to produce the follicle stimulating hormone at the end of the luteal phase, which acts on the ovary to stimulate the growth and development of the follicles; and the luteinising hormone during the follicular phase to induce ovulation. This in turn causes the follicles to produce estradiol, thus feeding back on the hypothalamus causing it to secrete the follicle stimulating and luteinising hormone. This process is necessary for the regulation of the menstrual cycle, and the preparation of the womb for pregnancy.

Human females are born with millions of primordial follicles (Wallace & Kelsey, 2010), which each contain a single primary oocyte (Fortune, 1994). After birth, the female will undergo a continual process of follicular loss throughout her life, leaving her with very few follicles when menopause occurs. The primary reason for the loss of follicles is not ovulation: while there is variation, fewer than 500 oocytes are usually lost via this route, with the majority of follicles being lost in situ by a process known as follicular atresia, which refers to the natural degeneration of follicles (Tilly et al., 1992; Hsueh et al., 1994). As the pool of follicles decreases, as does the ovaries ability to produce estradiol, and so the feedback system between the ovarian steroids and hypothalamus breaks down. Once a woman's follicular reserve declines to approximately 1000, menstrual cycles become irregular and hormone levels change, signalling entry into the peri-menopausal period. Menopause itself occurs when the follicular reserve is exhausted.

1.3.2 Global variation

1.3.2.1 Age of menopause

Cross-culturally, there is a large amount of variation in age of natural menopause, with population means ranging from 44.6 to 54.5 years (Laisk et al., 2019). Variation in menopause timing is partially the result of genetic factors, with heritability estimates ranging between ~40-80% (Peccei, 1999; de Bruin et al., 2001; van Asselt et al., 2004; Murabito et al., 2005). However, menopause timing also consistently covaries with a number of intrinsic factors, such as ethnicity and age of first

menstrual period, in addition to a number of socioeconomic and lifestyle factors – some of which are highly modifiable – such as smoking habits, level of physical activity, alcohol consumption, use of oral contraceptives, number of pregnancies, educational attainment, and body mass index (Gold et al., 2000; Gold et al., 2001; Sievert et al., 2001; Hardy & Kuh, 2002; Reynolds & Obermeyer, 2003; Ozdemir & Col, 2004; Ayatollahi et al., 2005; Aydin et al., 2005; Nagel et al., 2005; Parazzini & Progetto Menopausa Italia Study, 2007; OlaOlorun & Lawoyin, 2009; Canavez et al., 2011; McKnight et al., 2011; Li et al., 2012; Perez-Alcala et al., 2013; Sapre & Thakur, 2014; Ceylan & Ozerdogan, 2015; Tao et al., 2015; Ahuja, 2016; Maru et al., 2016; Ruth et al., 2016; Mishra et al., 2017; Bjelland et al., 2018; Bovet et al., 2018; Wang et al., 2018; Zhu et al., 2018). Cross-cultural multi-ethnic studies have found that women of East Asian ancestry often experience a later menopause than women of African ancestry, and that Latina women have an earlier menopause than white women (Gold et al., 2001; McKnight et al., 2011). Nulliparous women have been estimated to be at a 30% increased risk of earlier menopause (menopause between 40-44 years old), with this effect being strongest in women who also started menstruating at a young age (Mishra et al., 2017). Smoking is one of the most consistent predictors of menopause timing, with women who smoke experiencing menopause at an earlier age (Bromberger et al., 1997; Gold et al., 2000; Gold et al., 2001; Gold et al., 2004; Ozdemir & Col, 2004; Reynolds & Obermeyer, 2005; Parazzini & Progetto Menopausa Italia Study, 2007; OlaOlorun & Lawoyin, 2009; Gold, 2011; Sapre & Thakur, 2014; Schoenaker et al., 2014; Ahuja, 2016; Oboni et al., 2016; Wang et al., 2018), possibly due to the antiestrogenic effects of tobacco toxins (Tanko & Christiansen, 2004). Though effect estimates vary, a high body mass index is generally associated with a later menopause (Li et al., 2012; Sapre & Thakur, 2014; Zhu et al., 2018), with levels of physical activity presenting a U-shaped relationship in which regularly engaging in light exercise delays menopause, while heavy physical activity and being sedentary associates with an earlier menopause (Gold et al., 2013; Sapre & Thakur, 2014). Early life stress (e.g. maternal smoking, parental divorce, lower social class) has also been found to predict an earlier age of menopause (Mishra et al., 2009), leading to suggestions that menopause timing might be indicative of one's life history strategy. However, as discussed previously it is not necessarily clear where an earlier or late menopause would be predicted to fall on the fast-slow life history continuum, and there is mixed empirical support for menopause

timing being predictive of life history strategy, with a sequence analysis showing that age of menopause does not predictably covary with other life history traits (Sheppard & Van Winkle, 2020).

Currently, the mechanisms underlying these sociodemographic correlates are not fully understood. Nonetheless, it does demonstrate that menopause timing – within reason – is flexible, and also responsive to a number of biological and lifestyle factors. Typically, discussion of this variation occurs within the epidemiological, rather than evolutionary, literature. As such, research into the timing of menopause has generally not considered whether the variation could be facultative or not.

1.3.2.1.1 Issues in data collection

Despite the clinical and functional importance of menopause, the timing of such is difficult to measure both through interviews and in a clinical setting. Much of the data collected on menopause is done through surveys and in longitudinal studies, where women are either asked when they experienced their final menstrual period, or when they experienced menopause. Generally, gauging an accurate response to these questions is only possible longitudinally. Retrospective reporting of age of menopause requires individuals to remember when their final menstrual period was, which can lead to approximations and recall inaccuracies (Bean et al., 1979; Colditz et al., 1987; Hahn et al., 1997). Additionally, if a woman is asked when she experienced menopause, this can lead to different interpretations of the question. As stated, menopause is often seen as a transition, and that might lead respondents to class themselves as having experienced menopause when they began experiencing symptoms, thus leading to inaccurate data. Even in clinical settings it is still hard to discern when a woman has experienced menopause, as there are factors that can conceal symptoms of being peri-menopausal (Hillard et al., 2017). Combined oral contraceptives can cause withdrawal bleeds which occur monthly and mimic that which occurs during a menstrual period, while progesterone-only contraceptives often cause amenorrhea, meaning a woman might be peri-menopausal, but the symptoms of such would be masked by contraceptive use.

1.3.2.2 Symptoms

In addition to the diversity of age of menopause, there is also a large amount of variation in experience of menopause symptoms both in terms of severity, frequency,

and also duration (Thurston et al., 2008; Avis et al., 2015). While menopause symptoms are often associated with peri-menopause, they have been observed to occur at all stages of the menopausal transition (Williams et al., 2008). A wide range of symptoms are reported by those going through menopause, including mood changes, vaginal and bladder problems, and changes in sexual function. Though, perhaps the hallmark of the menopausal transition is vasomotor symptoms, which include hot flashes and night sweats, with a systematic review finding that cross-culturally 41% of women report hot flashes during the peri-menopause (Freeman & Sherif, 2007). The ubiquity of vasomotor symptoms has even led some to suggest that they have evolutionary origins, through being the result of intragenomic conflict (Ubeda et al., 2014; see Chapter 5) or to allow for the postpartum warming of infants (Naftolin et al., 1994; Pollycove et al., 2011); though these theories have received little empirical support (Sievert & Masley, 2015; Yang et al., 2019).

Biologically, vasomotor symptoms are thought to be caused by the fluctuations of hormones, such as estrogen and progesterone, during the menopausal transition. However, as stated, not everyone experiencing menopause will have vasomotor symptoms, and amongst those that do, the experience is not uniform. This suggests that symptoms experienced during the menopausal transition are not simply an inevitable result of the hormonal changes that accompany the menopausal transition, but also influenced by aspects of the woman's environment.

The contingent nature of vasomotor symptoms is seen in cross-cultural and multi-ethnic studies, in which it is consistently reported that women who reside in East Asia, or are of East Asian ancestry, experience less severe and more infrequent vasomotor symptoms for a shorter period of time (Gold et al., 2001; Anderson et al., 2004; Gold et al., 2004; Gold et al., 2006; Anderson & Yoshizawa, 2007; Chung et al., 2018; Dunneram et al., 2019). Conversely, women of African ancestry are often observed to experience more severe symptoms that persist for a longer duration of time (Gold et al., 2004; Gold et al., 2006; Avis et al., 2015). The relationship between ethnicity and menopause symptoms has been replicated in multiple different datasets and still persists after controlling for relevant factors. Despite this, why ethnicity associates so strongly and consistently with vasomotor symptom experience is currently unknown.

Another factor consistently associating with vasomotor symptoms is smoking, with research from North America demonstrating that women who smoke

are 60% more likely to report experiencing vasomotor symptoms than those who do not (Gold et al., 2006). Here, active smoking is not the only risk factor, as those who inhale smoke passively also report more frequent and severe vasomotor symptoms (Gold et al., 2004). This relationship may be partially due to the anti-estrogenic effect of smoking (Michnovicz et al., 1986; Tanko & Christiansen, 2004), but also due to the socioeconomic correlates associated with the uptake of smoking (Gold et al., 2004), with many measures of socioeconomic position also being observed to associate with vasomotor symptom frequency. Here, women of a lower socioeconomic position – whether this is measured through employment, education, or income – are consistently found to experience worse vasomotor symptoms (Gold et al., 2006). Further, a higher body mass index consistently predicts more hot flashes, though it is thought that this may be due to the increased levels of adipose fat preventing heat loss, thus worsening the perception of hot flashes (Freedman, 2005).

As well as these demographic factors, culturally specific traits have also been found to predict vasomotor symptoms. The wearing of head scarves – common in many world religions – is associated with increased reporting of hot flashes, and this has been attributed to the insulating property of the scarves (Sievert, 2014). Further, increased soy consumption has been linked to less severe vasomotor symptoms (Dunneram et al., 2019), with this observation often being used to explain some of the symptom patterning in East Asian women. Further, culturally specific attitudes towards ageing have been found to predict symptom severity, in which women from cultures with higher levels of elder-respect report being less bothered by vasomotor symptoms (Minkin et al., 2015).

Taken together, the current literature on menopause symptom experience demonstrates that they are not an inevitability of the menopausal transition, but rather are partially the product of sociodemographic and cultural factors – some of which are modifiable.

1.3.3 Why do we have menopause?

Proximately, it can be understood that menopause occurs because women are born with a finite number of eggs, which deplete throughout her life due to ovulation and atresia. However, this explanation does not address why selection did not favour a phenotype that allows life-long reproduction. Women spend a relatively large

proportion of their lives post-reproductive, which is a clear detriment to their lifetime reproductive success. In addition to the fitness costs of being infertile, we have also seen that the menopausal transition is a turbulent time for women as it is often accompanied by many unpleasant symptoms. Further, humans are the only primate to exhibit an extended post-reproductive lifespan. So why, at the ultimate level, do we exhibit this confusing life history trait?

Multiple evolutionary hypotheses for the origins of the menopause and post-reproductive lifespan have been proposed, which can be broadly split into non-adaptive and adaptive hypotheses, where the former generally states that menopause is a by-product of some other selected-for trait, while the latter proposes that evolution has selected for the trait and that there are hidden fitness benefits. However, before introducing these hypotheses, it should first be clarified what the evolutionary puzzle is in humans. So far, I have spoken about menopause as a trait being maladaptive; however, this statement is not necessarily precise. Rather, the puzzle is: *why do humans experience menopause so early on in the life course and then have such long post-reproductive survival*? As I will show shortly, the females of some other primate species do experience menopause if they live long enough (e.g. in captivity), but generally their reproductive systems senesce at the same rate as other bodily systems meaning that the length of their reproductive lifespan and actual lifespan are correlated with one another (Walker & Herndon, 2008; Alberts et al., 2013). If menopause occurs at the end of the lifespan, the trait is not confusing, because the reproductive system would then be ageing at the same rate as the rest of the body and the female would not be left infertile for any portion of her life. Therefore, evolutionary theorists are looking at the disconnect between age of menopause and life expectancy in humans, rather than menopause as a trait being confusing in and of itself.

1.3.3.1 Menopause outside of humans

All female mammals will experience reproductive ageing and a decline in fertility, but this process occurs alongside the general ageing process at a similar rate (Jones et al., 2014). This means that any lifespan beyond reproduction is very short, and that other mammals spend a very small proportion of their lives – if any at all – post-reproductive. To quantify post-reproductive lifespans across mammals, Levitis & Lackey (2011) developed a measure of post-reproductive representation (PrR), which

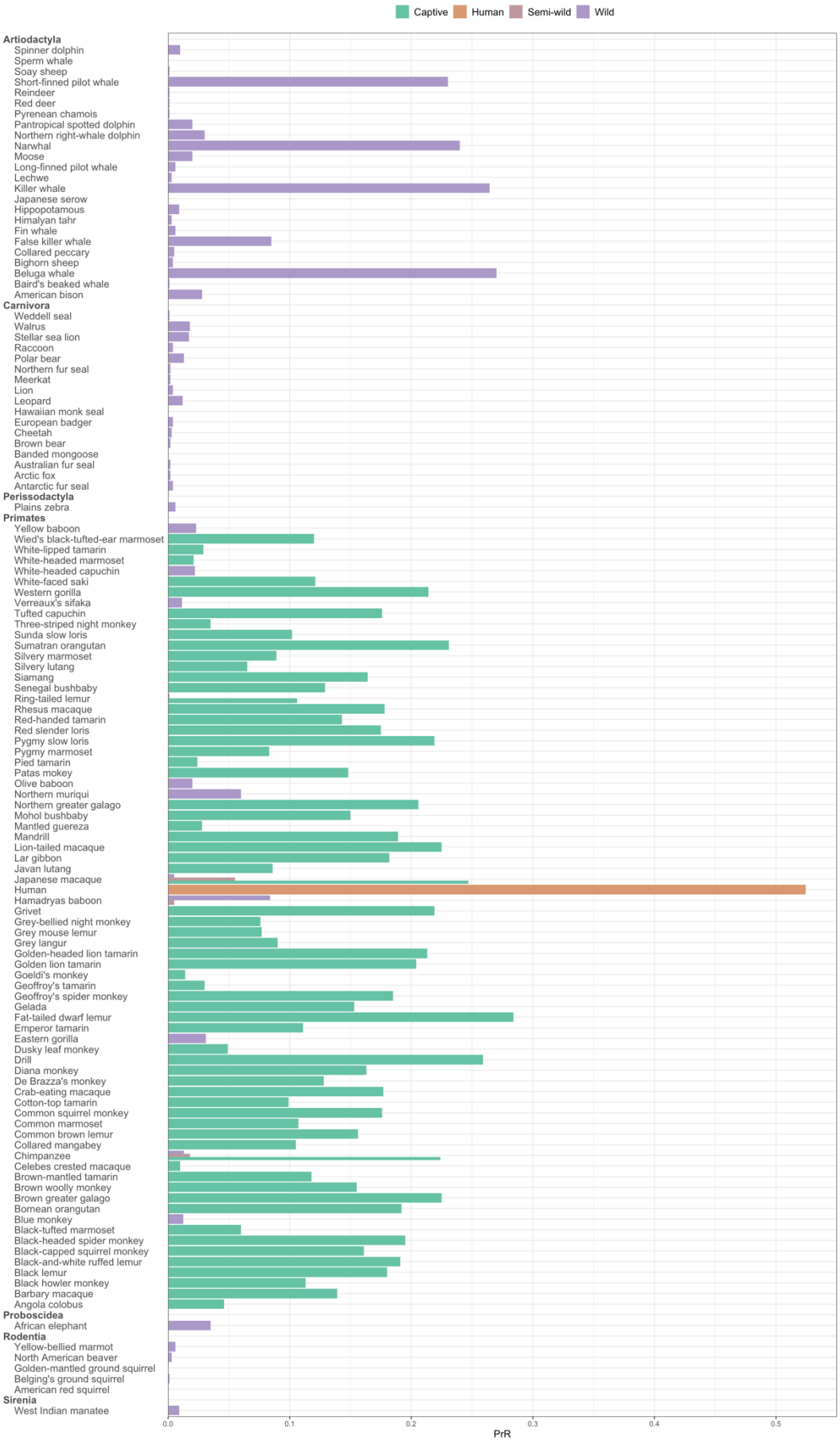

Figure 1.1. *Proportion of adult years spent post-reproductive in selected mammal species (post-reproductive representation; PrR). Data sources shown in Appendix A (Table A1).*

describes the proportion of adult years lived post-reproductive. The PrR of 124 mammals is shown in Figure 1.1, where it can be seen firstly that humans have a significantly longer PrR than most other mammal species; secondly, that there is a difference in PrR between captive and wild species, with captivity appearing to lengthen the PrR; and lastly, that the majority of data comes from captive species. The latter observation is due to the difficulties associated with data collection on menopause in natural environments (Walker & Herndon, 2008). In humans, one of the key markers of menopause is the cessation of menstrual periods, which (in the absence of external forces such as contraceptives) is an easily observable phenomenon. However, many non-human primates and other mammals do not experience regular patterns of menstrual bleeding, making the termination of ovulation less easy to measure. To deal with this, Walker & Herndon (2008) have proposed that for comparative analysis across species, multiple parameters that signal menopause should be utilised, including menstrual bleeding patterns, follicular depletion, hormonal changes, and perineal swelling. However, if we were collecting data on follicular depletion and hormonal changes, biological samples would have to be obtained; plus, longitudinal data is generally required to accurately gauge information on the menopausal transition, and outside of captivity these two things are generally tricky. In addition to this, there are a number of outside influences that might affect the ovulatory function of the organism that researchers need to account for, such as predation pressures (Hamilton et al., 1982), food availability (van Noordwijk & van Schaik, 2005), and social group dynamics (Machatschke et al., 2006; Atsalis & Videan, 2009). Taken together, this can make the study of reproductive cessation in wild animals extremely difficult, resulting in a great deal of data coming from captive animals.

In captivity, the lifespans of animals are generally longer than they would be in the wild as a result of better provisioning, a lack of predation, and veterinary care. As such, under these conditions many non-human primates have been observed to experience a process analogous to the menopausal transition, with older female primates experiencing irregular menstrual cycles, a decline in estrogen levels, and occasionally complete reproductive cessation (Graham et al., 1979; Gould et al., 1981). In a sample of 15 captive rhesus macaques aged 8-34 years, Walker (1995) found that those aged 24-26 had high levels of luteinising hormone and low levels of estradiol, signalling that they had entered the menopausal transition. Similar results

have been found in free-ranging rhesus macaques (*Macaca mulatta*) (Johnson & Kapsalis, 1998), free-ranging Japanese macaques (*Macaca fuscata*) (Itoigawa et al., 1992), and captive cynomolgus macaques (*Macaca fascicularis*) (Kavanagh et al., 2005). In a small sample of captive common chimpanzees (*Pan troglodytes*) and pygmy chimpanzees (*Pan paniscus*) aged between approximately 40-50 years, menstrual cycles were observed, with cycle length increasing with age (Gould et al., 1981). Similar results were found by Lacreuse et al. (2008) in a study of captive *P. troglodytes*, where it was found that, while they appeared to menstruate beyond the age of 50, the length of menstrual cycles became longer after age 20. In wild chimpanzees, research has shown that females cease ovulation around the age of 30 (Nishida et al., 2003), while data from healthy free-ranging chimpanzees has shown that females are capable of reproduction beyond 40 years (Thompson et al., 2007), suggesting that timing of menopause onset is the result of many things, such as ill-health. Histological examination of the ovaries of *P. paniscus* showed similar characteristics to aged ovarian tissue in humans, suggesting that their reproductive system does age in a similar way to ours (Gould et al., 1981), a conclusion that is further supported by research showing a greater incidence of foetal loss in chimpanzees as their age increased (Roof et al., 2005). Further, endocrinological data from captive gorillas (*Gorilla gorilla*) has shown that, similar to humans, they have a cyclic pattern of hormonal secretion that becomes irregular with increasing age (Atsalis & Margulis, 2006), with both peri-menopause and menopause having been observed in zoo populations of gorillas (Margulis et al., 2007). Captive orangutans (*Pongo pygmaeus*) have also demonstrated an age-specific decline in fertility (Caro et al., 1995), however, studies from wild orangutans (*Pongo abelii*) have failed to replicate this based on data on inter-birth interval length (Wich et al., 2004).

Though there is evidence for a post-reproductive lifespan in non-human primates amongst captive individuals, data outside of this setting is limited. However, as can be seen in Figure 1.1, a number of cetaceans appear to spend a large proportion of their lives post-reproductive in the wild, which include beluga whales (*Delphinapterus leucas*), false killer whales (*Pseudorca crassidens*), killer whales (*Orcinus orca*), narwhals (*Monodon monoceros*), and short-finned pilot whales (*Globicephala macrorhynchus*) (Olesiuk et al., 2005; Croft et al., 2017; Photopoulou et al., 2017; Ellis et al., 2018b). Collecting data on the reproductive status of cetaceans comes with many difficulties, as menopause status is generally inferred from post-mortem

anatomical examination of the organism (Perrin et al., 1984). Phylogenetic analysis suggests that menopause evolved multiple times within cetaceans (Ellis et al., 2018b), with species ceasing reproduction around the age of 40, but living decades beyond this (Croft et al., 2015). As these are the only other mammals to commonly display a pronged post-reproductive lifespan under 'natural' conditions, they are often used as models for the evolution of menopause in humans, despite their phylogenetic distance from us and difference in physical environment.

1.3.3.2 Non-adaptive explanations

1.3.3.2.1 Lifespan artefact hypothesis

Based on the evidence from primates that shows menopause sometimes occurs in captivity where lifespans are longer, some theorists have proposed that the human menopause is the result of a similar process, and a by-product of our long lifespans that are modelled here to be a recent phenomenon (Austad, 1997; Peccei, 2001). Here, it is suggested that menopause is simply an epiphenomenon, and that the capabilities of the female reproductive system have not yet 'caught up' with our longer lifespans, as evolutionary processes have not yet had time to alter the female phenotype to its most adaptive form. This therefore implies that menopause arose following the elongation of the human lifespan, with the latter happening due to a decreased risk of extrinsic mortality, improved medical care, better living conditions, and industrialisation.

While this theory seems intuitive at first glance, it has many theoretical and empirical issues. Firstly, it fails to explain why reproductive senescence in females happens so much earlier than other bodily systems, and also why it only occurs in females. Secondly, there is little empirical evidence that our ancestors had lifespans that significantly differed from ours today: it is thought that our maximum lifespan has remained constant for approximately 100,000 years (Cutler, 1975), meaning that – while in our history a large proportion of the population would not have lived to old age – many of our ancestors would have lived well into their seventh decade or older. This observation is supported by evidence from contemporary hunter-gatherer groups (e.g. the !Kung, Hadza, Yanomami, and Aché), where a large proportion of women experience extended post-menopausal survival in the absence of Western technology and medicine (Lancaster & King, 1985; Hill & Hurtado, 1991; Gurven & Kaplan, 2007; Croft et al., 2015). Lastly, this hypothesis implies menopause is a

recent trait. Though it is impossible to be sure as menopause does not fossilise, maximum estimates for the emergence of menopause coupled with a prolonged post-reproductive lifespan are around 1.8 million years ago (Bogin, 1999). At this point in hominin evolution, brain sizes were becoming larger, which would therefore result in a longer period of dependency (i.e. childhood) and therefore cooperative breeding. Thus, as life history patterns were becoming similar to that of modern *Homo sapiens*, it is likely that menopause and a post-reproductive lifespan would have been present (Hawkes et al., 1998).

1.3.3.2.2 Antagonistic pleiotropy and mutation accumulation

Antagonistic pleiotropy is currently one of the most accepted explanations for why we age (Williams, 1957). Pleiotropy refers to the phenomenon in which a single gene controls for more than one trait in an organism, and so antagonistic pleiotropy is specifically when one of these traits has a detrimental effect on the organism. For example, the BRCA1 and BRCA2 genes which account for 1-13% and 1-5% of all instances of ovarian and breast cancer, respectively (Risch et al., 2006), are thought to have been maintained due to the fertility benefits associated with the same genes. In a sample of women from the US from the early 1900s, it was found that those carrying the BRCA1 gene produced an average of two more children, meaning that there would be selection for the gene despite its detrimental effects in later life (Pavard & Metcalf, 2007). Additionally, the increased fertility as a result of the gene may have further mitigated the effects of the harmful gene, as multiple pregnancies have been found to be protective against breast cancer (Corbett et al., 2018).

Antagonistic pleiotropy is thought to explain the presence of many non-communicable diseases in post-industrial societies (Corbett et al., 2018), and it has also been used to explain the presence of menopause (Wood et al., 2000). Here, menopause is viewed as the endpoint of decades worth of follicular depletion, with the rate at which this depletion occurs thought to be necessary to maintain regular menstrual cycles. The regularity at which human females are fertile is thought to then offset the costs of terminating reproduction at the age of 50 (Wood et al., 2000). In summary, if follicular depletion is beneficial for pre-menopausal women and necessary to maintain ovarian cycles in early life, then it could be that menopause is just simply the product of this process. However, this explanation does not identify why the selection for follicular depletion as a means of maintaining regular menstrual

cycles led to the evolution of menopause well before the end of a woman's life (Laisk et al., 2019), and it does not explain why this process only happens in humans and not in other mammals, like elephants and chimpanzees, whose lifespans resemble that of humans (Lahdenpera et al., 2014).

1.3.3.2.3 Mate choice

Under the mate choice hypothesis of menopause, it is thought that menopause is the result of men preferring to mate with younger women (Morton et al., 2013). This model assumes that in our evolutionary history, males showed a preference for younger females. This preference then would have resulted in reproduction being unlikely beyond a certain age, which created a selection shadow that resulted in an accumulation of genetic mutations, including one which coded for reproductive senescence. Therefore, this model suggests that menopause evolved due to male mate choice preferences that resulted in an accumulation of genetic mutations in females (Morton et al., 2013).

However, this model is based on assumptions that have little theoretical grounding. Firstly, it suggests that a male preference for younger females was present prior to menopause, causing it to evolve. A preference for younger females is rare within the animal kingdom, with chimpanzee males showing a preference for mating with older females (Muller et al., 2006). Though mate choice research has found a universality for a male preference for youth (Buss, 1989), it is possible that this preference emerged as a consequence of declining fertility and menopause, rather than the other way around. Secondly, just because men show a preference for younger females, it does not mean that they are mated with exclusively, nor that the younger females themselves want to mate with these males. Just because males state their preference is younger females, it does not mean that this always translates into real life mating behaviours. Further, due to age-related assortative mating coupled with long-term pair-bonding (Van de Putte et al., 2009), post-menopausal women often do have a partner and, just because they are post-reproductive, they are not simply disregarded as sexual partners at this point seeing as humans engage in sex for recreational purposes as well as reproduction (Diamond, 1997; Harder et al., 2019). Finally, this hypothesis addresses only why menopause might emerge, but not why such a long post-reproductive life is present in humans.

1.3.3.3 Adaptive explanations

At present, there is limited empirical evidence in favour of the non-adaptive hypotheses relating to the evolution of menopause. Further, these explanations fail to distinguish between menopause and the extended post-reproductive lifespan, with most only attempting to explain the former rather than the latter (or both). As such, multiple adaptive hypotheses have been proposed. Prior to introducing these ideas, it should be noted that it is not always clear whether the authors are suggesting whether menopause is a trait that is *maintained* by selective pressures, or one that *evolved* as a result of selective pressures (Thouzeau & Raymond, 2017). These two 'pathways' to an adaptive menopause can be understood as follows:

1) *A maintained menopause.* This pathway can be seen as a combination of non-adaptive and adaptive hypotheses. In this scenario, the female life expectancy would have gotten longer, but some ecological factor (e.g. communal breeding) created a selective pressure that meant early reproductive senescence was *maintained* as the women were able to offset the fitness costs of not directly reproducing. Therefore, here there was possible selection against life-long reproduction.
2) *An evolved menopause.* In this pathway to menopause, the rate of senescence of the female reproductive system would have once been in line with other bodily systems, therefore allowing for prolonged reproduction. However, a selective pressure would have then caused the female reproductive lifespan to shrink and end early, therefore causing *menopause to evolve*.

Most literature refers to the evolution of menopause, which therefore alludes to the second pathway; however, it is not always explicit. It is not currently possible to know whether ancestral human females were reproducing into old age and then experienced selective pressures against this (Pathway 2) or whether there has just been a selective pressure against lifetime reproduction evolving (Pathway 1). Further discussion here on adaptive theories of menopause will refer back to such differentiation.

1.3.3.3.1 The Mother Hypothesis

For all animals, there are costs and benefits associated with reproduction. In mammals, the costs are particularly great for females who are responsible for gestating, giving birth, and lactating. In humans, the costs of reproduction further

increase for females due to our narrow birth canals and the large heads of babies that make birth an especially difficult process, with such difficulty increasing alongside the age of the mother (Cavazos-Rehg et al., 2015). Older women experience a greater risk of both maternal and neonatal mortality, pregnancy-related illnesses (e.g. preeclampsia, hypertension), and poor foetal growth, with older mothers also being more likely to have an infant born with birth defects (Yerushalmy et al., 1940; Grimes, 1994; Pavard et al., 2008; Cavazos-Rehg et al., 2015). As such, it is thought that menopause may have been selected for to protect against the costs associated with late-life pregnancy (Williams, 1957; Alexander, 1974). Reproduction in late life may not only negatively affect the fitness of the mother, but it might also be detrimental to any existing children she has. For example, if a woman with young children already dies during childbirth, then this does not only affect her current pregnancy, but also any children she already has that are still dependent on her. In summary, menopause is thought to ensure the prolonged investment in existing offspring, and so it is thought that, in our evolutionary history, there would have been a selective pressure against women who continue to reproduce later in life to protect against neonatal and maternal mortality (Figure 1.2a).

While this hypothesis seems intuitive and logical, for selection to have favoured menopause under these conditions the risk of maternal mortality would have to be substantive enough to outweigh the fitness benefits of continued reproduction, and there is little empirical support for this. Firstly, data from contemporary hunter-gatherer groups suggests that maternal mortality is not as much

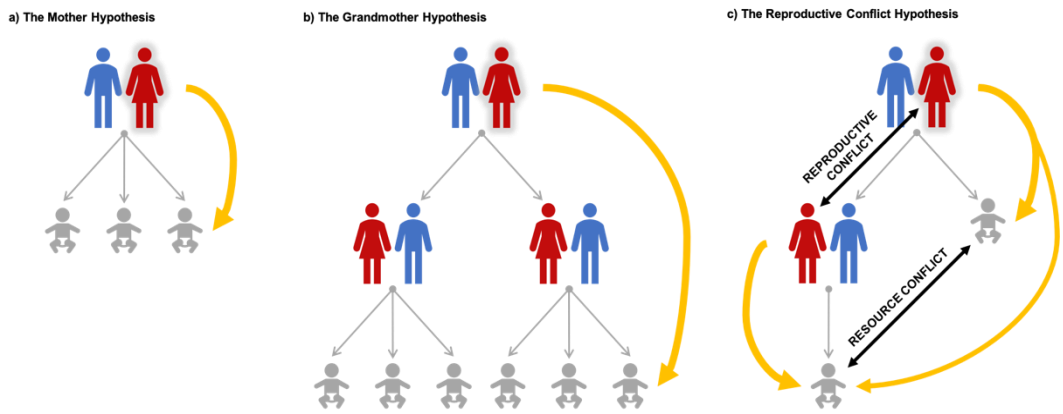

Figure 1.2. *Graphical description of the three most popular adaptive explanations for the evolution of menopause and the prolonged post-reproductive lifespan. a) shows the Mother Hypothesis, b) the Grandmother Hypothesis, and c) the Reproductive Conflict Hypothesis. Yellow arrows symbolise resource/energy transfers from a menopausal woman to children or grandchildren, grey arrows show relatedness, and black arrows conflict. Post-menopausal women are shown with a shadow behind them. Adapted from Laisk et al. (2019).*

of a risk factor as previously thought. Even amongst the Aché hunter-gatherers who traditionally had little access to 'modern' medicine, the odds of dying during childbirth were only 1 in 150 (Hill & Hurtado, 1996). Moreover, if the risk of maternal mortality *was* high in our evolutionary history, one could question why there was not a strong selective pressure favouring phenotypes that were able to successfully carry and birth healthy infants later in life, given it could be assumed that the fitness benefits of such would be large. It may be that elements of the Mother Hypothesis contributed to the evolution or maintenance of menopause coupled with a prolonged post-reproductive lifespan. However, as a standalone hypothesis, there is limited evidence that these conditions would neither maintain the menopause, nor facilitate its emergence (Thouzeau & Raymond, 2017). Further, artificial neural network models have demonstrated that menopause is able to evolve without maternal mortality as a selective pressure, thus offering limited support for the Mother Hypothesis (Aime et al., 2017)

1.3.3.3.2 The Grandmother Hypothesis

Perhaps the most famous hypothesis associated with menopause is the Grandmother Hypothesis (Figure 1.2b). This builds upon the Mother Hypothesis by acknowledging that the risk of maternal mortality alone might not be a sufficient adaptive explanation, but that the kin selected benefits of the trait coupled with the risk associated with late life birth might be (Hawkes et al., 1998; Hawkes, 2003; Hawkes, 2004; Kim et al., 2012).

As stated, humans display an interesting life history: despite presenting many classically slow life history traits such as a long lifespan and low mortality, people in natural fertility populations also display many traits characteristic of a fast life history, such as short inter-birth intervals and high fertility rates (Hill, 1993; Hawkes et al., 1998). It is likely that this is due to the presence of communal breeding, in which allomothers (a carer other than the genetic mother or father) assist in the provisioning and protection of children (Emmott & Page, 2019). The presence of alloparents means that women are able to wean earlier and reduce the period of lactational amenorrhea, thus reducing inter-birth intervals; and also allows for multiple generations of children to be cared for simultaneously, despite the great deal of investment human children require (Crognier et al., 2001; Bereczkei & Dunbar, 2002; Lahdenpera et al., 2004; Szabó et al., 2017). Genetic relatedness and possible

benefits from helping are thought to primarily predict care from kin (Hamilton, 1964), and therefore, a common alloparent is the grandmother. Hence, it is modelled that post-reproductive women are able to offset the lack of direct reproduction in later life through investing in their grandchildren, which will result in indirect fitness gains (Hawkes et al., 1998; Hawkes, 2003; Hawkes, 2004; Kim et al., 2012).

For post-reproductive women to increase their inclusive fitness, they would have to increase the survival and fertility of close kin, such as grandchildren. The effects of grandmother presence on grandchild survival have been primarily studied in natural fertility societies (both historical and contemporary). Evidence from these populations has found that – in some circumstances – the presence of a grandmother can increase grandoffspring health and survival, and also increase the fertility of their daughters; though this is contingent on factors such as grandoffspring sex and lineage (Sear et al., 2000; Sear et al., 2002; Sear et al., 2003; Lahdenpera et al., 2004; Gibson & Mace, 2005; Sear & Mace, 2008; Engelhardt et al., 2019; Nenko et al., 2020; Chapman et al., 2021). In a review of 45 studies looking at the effects of kin-presence on child outcomes, Sear & Mace (2008) found that in 69% of reviewed studies (9 out of 13), maternal grandmothers were associated with an increased probability of grandchild survival, with paternal grandmothers having the same effect in 53% of studies (9 out of 17). For example, maternal grandmothers in Ethiopia who assisted their daughters with heavy domestic tasks were found to increase the probability of a grandchild surviving to three years by 25% (Gibson & Mace, 2005), while in the Gambia they were observed to have a positive effect on the nutritional status of their grandchildren (Sear et al., 2000). Data from pre-industrial Finland has also shown that maternal grandmothers are able to protect against the possible detrimental effects of short interbirth intervals (Nenko et al., 2020), and that their presence had a positive effect on survival between the ages of two and five (Chapman et al., 2021).

However, the positive effect of maternal grandmothers is not universal. In rural Malawi, child mortality rates were found to be higher in the presence of maternal grandmothers (Sear, 2008), and in Greece and Pakistan it was found that paternal grandmothers invested more in their grandchildren than maternal kin (Pashos, 2000; Chung et al., 2020). Though paternal grandmothers have been found to sometimes be a positive presence (Sear & Mace, 2008); as with maternal grandmothers, it appears the level of investment from paternal grandmothers

depends on many external factors such as the sex (Gibson & Mace, 2005) and age (Beise & Voland, 2002) of the grandchild, conflict levels in the household (Sheppard & Sear, 2016), and the ecological conditions (Borgerhoff Mulder, 1990). Further, many studies have found that the presence of paternal grandmothers can increase the likelihood of mortality in grandoffspring (Jamison et al., 2002; Voland & Beise, 2002; Sheppard & Sear, 2016; Chapman et al., 2019), such as in historical Germany, where the presence of a mother-in-law was found to increase the risk of stillbirth in grandchildren by 35% and infant mortality by 85% (Beise & Voland, 2002; Voland & Beise, 2005).

It is thought that some of the differences in investment between maternal and paternal grandmothers can be explained by paternity uncertainty. While maternal grandmothers can always be sure of their genetic relatedness to their daughter, and therefore their grandchildren via their daughter; grandchildren born via a son can be modelled to have a lower relatedness coefficient due to the inclusion of paternity uncertainty, thus lowering their inclination to invest in these grandchildren. However, recent empirical research casts doubt on this assumption. It was found that grandmothers who are both maternal and paternal grandmothers (i.e. have grandchildren via both sons and daughters) increased the survival of grandoffspring born via their daughters *and* their sons; whereas grandmothers who were only paternal grandmothers had no effect on grandoffspring survival (Chapman et al., 2021), suggesting there may an unidentified force other than relatedness and paternity uncertainty driving lineage related differences in investment.

The positive effects of grandmothering have also been documented in WEIRD populations, despite the possible confounding effects of contraception, better medical care, schooling, and neolocal residence patterns. Data from the US and Australia showed that post-menopausal women spent more time caring for their grandchildren, when compared to an activity that had a neutral effect on fitness (Hofer et al., 2019). Further, findings from the Millennium Cohort Study (an ongoing longitudinal study being carried out in the UK) have shown that maternal grandparents spend more time with their new-born grandchildren than paternal grandparents, and were also more likely to provide their grandchildren with financial assistance and gifts (Pollet et al., 2009). In the US, low birthweight infants born to teen mothers who lived with their grandmother (in addition to their own mother) had better cognitive and health outcomes compared to babies who just lived with

their mothers, even after controlling for maternal age (Pope et al., 1993). Similarly, positive behavioural outcomes have been associated with grandmother presence, with research from the US finding that grandchildren who receive care from their grandparents up to the age of two are 28% less likely to score highly on the hyperactivity scale than children who received no grandparental care (Fergusson et al., 2008).

The large amount of data showing the tendency of grandmothers to invest heavily in their grandchildren has resulted in the Grandmother Hypothesis becoming one of the most popular adaptive explanations for the evolution of menopause. Another advantage of this hypothesis is that it also appears to be applicable to the species of cetacean who experience menopause. Amongst killer whales, it has been found that post-reproductive females act as important hubs of ecological knowledge by leading their families to salmon (Brent et al., 2015). Additionally, post-reproductive grandmothers also influence grandoffspring survival, as calves whose grandmother has died in the past two years are at a 4.5 times greater risk of dying than calves with living grandmothers (Nattrass et al., 2019).

Despite the wealth of evidence in favour of the Grandmother Hypothesis, some have argued that positive grandmothering alone is not enough to have selected for a post-reproductive lifespan over continued reproduction (Rogers, 1993; Thouzeau & Raymond, 2017). A game theory model has shown that positive grandmothering combined with high maternal mortality (the Mother Hypothesis) can create the conditions necessary for menopause and a post-reproductive lifespan to be maintained, but finds no evidence for the grandmother effect alone to be sufficient to account for the emergence of menopause (Thouzeau & Raymond, 2017). Further, based on this hypothesis alone, it is unclear why women cannot simultaneously be grandmothers and carry on reproducing themselves. Older elephants have been observed to increase the survival of their grand-calves despite not experiencing a post-reproductive lifespan comparable to ours, suggesting that grandmothers are able to invest in their grandchildren in a positive way without complete reproductive cessation (Lahdenpera et al., 2016).

Taken together, it seems that if menopause is a maintained trait as opposed to one that was selective for in and of itself, then the Grandmother Hypothesis (possibly in combination with the Mother Hypothesis) may be sufficient, as it acts as a selective pressure against the evolution of life-long reproduction in women.

1.3.3.3.3 The Reproductive Conflict Hypothesis

Drawing on evidence for the Grandmother Hypothesis, Cant & Johnstone (2008) proposed the Reproductive Conflict Hypothesis, which concentrates less on the kin-selected benefits of a post-reproductive lifespan, and more on the fitness costs associated with intergenerational co-breeding under different residence patterns (Figure 1.2c).

Humans display a wide range of sex-specific dispersal patterns that result in different levels of relatedness within the residential group. Typically, human kinship systems are classified based on whether males or females remain in their natal group as follows:

i. *Patrilocality*, also known as female-biased dispersal, describes a system where males remain with their natal group for marriage/mating, with females leaving their current residence to join them. This is the most common human residence system, with 71% of societies listed as such in the Ethnographic Atlas (Murdock, 1967), and is associated with a patrilineal inheritance system. Typically, patrilocality occurs when the ecological conditions allow resources to be monopolised by males, resulting in the wealthiest males marrying polygynously (Hartung, 1982).
ii. *Matrilocality* is the converse of patrilocality, and – while rare – is observed on all continents (Schneider & Gough, 1961). Here, women remain in their natal groups, with their husbands joining them when married. This system is associated with matriliny, in which descent is traced through the female line, and often occurs in areas where there are few resources that can be monopolised (Jones, 2011)
iii. *Neolocality*, which is the norm in Western societies, refers to a residence pattern where both the male and female leave their natal homes for marriage and reproduction.
iv. *Duolocality* describes a residence pattern where neither sex disperses. This is relatively rare, but does occur when couples practice 'walking marriages', as seen among the Mosuo of China (He et al., 2016).

It should be noted that there is a huge amount of variability and flexibility in kinship rules and, while a population may be classified as patrilocal – for example – it does not mean everyone within that group adheres to this rule (Hill et al., 2011), which is

something that should be bared in mind when considering the Reproductive Conflict Hypothesis.

In the original Reproductive Conflict Hypothesis model (Cant & Johnstone, 2008), it is predicted that menopause evolved under a patrilocal residence system. This system results in an age dependent relatedness asymmetry that makes it adaptive for younger females to invest in reproductive competition, and for older females to stop reproducing. In patrilocal societies, when a young female leaves her natal group to join a new one, she will be related to no one, which contrasts with the established older females in the group who will have high relatedness by virtue of having been residing and reproducing there for a long time. The young female has two options upon joining the group: 1) she can help the older females reproduce (e.g. assist in childcare); or 2) she can reproduce herself. As the young female is related to no one, she would not gain any inclusive fitness benefits from helping in the group others reproduce. As a result, the adaptive strategy would be for her to invest energy in her own reproduction. The older female also has two options: 1) she can forgo reproduction, help the younger female reproduce, and invest more energy in kin she already has, or 2) she can reproduce herself. As the older female has less to lose from not reproducing due to having higher relatedness to the group, it makes more sense for her to invest less energy into reproductive competition with the younger female, and to invest more in increasing her inclusive fitness via her existing kin. Further, there is a chance that the older female would be related to the new female's offspring, so she would not benefit from generating competition with her new grandchildren (Figure 1.2c). Hence, due to the asymmetry in relatedness between the women of the group and the associated differences in benefits to investing in reproductive competition, it is modelled that the younger females will 'win' the right to directly reproduce, resulting in selection favouring the evolution of an adaptive menopause in older women (Cant & Johnstone, 2008; Johnstone & Cant, 2010).

This model was originally developed to explain menopause and an extended post-reproductive lifespan in humans; however, following the discovery of an analogous reproductive senescence in some cetaceans, the model was amended slightly (Johnstone & Cant, 2010). The original model looked at age-specific reproductive conflict specifically under a patrilocal residence pattern, but whales are not patrilocal. Due to difficulties in data collection, it is not completely clear what the social structure of whales that experience menopause is; however, it is thought they

primarily display natal philopatry with non-local mating (Kasuya & Marsh, 1984; Bigg et al., 1990; Heimlich-Boran, 1993; O'Corry-Crowe et al., 1997; Palsbøll et al., 1997; Ellis et al., 2021). This social structure clearly differs from a patrilocal post-residence pattern predicted to cause the age-specific relatedness symmetries modelled to have caused menopause and a post-reproductive life span to have evolved (Cant & Johnstone, 2008), and was therefore adjusted accordingly (Johnstone & Cant, 2010). In humans, it was modelled that patrilocality combined with local mating resulted in increased female relatedness to the group with age, which gives younger females (with low relatedness) the upper hand when it comes to reproductive competition. In philopatric cetaceans, female-female relatedness remains constant over time, but relatedness to male group members increases with age, creating a relatedness structure where younger females should invest more in competition than older females (Johnstone & Cant, 2010; Croft et al., 2017). The reproductive conflict here occurs between co-breeding mothers and daughters, with empirical findings here showing that simultaneous breeding in killer whales results in the calves of older females having a mortality risk 1.7 times greater than calves of younger females (Croft et al., 2017). It has also been observed that, because older females are not in direct reproductive competition with their sons due to non-local mating, they tend to direct more help towards male offspring as this allows them to increase their inclusive fitness without incurring any costs via within-group competition (Foster et al., 2012; Brent et al., 2015).

Though there is some empirical support for the reproductive conflict hypothesis in cetaceans (Croft et al., 2017), in humans, few studies have been carried out to explicitly test this model. Data from pre-industrial Finland found that simultaneous reproduction with in-laws associates with declines in offspring survivorship of up to 66% (Lahdenpera et al., 2012). However, costs have been associated with co-breeding with many group members: amongst the matrilineal Mosuo, living with a sister associated with decreased fertility (Ji et al., 2013); and in Finland, the risk of offspring mortality as a juvenile increased by 23% if co-residing women reproduced within 2 years of one another (Pettay et al., 2016). Research from the Gambia found results contrary to those predicted by the reproductive conflict hypothesis, in which younger women 'lost' the competition with older, unrelated women (Mace & Alvergne, 2012) – though here, many of the unrelated women were sisters-in-law or other unrelated individuals rather than mothers-in-law, and so the

Reproductive Conflict Hypothesis was not necessarily being directly tested. In the same study, it was also found that, if the reproductive span of mothers and daughters overlapped, the mother reduced her fertility while the daughters' was left unaffected (Mace & Alvergne, 2012). Further, data from historical Norway found results converse to that predicted by the reproductive conflict hypothesis, and showed that women of different generations who co-bred with one another had greater reproductive success than those who did not, possibly due to the learning and allocare opportunities that arise alongside co-breeding (Skjaervo & Roskaft, 2013).

Researchers have also looked at age of menopause when testing this hypothesis, predicting that menopause in patrilocal societies should be earlier as a result of reproductive conflict; however, results from Indonesia found no relationship between level of female dispersal and menopause timing (Snopkowski et al., 2014). Here, it may be that age of menopause is not a reliable metric to measure currently as a proxy of how menopause evolved. As discussed previously, there is a degree of variation in menopause timing, and this variance has been used to model the evolution of menopause in many studies (e.g. Chan et al., 2020). However, for this to be a valid way of studying the evolution of menopause, menopause timing has to be plastic in a facultative way now. It might be that menopause timing – like menarche (Chisholm et al., 2005) – is facultatively plastic, and adaptively responds to various ecological cues to result in an optimal age of menopause; or it may also be that timing of menopause has no adaptive value and is just the result of various hormonal processes. Most of the research into the between and within population differences in age of menopause comes from epidemiology, and therefore it is not discussed from an evolutionary framework. At present, not much research has directly tested whether there is evidence for menopause being facultative today, but evidence for such would add validity to studies where variation in age of menopause is used to test evolutionary hypotheses relating to menopause.

In addition to there being little empirical evidence in favour of it, the Reproductive Conflict Hypothesis can also be brought into question as it is reliant on the assumption that our ancestral residence pattern was patrilocal. However, not only do we not know what the ancestral social structure of humans was, but we also do not know exactly at what point in our evolutionary history menopause and the post-reproductive lifespan emerged, meaning we cannot even begin to model the co-evolution of residence patterns and menopause. Contemporary hunter-gatherer

groups are often used as models of human evolution, but there is not one universal hunter-gatherer social structure, with research showing that cross-culturally either sex may disperse, and that the majority of hunter-gatherer groups have low genetic relatedness (Hill et al., 2011). Further, even in cultures that are classified as being patrilocal, there is always a great deal of flexibility, with many individuals not adhering strictly to the residence rules. The model also assumes that if we did live patrilocally in our ancestral history, then women were cut off from any relatives (and so any way to increase inclusive fitness) completely, when this would not be the case. Even if a woman does move to live with her husband following marriage, there is generally still communication and interaction between the families (Hill et al., 2011), meaning the residence patterns are not as strict as the model may require for the selective pressures causing menopause and a post-reproductive lifespan to evolve to be in force. As a result, we cannot be sure whether the conditions required to create the required age-specific levels of reproductive conflict were present in our evolutionary history.

Something else that could be perceived to be a problem with the hypothesis is the post-hoc amendment of the conditions required for menopause to evolve following the discovery of a post-reproductive lifespan in cetaceans. In Cant and Johnstone's 2008 paper, they propose that the reproductive conflict between young women and older unrelated women that emerges under a patrilocal residence pattern creates the conditions required for menopause to evolve. However, as stated, cetaceans are not patrilocal, and thus this model would not work with them. As such, after menopause was described in cetaceans the model was amended and a new paper published in 2010 explaining how reproductive conflict between mothers and daughters may also facilitate the evolution of menopause and a post-reproductive lifespan. What they did not address was the fact that many residence systems can result in this kind of conflict, with co-residing mothers and daughters being common in matrilocal and duolocal human populations. If this conflict is enough for menopause to evolve in cetaceans, then why was only conflict generated through patrilocality included in Cant and Johnstone's original model? At present, it is unclear why in humans it is conflict between unrelated females that is predicted to cause a post-reproductive lifespan in humans yet in cetaceans it is conflict between related females, which is a large downfall of the hypothesis.

Agent-based models have suggested that resource/reproductive conflict in combination with positive grandmothering might explain how menopause emerged as a trait (Thouzeau & Raymond, 2017). However, the Grandmother Hypothesis and the Reproductive Conflict Hypothesis are not necessarily compatible as they rely on two different migration patterns. The latter hypothesis models that menopause evolved under a patrilocal residence pattern, while the former relies on the positive effect of grandmothering. As discussed earlier, much of the beneficial care comes from maternal grandmothers, with proximity to paternal grandmothers sometimes having a negative effect on offspring fitness. If menopause evolved under a patrilocal residence pattern, as Cant & Johnstone (2008) suggest, then maternal grandmothers would be around less to help with alloparenting, and the reproducing female would instead be living with (or nearer to) her mother-in-law, who has been found to negatively impact grandoffspring survival and health in some populations. Therefore, the reconciliation of the two hypotheses can be brought into question.

1.4 Thesis aims and outline

In this thesis, I will utilise the literature summarised in this chapter so far to generate new hypotheses regarding variation in the menopausal transition, and also to test existing theories about menopause. I do not aim to offer a new evolutionary explanation for why humans have menopause and a post-reproductive lifespan. Rather, I look at the demographic trends of menopause timing and symptoms and interpret them from an adaptive framework, with the hopes that I will make a valuable contribution to the evolutionary literature on menopause and the female post-reproductive lifespan. Therefore, in this thesis I investigate:

1) Whether variation in age of menopause can be understood to be facultative in line with proximate determinants relevant to fitness
2) Whether menopause symptoms require an evolutionary explanation
3) Caring behaviours relative to menopause status

Through discussing current variation in menopause timing from an adaptive framework, it will help us to understand whether current trends can be used to test evolutionary hypotheses about the original emergence of menopause. Further, menopause symptoms are typically discussed within the epidemiological literature and neglected in evolutionary research, despite them being an important aspect of the menopausal transition. Therefore, I test an evolutionary hypothesis regarding the

presence of menopause symptoms, and also use a behavioural ecology framework to analyse some of the variation we see. Finally, though grandmothering is well studied in traditional populations, it has not been researched as much in WEIRD populations. As WEIRD populations have different constraints on grandparenting behaviours to natural fertility populations, such as employment outside of one's social network, neolocal residence patterns, and also the responsibility to care for elder kin, I aim to look at grandmothering in this context.

The remainder of this thesis contains seven chapters, five of which are analytical and each answer and individual research question. In Chapter 2, I introduce the three datasets used throughout the thesis, and the rationale underlying why this data was chosen. Chapter 3 investigates whether age of menopause is facultative relative to the likelihood of future pregnancy. Chapter 4 looks at age of menopause and menopause symptoms relative to residence pattern in a traditional society, and Chapter 5 draws on the findings from Chapter 4 to look at the relationship between menopause symptoms, stressful events, and social support. Chapter 6 tests whether caring behaviours are facultative relative to menopause status, and Chapter 7 looks at the relationship between menopause timing and number of grandoffspring. Finally, Chapter 8 presents the final discussion and summarises the conclusions made throughout this thesis.

CHAPTER TWO

Introducing the data

2.1 Introduction

Throughout this thesis, three different datasets will be used: the Study of Women's Health Across the Nation (SWAN), the National Child Development Study (NCDS), and data collected from China. Choices regarding data usage were primarily made based on variable availability. While there are many longitudinal cohort studies available to use data from, not all collect data on age of menopause or menopause symptoms. For example, of the nine UK cohort studies that form CLOSER (Cohort and Longitudinal Studies Enhancement Resources), only four include questions that mention the word "menopause", with the usage of datasets further being limited by the relevant covariates required to test the hypotheses in this thesis. After evaluating the various datasets available based on the variables they contain, I settled on using SWAN – which is a cohort study designed to collect data on the menopausal transition – and the NCDS. In addition to these datasets, I also had the opportunity to use primary data collected by students and researchers at Lanzhou University to answer one of my research questions. Therefore, this thesis contains a mixture of both primary and secondary data sources. Both have their advantages: the longitudinal cohort studies allow access to a large sample of data collected over decades, however, as it is pre-collected, I am limited in terms of what questions have been asked; conversely, the data from China was somewhat tailored to this project but is far smaller in sample size. In this thesis, Chapters 3 and 5 use data from SWAN, Chapter 4 data from China, and Chapters 6 and 7 data from NCDS. This chapter will give an overview of these different datasets.

2.2 The Study of Women's Health Across the Nation

SWAN is an ongoing, multisite, multi-ethnic, longitudinal cohort study, designed to study the menopausal transition. This study emerged firstly because the menopausal transition was poorly understood, and secondly because the little data that was available on menopause was racially biased towards white women who had either self-referred to menopause clinics or were already being seen in a clinical setting for

other health problems (Sowers et al., 2000). Funded by the National Institute on Aging, the National Institute of Nursing Research, and the Office of Research on Women's Health, SWAN aims to understand the biological and psychosocial changes that characterise the menopausal transition, and the predictors of poor health post-menopause. As the majority of research into menopause prior to SWAN had focussed on white women, SWAN placed a special emphasis on the recruitment of women from minority populations to try and understand the relationship between characteristics of the menopausal transition and lifestyle factors. As such, SWAN's recruitment process was community-based – rather than volunteer or clinically based – as a means of trying to ensure the sample is representative of all socioeconomic and cultural backgrounds (Sowers et al., 2000).

SWAN has multiple sites across the US, with each site recruiting white women in addition to a sample of women from a predetermined minority group (African Americans in Boston, Chicago, Detroit, Michigan, and Pittsburgh; Chinese Americans in California; Japanese Americans in Los Angeles; and Hispanic women in Newark) (Gold et al., 2001). Beginning in 1994, SWAN has been conducted in three phases. Firstly, researchers recruited a focus group of women and held discussions that focussed on how best to develop an effective and culturally sensitive study design, that would successfully be able to recruit and retain a diverse group of women. After this, a cross-sectional survey was carried out between January 1995 and January 1997. Eligibility criteria included being able to speak and read in English, Cantonese, Japanese or Spanish, being aged between 40 and 55, living in one of the previously mentioned sites, and self-identifying with one of the two ethnic groups being targeted at each site. Data collection was carried out through 15-minute interviews, held either on the phone or in-person; and women were recruited for this phase using telephone numbers randomly generated from random digit dialling[1] and through census records (Sowers et al., 2000). In total 202,985 households were screened for potential participation in the cross-sectional survey, with 34,985 fitting the eligibility criteria, and 16,065 completing the interview. This cross-sectional screener allowed the researchers to identify women who were eligible for further longitudinal study, and also gain an initial understanding of the cross-sectional

[1] Randomly generated phone numbers were first screened to ensure it corresponded with an actual household, and these households then also screened to determine whether it contained a woman eligible to participate in the cross-sectional study (e.g. right age, ethnicity, and so on).

associations of age of natural menopause. In the final phase, women from the cross-sectional survey who were between 42 and 52 years old, had menstruated within the last three months, still had a uterus, had at least one ovary, and had not taken hormonal medication (e.g. hormone replacement therapy, birth control) in the last three months were invited to take part in a longitudinal follow up study. In total, 6,521 women were deemed eligible for participation. Enrolment to the study began in 1996, and by December 1997 all sites had completed the baseline visit. 3,302 women completed SWAN's baseline interview, of which 46% were white, 28% African American, 9% Hispanic, 9% Japanese American, and 8% Chinese American (Sowers et al., 2000; Santoro & Sutton-Tyrrell, 2011).

At present, SWAN has completed 16 rounds of interviews (in addition to the cross-sectional survey and baseline visit) (SWAN Study, 2021). Questionnaires are translated into Spanish, Cantonese, and Japanese, and collect data through both interview-administered and self-administered questionnaires. Physical measures are also taken, such as weight, height, and blood pressure, and a fasted morning blood draw. To collect data on menstruation, women are also provided with calendars to complete on a monthly basis (Santoro & Sutton-Tyrrell, 2011).

As SWAN is a community-based sample, rather than participants having been chosen as a national probability sample, results cannot be generalised to the national population of midlife women (Sowers et al., 2000). However, only a limited number of studies have looked at menopause timing and menopause symptoms in a large multi-ethnic and multi-cultural sample, and the frequency and extent that SWAN has. The large number of variables collected allows researchers to look at social and biological aspects of the menopausal transition, and since conception, has produced a plethora of research into the determinants of menopause timing and menopause symptoms (for example: Gold et al., 2000; Gold et al., 2004; Avis et al., 2005b; Bromberger & Kravitz, 2011; Santoro & Sutton-Tyrrell, 2011; Thurston & Joffe, 2011; Avis et al., 2015; Hedgeman et al., 2018). At present, 10 years' worth of data – in addition to the baseline and cross-sectional interviews – is available to the public for analyses, with subsequent years of data currently being analysed by the SWAN investigators (SWAN Study, 2021). This thesis therefore utilises the publicly available data only, with the selected variables and the years of interviews from which these were taken shown in Table 2.1 (Sutton-Tyrell et al., 2018a; Sutton-Tyrell et al., 2018b; Sutton-Tyrell et al., 2018c; Sutton-Tyrell et al., 2018e; Sutton-Tyrell et al.,

2018d; Sutton-Tyrrell et al., 2018; Sutton-Tyrrell et al., 2019f; Sutton-Tyrrell et al., 2019a; Sutton-Tyrrell et al., 2019e; Sutton-Tyrrell et al., 2019d; Sutton-Tyrrell et al., 2019c; Sutton-Tyrrell et al., 2019b).

Table 2.1. *List of variables used from the longitudinal datasets in each chapter of this thesis, and the dataset and interview they were taken from.*

Variable	**SWAN interview number**	**NCDS interview number**	**Chapter**			
			3	**5**	**6**	**7**
Age	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	Perinatal mortality study, NCDS1, NCDS2, NCDS3, NCDS4, NCDS6, NCDS7, NCDS8, NCDS9	✓	✓		
Age first birth		NCDS4, NCDS5, NCDS6, NCDS7, NCDS8				✓
Age left education		NCDS6			✓	
Body mass index	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10		✓			
Employment status		NCDS9			✓	✓
Ethnicity	Baseline interview		✓	✓		
Health	Cross-sectional screener interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	NCDS9	✓	✓	✓	✓
Hours spent caring for grandchildren		NCDS9			✓	
Hours spent caring for parents		NCDS9			✓	
Male cohabitation	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10		✓			

Marital status	Cross-sectional screener interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	NCDS9	✓	✓	✓	✓
Maximum education	Cross-sectional screener		✓	✓		
Menarche	Baseline interview		✓			
Menopause status	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	NCDS6, Biomedical interview, NCDS8, NCDS9	✓	✓	✓	✓
Menopause symptoms	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10			✓		
Number of children	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	NCDS4, NCDS5, Biomedical interview, NCDS7, NCDS8, NCDS9	✓		✓	✓
Number of grandchildren		NCDS7, NCDS9			✓	✓
Parental age		Perinatal mortality study			✓	
Parental mortality status		NCDS1, NCDS2, NCDS3, NCDS4, NCDS6, NCDS7, NCDS8, NCDS9			✓	
Sexual frequency	Baseline interview, 1, 2, 3, 4, 5, 6, 8, 10		✓			
Smoking	Baseline interview, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10	NCDS3, NCDS4, NCDS5, NCDS6, Biomedical interview, NCDS7, NCDS8, NCDS9	✓	✓		
Stressful events	1, 2, 3, 4, 5, 6, 8, 9, 10			✓		
Support	1, 2, 3, 4, 5, 6, 8, 9, 10			✓		

2.3 The National Child Development Study

Also known as the 1958 British birth cohort study, the NCDS is an ongoing, multidisciplinary, longitudinal study. It began as the study of Perinatal Mortality

(Power & Elliott, 2006), focussing on the births of 17,205 women from England, Scotland, and Wales during the week of the 3rd to 9th of March 1958 (n = 17,415 births). Initially designed as a one-off study, the Perinatal Mortality Study came about to research why Great Britain had a relatively high rate of stillbirths compared to other 'developed' countries. Five years later, a follow up of NCDS was commissioned by the National Children's Council for Education (the Plowden Committee), who were researching school children. Data collection for this took place in 1965 when the cohort members were 7 years old, and looked at their educational, physical, and social development (Davie et al., 1972). The results of this survey are published under the title *Children and Their Primary Schools* (Central Advisory Council for Education, 1967). The children were once again followed up at the ages of 11 and 16. For these surveys, data was collected both through interviews with the cohort member, in addition to interviewing a parent or guardian (usually the mother) and employees of the cohort member's school. During the time at which this cohort was at school, there was significant debate about the schooling system, as the 'eleven-plus' exam was in the process of being abolished, and the school leaving age was being raised to 16 (Power & Elliott, 2006). To understand the transition from adolescence and dependency to adulthood and independent living, the cohort was once again surveyed in 1981 (age 23). This is the first of the follow-ups in which all of the data was collected through just interviewing the cohort member, rather than a parent or a guardian. From here, participants were followed up a further seven times at ages 33, 42, 44, 46, 50, 55 and 62. This includes a biomedical survey that was carried out at age 44 to examine how aspects of development, lifestyle and the environment affect health and psychological functioning in early mid-age; and a special online survey conducted during 2020 (at age 62) to examine the effects of COVID-19 on various aspects of life. The 'official' 10th sweep of interviews was due to be carried out in 2020 to examine the cohort members lives in their early 60s, however, it has currently been postponed due to the pandemic. Table 2.2 gives an overview of the timing of each interview and its intended purpose.

As stated, the participants are those who were born during one week in 1958, with the baseline sample amassing to over 17,000 individuals. Despite the large sample size, it does not represent the ethnic diversity of Great Britain's current population. For example, at 11 – when ethnicity was first recorded – of a sample of

15,335 children, 81.43% were white, 16.71% had missing data, with the remaining 1.86% being classified as Black, Asian, mixed race, or 'other'. This is a major

Table 2.2. *National Child Development Study dates of contact and aims. Adapted and updated from Power and Elliott (2006).*

Sweep	Year	Age	Data collected from	Aims
Perinatal mortality survey	1958	Birth	Mother and medical records	Study the health of mothers of new-born babies, and new-born babies
NCDS1	1965	7	Parents; school; tests; medical exam; cohort member	Measure the educational, social, and physical development of children
NCDS2	1969	11	Parents; school; tests; medical exam; cohort member	To track children's development as they transition from primary to secondary school
NCDS3	1974	16	Parents; school; tests; medical exam; cohort member; census	Continue measuring the educational, social, and physical development of the children as they go through adolescence
Exams	1978	20	Schools attended by cohort member at age 16	Gauge academic outcomes
NCDS4	1981-1982	23-24	Cohort member; census	To understand the transition from adolescence to adulthood
NCDS5	1991	33	Cohort member; spouse/partner; children; children's mother	Continue measuring the cohort members journey through adulthood
NCDS6	2000	42	Cohort member	Continue measuring aspects of the cohort members life, such as living arrangements, relationships, employment, smoking, and drinking
Biomedical interview	2002-2003	44-45	Cohort member	Understand factors associated with health in early middle age

NCDS7	2004	46	Cohort member	Understand lives, relationships, technology use, smoking, drinking habits, and experience of crime in middle age
NCDS8	2008	50	Cohort member; tests	Find out what the cohort members lives are like as they hit a big milestone
NCDS9	2013-2014	55-56	Cohort member	Understand the cohort members relationships, work, expectations of retirement, and caring responsibilities
NCDS10	2020 (post-poned)	62	Cohort member	Continue collecting data on relationships and work, in addition to memories from childhood
COVID-19 survey	2020-2021	62-63	Cohort member	Examine the impact of the pandemic on work, health, relationships, and social life

limitation of this study, and means that any findings using this data are not generalisable to the whole population. Despite this, the NCDS has been a highly influential study with over 1000 publications to date, primarily in the fields of health research and social sciences. The wealth of data and the plethora of variables available make it possible to test hypotheses from many different disciplines, including evolutionary anthropology.

2.4 Primary data collected from China

2.4.1 Study area

The final source of data used in this thesis was primary data collected in Southwestern China from women of four ethnic groups: the Mosuo, Zhaba, Han, and Yi. The Mosuo and Zhaba, who reside around Lugu Lake and in Daofu County, respectively, display an uncommon duolocal residence pattern coupled with matrilineal descent. This means that neither sex leave the natal household for marriage, and descent is traced down the maternal line (Wu et al., 2013). The Mosuo are perhaps most famous for this social structure, having been dubbed the 'Kingdom of Women' (Choo, 2017). The Han and Yi also live around Lugu Lake, but display patrilocality with patriliny, meaning that women typically join the husband's family following marriage, and descent is traced down the male line.

In the Mosuo and Zhaba, families live in large households of up to three generations of brothers, sisters, and any matrilineal offspring. One of the most famous aspects of the cultures of these ethnicities is the practice of 'walking marriage'. Here, a woman and her partner live separately, with the man only visiting his 'girlfriend' at night, and then returning to his natal home in the morning. It is thought that this mating system in combination with matriliny will lead to high levels of paternity uncertainty that would result in men investing more in their sisters' children – through whom their relatedness is certain – than their offspring. While quantitative evidence showing high levels of avuncularity amongst the Mosuo and Zhaba has not yet been found (Mattison et al., 2019), ethnographies have reported that men in matrilineal societies give heritable possessions, such as land, to his sisters' children (through whom relatedness is certain) (Richards, 1950), but are still involved with the day-to-day upbringing of their own children (Mattison et al., 2014). In general children in duolocal societies generally receive high levels of investment, due to the large number of alloparents available in their own household (Mattison et al., 2014). Sisters reproduce communally, with older sisters investing more time in household work, and also having greater reproductive success than their younger sisters (Ji et al., 2013).

All ethnic groups included in this sample use agriculture as their primary means of subsistence; however, suitable land for this practice is constrained by the steep, forested hills that surround Lugu Lake and Daofu County. Further, for those living around Lugu Lake their traditional way of living is further being affected by the increasingly polluted lake and growing tourism industry. Since 1990, Lugu Lake has become a beauty spot for both domestic and international tourists, receiving over 90 million visitors in 2007 (Mattison, 2010). This tourism has had an impact on the Mosuo's way of life, with cultural diffusion and increased rates of intermarriage with Han individuals causing many to deviate from the traditional matrilineal, duolocal norms. In addition to this, a family planning policy established in China in the 1980s meant that marriage became a requirement for reproduction, and therefore partnerships in the Mosuo and Zhaba are slightly more formal now; however, in the majority of cases, the husband and wife still live apart (Thomas et al., 2018). It should be noted that this family planning policy only allowed most ethnic groups in China (including the Han) to only have one child at a time, but was not applicable to most

ethnic minorities (such as the Zhaba and Mosuo) who were allowed to have up to two or three children while the policy was being implemented.

2.4.2 Data collection

Data collection in China was carried by Yuping Yang, Yuan Chen, Erhao Ge, Jiayu Huang, Yu Tang, Almira Ahmed, Xin Zhu, Hanzhi Zhang, and Jiajia Wu over the course of two field seasons in 2018 and 2019, during which Lugu Lake and Daofu County were both visited. Eight villages across both sites were visited in total, with all households being invited to take part in the research. Of those that agreed to participate, adult women from within the household were interviewed. Consent was sought from the interviewee and from the Local People's Government at each site, and the research was approved by Lanzhou University Life Sciences and UCL Research Ethics committee.

All women who agreed to participate were asked a standard demographic survey, which included questions about their age, post-marital residence patterns, family structure, socioeconomic characteristics, smoking status, living arrangements, fertility status, and so on. In addition to this, the Menopause Rating Scale (MRS; see Appendix B Figure B1) was used to collect data on menopause symptoms. The MRS was developed to ensure both clinicians and researchers had a standard method of measuring women's quality of life when going through the menopausal transition (Heinemann et al., 2003). It was initially constructed in a Western medical setting, but it has since been cross-culturally validated elsewhere (Heinemann et al., 2003; Heinemann et al., 2004; Dinger et al., 2006; Wu et al., 2015a). The MRS measures the severity of 11 symptoms commonly experienced throughout the menopausal transition on a Likert scale ranging from 0 to 4 (0 = none, 1 = mild, 2 = moderate, 3 = severe, 4 = very severe). These 11 symptoms can then be further divided into four sub-groups: psychological symptoms (depression, irritability, anxiety, exhaustion), urogenital symptoms (sexual problems, bladder problems, vaginal dryness), and somato-vegetative symptoms (hot flashes, heart discomfort, sleep problems, joint/muscular discomfort). By using the values derived from the Likert scale, symptoms scores can then be calculated, with an overall symptoms score ranging from 0 to 44, a psychological symptoms score from 0 to 16, a urogenital symptoms score from 0 to 12, and a somato-vegetative symptoms score from 0 to 16. This data

was collected both retrospectively (if the woman was post-menopausal) and presently (if the woman was peri-menopausal).

2.5 Closing remarks

In the following four chapters, results are presented of analyses using the data previously outlined. SWAN is used in Chapters 3 and 5, NCDS in Chapters 6 and 7, and the primary data from China used in Chapter 4. Data were chosen specifically for these chapters based on the availability of the required variables. Though it might have been preferable to use fewer datasets in this thesis for cohesiveness and to make the results directly comparable to one another; through utilising the wide range of data available, it has allowed me to answer research questions that have not yet been addressed within the literature.

CHAPTER THREE

Age of menopause as a facultative trait

3.1 Chapter summary

Many factors have been observed to associate with age of natural menopause, including marital status. Here, women who are married tend to experience menopause later than those who are single, separated, or widowed, and the reasons for this relationship are yet to be understood. In this chapter, I test an original hypothesis that sexual frequency acts as a bio-behavioural mediator between marital status and age of natural menopause. I hypothesise that a trade-off occurs between continued ovulation and menopause based on the woman's chances of becoming pregnant. If a woman is sexually inactive, then pregnancy is impossible, and continued ovulation can be seen as a non-optimal strategy and therefore not adaptive. In addition to this hypothesis, I test an existing model that proposes that the relationship between marital status and menopause timing is due to exposure to male pheromones (Sievert et al., 2001). Data used in this chapter are taken from SWAN, and time-varying Cox regression is used to test the two hypotheses. As a proxy of male pheromones, male household presence was measured, and no relationship was found between cohabiting with a male and menopause timing. However, results did demonstrate that women who had sex weekly during the study period were 28% less likely to experience menopause at any given age than women who had sex less than monthly. This suggests that menopause timing may be somewhat facultative in response to the likelihood of pregnancy, if sexual frequency is taken as a proxy of such. Further, this is evidence that age of natural menopause is responsive to behavioural factors, which gives grounding to the notion that menopause timing may vary according to ecology.

The results presented in this chapter have been peer-reviewed and published in *Royal Society Open Science* under the title "*Sexual frequency is associated with age of natural menopause: results from the Study of Women's Health Across the Nation"* and co-authored by my supervisor, Ruth Mace (Arnot & Mace, 2020).

3.2 Introduction

As shown in Chapter 1, there is a great deal of current cross-cultural variation in age of natural menopause (Laisk et al., 2019). While menopause timing is somewhat governed by genetic factors (de Bruin et al., 2001), it is estimated that up to half of the population variance in menopause timing is the result of non-genetic influences (Mishra et al., 2009). Menopause and the female post-reproductive lifespan are thought to have evolved or been maintained through selective pressures, which has resulted in people using the variance in menopause timing as a proxy of the evolution of the trait (e.g. Snopkowski et al., 2014). However, the validity of using current age of menopause as a proxy of the evolution of the trait is seldom discussed. As menopause timing appears to be affected by many socioeconomic and lifestyle factors, it may just be that the variation we see now is a product of these factors, and not governed by evolutionary principles at all. For example, while selective pressures may have selected for a menopause that occurs at some point in mid-life, any variation we see around this mean point could just simply be random and not a reflection of the effects of the selective pressures that resulted in the evolution of menopause in the first place. Therefore, in this chapter, I aim to examine whether menopause still does vary in the way one would predict based on frameworks taken from evolutionary theory; specifically, how energetic trade-offs might impact menopause timing.

Menopause is integrally a biological process and the result of ovulation and follicular depletion, a number of behavioural and lifestyle factors have been found to associate with menopause timing, including smoking habits, socioeconomic status, educational attainment, and body mass index (Gold et al., 2000; Gold et al., 2001; Sievert et al., 2001; Ozdemir & Col, 2004; Ayatollahi et al., 2005; Nagel et al., 2005; Parazzini & Progetto Menopausa Italia Study, 2007; McKnight et al., 2011; Li et al., 2012; Sapre & Thakur, 2014; Tao et al., 2015; Ruth et al., 2016; Bjelland et al., 2018; Wang et al., 2018). One puzzling association reported in epidemiological literature is the relationship between marital status and menopause timing, in which married women attain menopause later than never married or divorced women (Gold et al., 2001; Sievert et al., 2001; Ayatollahi et al., 2005; OlaOlorun & Lawoyin, 2009; Sapre & Thakur, 2014; Ahuja, 2016; Shadyab et al., 2017) – a relationship that still persists even after controlling for possible confounders. Currently, there is little understanding of why marital status would present this relationship with menopause

timing. One existing hypothesis postulates that the relationship between menopause timing and marital status is the result of male-female cohabitation (Sievert et al., 2001). This is based on the idea that increased exposure to male pheromones (as a result of being married, and therefore cohabiting) may increase the likelihood of having a regular menstrual cycle, with regular menstrual cycles having been observed to delay the menopause (Whelan et al., 1990). As an alternative to this, I propose an adaptive explanation based on an energetic trade-off. As married people typically have sex more often than those who are uncoupled (Addis et al., 2006; Flynn & Gow, 2015), I suggest that the observed relationship between marital status and menopause timing may be capturing the effect of sexual frequency during pre- and peri-menopause on menopause timing. Ovulation can be seen as a costly process, both in terms of energetics and due to its impairing effect on the immune system (Alvergne & Tabor, 2018; Lorenz et al., 2018). Hence, should a woman be having little or infrequent sex when approaching midlife, then the body will not be receiving the physical cues of a possible pregnancy, and it may therefore not be optimal to invest resources into continued ovulation. Rather, it would be better from a fitness-maximising perspective for the woman to cease fertility, and invest energy into any existing kin she has (Hawkes et al., 1998; Shanley et al., 2007; Sear & Mace, 2008). Conversely, if the woman is still engaging in sex regularly, then it may be better for her to continue ovulating for slightly longer, allowing her to increase her direct fitness.

3.2.1 Aims and hypotheses

Currently, there is no clear reason why married women experience a later menopause, and I propose a functional reason for this relationship. Here, I test the two following hypotheses:

Hypothesis 1. Increased sexual frequency lowers the risk of entering menopause;

Hypothesis 2. Exposure to male pheromones delays menopause.

I acknowledge that this study is largely correlative, but nonetheless, if positive results are found in favour of Hypothesis 1, then would suggest that menopause timing could be facultative, and that the within and between population differences in age of natural menopause could be understood from an evolutionary framework.

3.3 Materials and methods

3.3.1 Data

Data from 3093 women were drawn from SWAN, which is an ongoing community-based, multi-site, longitudinal cohort study currently being carried out in the US, specifically designed to collect data on the biological and psychosocial changes that occur alongside menopause. Despite being a community-based sample, SWAN is thought to be the largest, most diverse, and most representative study currently available to research aspects of the menopausal transition (Bromberger & Kravitz, 2011). Criteria to be part of the baseline cohort (recruited in 1996/97) included being aged between 42 and 52, having an intact uterus, at least one ovary, not being pregnant, having experienced a menstrual cycle within the past three months, and self-identifying as one of the five pre-specified racial/ethnic groups (African-American, Chinese or Chinese American, Japanese or Japanese American, Hispanic, or white) (Sowers et al., 2000).

The current analysis uses data from the baseline interview and follow up visits 1, 2, 3, 4, 5, 6, 8 , and 10. Data from interviews 7 and 9 were omitted from this chapter as questions regarding sexual frequency were not asked in those years (Sutton-Tyrell et al., 2018a; Sutton-Tyrell et al., 2018b; Sutton-Tyrell et al., 2018c; Sutton-Tyrell et al., 2018e; Sutton-Tyrell et al., 2018d; Sutton-Tyrrell et al., 2018; Sutton-Tyrrell et al., 2019f; Sutton-Tyrrell et al., 2019a; Sutton-Tyrrell et al., 2019e; Sutton-Tyrrell et al., 2019d; Sutton-Tyrrell et al., 2019c; Sutton-Tyrrell et al., 2019b).

3.3.2 Analyses

Time-varying Cox proportional hazards modelling was used to conduct an event history analysis, which is a powerful method of regression modelling that allows the isolation of precise effects over the risk of an event happening (Cox, 1972). Typically one's risk of death is modelled; however, in this analyses, risk of menopause serves as the hazard. Unlike standard regression models, it is able to deal with time-series and censored data, and produces a hazard ratio (HR), which is a measure of the risk of the event happening. The HR can be expressed as follows:

$$h(t) = \frac{f(t)}{S(t)}$$

Where t is the time interval, and so $f(t)$ the probability of the event at a given t, and $S(t)$ the probability of the event happening at that point in time. In all models, the

age of the participant was used as the time-scale (Korn et al., 1997), with participants being left censored, or entering the model, at the age of 40. Once a woman had experienced menopause she was no longer retained in the dataset, and women who did not experience menopause during the study period or who stopped menstruating for a reason other than menopause were right censored. Analyses were carried out in *R* (version 4.0.3) (R Core Team, 2020) using the packages *survival* (Therneau, 2015), *survminer* (Kassambara & Kosinski, 2018), and *AICcmodavg* (Mazerolle, 2020). Visualisations were created using *ggplot2* (Wickham, 2016).

3.3.3 Variables

3.3.3.1 Age of menopause

Menopause timing was the primary variable of interest within the study, with the risk of menopause at any given age being measured in the regression models. Biomedically, a woman is defined as having experienced menopause once she has experienced 12 months of amenorrhea in the absence of external influence over menstruation (e.g. breastfeeding, hormonal contraceptives) (Kirchengast & Ruhli, 2013). SWAN provides an existing derived variable that conforms to this definition, and this was used to code women as having experienced menopause, not entered menopause (e.g. still menstruating, peri-menopausal), or ceased menstruation for another reason (e.g. hysterectomy). Women who did not experience menopause during the study period (either because they were still menstruating, or had stopped menstruation for another reason) were right censored; and women who did experience menopause left the study sample once they had experienced menopause.

3.3.3.2 Sexual frequency

To test the first hypothesis, I looked at age of natural menopause in relation to sexual frequency. A time-varying "sex index" was derived from the woman's responses to questions about her sex habits, which included:

- "During the past 6 months, have you engaged in sexual activities with a partner?" (yes; no)
- "During the past 6 months, how often, on average, have you engaged in each of the following activities: sexual touching or caressing; oral sex; sexual intercourse?" (not at all; once or twice a month; about once a week; more than once a week; daily)

- "On average, in the last 6 months, how often have you engaged in masturbation (self-stimulation)?" (not at all; less than once a month; once or twice a month; about once a week; more than once a week; daily).

An aggregated 'sex index' was constructed as preliminary analyses looking at each individual measure of sexual engagement and menopause timing showed that they all presented a similar relationship with age of menopause. Hence, for each woman, the maximum amount of sexual activity from any of the aforementioned questions was taken as being her sexual frequency. For example, if a woman reported having intercourse 'once or twice a month', but oral sex 'daily', 'daily' was recorded as her sex index. Sexual activity other than intercourse was used to create the sex index as the hypothesis is predicting that cues from sex will result in a trade-off, and the underlying mechanism of sexual touching, oral sex, and masturbation could all signal possible pregnancy to the body. Due to the small number of responses in some of the categories they were aggregated into three new categories, with '*less than monthly*' comprising of women who reported to not have had any sex in the past 6 months, as well as those who responded 'not at all' or 'less than once a month' within the sexual activity questions. '*Monthly*' sex was used to code women who have engaged in any form of sexual activity 'once or twice a month'; and '*weekly*' if a woman reports engaging in any form of sexual activity 'about once a week', 'more than once a week' or 'daily'.

3.3.3.3 Male pheromones

It is proposed that women who cohabit with men have a later menopause because male pheromones regulate menstrual cycles, with a more regular menstrual cycle predicting a later menopause. There is no direct measure of male pheromone exposure within the dataset; however, SWAN offers two options as an alternative: menstrual cycle stability, and presence of men in the household (through looking at household composition). While the former variable would be desirable to use in analyses (as it is proposed that it is a stable menstrual cycle length that contributes to a later menopause (Whelan et al., 1990)) there is a large amount of missing data in this variable (Figure 3.1), and including it in the analyses would have resulted in a significantly reduced sample size. Due to this, I use male household presence is used as an alternative. However, it should be noted that the graph presented in Figure 3.1 shows that there is no significant difference in cycle regularity between women who

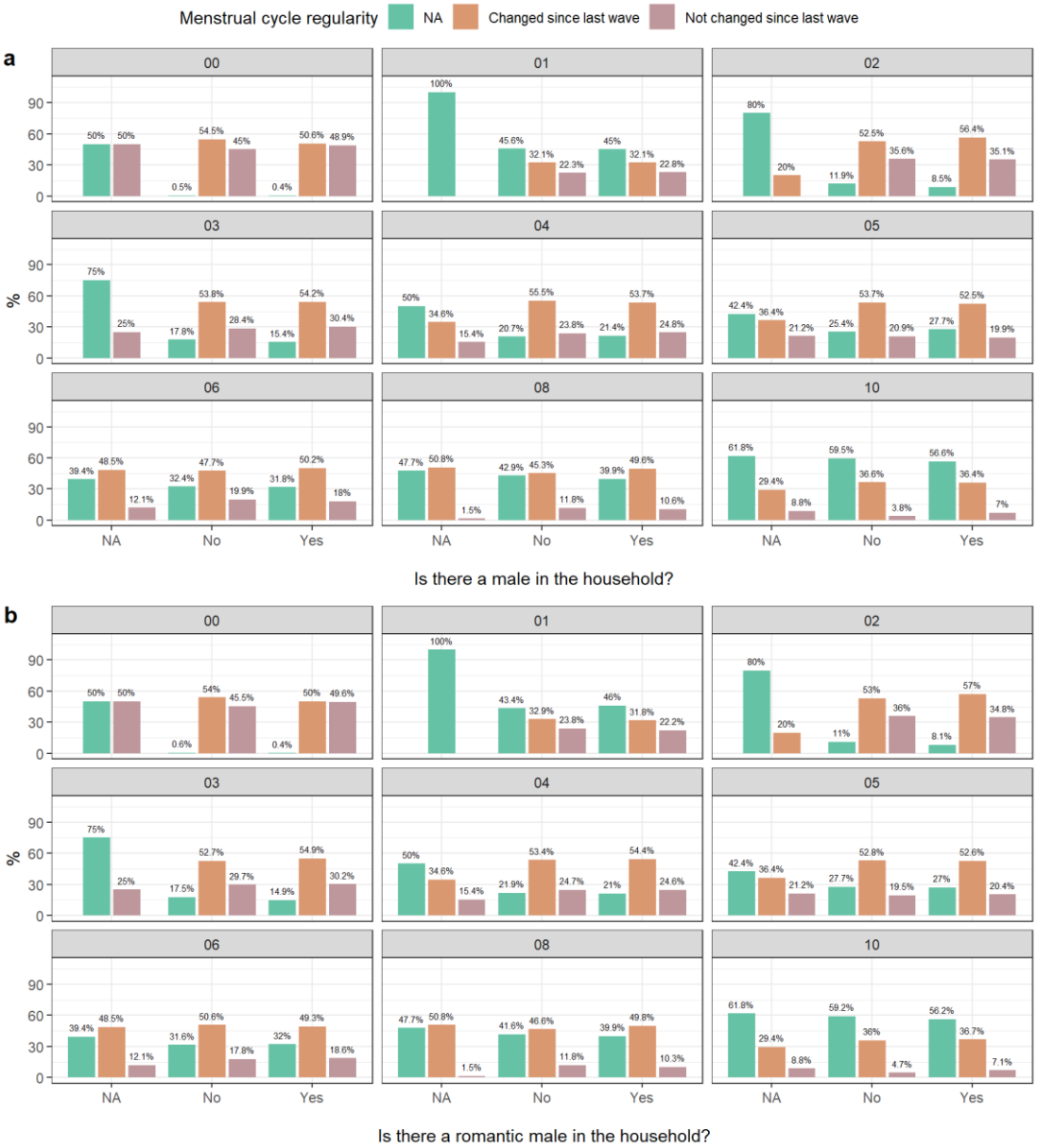

Figure 3.1. *Male cohabitation and menstrual cycle stability. Plot a) shows menstrual cycle stability by general male-female cohabitation, and plot b) shows menstrual cycle stability by female cohabitation with a romantic male partner.*

cohabit with a man versus those who do not. Results from a chi-square test confirm the insignificance of this difference ($p > 0.05$), and this will be addressed further in the discussion.

To measure male-female cohabitation, three variables were derived from SWANs questions regarding household composition. Firstly, a binary variable was created based on whether the respondent reported living with a romantic male partner, such as a husband, boyfriend, fiancé, or similar. A second binary variable was made which then coded whether the woman lived with any male (e.g. husband,

son, male friend), and this was also included as a variable that counted the number of males living in the house.

3.3.3.4 Covariates

Marital status was included as a covariate when looking at the relationship between sexual frequency and age of natural menopause, based on previous literature isolating it as being associated with menopause timing. It was not included when testing the pheromone hypothesis due to the high degree of collinearity with male household presence. To create this variable, women's responses to questions regarding their relationship status and living arrangements were used, with women subsequently being coded as 'divorced, separated or single', 'married or in a relationship', or 'widowed'.

Additional variables were selected based on existing research looking at what influences age of natural menopause (see Section 1.3.2 for a detailed review of their relationship with menopause timing). Time-varying covariates, which were measured at every interview, included the woman's smoking habits (never smoked; ever smoked) and body mass index. Ethnicity (self-identified as black or African American; Chinese; Japanese; white; Hispanic), educational attainment (less than high school education; high school education; some college/technical school; college degree; post-graduate education), number of live births, and age at menarche were included as time invariant covariates, and were therefore held constant in analyses (Gold et al., 2000; Gold et al., 2001; Sievert et al., 2001; Hardy & Kuh, 2002; Reynolds & Obermeyer, 2003; Ozdemir & Col, 2004; Ayatollahi et al., 2005; Nagel et al., 2005; Parazzini & Progetto Menopausa Italia Study, 2007; OlaOlorun & Lawoyin, 2009; Gold, 2011; McKnight et al., 2011; Li et al., 2012; Perez-Alcala et al., 2013; Sapre & Thakur, 2014; Tao et al., 2015; Ahuja, 2016; Ruth et al., 2016; Tao et al., 2016; Mishra et al., 2017; Bjelland et al., 2018; Wang et al., 2018; Zhu et al., 2018). Self-perceived health was also included as a time-varying covariate to adjust for the physical changes that occur throughout the menopausal transition, which may also affect a woman's likelihood and desire to engage in sex (Bach et al., 2013).

3.3.4 Sample

A complete case analysis was carried out, meaning if a participant had missing data for a particular variable in a particular wave, their data from that wave of interviews

was not included in the analysis. As the data are longitudinal, it does mean that they would not be dropped from the whole analysis, only the specific year of interviews where there is missing data; however, two women with no participant number were removed from the whole analyses as it was not possible to link the data to the individual, making the possible sample of women 3300.

As can be seen in Figure 3.2a, there are generally low levels of missing data throughout the dataset. A full breakdown of missingness is presented in Appendix C Table C1, but on average 262 women were lost per wave due to missing data. Figure 3.2a shows how missingness was clustered across variables, and it can be seen here that there are groupings of missingness, suggesting that it was the same women who

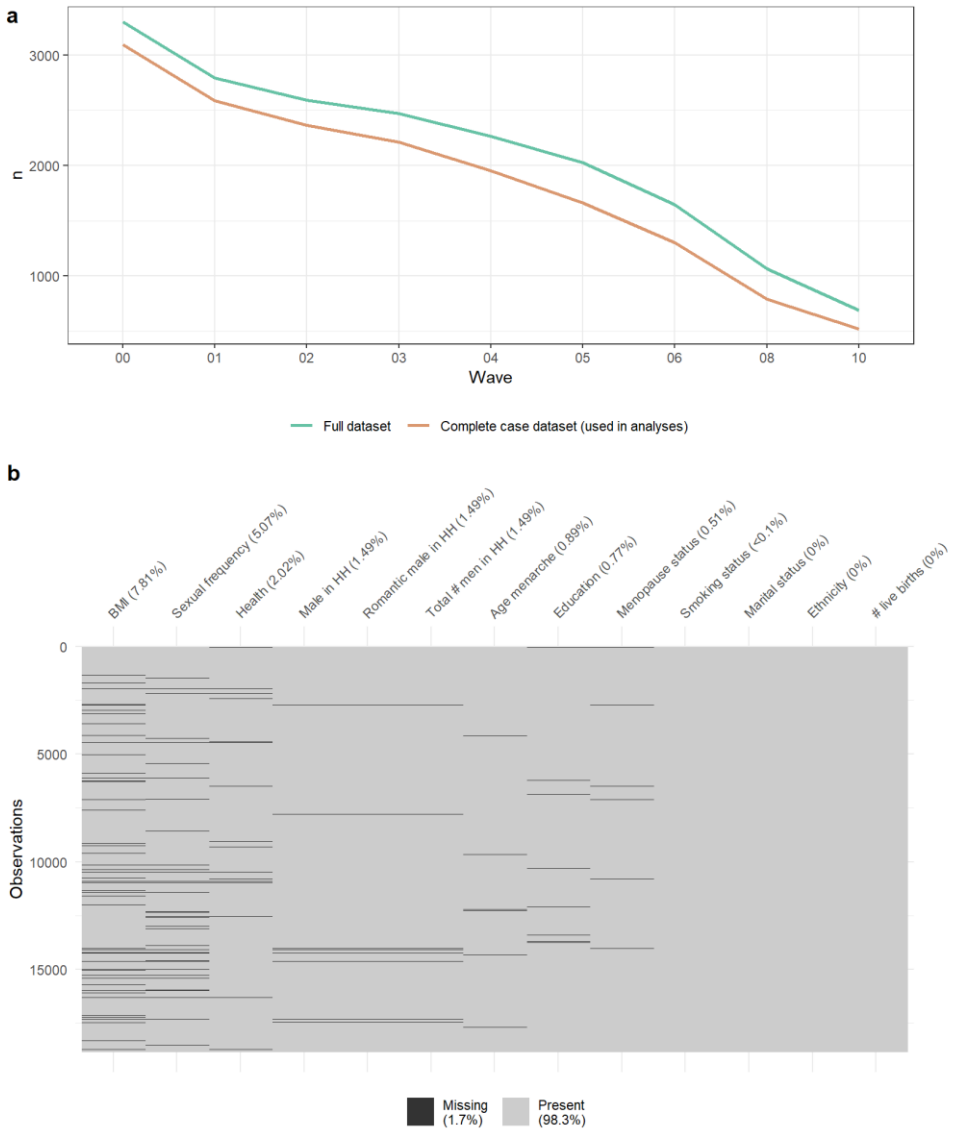

Figure 3.2. *Plot a) shows the possible number of participants (green line), versus the number of participants included the analysis following excluding missing data (orange line). Plot b) shows the percent of missing data by variable across all the interviews.*

did not answer multiple questions. This will be further addressed in the discussion, but as a result of this it is likely that the sample is somewhat biased, and these biases should be considered when interpreting the results.

3.3.5 Model selection

A base candidate model was created which included all the aforementioned covariates (education, body mass index, ethnicity, smoking habits, number of live births, age at menarche, overall health, age at first interview), with the variables of interest being subsequently added to create seven models in total. A list of candidate models is shown in Table 3.1. Sexual frequency and marital status are not included in models with measures of household composition due to a high degree of collinearity. Similarly, the measures of household composition are all included in separate models as each of them capture similar information.

To compare model fit, I utilise Akaike Information Criterion (AIC), which is calculated by:

$$AIC = 2k - 2\ln(\mathcal{L})$$

Where k is the number of model parameters, and $\mathcal{L}$ is the maximum value of the likelihood function in the model (Akaike, 1973). AICs are calculated for every candidate model and then compared to one another, with the best fitting model being the one with the lowest AIC value. The ΔAIC is also calculated, which is the difference between the candidate models AIC and the AIC value of the best fitting candidate model. If the ΔAIC value is ≤ 2, then it indicates that there is still good evidence to support the candidate model, meaning that a candidate model with a ΔAIC of ≤ 2 is almost as good as the best fitting model. A ΔAIC value of between 4 and 7 is taken to indicate the candidate model has considerably less support, and a ΔAIC of greater than 10 indicates there is no support for the candidate model (Burnham & Anderson, 2002). The Akaike weights (w_i) are also calculated and compared to measure model fit. These give the probability that the candidate model is the best among the set of presented candidate models. For example, a w_i of 0.70 would mean that, given the data, the candidate model has a 70% probability of being the best one (Burnham & Anderson, 2002).

Table 3.1. *List of candidate models use in analyses.*

Model	Hypothesis	Covariates
1	Base model	Education + Body mass index + Ethnicity + Smoking habits + Parity + Age at menarche + Overall health + Age at first interview
2	1	Marital status + Base model
3	1	Sexual frequency + Base model
4	1	Sexual frequency + Marital status + Base model
5	2	Whether woman lives with a male partner + Base model
6	2	Whether woman lives with a male + Base model
7	2	Total number of males living in household + Base model

3.4 Results

3.4.1 Descriptive statistics

A total of 3,093 women were included in the baseline sample, with the mean age at this interview being 45.86 (standard deviation [SD]: 2.69). Due to the requirements to be part of SWAN, no one had yet entered menopause, but 46% were in early peri-menopause, and 54% were pre-menopausal. Across the ten years of follow-up interviews used within this study, 1,236 (45%) women experienced a natural menopause at an average age of 52 (SD: 2.60).

At entry to the study, most women were either married or in a relationship (78%), and 68% of women lived with their partner, and the most frequent pattern of sexual activity was weekly (64%). White women were most represented within the sample as 47% of women identified as being such, in addition to the majority of women being educated to above a high school level (some college/technical school: 32%, college degree: 20%, post-graduate education: 23%). The median body mass index was 28 (IQR: 23, 32), and most women reported to having never smoked regularly (57%). Women on average had two children (SD: 1.41), and experienced menarche at the age of 13 (SD: 1.68). I present full descriptive statistics of the baseline cohort in Table 3.2.

3.4.2 Hypothesis 1

To test Hypothesis 1 – that sexual frequency associates with menopause timing – four models were compared (Table 3.3). The best fitting model was Model 3, which includes the covariates and sexual frequency (w_i: 0.64), followed by Model 4 which

Table 3.2. Participant characteristics at the baseline interview. SD = standard deviation; IQR = interquartile range.

	n (%)	**Mean (SD)**	**Median (IQR)**
n	3093		
Age		45.86 (2.69)	46.00 (44.00, 48.00)
Age at first interview		45.86 (2.69)	46.00 (44.00, 48.00)
Menopause status			
Unknown	6 (0.19)		
Post-menopausal	-		
Late-peri	-		
Early-peri	1418 (45.85)		
Pre-menopausal	1669 (53.96)		
Pregnant/breastfeeding	-		
Sexual frequency			
Less than monthly	461 (14.90)		
Monthly	647 (20.92)		
Weekly	1985 (64.18)		
Lives with a romantic male partner			
No	1004 (32.46)		
Yes	2089 (67.54)		
Lives with a male			
No	540 (17.46)		

Table 3.2. Participant characteristics at the baseline interview. SD = standard deviation; IQR = interquartile range.

	n (%)	Mean (SD)	Median (IQR)
Yes	2553 (82.54)		
Total number of males in household		1.55 (1.13)	1.00 (1.00, 2.00)
Marital status			
Divorced/Separated/Single	695 (22.47)		
Married/In a relationship	2398 (77.53)		
Education			
Less than high school	221 (7.15)		
High school	545 (17.62)		
Some college/technical school	999 (32.30)		
College degree	627 (20.27)		
Post-graduate education	701 (22.66)		
Body mass index		28.25 (7.25)	26.57 (22.84, 32.18)
Ethnicity			
Black	859 (27.77)		
Chinese	238 (7.69)		
Japanese	272 (8.79)		
White	1465 (47.37)		
Hispanic	259 (8.37)		
Smoking status			
Never smoked	1774 (57.36)		

Table 3.2. Participant characteristics at the baseline interview. SD = standard deviation; IQR = interquartile range.

	n (%)	Mean (SD)	Median (IQR)
Ever smoked	1319 (42.64)		
Number of live births		2.01 (1.41)	2.00 (1.00, 3.00)
Menarche		12.56 (1.68)	12.00 (12.00, 13.00)
Self-perceived health			
Poor	59 (1.91)		
Fair	344 (11.12)		
Good	894 (28.90)		
Very good	1128 (36.47)		
Excellent	668 (21.60)		

includes the covariates, marital status, and sexual frequency (ΔAIC: +1.12, w_i: 0.36). All other models had ΔAIC of greater than 10, indicating a significantly worse model fit.

In the best fitting model (Model 3), increased sexual frequency associated with a decreased risk of entering menopause, suggesting that women in this study who had sex more frequently experienced a later age of natural menopause (see Figure 3.3). At any given age within the study period, women who had sex monthly were 18% less likely to experience menopause (95% confidence interval [CI]: 0.70-0.96), and those who had sex weekly were 28% less likely to experience menopause (95% CI: 0.63-0.82). Results from this model are plotted in Figure 3.4, where it can be seen that a college education associates with a later age of menopause (HR: 0.71, 95% CI: 0.24-0.96), as does having more live births (HR: 0.96, 95% CI: 0.92-1.00), a higher body mass index (HR: 0.99, 95% CI: 0.98-1.00) and never having smoked regularly (ever smoked HR: 1.24, 95% CI: 1.10-1.39). Being Japanese weakly predicted having a later menopause (HR: 0.81, 95% CI: 0.64-1.01). There was no relationship between self-reported health or menarche and age of menopause.

Table 3.3. *Results from model fitting based on Akaike Information Criterion (AIC) value. The lowest AIC value is deemed to best fit the data, and a ΔAIC of more than two demonstrating a significantly poorer model fit. w_i shows the model probability. The best fitting model has been italicised. See Table 3.1 for model composition.*

Model	K	AIC	ΔAIC	w_i
Hypothesis 1 (Models 1-4)				
Model 1	17	16550.63	17.95	0.00
Model 2	19	16553.48	20.81	0.00
Model 3	*19*	*16532.68*	*0.00*	*0.64*
Model 4	21	16533.79	1.12	0.36
Hypothesis 2 (Models 1, 5-7)				
Model 1	*17*	*16550.63*	*0.00*	*0.38*
Model 5	18	16552.01	1.38	0.19
Model 6	18	16552.12	1.49	0.18
Model 7	18	16551.53	0.90	0.24

In the second-best fitting model, this relationship was maintained (monthly sex HR: 0.79, 95% CI: 0.67-0.93; weekly sex HR: 0.68, 95% CI: 0.58-0.79). Marital status presented a little relationship with menopause timing, with married or partnered women (HR: 1.14, 95% CI: 0.98-1.33) and widowed women (HR: 1.11, 95% CI: 0.80-1.54) experiencing menopause slightly earlier than those who reported being divorced/separated, but not to a significant degree (results from variables of interest are shown in Table 3.4). Full results are shown in Appendix C Table C2.

3.4.3 Hypothesis 2

Four models were again compared against one another to test whether male household presence, as a proxy of male pheromones, associates with menopause timing (Table 3.3). The best fitting model here was the base model (Model 1) that included only the covariates (w_i: 0.38). The inclusion of the different variables

Table 3.4. *Results from Cox regression predicting risk of menopause, showing the hazard ratio and 95% confidence interval from the variables of interest. A hazard ratio of > 1 indicates a greater risk of entering menopause. All models adjust for education, body mass index, self-identified ethnicity, smoking status, number of live births, menarche, and self-perceived health.*

	Model 2	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**
Sexual frequency (ref.: Less than monthly)						
Monthly	-	0.82 (0.70-0.96)	0.79 (0.67-0.93)	-	-	-
Weekly	-	0.72 (0.63-0.82)	0.68 (0.58-0.79)	-	-	-
Marital status (ref.: Divorced/Separated)						
Married/In a relationship	0.97 (0.84-1.11)	-	1.14 (0.98-1.33)	-	-	-
Widowed	1.14 (0.83-1.58)	-	1.11 (0.80-1.54)	-	-	-
Lives with a romantic male partner (ref.: No)						
Yes, lives with a romantic male partner	-	-	-	0.95 (0.84-1.08)	-	-
Lives with a male (ref.: No)						
Yes, lives with a male	-	-	-	-	1.05 (0.91-1.22)	-
Total number of males in household	-	-	-	-	-	1.03 (0.97-1.10)

indicating male household presence all worsened the model fit, but had ΔAIC values of less than 2, suggesting that the fit was not significantly poorer. The next best fitting model after Model 1 was Model 7, which included the total number of males in the household (ΔAIC: +0.90, w_i: 0.24), followed by Model 5 (whether the woman

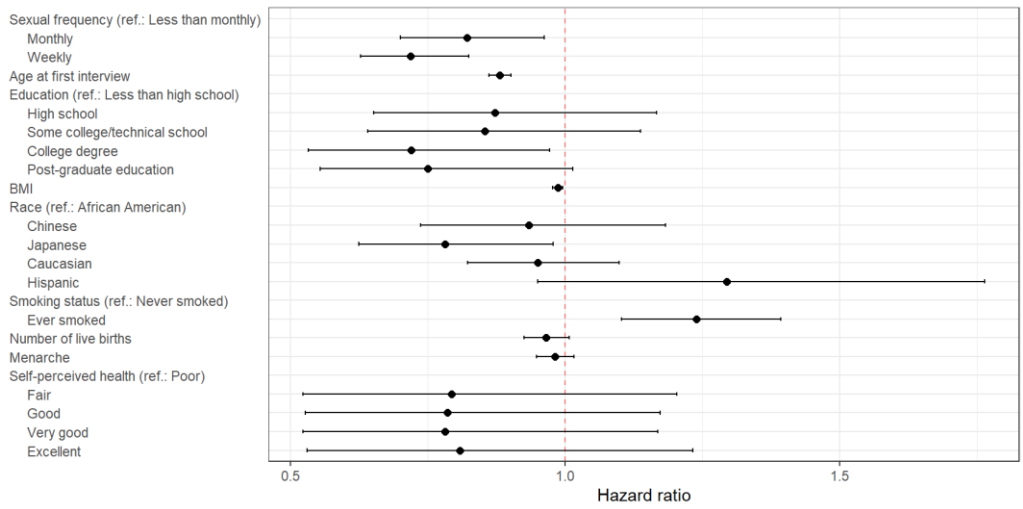

Figure 3.3. *Forest plot showing the results from Cox regression predicting the risk of entering menopause. Hazard ratios and 95% confidence intervals from the best fitting model (Model 3) are shown. A hazard ratio of >1 indicates an increased risk of entering menopause.*

lives with a male partner, ΔAIC: +1.38, w_i: 0.19), and then Model 6 (whether the woman lives with any male, ΔAIC: +1.49, w_i: 0.18). In each of these models, male-female cohabitation (HR: 1.05, 95% CI: 0.91-1.22) and the number of males in the household (HR: 1.03, 95% CI: 0.97-1.10) predicted an earlier age of menopause; while cohabiting with a romantic male partner predicted a later menopause (HR: 0.95, 95% CI: 0.84-1.08). However, all of these effect sizes are negligible and non-significant (Table 3.4). Full results from all models are shown in Appendix C Table C2.

3.4.4 Post-hoc analysis

To strengthen support for the notion that sexual frequency affects menopause timing, I also conduct a post-hoc analysis in which I lag the value of sexual

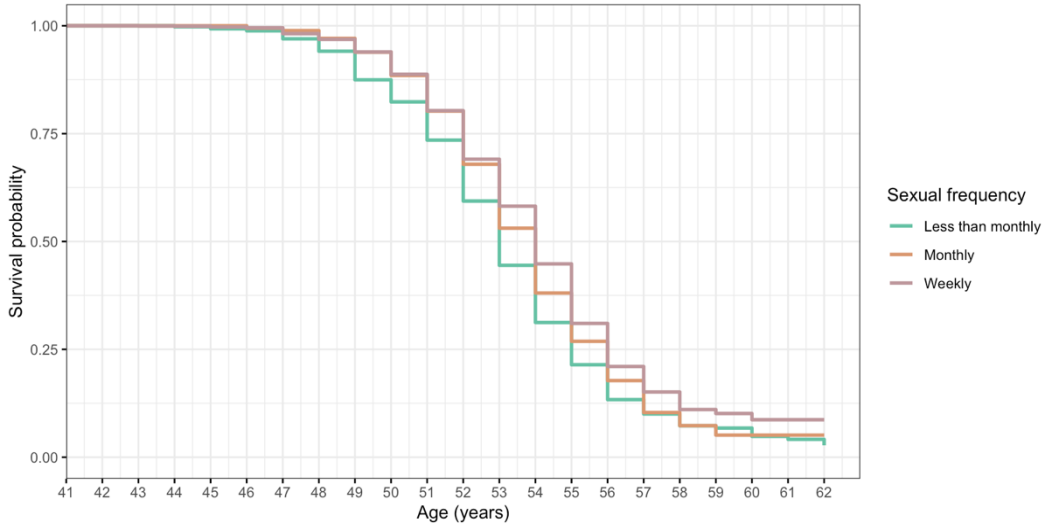

Figure 3.4. *Kaplan-Meier curves of the survival function stratified by sexual frequency.*

frequency, meaning that menopause status (or risk of menopause) is being predicted by sexual frequency in the prior year. If menopause timing is affected by sexual frequency approaching menopause, then I would expect that sexual frequency measured one year prior to the measurement of menopause status would also be a good predictor of the risk of menopause. Hence, I replicate the two best fitting models using a lagged version of the sexual frequency variable. As a further sensitivity analysis, I also replicate models using sexual frequency at the baseline interview (and therefore a time invariant measure) to predict menopause timing. The baseline measure of sexual frequency would be predicted to have no relationship with menopause timing, as the women in the sample experienced menopause at varying age distances from the baseline wave, and therefore while the baseline measure of sexual frequency may be just before menopause for some women, for other women this measure of sexual frequency may be years prior to menopause. Therefore, this measure of sexual frequency is essentially a random measure relative to menopause timing.

Results from these post-hoc analyses are presented in Appendix C Table C3. In the lagged models, sexual frequency is still a significant predictor of menopause occurring, with women who have weekly sex being at the lowest risk of menopause. Conversely, in the analyses looking at sexual frequency from the baseline interview (i.e., a random measure of sexual frequency relative to time until menopause), sexual frequency no longer predicted menopause timing well, suggested that it is sexual frequency in the run-up to menopause that might influence menopause timing.

3.5 Discussion

Women typically stop reproducing many years prior to menopause, even in societies where contraceptives are not the norm (Towner et al., 2016), but nonetheless, menopause signals permanent reproductive cessations and means that women no longer have the option to increase their fitness directly. Multiple lifestyle factors have been found to predict menopause timing; however, they are seldom discussed from an evolutionary framework. Here, I have focussed on unpacking the relationship between marital status and age of natural menopause through testing an original hypothesis that sexual frequency acts as a bio-behavioural mediator which signals the possibility of getting pregnant. In addition to this, I test an existing hypothesis that

married women enter menopause later due to exposure to male pheromones (Sievert et al., 2001).

To test the latter hypothesis, three measures of male-female cohabitation were used as a proxy of male pheromones. As stated previously, it would have been preferable to use a measure such as menstrual cycle stability to predict menopause timing, however there was a great deal of missing data present in this variable. Further, as shown in Figure 3.1, there is little relationship between male-female cohabitation and cycle regularity. It was initially predicted that marital status associates with menopause timing, because the presence of a man, and therefore male pheromones, in the household result in a more regular menstrual cycle, which itself results in a later menopause (Whelan et al., 1990; Sievert et al., 2001); but it seems this proposal has little empirical support within this sample. As well as this issue, the hypothesis may have other flaws, as currently there is no conclusive evidence either that humans produce pheromones, or that they are capable of detecting them (Wyatt, 2015). Nonetheless, this is the first study addressing the pheromonal hypothesis since it was originally proposed, and while male household presence is merely a proxy of pheromones – and it may be that the hypothesis is moot due to the absence of evidence for human pheromones and the lack of relationship between male-female cohabitation and menstrual cycle regularity – it is an indication that the relationship between marital status and age of natural menopause is not capturing the effect of male pheromones on the menstrual cycle.

This study did not replicate findings from previous research that married women enter menopause later. In fact, following complete adjustment, the converse was found with women who were married or in a relationship having an increased risk of entering menopause compared to divorced, separated, and single women (though this relationship was not statistically significant). Conflicting results regarding marital status' effect on age of natural menopause have been found elsewhere (Reynolds & Obermeyer, 2005), and one reason for this may be the way in which the researcher chooses to code the variable. In this analysis, romantic partnerships that may not have been acknowledged in previous studies due to having not been formalised by a marriage ceremony (e.g. cohabiting but unmarried), were taken into account. In addition, some prior studies have not included marital status as time-varying, and dichotomised the variable as 'ever married' or 'never married' (Parazzini

et al., 1992). Hence, the responsive way in which this study coded marital status may account for the difference in results.

Another reason for this difference may be the cultural setting of previous studies. For example, research originating from Iran found that ever married women experience a later menopause than those who never married (Ayatollahi et al., 2005). However, in the case of Iran where dowry is still common practice, it means marriage is contingent upon family wealth (Sadeghi-Fasaei, 2017). Therefore, the effect of marital status on age of natural menopause would be confounded by a woman's socioeconomic position, which itself would relate to other aspects of her health and life history that have been associated with menopause timing – such as body mass index and age at menarche – therefore resulting in a significant difference in age of natural menopause between those who have and have not been married. Within Iran, sex outside of marriage is prohibited both legally and socially, meaning marital status would be highly correlated with sexual behaviour (Farahani et al., 2011). Hence, it may be that previous studies identifying married women enter menopause later are simply capturing the effect of health and lifestyle patterns that themselves associate with both marital status and menopause timing, rather than demonstrating that marital status itself is a cross-cultural correlate of age of natural menopause.

Evidence supporting the notion that age of natural menopause associated with sexual frequency during the pre- and peri-menopause was found. Even following complete adjustment, results still indicated that women who engage in sexual activity weekly or monthly have a lower risk of entering menopause relative to those who report having some form of sex less than monthly. While I am not directly making causal claims, the post-hoc lagged analysis (section 3.3.4) adds weight to the idea that sexual frequency when approaching have some influence over menopause timing. The fact that a lagged version of sexual frequency is still a good predictor of menopause timing suggests that results are not just a spurious relationship, and not the result of reverse causality (e.g. women who are post-menopausal having less sex). Causal claims should always be made with caution, but this analysis is evidence that a behaviour in the approach to menopause might affect the timing at which it occurs. But why would increased sexual frequency be expected to associate with a later menopause? Interpreting these resulted from a fitness-maximising framework, it may be that the physical cues of sex signal to the body that there is a possibility of becoming pregnant, and therefore a trade-off may occur

between continued energetic investment in ovulation and reproductive cessation. The maintenance of menstrual function is highly energetically costly, plus during ovulation the woman's immune function is impaired making the body more susceptible to disease (Alvergne & Tabor, 2018; Lorenz et al., 2018). Hence, if a pregnancy is unlikely due to a lack of sexual activity, then it would not be beneficial to allocate energy to a costly process, especially if there is the option to invest resources into existing kin (Hawkes et al., 1998; Sear & Mace, 2008). On the other hand, if the chance of becoming pregnant is high due to engaging in regular sex, then the costs of menopause may outweigh the possible fitness benefits. In other words, if she is still able to increase her direct fitness, then it may be better to maintain the function of her menstrual cycle for slightly longer. Of course, it is unusual for women to reproduce right before menopause (Towner et al., 2016), but given the possible fitness benefits it could pay off to maintain ovulatory function for slightly longer. Previous research has shown that women sexual inactivity is associated with irregular menstrual cycles, and lower levels of estrogen and mid-cycle luteinising hormone, with evidence that sexual activity, reproductive hormones, are closely intertwined (Prasad et al., 2014). As regular menstrual cycles have been found to associate with a later menopause, it may be that, proximately, menopause is delayed by increased sexual frequency in this way.

It should be noted that, despite those organising SWAN making efforts to target individuals in under-researched groups (e.g. ethnic minority groups), the women in the study are not representative of the general US population. The sample is largely white (with attrition being higher in non-white groups), highly educated, and healthy, which is likely reflected in the fact that many experienced menopause at a relatively old age (see Figure 3.3). While the retention rate of SWAN is generally high, as stated women of certain demographics are more likely to drop out, including those who are not white and those of poorer health (Harlow et al., 2017) This bias is also likely reflected in the patterns of missing data throughout, with clusters of missing data generally coming from the same few women. While previous research has shown that results of analyses using SWAN's data are comparable regardless of whether a complete case analysis or imputed data is used (Harlow et al., 2017), the possible biases that occur from certain demographic groups being more likely to not respond to questions/interviews and/or leave the study all together (e.g. those on a

lower income, less well educated) (Watson & Wooden, 2009) means that how generalisable these results are could be brought into question.

3.6 Conclusion

In this chapter, I have demonstrated that sexual frequency is associated with age of natural menopause, and also tested the relationship between male pheromone exposure and menopause timing. While only a proxy of male pheromones was used, no association between male cohabitation and menopause timing was found, indicating that male-female cohabitation is not the driving force behind the relationship between marital status and menopause timing, and that pheromones likely do not influence age of natural menopause. I did not replicate the findings from previous research showing that simply being married is associated with a later age of natural menopause, most likely due to the variable cultural and temporal settings of previous studies. However, I did demonstrate that increased sexual frequency during the pre- and peri-menopause decreased the risk of experiencing menopause. While causation cannot be conclusively inferred, I hypothesise that this relationship is the result of an adaptive trade-off relative to the likelihood of pregnancy when approaching menopause. Of course, menopause is an inevitability for women, and there is no behavioural intervention that will prevent reproductive cessation; nonetheless, these results are an initial indication that menopause timing may be adaptive in response to sexual behaviour, and can possibly be used within evolutionary models.

CHAPTER FOUR

Residence patterns and menopause

4.1 Chapter summary

This chapter looks at the relationship between post-marital residence pattern, menopause symptoms, and age of menopause. Using data from four ethnic groups in China, I test an existing hypothesis that menopause symptoms and timing are the product of intragenomic conflict between maternally and paternally inherited genes, with the outcome of such conflict predicted to be contingent on the ancestral post-marital residence pattern of the female (Ubeda et al., 2014). The intragenomic conflict model predicts that ancestral patrilocality would be associated with less intragenomic conflict, which in turn predicts a shorter, less symptomatic peri-menopause, which terminates in a later menopause. The findings presented in this chapter offer no support for this hypothesis. Rather, I find that current residence pattern (rather than the woman's ancestral residence pattern) predicts menopause symptoms, with current patrilocality associating with reporting of worse menopause symptoms. No relationship between residence pattern and menopause symptom duration or menopause timing was found. I propose that the differences in menopause symptoms between patrilocal and duolocal households are the result of differing levels of social support and sexual or household conflict relative to residence pattern, rather than intragenomic conflict.

This chapter has been published in *Ecology and Evolution* (Yang et al., 2019), and co-first-authored with Yuping Yang, who is currently a PhD student at Lanzhou University and was part of the group who collected the data used in this chapter's analyses, and my supervisor Ruth Mace. I was not involved in the data collection for this chapter, and I joined the project after the fieldwork had been carried out, meaning I was not responsible in any part of the designing of the questionnaire or the data collection. Once I joined the project, Yuping and I discussed the possibility of testing the hypothesis presented in this chapter together using the data collected by her and the rest of the research group in Lanzhou. I was then responsible for carrying out all analyses and interpreting the results. I also led the writing of the published version of this chapter.

4.2 Introduction

Cross-culturally, there is a wide range of dispersal patterns following marriage, with such patterns structuring the relatedness of different individuals to the rest of the group, which can influence patterns of cooperation and conflict (Johnstone & Cant, 2010). Sex-specific dispersal at marriage means that male and female relatedness to their residential group will vary with age, which means they may experience different evolutionary pressures over their lifespan, and it is thought that these evolutionary pressures may have contributed to the evolution of the female menopause. As highlighted in Chapter 1, menopause is still considered an evolutionary puzzle; however, many agree that post-reproductive women may be able to increase their indirect fitness through consanguineal cooperative breeding (e.g. grandmothering) when there are age-specific relatedness asymmetries between the women in the group (Hawkes et al., 1998; Sear et al., 2000; Hawkes, 2003; Hawkes, 2004; Cant & Johnstone, 2008; Johnstone & Cant, 2010; Kim et al., 2012), Further, some have argued that dispersal patterns would have been crucial to setting up the reproductive conflict that facilitated the evolution of the menopause itself (Cant & Johnstone, 2008; Johnstone & Cant, 2010). Here, it is thought that under a patrilocal post-marital residence system, the age-specific patterns of relatedness amongst women would result in reproductive conflict that would favour older women ceasing reproduction and investing in existing kin (see Chapter 1 for a more detailed explanation). As such, it was predicted that menopause evolved under a residence system where females disperse for reproduction (Cant & Johnstone, 2008). Building on this, it was also recently modelled that the ecological conditions hypothesised to have driven the evolution of menopause might also drive intragenomic conflict over reproductive schedules, which in turn may explain the negative symptoms experienced by many women throughout the menopausal transition (Ubeda et al., 2014). This theory will form the basis of this chapter, and is elaborated upon below.

It has been proposed that a woman's experience during the menopausal transition, and the age at which she reaches reproductive cessation, may be the result of intragenomic conflict. Intragenomic conflict refers to the evolutionary phenomenon in which selection favours different levels of expression for maternally and paternally inherited genes at the same locus in the same individual. In the case of menopause, a model by Ubeda et al. (2014) suggests that genes at loci related to

fertility will have parent-of-origin differential expression. Specifically, assuming that dispersal is female-biased, that paternally inherited genes will favour an earlier menopause for the benefit of the woman's social group due to the inclusive fitness gains associated with post-reproductive care (in line with the Grandmother Hypothesis), while maternally inherited genes will favour a later menopause for the benefit of her own direct fitness. This is because, when there has been generations of female dispersal, women will be more related to the group on average through their paternally inherited genes compared to their maternally inherited genes. As the maternally inherited genes are less related to the group on average, the 'best' way for them to increase their fitness is to continue reproducing directly (rather than investing in existing kin), and to experience menopause later. Conversely, the paternally inherited genes would benefit from an earlier menopause so that the social group benefits from increased levels of post-reproductive grandparental care. Further to this, Ubeda et al. (2014) predict that the conflict that occurs as a result of the different optimal ages of menopause for the paternally and maternally inherited genes leads to genomic imprinting, which explains the unpleasant symptoms that occur during peri-menopause. Specifically, they predict that genes controlling the stock of follicles will be maternally expressed (Haig, 1996), whereas others controlling the duration of the menstrual cycle will be maternally expressed at a constant rate while the paternal expression would be stochastic. Moreover, the model predicts that the optimal ages of menopause for both the maternally and paternally inherited genes are contingent upon how biased the female dispersal is within the population. Holding male dispersal constant, the higher the female dispersal is the later the age of menopause, as maternally inherited genes related to fertility are predicted to be expressed. Finally, when there are high levels of female dispersal, the optimal ages of menopause for the paternally and maternally inherited genes are more similar, as the difference in relatedness between the maternally and paternally inherited genes declines with age (as is the case in the Reproductive Conflict Hypothesis (Cant & Johnstone, 2008)), and so there is a shorter window of conflict between the two genes, resulting in a shorter peri-menopause. In sum, the predictions of this model are that greater rates of female dispersal will result in a less symptomatic, shorter peri-menopause, and later menopause.

This model has not yet been directly empirically tested; however, some have taken evidence of ethnic differences in aspects of the menopausal transition as

indirect evidence in favour of it (Ubeda et al., 2014). Women of Japanese descent in the US typically report less vasomotor symptoms (e.g. hot flashes) and a later age of natural menopause, whereas African American women experience the converse (Gold et al., 2000; Avis et al., 2001b; Gold et al., 2001; Avis et al., 2005b; Im, 2009). Assuming that, ancestrally, there were ethnic differences in post-marital residence pattern, this theory would contribute to explaining why we see these different experiences of the menopausal transition. Some ethnographic reports state that Japan was traditionally patrilineal and patrilocal (Befu, 1968), offering some support for the hypothesis. However, the ancestry of African Americans is diverse, and there are conflicting reports on the ancestral residence pattern of various African groups (e.g. opposing findings on Bantu residency see: Opie et al., 2014; Hage & Marck, 2015). In addition to this, all groups would have experienced a great deal of admixture in their past, meaning it would be hard to categorise any population by residence pattern.

As the idea that intragenomic conflict drives differences in the menopausal transition has not been formally tested, in this chapter I empirically test its predictions which are that high rates of female dispersal should associate with a less symptomatic peri-menopause (H1), a shorter peri-menopause (H2), and a later menopause (H3). To do so, I use primary data collected from Southwestern China. This area was chosen due to the variation in residence patterns, which determine the degree of female dispersal at marriage (Wu et al., 2015b). In most Chinese kinship systems males never disperse, but there is some variation in female dispersal: in patrilineal groups females disperse at marriage, thus displaying patrilocality; whereas in matrilineal groups they usually stay in their natal households and live under a duolocal residence pattern, where neither males nor females disperse. However, there is always variability in residence patterns around the norm (Ly et al., 2018), and many women who are ancestrally duolocal no longer live in this way due to cultural change in the region (Mattison, 2010; Ji et al., 2016). Therefore, in addition to collecting data on ancestral residence pattern, data on current residence pattern was collected as well. This second variable allows us to attempt to capture whether the ancestral or current residence pattern is more influential over the menopausal transition. The intragenomic conflict hypothesis predicts that the former would be most important, whereas if conditions specific to current residence pattern (e.g. different degrees of sexual and social conflict between patrilocal and duolocal groups

(Leonetti et al., 2007)) are more important, then menopause symptoms may be more related to current living arrangements.

4.3 Materials and methods

4.3.1 Study area

Data collection was carried out over two field sessions in the Sichuan Province of China in 2018 and 2019. Consent was sought from each individual interviewed and from local People's Government at each site, and the research was approved by Lanzhou University Life Sciences and UCL Research Ethics committee. Two sites were visited: Lugu Lake and Daofu County. In each of these sites, different ethnic groups were interviewed, with Mosuo, Han and Yi women being interviewed around Lugu Lake, and Zhaba women in Daofu County. While the Han and Yi typically display a patrilocal residence pattern, the Mosuo and Zhaba traditionally have a duolocal post residence pattern. It is thought that these groups have likely been living the way they do for thousands of years (Dong et al., 2008), which means they serve as good models for testing the intragenomic conflict hypothesis of menopause.

As stated, the Mosuo and Zhaba display an uncommon duolocal residence pattern coupled with matrilineal descent. This means neither sex leaves the natal group for marriage and reproduction, and descent is traced through the female line. Here, males and females live in their natal households that comprise many generations of genetically related family members (Ji et al., 2013; Wu et al., 2015b; He et al., 2016). Further, as an alternative to cohabiting marriage, they also engage in the practice of *zǒu hūn* ('walking marriage', 走婚) where men only visit their wife or girlfriend during the night (Cai, 2001). Men have little or no financial obligations to their spouses or children, and they tend to invest heavily in their sisters' offspring instead of their own (He et al., 2016), possibly due to the paternity uncertainty that results from living separately from your partner (Mattison et al., 2019). A family planning policy established in the 1980s meant that marriage became a requirement for reproduction, and therefore partnerships in the Mosuo and Zhaba are slightly more formal now than they were in previous years; however, in most cases, the husband and wife still live apart (Ji et al., 2016; Thomas et al., 2018). In contrast with the Mosuo and Zhaba, the Han and the Yi display patrilocality accompanied by patriliny where descent is traced down the male line, and women leave their natal home to join that of their husbands' kin at marriage.

Residence patterns are always somewhat flexible (Ly et al., 2018), and, amongst the Mosuo, Zhaba, Han and Yi, they have become more fluid in recent years due to policy changes and increased tourism (Mattison, 2010). For example, while the Mosuo are traditionally duolocal, there is a proportion of individuals who no longer adhere to this lifestyle. Therefore, as there is a difference between current and ancestral residence patterns, I am able to compare the relationship between their ancestral residence patterns (based on ethnic group) and current residence patterns (how the individual woman is currently living). Based on Ubeda et al. (2014)'s model, it is predicted that ancestral residence patterns (i.e. ethnic group) should associate with aspects of the menopausal transition. However, by also looking at current living arrangements it allows me to consider whether something else might be responsible for the diversity observed throughout the menopausal transition.

4.3.2 Data collection

Data were collected using a standard demographic survey, which was conducted in eight villages around Lugu Lake and five villages in Daofu County. All houses in these areas were visited, and in households that agreed to participate all adult women were interviewed. Demographic data was collected, including information on their ethnicity, current living arrangements, marital status, fertility status, height, weight, financial security, and number of children. The MRS (see Appendix B Figure B1) was also used to collect data on menopause symptoms (see Chapter 2 for more detail on data collection) (Heinemann et al., 2003). The MRS measures a total of 11 symptoms, with each symptom being rated by the woman according to its severity using a Likert scale ranging from 0 to 4 (0 = none; 1 = mild; 2 = moderate; 3 = severe; 4 = very severe). These symptoms can be divided into three sub-groups: psychological symptoms (depression, irritability, anxiety, exhaustion), urogenital symptoms (sexual problems, bladder problems, vaginal dryness), and somato-vegetative symptoms (hot flushes, heart discomfort, sleep problems, joint and muscular discomfort). Using the measures derived from the Likert scale, an overall symptoms score can be calculated (ranging from 0 to 44), in addition to individual symptom scores (psychological: 0 to 16; urogenital: 0 to 12; somato-vegetative: 0 to 16). Symptom reporting could either be current or retrospective, with women who had already passed through the transition being asked how severe their symptoms were when they were experiencing peri-menopause, and women currently in peri-menopause being asked about their

current experience. This means when modelling symptom severity in peri-menopausal and post-menopausal women slightly different things will be captured: for post-menopausal women, their memory of the menopausal transition is being modelled, whereas for peri-menopausal women it is their current experience.

4.3.3 Variables

4.3.3.1 Characteristics of menopausal transition

In order to test the intragenomic conflict hypothesis of the menopausal transition, three variables of interest were collected: severity of peri-menopause symptoms, the duration of the peri-menopause symptoms, and the age of natural menopause.

The MRS was used to capture information regarding symptom severity. Though the MRS asks questions about psychological, urogenital, and somato-vegetative symptoms (see Chapter 2), here I only utilise data pertaining to somato-vegetative symptoms. This is because the hypothesis being tested in this chapter models the effect of residence on vasomotor symptoms, which are symptoms that occur due to the constriction or dilation of blood vessels. Typically, vasomotor symptoms are experienced through hot flashes and night sweats. However, the MRS measures somato-vegetative symptoms, includes hot flashes as well as heart discomfort, sleeping issues, and joint and muscle complaints. While these are not necessarily usually classes as vasomotor symptoms, all of these symptoms have been found to be both exacerbated by, and symptomatic of, vasomotor issues (Avis et al., 2001a; Ashraf et al., 2015; Islam et al., 2016; Chung et al., 2018). Therefore, to adhere to the assumptions of the model as much as possible, the analysis is limited to modelling somato-vegetative symptom severity. This was done by summing the responses to the relevant questions (questions 1, 2, 3 and 11 in the MRS), which gives a score ranging from 0 to 16, where a higher value indicates more severe symptoms.

Symptom duration was measured by asking women at what age or in what year they began experiencing menopause symptoms. If they had already entered menopause then their age at menopause was used as their symptom finishing date, and if they had not yet entered menopause and were experiencing symptoms, then they were coded as ongoing. I acknowledge that menopause symptoms are not confined to the pre- and peri-menopause and can often persevere following the termination of fertility (Avis et al., 2015); however, the hypothesis of interest

specifically models symptoms up until the final menstruation. Therefore, while some women did report that they were still experiencing symptoms following the cessation of fertility, the duration of their symptoms was only included until menopause.

Finally, age of natural menopause was measured by asking women whether or not they were still experiencing regular menstruation, and when they had experienced their last period. If a woman had not experienced a period for 12 months or more in the absence of extenuating factors (e.g. pregnancy, breastfeeding), then she was considered to have experienced menopause (Kirchengast & Ruhli, 2013). Furthermore, if a woman was reporting irregular periods then she was classed as being peri-menopausal, and anyone with regular periods or reproducing (i.e. pregnant) as pre-menopausal.

4.3.3.2 Residence pattern

The intragenomic conflict hypothesis predicts that more female dispersal will result in a menopausal transition characterised by less severe symptoms that occur for a shorter period of time and terminate in a later menopause. In order to test this, two variables are used to indicate the individual's residence pattern. Firstly, a woman's ancestral residence pattern, which was derived from self-reported ethnicity. Here, women who reported being Mosuo or Zhaba were coded as having a duolocal residence pattern, while those who identified as Han or Yi were coded as patrilocal. In addition to these groups, a small number of women reported having mixed ancestry. In the context of the model of interest, it can be seen that the ancestrally patrilocal groups would have experienced high rates of female dispersal, whereas the duolocal groups would have been characterised by low rates of female dispersal. Secondly, I also included a second variable of current residence pattern, which captures the way the woman currently lives regardless of her ethnicity. Here, women were coded as either living with their natal household or away from their natal household.

4.3.3.3 Covariates

Covariates were held constant in each analysis and selected based on existing literature on the demographic correlates of menopause symptoms and timing, and also based on the data available. These included parity, which has been associated with worse menopause symptoms and a later menopause (Parazzini & Progetto

Menopausa Italia Study, 2007; Li et al., 2012; Mishra et al., 2017; Wang et al., 2018); whether the woman was financially secure over the past year (as a measure of socioeconomic position/financial stress), with a lower socioeconomic position being associated with worse menopause symptoms and an earlier menopause (Lawlor et al., 2003; Schoenaker et al., 2014); body mass index, which is thought to have a U-shaped relationship with age of menopause and a positive correlation with menopause symptoms (Parazzini & Progetto Menopausa Italia Study, 2007; Li et al., 2012; Sapre & Thakur, 2014; Tao et al., 2015; Ahuja, 2016; Maru et al., 2016; Wang et al., 2018; Zhu et al., 2018); and smoking habits, which predict an earlier menopause and worse menopause symptoms (Gold et al., 2000; Gold et al., 2001; Ozdemir & Col, 2004; Ayatollahi et al., 2005; Nagel et al., 2005; Parazzini & Progetto Menopausa Italia Study, 2007; McKnight et al., 2011; Sapre & Thakur, 2014; Tao et al., 2015; Bjelland et al., 2018; Wang et al., 2018). In addition, age was included as a covariate when modelling symptom duration and symptom severity; and menopause status was adjusted for when looking at symptom severity, to control for the possibility that retrospective accounts of menopause may differ from current reporting (see Appendix D Tables D1-D3 for model composition).

4.3.4 Analyses

Different datasets with different sample sizes were used for each analysis (see Table 4.1). This is in part due to data being limited as a result of fieldwork constraints, in addition to the fact that different samples of women are required to test each hypothesis. When looking at symptom severity, only women who were peri-menopausal and postmenopausal were included in analyses. For models predicting symptom duration, women who could not recall when they started experiencing menopause symptoms or had never experienced menopause symptoms were excluded when looking at symptom duration. No women were removed from the dataset when looking at menopause timing. As there are varying sample sizes that included different women, it means the results from each analysis are not necessarily directly comparable to one another. Furthermore, the different sizes of dataset mean that each analysis has different levels of statistical power. With alpha set at 0.05 and power at 0.80, the datasets used to look at age of natural menopause (n = 876), symptom severity (n = 445), and symptom duration (n = 83) have enough power to capture small, medium, and large effect sizes, respectively (Cohen, 1992).

To test the hypothesis that women from ancestrally patrilocal societies should experience less severe menopause symptoms, Poisson regression was used to account for the fact that the outcome variable is a count variable with a non-normal distribution (Figure 4.1). To look at whether ancestral patrilocality predicts a shorter duration of menopause symptoms and a later menopause, Cox proportional hazards models were used to calculate a HR for the risk of the peri-menopause ending, and menopause occurring, respectively (Cox, 1972). To test if patrilocality predicts a shorter peri-menopause, symptom duration in years was used as the time-scale, with women being left censored at $t = 0$. Women were right censored if they were still reporting menopause symptoms when the data were being collected, with their symptom duration stopping once they entered menopause. When looking at whether patrilocality associates with an earlier menopause, the age of the woman was used as the time-scale (Korn et al., 1997), with women being left censored at the age of 20, meaning that the model began at this age, rather than measuring risk from birth. This is because women only become at risk of menopause once they have started menstruating and, given the mean age of menarche in China is 12 (Lei et al., 2021), it can be assumed that women will have begun menstruating by the age 20. Further, by starting the analysis at this age, it allows for the retention of younger right censored women (i.e. women in their 20s and 30s who have not yet experienced menopause) who would have otherwise been lost if the analysis was left censored at 40, for example. Women who had not yet entered menopause (pre-menopause and peri-menopause) or ceased fertility for another reason (e.g. pregnancy) were right censored.

When testing each hypothesis, a univariable analysis was initially carried out. A model just containing the covariates for each hypothesis was then made, with ancestral and current residence pattern subsequently being added to the model of covariates together, and then individually (see Appendix D Tables D1-D3 for model composition). Models were then compared based on AIC value, in which the model with the lowest AIC value is the best fit for the data, with a ΔAIC value of greater than two indicating a significantly poorer model fit (see Chapter 3 for more detail) (Burnham & Anderson, 2002). All analyses were carried out in *R* (version 4.0.3) using the packages *AICcmodavg*, *survival*, and *survminer* (Therneau, 2015; Kassambara & Kosinski, 2018; Mazerolle, 2020; R Core Team, 2020), with *ggplot2* being used for all visualisations (Wickham, 2016)

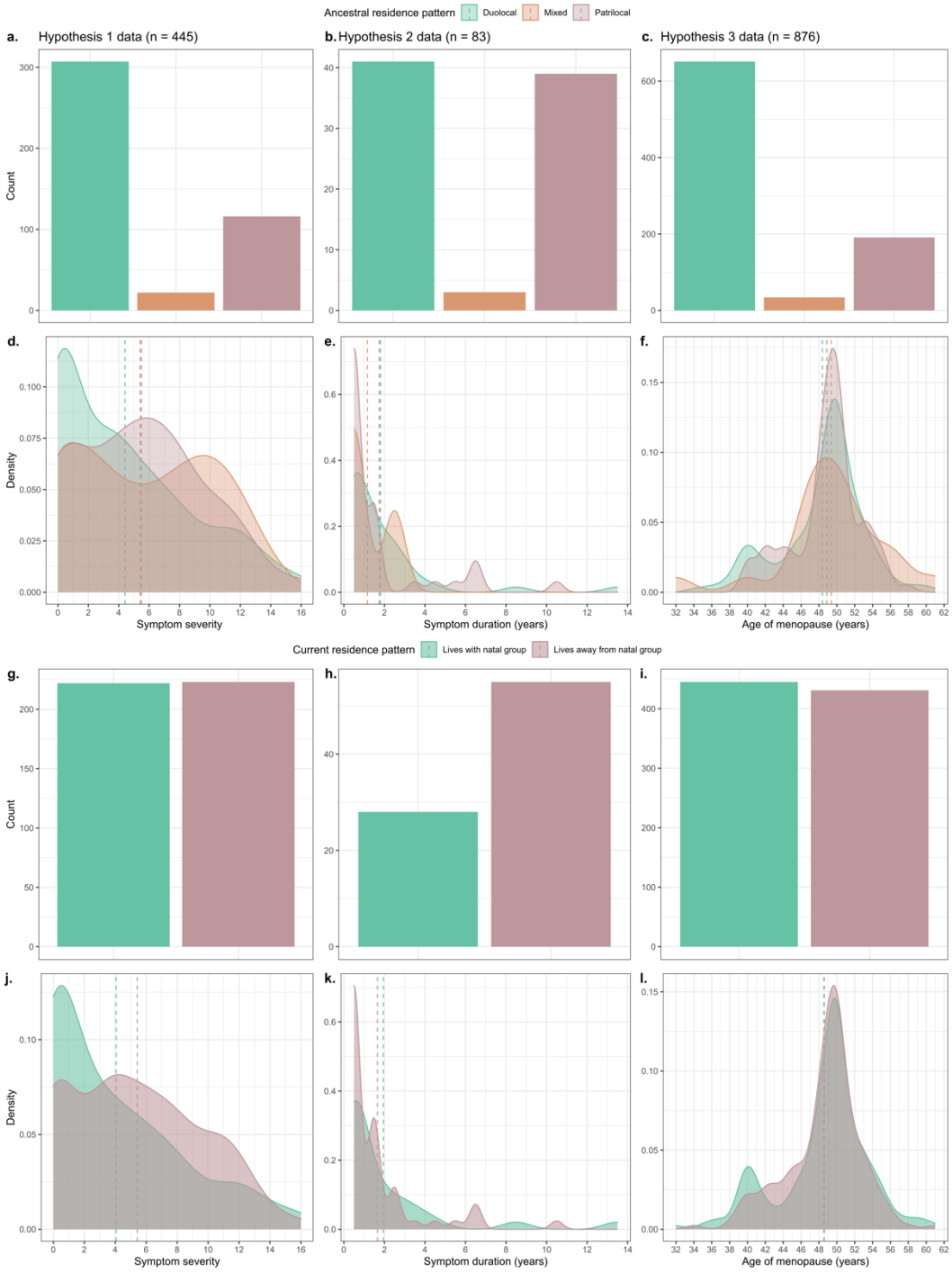

Figure 4.1. *Bar and density plots showing the relationship between the variables of interest within each dataset. a) and d) show symptom severity by ancestral residence pattern; b) and e) show symptom duration by ancestral residence pattern; c) and f) show menopause timing by ancestral residence pattern; g) and j) show symptom severity by current residence pattern; h) and k) show symptom duration by current residence pattern; and i) and l) show menopause timing by current residence pattern.*

4.4 Results

4.4.1 Hypothesis 1. More female dispersal associates with a less symptomatic peri-menopause

To test whether patrilocality associates with a less symptomatic peri-menopause, data from 445 women were used. In this sample, the mean age was 60.49 (SD: 9.06), and the women were primarily post-menopausal (90%), meaning that the majority of the symptom reporting was retrospective. The sample here was biased towards women who are ancestrally duolocal (69%; Figure 4.1a), and these women reported less severe menopause symptoms (mean: 4.41, SD: 4.33) than women who were patrilocal (mean: 5.49, SD: 4.09) or of mixed descent (mean: 5.41, SD: 4.52) (Figure 4.1d). The majority of women in the sample had never smoked (93%); however, a larger proportion of ancestrally patrilocal women reported having ever smoked (16%) than women from duolocal (4%) or mixed ancestry (4%). Full descriptive statistics are presented in Table 4.1.

Univariable Poisson regression indicates that patrilocality both currently (IRR: 1.34, 95% CI: 1.23-1.46) and ancestrally (IRR: 1.24, 95% CI: 1.13-1.37) associates with worse menopause symptoms. However, following complete adjustment, ancestral residence is no longer a strong predictor of menopause symptoms (IRR: 1.06, 95% CI: 0.94-1.18); but women who live away from their natal group still report worse menopause symptoms (IRR: 1.26, 95% CI: 1.14-1.39). Model fitting (Table 4.2) shows that the model just including current residence pattern (in addition to the covariates) best fits the data, and that the inclusion of ancestral residence pattern worsens the model fit. Based on AIC value, the model just including the covariates was the worst fit. In the best fitting model, women who lived away from their family were predicted to report worse menopause symptoms (IRR: 1.28, 95% CI: 1.17-1.40). Results also show that worse menopause symptoms associate with having more children (IRR: 1.08, 95% CI: 1.06-1.11) and experience of financial insecurity in the past year (IRR: 0.81, 95% CI: 0.74-0.89). Full model results for this hypothesis are presented in the Appendix D (Table D1), and best model results displayed graphically in Figure 4.2.

Though the model of interest only offered predictions for vasomotor symptoms, I also carry out a post-hoc robustness test looking at how severe urogenital and psychological symptoms are relative to current and ancestral residence pattern. Results from this analysis are presented in Table 4.3, and the modelling

Table 4.1. *Descriptive statistics of the datasets used for each hypothesis, showing n and % or mean and standard deviation*

	Hypothesis 1 data	**Hypothesis 2 data**	**Hypothesis 3 data**
n	445	83	876
Somato-vegetative symptom score	4.74 (4.30)	-	-
Urogenital symptoms score	1.08 (1.92)	-	-
Psychological symptoms score	1.09 (1.93)	-	-
Symptom duration (years)	-	1.75 (2.32)	-
Age of menopause (years)	-	-	48.57 (4.54)
Age	60.49 (9.06)	60.24 (7.17)	51.04 (12.85)
Menopause status			
Pre-menopausal	-	-	446 (50.9)
Peri-menopausal	46 (10.3)	13 (15.7)	46 (5.3)
Post-menopausal	399 (89.7)	70 (84.3)	382 (43.6)
Don't know	-	-	2 (0.2)
Current residence pattern			
Lives with natal group	222 (49.9)	28 (33.7)	445 (50.8)
Lives away from natal group	223 (50.1)	55 (66.3)	431 (49.2)
Ancestral residence pattern			
Duolocal	307 (69.0)	41 (49.4)	651 (74.3)
Mixed	22 (4.9)	3 (3.6)	34 (3.9)
Patrilocal	116 (26.1)	39 (47.0)	191 (21.8)
Parity	3.86 (1.92)	3.99 (1.82)	3.24 (1.73)
Experienced financial difficulty in the past year			
Yes	266 (59.8)	57 (68.7)	486 (55.5)

Table 4.1. *Descriptive statistics of the datasets used for each hypothesis, showing n and % or mean and standard deviation*

	Hypothesis 1 data	Hypothesis 2 data	Hypothesis 3 data
No	179 (40.2)	26 (31.3)	390 (44.5)
Smoking			
Ever smoked	31 (7.0)	14 (16.9)	32 (3.7)
Never smoked	414 (93.0)	69 (83.1)	844 (96.3)
Body mass index	22.91 (4.79)	21.93 (3.20)	23.10 (4.58)

procedure was the same as the prior analysis just with urogenital and psychological menopause symptoms serving as the dependent variable, rather than somato-vegetative symptoms. Here, it can be seen that women living away from their natal group experienced worse psychological (IRR: 1.44, 95% CI: 1.19-1.73) and urogenital (IRR: 1.43, 95% CI: 1.19-1.73) menopause symptoms. Covariate results from previous models were also robust following a change in dependent variable, as women with more children and greater financial insecurity also reported worse symptoms in these two domains.

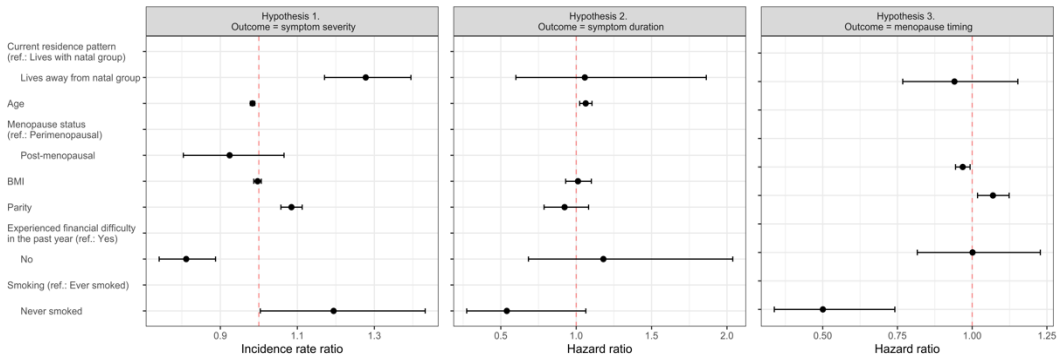

Figure 4.2. *Results from the best models testing each hypothesis. Hypothesis 1 uses Poisson regression to predict menopause symptoms severity; Hypothesis 2 uses Cox regression to predict duration of menopause symptoms; and Hypothesis 3 uses Cox regression to predict menopause timing. Incidence rate ratio (Hypothesis 1) and hazard ratios (Hypotheses 2 and 3) reported, with error bars showing the 95% confidence interval. A higher incidence rate ratio when testing Hypothesis 1 indicates more severe symptoms, and a higher hazard ratio when testing Hypotheses 2 and 3 indicate a shorter symptom duration and an earlier menopause, respectively.*

Table 4.2. *Results from model fitting based on Akaike Information Criterion (AIC) value. The lowest AIC value is deemed to best fit the data, and a ΔAIC of more than two demonstrating a significantly poorer model fit. The best fitting model has been italicised. All models adjust for parity, financial security, smoking status, and body mass index, with H1 and H2 additionally adjusting for age, and H1 also adjusting for menopause status.*

Model	K	AIC	ΔAIC	w_i
H1. Ancestral patrilocality results in less severe menopause symptoms				
Covariates	7	3092.21	28.25	0.00
Current residence pattern	*8*	*3063.97*	*0.00*	*0.51*
Ancestral residence pattern	9	3081.30	17.34	0.00
Current and ancestral residence pattern	10	3064.07	0.10	0.49
H2. Ancestral patrilocality results in a shorter duration of menopause symptoms				
Covariates	*5*	*496.53*	*0.00*	*0.60*
Current residence pattern	6	498.49	1.96	0.23
Ancestral residence pattern	7	499.68	3.15	0.12
Current and ancestral residence pattern	8	501.68	5.15	0.05
H3. Ancestral patrilocality results in a later age of menopause				
Covariates	*4*	*4151.59*	*0.00*	*0.61*
Current residence pattern	5	4153.24	1.65	0.27
Ancestral residence pattern	6	4155.56	3.97	0.08
Current and ancestral residence pattern	7	4157.16	5.57	0.04

4.4.2 Hypothesis 2. More female dispersal associates with a shorter peri-menopause.

To test Hypothesis 2, data from a sample of 83 women was used, of which 49% were ancestrally duolocal (Figure 4.1b). The majority were post-menopausal (84%) with a mean age of 60.24 (SD: 7.17). On average, women experienced menopause symptoms for 1.75 years (SD: 2.32), with ancestrally duolocal women experiencing symptoms for slightly longer (Figure 4.1e). See Table 4.1 for full descriptive statistics.

Table 4.3. *Results from multivariate Poisson regression models, predicting severity of psychological and urogenital menopause symptoms. Results show the incidence rate ratio and 95% confidence interval.*

	Psychological symptoms			**Urogenital symptoms**		
	Current and ancestral residence	**Ancestral residence**	**Current residence**	**Current and ancestral residence**	**Ancestral residence**	**Current residence**
Ancestral residence pattern (ref.: Duolocal)						
Mixed	1.51 (1.05-2.12)	1.48 (1.03-2.07)	-	1.51 (1.05-2.12)	1.48 (1.03-2.07)	-
Patrilocal	1.01 (0.80-1.28)	1.26 (1.02-1.54)	-	1.01 (0.79-1.27)	1.25 (1.02-1.54)	-
Current residence pattern (ref.: Lives with natal group)						
Lives away from natal group	1.47 (1.19-1.82)	-	1.44 (1.19-1.73)	1.47 (1.19-1.82)	-	1.43 (1.19-1.73)
Age	0.99 (0.97-1.00)	0.98 (0.97-1.00)	0.99 (0.97-1.00)	0.99 (0.97-1.00)	0.98 (0.97-1.00)	0.99 (0.97-1.00)
Menopause status (ref.: Perimenopausal)						
Post-menopausal	1.21 (0.88-1.70)	1.18 (0.86-1.65)	1.24 (0.90-1.73)	1.22 (0.89-1.70)	1.19 (0.86-1.66)	1.24 (0.91-1.74)

Table 4.3. *Results from multivariate Poisson regression models, predicting severity of psychological and urogenital menopause symptoms. Results show the incidence rate ratio and 95% confidence interval.*

	Psychological symptoms			**Urogenital symptoms**		
	Current and ancestral residence	**Ancestral residence**	**Current residence**	**Current and ancestral residence**	**Ancestral residence**	**Current residence**
Body mass index	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.97-1.01)	0.99 (0.97-1.01)
Parity	1.13 (1.07-1.19)	1.14 (1.08-1.20)	1.14 (1.08-1.20)	1.13 (1.07-1.19)	1.14 (1.08-1.20)	1.14 (1.08-1.20)
Experienced financial difficulty in the past year (ref.: Yes)						
No	0.70 (0.58-0.85)	0.71 (0.58-0.86)	0.69 (0.57-0.84)	0.70 (0.58-0.85)	0.71 (0.58-0.86)	0.69 (0.57-0.84)
Smoking (ref.: Ever smoked)						
Never smoked	2.07 (1.33-3.43)	2.05 (1.32-3.39)	2.15 (1.39-3.56)	2.16 (1.38-3.57)	2.13 (1.37-3.52)	2.24 (1.45-3.70)

Cox regression was used here to measure the differences in symptom duration. There was no evidence for current or ancestral female dispersal associating with a shorter duration of menopause symptoms (Appendix D Table D2). Model fitting (Table 4.2) shows that the model just including the covariates best fit the data. The inclusion of current residence pattern did not significantly worsen the model fit as it had a ΔAIC value of less than two (ΔAIC: +1.96), however, current residence pattern's relationship with the duration of menopause symptoms is negligible (HR:

1.06, 95% CI: 0.60-1.86). Full model results are presented in Appendix D (Table D2).

4.4.3 Hypothesis 3. More female dispersal associates with a later menopause.

The dataset used to test the final hypothesis comprised of 876 women, who were primarily of duolocal descent (74%; Figure 4.1c). As this dataset also included women who were pre-menopausal, it had a lower average age of 51.04 (SD: 12.85). As in the previous datasets, the majority of women had never smoked (96%) and had experienced some degree of financial insecurity (56%).

Cox regression was used to measure the 'risk' of entering menopause at any given age. Model results do not suggest that dispersing females (either ancestrally or currently) have a later menopause. As with Hypothesis 2, the model just including the covariates best fit the data (Table 4.2), with the addition of current residence pattern only slightly worsening model fit (ΔAIC: +1.65). Here, women currently living away from their natal group had a decreased risk of entering menopause at any given age, meaning they had a slightly later age of menopause (HR: 0.94, 95% CI: 0.77-1.15), but not to a significant degree (see Figure 4.2). Full model results are shown in Appendix D Table D3.

4.5 Discussion

Ubeda et al. (2014)'s model predicted that, due to intragenomic conflict, women from populations that were ancestrally patrilocal should present a shorter and less symptomatic peri-menopause that terminates in a later menopause. However, using a sample of women from four ethnic groups in China, I find no support for this hypothesis. Rather, I find that women from ethnic groups that are traditionally patrilocal and women who are currently living patrilocally report more severe menopause symptoms, and find little significant difference between symptom duration or menopause timing based on current or ancestral residence pattern.

Firstly, it should be that the predictions made by the model may theoretically unjustified. Ubeda et al. (2014) state that, if a prolonged post-reproductive lifespan is adaptive (as many suggest it is) then it is unclear why the peri-menopausal period is not a "rapid and smooth transition from the reproductive to the non-reproductive phases of a woman's life" (Ubeda et al., 2014:165). However, just because

menopause and the post-reproductive lifespan possibly have evolutionary origins, it does not mean that any physiological side effects that accompany this life history trait also have to be adaptive. Many biological transitions seen in nature are difficult, and there is no reason to assume that the menopausal transition would be any different. In fact, many turbulent life events – such as the birthing process and puberty – are important for fitness and selection. The model is also reliant on strict dispersal patterns when, in reality, dispersal patterns are often very flexible, and this would have likely been the case in our evolutionary history. It is not known what the ancestral dispersal pattern of humans was, but it is likely to have been responsive to ecological cues and environmental factors, meaning that there was probably not one universal 'ancestral *Homo sapiens* residence pattern'. Therefore, it is likely the social system required for the model to work was not present in our history.

Secondly, we do not necessarily know how prevalent menopause symptoms were in our evolutionary history. As reviewed in Chapter 1, there is a great amount of variance in menopause symptom experience, and there are many culturally and temporally specific factors that have been found to influence symptom severity, such as diet, clothing, body mass index, and so on. Hence, it might be that bothersome menopause symptoms are a relatively new phenomena in response to various aspects of 'modernity' that were not present in our evolutionary history.

No evidence was found for a difference in menopause symptom duration relative to residence pattern, whether this be current or ancestral. This could be for a number of reasons. Firstly, as stated previously, the sample size when looking at symptom duration is not large enough to capture small effects. Secondly, it may be that the data are not accurate enough to capture differences in symptom duration. As the data are not longitudinal, women had to remember when they began experiencing symptoms (which may have been many years ago). Relying on memory can lead to inaccurate data, and evidence for this can be seen in Figure 4.2, in which older women reported a shorter peri-menopausal period. This could be because older women are unsure about when they started symptoms and so estimated their symptoms beginning shortly before they went through menopause. Though equally, it could be that older women did have a shorter peri-menopause and that symptoms did not last so long a decade or so ago. It is likely that non-retrospective longitudinal data is required to gain a more accurate representation of how long women experience symptoms for. However, it may also be that the initial model has some

inaccuracies regarding its predictions about menopause symptoms. As previously mentioned, the model only addresses menopause symptoms experienced prior to menopause, and fails to address any symptoms experienced by post-menopausal women, despite them being frequently reported (Thurston et al., 2008; Avis et al., 2015). As the data here may not be completely accurate, there is scope for future research into the proposed effect of intragenomic conflict on menopause symptom duration. Additionally, there is currently little research into what causes women to experience menopause symptoms for different durations of time, so this is a possible avenue for future studies. However, at present, the data analysed here showed no evidence for a difference in symptom duration between groups with different residence patterns.

I also found no support for the prediction that greater levels of female dispersal will associate with a later menopause. As with symptom duration, it might be that there were some inaccuracies in reporting of age of natural menopause. In Figure 4.1f and 4.1l, it can be seen that there are peaks in reported age of menopause around the ages of 40 and 50, which could be an indicator that some women were unsure of exactly when they experienced their final menstrual period, and were therefore just approximating the timing. Hence, it might be that the insignificant relationship is due to the lack of clarity in the data. Though, the results do show that women who have smoked and have a lower body mass index had a greater 'risk' of entering menopause, which is in line with previous research (Gold et al., 2000; Gold et al., 2001; Ozdemir & Col, 2004; Ayatollahi et al., 2005; Nagel et al., 2005; Parazzini & Progetto Menopausa Italia Study, 2007; McKnight et al., 2011; Sapre & Thakur, 2014; Tao et al., 2015; Bjelland et al., 2018; Wang et al., 2018), suggesting that the data here is – to an extent – capturing significant behavioural and lifestyle factors that influence menopause timing, even if the women were only estimating when they experienced menopause. Results from this data also demonstrate that women who have more children experience and earlier age of natural menopause. Currently, there is conflicting data on the relationship between menopause timing and fertility, with some research indicating that nulliparity or low fertility associates with an earlier menopause (Mozumdar & Agrawal, 2015; Mishra et al., 2017), and while other studies have reported a negative relationship between number of children and age of natural menopause (Ahuja, 2016).

The final prediction of Ubeda et al. (2014)'s model is that higher rates of female dispersal should predict less severe menopause symptoms; however, in this data, the opposite was found to be the case. While current living arrangements better fit the data than ancestral ones, both variables predicts that women would experience worse menopause symptoms under a patrilocal residence system, thus negating the predictions made in the model of interest. These findings were also replicated when looking at other domains of menopause symptoms. Though the model of interest only made predictions about vasomotor symptoms, I also modelled the effect of ancestral and current residence pattern on psychological and urogenital symptoms, and found that women who dispersed from their natal group reported more severe symptoms in these two areas also, suggesting that women who live more patrilocally experience more severe menopause symptoms across the board. This could partially be due to the confounding variables. Patrilocal women within this sample were both more likely to smoke and had more children: two factors commonly associated with a more turbulent peri-menopause (Blumel et al., 2000; Olofsson & Collins, 2000; Chedraui et al., 2010; Jaber et al., 2017; Avis et al., 2018; Chlebowski et al., 2018). In addition to these possible confounding effects, it may be that the relationship between residence patterns and symptom severity is driven by the social differences that occur as a result of different living arrangements. Within the study sample, the majority of women who reported to still live with their natal group (i.e. not disperse) are Mosuo, and – while there is occasionally a degree of conflict between and within households – they are generally known for being an extremely peaceful group (Wu et al., 2015b; Mace et al., 2018). Ethnographic reports state that harmony is the ideal within the household, and that fissions between genetic kin are seldom observed and often consciously avoided (Shih & Jenike, 2002; Shih, 2009). Furthermore, Mosuo women are highly autonomous (Liu & Zuo, 2019), which decreases their stress load and may make them better able to manage any negative menopause symptoms (Ahmad & Zakaria, 2015). This autonomy has been observed to have positive health outcomes, with matrilineal Mosuo found to have lower levels of C-reactive protein compared to Mosuo women who have adopted a patrilineal descent pattern, and therefore a decreased risk of inflammation and hypertension (Reynolds et al., 2020). Amongst the Han and Yi there are more reports of social conflict (Link, 2016), though this has not been linked to any health outcomes.

The different levels of relatedness within households that result from different residence patterns may also contribute to women experiencing menopause symptoms differently. Compared to duolocal households, patrilocal households have lower levels of relatedness, which may decrease the drive to cooperate with one another. Sexual conflict (Trivers, 1972) can lead to tension between husbands and wives or women and their patrilineal relatives over fertility and mating decisions, which are more pronounced in patrilocal than duolocal groups (Leonetti et al., 2007). It has been observed that when women live patrilocally, there is an increased level of spousal conflict that is often exacerbated by in-laws, possibly because her husband's kin have less of an evolutionary interest in her (Skinner, 1997; Leonetti et al., 2007). This is exemplified in studies demonstrating that women who live with their matrilineal kin have higher fertility than women who do not, and that women who live with their mothers are less likely to get divorced than those who live patrilocally (Sear et al., 2003; Leonetti et al., 2007). Furthermore, intimate partner violence is more often experienced by women in patrilineal and patrilocal societies (Sedziafa et al., 2016; Sedziafa et al., 2018). Hence, sexual conflict and household dissonance may increase the woman's stress load, and so worsen a woman's menopausal transition.

Differences in perceptions of the menopausal transition between the groups may also contribute to the difference in symptom reporting. Compared to the Han, the Mosuo have been found to have a more positive attitude towards the menopausal transition, with the latter group perceiving it to be an inevitable natural event, with pleasure being derived from being post-menopausal (Zhang et al., 2016). Given previous research has found that a more negative attitude towards the menopausal transition is associated with increase symptom reporting throughout (Ayers et al., 2010), it may be that the increased symptoms reporting amongst patrilocal groups are somewhat the product of a less positive approach to the menopausal transition.

4.6 Conclusion

In this chapter, I have tested the predictions made in the intragenomic conflict hypothesis of menopause symptoms and age, with current residence pattern better predicting variation in aspects of the menopausal transition than ancestral residence pattern, and results converse to the models predicted being found for menopause symptom severity. I propose that this may in part be the result of sexual and social conflict, rather than intragenomic conflict, with a more stressful environment

exacerbating the peri-menopausal experience. The results presented here are based on a small sample of data, but nonetheless, they suggest that ecology and social organisation may be an important predictor of menopause symptoms but not timing, but in the direction predicted from sexual and social conflict, rather than intragenomic conflict.

CHAPTER FIVE

Support, stress, and menopause symptoms

5.1 Chapter summary

Many women going through the menopausal transition report experiencing vasomotor symptoms. While these symptoms are common, they are by no means universal, and there is a great deal of variation in their frequency and severity. Many factors have been documented to covary with symptom severity, including levels of social support and stress. Here, stress has been found to worsen menopause symptoms, and there is also some evidence that support somewhat eases them. However, there is little research testing whether support is an effective buffer against the negative effects of stress. In this chapter, using data from SWAN, I use multilevel Poisson regression with random effects to test 1) if more social support is associated with decreased vasomotor symptoms frequency, 2) if increased life stress worsens vasomotor symptoms, and 3) if support acts as a buffer against stress. After adjusting for age, marital status, smoking, self-perceived overall health, ethnicity, and menopausal status, I find that stress increases the frequency of vasomotor symptoms. However, contrary to my hypothesis, I did not find strong evidence that increased support led to lower vasomotor symptoms frequency, or that support buffered against the effects of stress. This research highlights that social factors impact the menopausal transition. This chapter has been published in *PLOS One*, under the title *The relationship between social support, stressful events, and menopause symptoms* (Arnot et al., 2021), and was co-authored by Emily Emmott and Ruth Mace.

5.2 Introduction

Menopause is often a significant event for women; and, for many, the process is not smooth. Rather, it is accompanied by a multitude of physical symptoms, with vasomotor symptoms (vasomotor symptoms) being the most frequently reported, which include hot flashes, night sweats, and cold sweats (Avis et al., 2005a). In addition to vasomotor symptoms being generally bothersome, they have also been observed to have a negative impact on women's mental health and wellbeing (Genazzani & Gambacciani, 2011). Typically, menopause symptoms are confined to

the peri-menopausal period; however, they are known to occasionally persist beyond final menstruation (Gold et al., 2006). They are thought to be primarily the result of hormonal fluctuations that occur during the menopausal transition, with higher levels of follicular stimulating hormone and lower estradiol being associated with a greater likelihood of reporting vasomotor symptoms (Randolph et al., 2005). However, while all women experience these hormonal changes during the menopausal transition, not all report vasomotor symptoms (Thurston & Joffe, 2011). Though population estimates vary (Melby et al., 2005), it is thought that up to 70% of all women experience vasomotor symptoms at some point during the menopausal transition (Gold et al., 2006; Williams et al., 2008). Further, even amongst the women who do report vasomotor symptoms, the symptomatology is not uniform within and between populations, with women reporting them to varying degrees of severity and frequency (Gold et al., 2000; Avis et al., 2001b; Anderson et al., 2004; Melby, 2005; Gold et al., 2006; Li et al., 2012; Yang et al., 2019). Given the negative impact of vasomotor symptoms reported by women (Thurston et al., 2008), it is important to understand what co-varies with vasomotor symptoms prevalence and severity, as it may better equip women to deal with the menopausal transition.

In recent years, different social and ecological factors have been found to associate with vasomotor symptoms, including ethnicity and socioeconomic position (Gold et al., 2004; Gold et al., 2006). Further, as I showed in Chapter 4, post-marital residence patterns are also associated with menopause symptoms, as duolocal women living with their own matrilineal relatives reported less severe menopause symptoms when compared with women who live with their husband's family (Yang et al., 2019). I hypothesised that this underlying relationship could possibly be attributed to the different levels of household conflict and social support present under the different social structures. This is based on ethnographies reporting that the Mosuo and Zhaba are generally very peaceful groups where the women have high levels of autonomy (Shih & Jenike, 2002; Shih, 2009; Liu & Zuo, 2019). This autonomy has been shown to have positive health outcomes, with the high levels of gender equality present in the Mosuo reversing the gender disparities in inflammation and hypertension that are found in populations with gender norms that favour men (Reynolds et al., 2020). Here, patrilineal Mosuo women were found to have higher levels of C-reactive protein than matrilineal Mosuo women, and therefore a greater risk of chronic inflammation and hypertension (Reynolds et al., 2020), possibly as a

result of the differing levels of stress and support associated with the two different kinship systems (Yang et al., 2014; Liu et al., 2017).

In addition to possibly having an impact on menopause symptoms, ones experience of stressors has been linked to higher levels of cortisol and fibrinogen, with the former being associated with the suppression of the immune, reproductive, and digestive systems, and the latter with increased inflammation (Segerstrom & Miller, 2004). As such, stress has causally been associated with a greater risk of cardiovascular disease (Steptoe & Kivimaki, 2012), faster progression of HIV (Leserman et al., 2000), delayed wound healing (Kiecolt-Glaser et al., 1998; Marucha et al., 1998), decreased receptivity to the influenza vaccine (Kiecolt-Glaser et al., 1996), and a greater risk of upper respiratory tract infection (Cohen et al., 1991; Cohen et al., 1998). Moreover, a number of studies have found that increased stress (both perceived stress and stressful experiences) can worsen the frequency and severity of menopause symptoms (Gold et al., 2006; Bauld & Brown, 2009; Nosek et al., 2010; Sood et al., 2019; Jalava-Broman et al., 2020); however, the causal pathway underlying this particular association is currently unclear (Falconi et al., 2016).

The potential effect of stress on menopause symptoms is of importance to understand: midlife is often a highly stressful time for women, with many major life events coinciding with one another, such as parental death, children leaving home, and divorce (Perrig-Chiello & Hopflinger, 2005; Sakraida, 2005; Dare, 2011). Further, going through menopause can itself be a stressful event, as it marks a transition to a new phase of life, with many women reporting a sense of loss accompanying the end of their fertility (Glazer et al., 2002). Previous research suggests that a strong social network and higher levels of social support throughout the menopausal transition may reduce menopause symptom frequency and severity (Gold et al., 2006; Dimkpa, 2011; Yazdkhasti et al., 2012; Zhao et al., 2019). Similar associations have also been observed between social relationships and other health outcomes (Uchino, 2006; Thoits, 2011), with strong social ties being related to an increased likelihood of cancer recovery (Ikeda et al., 2013; Boen et al., 2018), a reduced risk of Alzheimer's and dementia (Fratiglioni et al., 2000; Dyer et al., 2020), and also protect against cardiovascular disease (Ford et al., 2000; Shaya et al., 2013). Exactly how social ties work to sustain and/or improve health and well-being is not completely clear (Thoits, 2011). However, possible pathways include lower social support manifesting

itself in more negative behavioural (i.e. poorer health behaviours) and psychological (i.e. depression) processes (Uchino, 2006).

In addition to the noted direct benefits of social support, it is also thought that support can act as a buffer against the negative effects of stress on health (Cohen & Wills, 1985). Here, a stronger support system is thought to lessen the impact of stress, through decreasing its perceived intensity, and also helping to correct any maladaptive behaviours that form as a result of stress (e.g. alcoholism) (Cohen & Wills, 1985). Hence, social support may weaken the association between stress and negative health outcomes by moderating the actor's reaction or perception of the stress they are experiencing. Despite the fact that stress has been widely associated with worse menopause symptoms, to my knowledge, the stress-buffering hypothesis for social support has not been explicitly tested in relation to vasomotor symptoms.

Based on the health differences observed between patrilineal and matrilineal populations in Southwestern China, as well as the relationship between stress, support, and health outcomes already present in the epidemiological literature, I did intend to build on research from the previous chapter by testing whether the relationship between residence pattern menopause symptoms is mediated by stress, and somewhat moderated and/or mediated by social support (Figure 5.1a). Here, I would predict that the high levels of social support and low levels of stress and

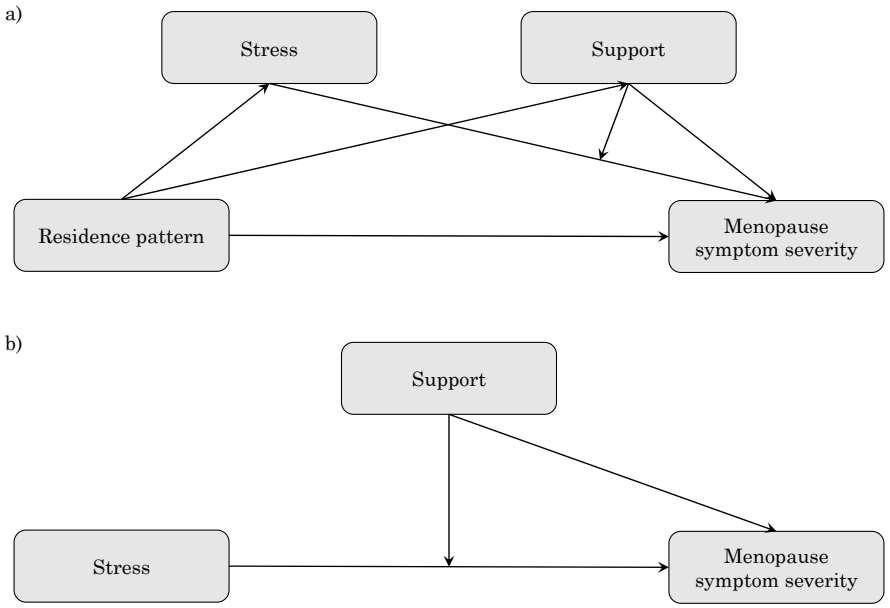

Figure 5.1. *Proposed model to be tested in this chapter (a) and the model that was tested due to data restrictions (b).*

conflict present in the duolocal households would ease one's symptoms throughout the menopausal transition; while the heightened levels of conflict in patrilocal households would result in increased stress and therefore worse menopause symptoms. To test these predictions, I had planned to collect additional data on how much support (both instrumental and emotional) the women felt they had, and also data on conflict (e.g. household arguments). Unfortunately, due to COVID-19 and its associated travel restrictions I was unable to go to China as planned at the beginning of 2020, and so could not plan, organise, or carry out any data collection to test this model. Therefore, I had to utilise the data I already had available. As SWAN collects data on menopause symptoms, experience of stress, and social support, I decided to use it for this chapter. However, as the US is predominantly neolocal, I was unable to include post-marital residence pattern within the model. I did explore whether there was variation in household structure through looking at whom the women surveyed reported living with, in case I was able to use living with parents or in-laws as a proxy of residence pattern. However, in the baseline of SWAN only ~5% of women reported living with either their mother or father, and ~2% lived with either their mother-in-law or father-in-law, so the sample size was not large enough to get a meaningful comparison between groups. There was also no data on distance from either set of parents, which meant I could not use this as an additional proxy of residence patterns either. Due to this, I was unable to test the model presented in Figure 5.1a, and so modified it based on the data available to me (Figure 5.1b). Therefore, in this chapter I firstly test whether higher levels of social support and stress associate with less frequent vasomotor symptoms and more frequent vasomotor symptoms, respectively. Secondly, I test whether social support buffers against the proposed negative impact of stress on vasomotor symptoms. This research aims to clarify how support and stress are associated with one another in regards to vasomotor symptoms.

5.3 Materials and methods

5.3.1 Data

This chapter uses data from nine years' worth of interviews from SWAN. The baseline wave (visit 0) was excluded as the stress measure was not comparable to the question patterning in subsequent interviews, and data from visit 7 was also not included as participants were not asked any questions about social support in that

year of interviews. Therefore, data from interviews 1, 2, 3, 4, 5, 6, 8, 9 and 10 are used in this chapter. See Chapter 2 for a detailed description of SWAN.

5.3.2 Variables

5.3.2.1 Vasomotor symptoms

Vasomotor symptoms served as the outcome variable in all analyses, and was measured through combining how often women experienced hot flashes, cold sweats and night sweats in the two weeks approaching the interview. These were measured individually on a Likert scale (not at all; 1-5 days; 6-8 days; 9-13 days; every day), and I then assigned each of these responses a value ranging from 0 to 4 (i.e. 0 = not at all; 1 = 1-5 days; and so on) and summed the woman's experience of the three symptoms. This left each respondent with a score between 0 and 12, in which a higher score indicated more frequent vasomotor symptoms.

5.3.2.2 Social support

SWAN provides four measures of social support which include how often the respondent feels she has someone to confide in, someone to listen to her, take her to the doctors, and help out when sick (see Figure 5.2). These were measured on a 5-point Likert scale (none of the time; a little of the time; some of the time; most of the time; all of the time), and taken from the Medical Outcome Study: Social Support Survey which is one of the most widely used instruments for measuring social support (Sherbourne & Stewart, 1991), and shows high levels of validity and

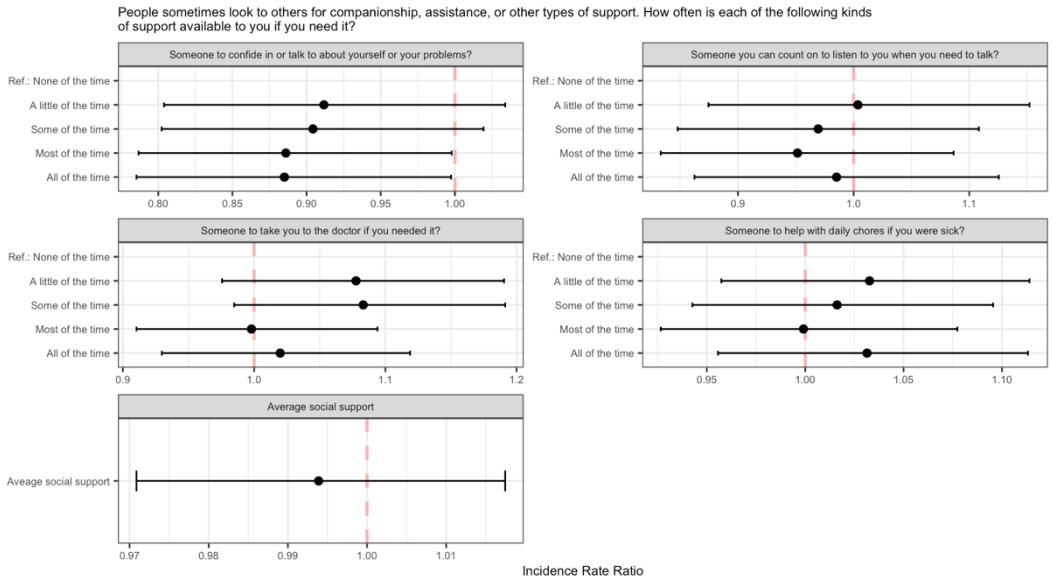

Figure 5.2. *Relationship between individual support measures and the aggregated average support measure, and vasomotor symptoms.*

reliability (Yu et al., 2004; Robitaille et al., 2011; Saddki et al., 2017; Khuong et al., 2018). An average measure of support was created, in which the Likert scales for each measure of support were converted to numeric (i.e. none of the time = 1; a little of the time = 2, and so on), then summed and averaged to create a value ranging from 1-5, where a higher value indicated greater social support (Sherbourne & Stewart, 1991).

5.3.2.3 Stress

For this chapter, experience of stressful events was used as a metric of how stressed the participant was. SWAN asks participants about the occurrence and perceived stressfulness, of 18 stressful life events that might have happened over the past year. These include the death of a close kin, changes in employment, and changes in household structure (see Figure 5.3 for the specific stressors and individual relationships). This is a modified version of the Psychiatric Epidemiology Research Interview life events scale (Bromberger et al., 2003), which has been validated across multiple racial and ethnic groups and shows high internal consistency (Vernon & Roberts, 1981). Participants are asked about whether they had experienced each stressor and whether/how much it upset them, with this being measured on a 5-point Likert scale (no – did not experience; yes – not upsetting; yes – somewhat

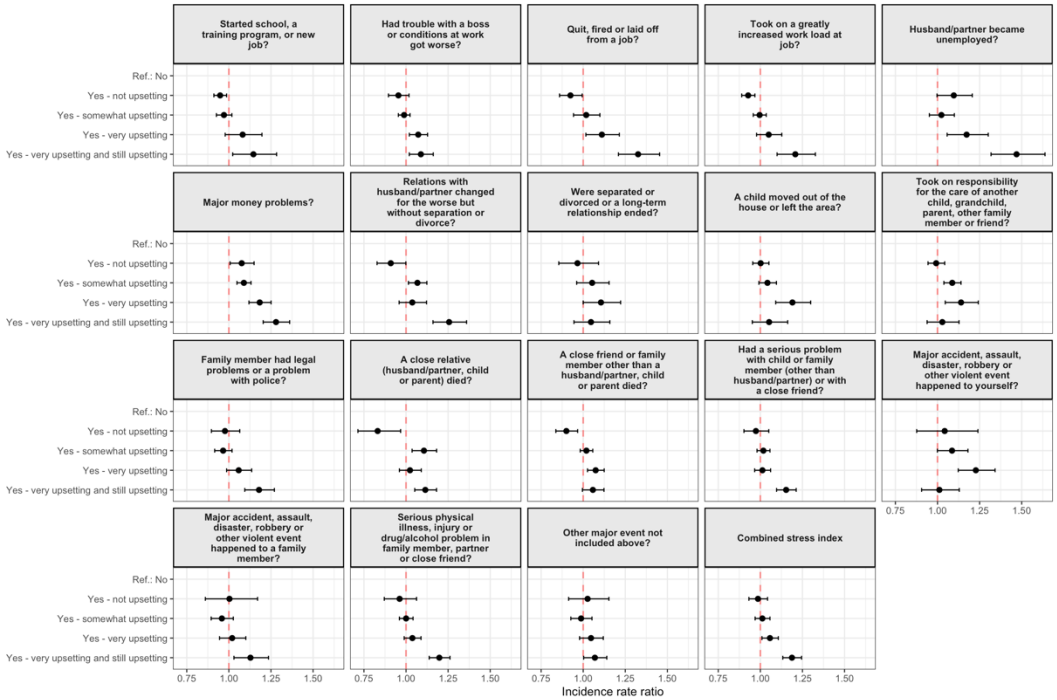

Figure 5.3. *Relationship between individual stressors and the stress index, and vasomotor symptoms.*

Table 5.1. *Example of how the stress index was derived using hypothetical data.*

Person ID	Stressor 1	Stressor 2	Stressor 3	Derived stress index
1	Yes – not upsetting	Yes – not upsetting	Yes – very upsetting	Yes – very upsetting
2	No	No	Yes – not upsetting	Yes – not upsetting
3	Yes – very upsetting and still upsetting	Yes – very upsetting	No	Yes – very upsetting and still upsetting
4	No	No	No	No
5	Yes – not upsetting	Yes – somewhat upsetting	Yes – not upsetting	Yes – somewhat upsetting

upsetting; yes – very upsetting; yes – very upsetting and still upsetting). As all of the individual life events presented a similar relationship with vasomotor symptoms (Figure 5.3), two variables to indicate stress levels were derived.

Firstly, a *stress index* was made, which is the maximum amount of stress the participant experienced across all of the 18 stressful events. As worse menopause symptoms appeared to associate with experience and upset of a stressful event (Figure 5.3), a composite measure was created to indicate the maximum amount of stress experienced by the respondent in the past year. For example, if a woman answered "No" to 17 of the events, and stated that one event had "Somewhat upset her", her stress index would be "Yes – somewhat upsetting", as this is the maximum amount of stress she experienced. A hypothetical example of the derivation of this variable can be seen in Table 5.1. In summary, a woman's stress index can be seen as: *Did the participant experience a stressful event over the past year, and was she upset by it?* With the possible response being either: no; yes – but she did not find it upsetting; yes – she found it somewhat upsetting; yes – she found it very upsetting; or yes – she found it very upsetting and is still upset by it. Secondly, a *stress dose* variable was created to measure how many stressors the woman had experienced over the past year. Here, any stressor that the woman experienced – regardless of whether it upset her or not – was counted. This variable therefore ranged from 0 to 18, with a higher number indicating she had experienced more stressful events. See

Table 5.2. *Example of how the stress dose variable was derived using hypothetical data.*

Person ID	Stressor 1	Stressor 2	Stressor 3	Stressor 4	Stressor 5	Derived stress dose
1	Yes – not upsetting	Yes – not upsetting	Yes – very upsetting	Yes – not upsetting	Yes – very upsetting	5
2	No	No	Yes – not upsetting	No	Yes – not upsetting	2
3	Yes – very upsetting and still upsetting	Yes – very upsetting	No	Yes – very upsetting	No	3
4	No	No	No	No	No	0
5	Yes – not upsetting	Yes – somewhat upsetting	Yes – not upsetting	Yes – somewhat upsetting	Yes – not upsetting	5

Table 5.2 for a hypothetical example of the derivation of this variable. In analyses, both the stress index and stress dose were included as time varying. Further information on the creation and selection of these variables can be found in Appendix E (Figure E1).

5.3.2.4 Covariates

Covariates were selected based on existing research into vasomotor symptoms and included as time varying, with data on them being collected every included wave. These included marital status (divorced/separated, married/in a relationship, widowed) based on previous findings that divorced women have more a more symptomatic menopausal transition (Lee et al., 2010); smoking habits (never smoked, ever smoked), as women who smoke are widely documented to experience worse vasomotor symptoms (Gallicchio et al., 2006); self-perceived overall health (poor; fair; good; very good; excellent) (Del Sueldo et al., 2018) as how healthy one feels could be seen to have a bidirectional relationship with menopause symptoms; and menopause status (pre-menopause; early peri-menopause; late peri-menopause; post-menopause; and not menstruating for another reason). As time invariant covariates, maximum level of education (less than high school, high school, some college/technical college, college degree, post-graduate degree) (Avis et al., 2018) and ethnicity (self-identified as Black or African American; Chinese American; Japanese

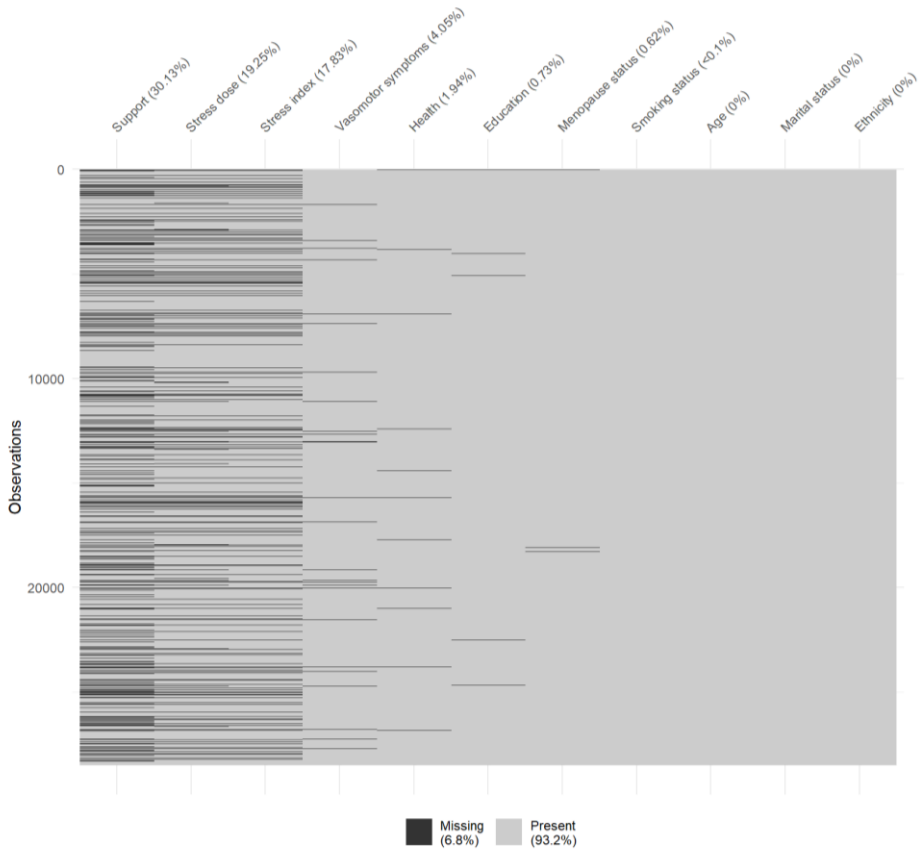

Figure 5.4. *Percent of missing data by variable for all years of interviews.*

American; white; Hispanic) (Gold et al., 2006) were also adjusted for, with education serving as a proxy of socioeconomic status, and ethnicity being controlled for due to the well documented relationship between ethnicity and vasomotor symptoms (reviewed in section 1.3.2.2). In all analyses, age at time of interview was also included as a covariate, and was centred on the mean, with cubic and quadratic terms also included.

5.3.3 Sample

A complete case analysis was once again carried out, with the first wave of interviews including 2718 individuals of a possible 2881 (94%). The missingness by wave and variable is presented in Appendix E (Table E1), and missingness by variable is plotted in Figure 5.4, where it can be seen that the majority of missing data is within the stress and support variables. As these variables are of interest within this chapter, I used binary logistic regression to see what predicted missingness in stress and support (Appendix E Table E2). Here I found that missingness in these variables was associated with having less severe menopause symptoms, being less well educated,

Table 5.3. *List of models used in analyses.*

Model name	Covariates
Base model	Age + Age^2 + Age^3 + Marital status + Smoking habits + Education + Ethnicity + Overall health + Menopause status
Support model	Social support + Base model
Stress model 1	Stress index + Base model
Stress model 2	Stress dose + Base model
Full model 1	Social support + Stress index + Base model
Full model 2	Social support + Stress dose + Base model
Interaction model 1	Social support*Stress index + Base model
Interaction model 2	Social support*Stress dose + Base model

having ever smoked and being of Hispanic ethnicity. This is further addressed in the discussion.

5.3.4 Analyses

To test whether support and stress influence vasomotor symptom experience throughout the menopausal transition, multivariable, multilevel Poisson regression (random-intercept). Multilevel modelling allows for a 'nested' structure of the data – as is the case here with each participant having multiple observations from different time points – and controls the fact that measurements taken from the same individual are going to be similar to one another (Singer & Willett, 2003). Poisson regression rather than linear regression was used as the data are skewed, with the majority of women reporting infrequent menopause symptoms. In total, 8 models were made, which are shown in Table 5.3. An initial model that comprised solely of the covariates (the *Base model*) was first created as a unit of comparison. Social support, the stress index, and stress dose variables were then subsequently added to the base model (the *Support model, Stress model 1* and *Stress model 2*, respectively) individually, and then together (*Full model 1* and *2*; stress measures were always included in separate models due to collinearity). Finally, to measure whether support buffers against stress, two models were made with an interaction effect between support and the two measures of stress (*Interaction model 1* and *2*).

Model fitting was then carried out based on AIC value, which has been shown to be appropriate for Poisson regression with random effects (Lian, 2012). The AIC shows which model is a relatively better fit to the data, and also penalises models for complexity. The model with the lowest AIC value is deemed to best fit the data, and a ΔAIC increase of more than two signifies weaker support for the model (Burnham & Anderson, 2002). Models were then weighted based on how much their AIC value changed relative to the best fitting model (Burnham & Anderson, 2002). All analyses were performed in *R* version 4.0.2 (R Core Team, 2020) using the packages *lme4* (Bates et al., 2015) and *ggeffects* (Lüdecke, 2018). Model fitting was carried out using *AICcmodavg* (Mazerolle, 2020), and data visualisations created using *ggplot2* (Wickham, 2016).

5.4 Results

5.4.1 Descriptive statistics

In the first round of interviews used in this analysis (n = 2718) women were aged 46.9 on average (SD: 2.69), with the majority of participants either being married or in a relationship (76.09%). A greater proportion of women had never smoked (58.06%), and very few women were not educated to a high school standard, with most women having attended some kind of college or technical school (32.38%). Half of the women in the sample were white (49.71%), and at this point in the study very few women were post-menopausal (1.51%), with most of them being in early peri-menopause (60.60%). Women were receiving high levels of social support on average (4.18, SD: 0.78), most had experienced a stressful event that upset them to some degree (76.49%), and the stress dose variable was positively skewed, with women experiencing on average 3 (SD: 2.45) stressful events in the past year. At this stage, the majority of women were not reporting frequent menopause symptoms (mean: 1.21, SD: 2.05). Full descriptive statistics are presented in Table 5.4.

5.4.2 Base model

The *Base model*, which comprised of all the covariates, was created to serve as a unit of comparison. Within this model, women who were married experienced more frequent vasomotor symptoms compared to divorced or separated women (IRR: 1.10, 95% CI: 1.05-1.16), as did women who had ever smoked (IRR: 1.22, 95% CI: 1.12-1.32). There was a near linear relationship between level of education and

Table 5.4. *Participant characteristics from the first year of interviews used in this analysis.*

Variable	n (%)	Mean (SD)
Vasomotor symptoms		1.21 (2.05)
Average among of support received		4.18 (0.78)
Stress index (experienced a stressful life event in past year)		
No	388 (14.3)	
Yes - not upsetting	251 (9.2)	
Yes - somewhat upsetting	766 (28.2)	
Yes - very upsetting	642 (23.6)	
Yes - very upsetting and still upsetting	671 (24.7)	
Stress dose		3.07 (2.45)
Age		46.91 (2.69)
Marital status		
Divorced/Separated/Single	601 (22.1)	
Married/In a relationship	2068 (76.1)	
Widowed	49 (1.8)	
Smoking habits		
Ever smoked	1140 (41.9)	
Never smoked	1578 (58.1)	
Education		
Less than high school	169 (6.2)	
High school	443 (16.3)	
Some college/technical school	880 (32.4)	
College degree	569 (20.9)	
Post-graduate education	657 (24.2)	
Ethnicity		

Table 5.4. *Participant characteristics from the first year of interviews used in this analysis.*

Variable	n (%)	Mean (SD)
African American	687 (25.3)	
Chinese American	220 (8.1)	
Japanese American	262 (9.6)	
White	1351 (49.7)	
Hispanic	198 (7.3)	
Self-perceived overall health		
Poor	50 (1.8)	
Fair	294 (10.8)	
Good	820 (30.2)	
Very good	1073 (39.5)	
Excellent	481 (17.7)	
Menopausal status		
Pre-menopausal	676 (24.9)	
Early peri	1647 (60.6)	
Late peri	112 (4.1)	
Post-menopause	41 (1.5)	
Other	242 (8.9)	

reporting of menopause symptoms, with a longer time spent in education being associated with decreased vasomotor symptoms frequency (IRR: 0.72, 95% CI: 0.59-0.88). As in previous studies (Gold et al., 2006), African Americans reported vasomotor symptoms most often, with Japanese American women experiencing them least (IRR: 0.41, 95% CI: 0.35-0.48); and women who perceived themselves to be healthier also reported less frequent vasomotor symptoms (IRR: 0.65, 95% CI: 0.58-0.72). Women who were in late peri-menopause (IRR: 1.87, 95% CI: 1.79-1.96) or had experienced menopause (IRR: 1.44, 95% CI: 1.37-1.51) reported symptoms more often. Full results are presented in Appendix E Table E3.

Table 5.5. *Results from model fitting based on Akaike Information Criterion (AIC) value. The lowest AIC value is deemed to best fit the data, and a ΔAIC of more than two demonstrating a significantly poorer model fit. The best fitting model has been italicised. Model composition is shown in Table 5.3.*

Model	K	AIC	ΔAIC	w_i
Base model	24	64259.50	91.52	0.00
Support model	25	64258.49	90.52	0.00
Stress model 1	*28*	*64167.97*	*0.00*	*0.58*
Stress model 2	25	64187.53	19.55	0.00
Full model 1	29	64168.64	0.66	0.42
Full model 2	26	64187.96	19.99	0.00
Interaction model 1	29	64184.11	16.13	0.00
Interaction model 2	25	64204.12	36.14	0.00

5.4.3 Support and stress

This chapter is testing the prediction that social support and stress influence symptom severity, and that support would act as a buffer against the effects of stress. Should this be the case, it can be expected that models including these variables would better fit the data, in addition to an interaction effect. Results from model fitting are presented in Table 5.5, where it can be seen that the best fitting model is *Stress model 1* (ΔAIC: 0.00), which included the stress index and covariates. The inclusion of social support (*Full model 1* ΔAIC: +0.66) and an interaction term (*Interaction model 1* ΔAIC: +16.13) worsened the model fit, though the former was not a significantly worse fit. The model that included just the covariates worst fit the data (*Base model* ΔAIC: +91.52).

In the best fitting model (*Stress model 1*) it can be seen that experiencing a stressor did worsen menopause symptoms, but the degree to which the woman was affected by the stressor was most critical. Figure 5.5 displays the predicted vasomotor symptoms at any given age based on experience of a stressor. Here, being still upset by a stressful event increased vasomotor symptoms frequency by 21% (95% CI: 1.15-1.26), and women who found the stressful event very upsetting at the time (but were no longer upset by it) experienced a 7% increase in vasomotor symptoms

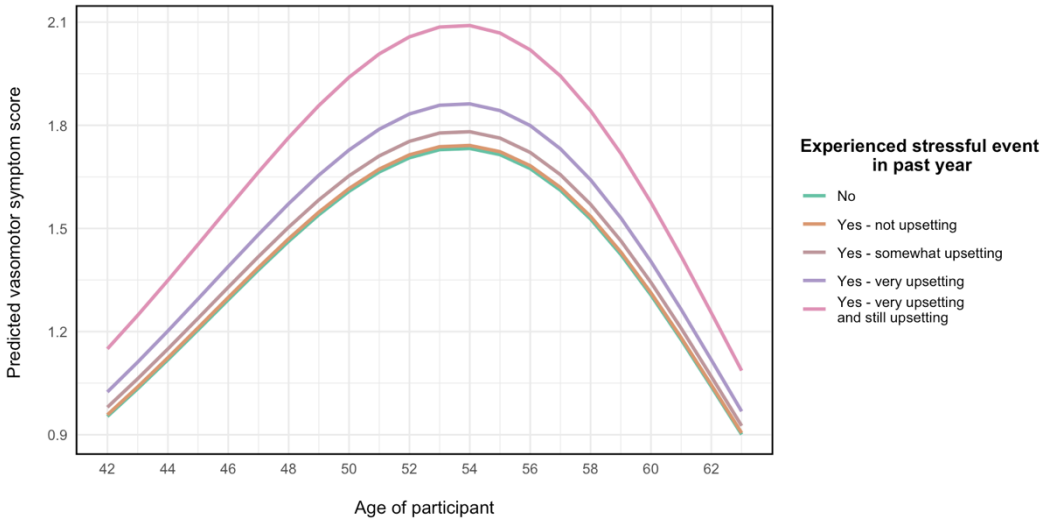

Figure 5.5. *Predicted vasomotor symptoms at any given age relative to experience of stress, based on the results from the best fitting model. Model adjusts for marital status, education, smoking habits, ethnicity, self-perceived health, and menopause status.*

frequency (IRR: 1.03-1.13). However, those who were not upset by the stressful event (IRR: 1.01, 95% CI: 0.95-1.06) or only found it somewhat upsetting (IRR: 1.03, 95% CI: 0.98-1.07) did not experience a statistically significant increase in vasomotor symptoms at the time of reporting (see Table 5.6).

As the current understanding of the physiological pathways around stress and vasomotor symptoms lead me to predict that current stress is an important determinant of vasomotor symptoms, I then also conducted an additional *post hoc* analysis with a time-lagged stress measure to test whether current stress is a better predictor of vasomotor symptoms than historical stress. Results presented in Appendix E (Table E3) show that stressful events from a year ago have less of a relationship with current vasomotor symptoms than current life stress, suggesting that vasomotor symptoms may be responsive to current life circumstances.

5.5 Discussion

This chapter is testing the idea that stress worsens menopause symptoms, and that support can act as a buffer against the hypothesised negative effects of stress. Existing research has shown that stress associates with more severe and more frequent vasomotor symptoms (Gold et al., 2006; Bauld & Brown, 2009; Nosek et al., 2010; Sood et al., 2019; Jalava-Broman et al., 2020), and some research has shown that a supportive environment is associated with decreased symptoms (Gold et al., 2006; Ayers et al., 2010; Yazdkhasti et al., 2012; Zhao et al., 2019). However, to my knowledge, whether support acts as a buffer against the negative effects of stress

Table 5.6. *Association between support, stress, and vasomotor symptoms. Results from multilevel Poisson regression, showing the incidence rate ratio and 95% confidence interval. All models adjust for age (linear, cubic, and quadratic), marital status, smoking status, education, ethnicity, health, and menopause status.*

	Support model	**Stress model 1**	**Stress model 2**	**Full model 1**	**Full model 2**	**Interaction model 1**	**Interaction model 2**
Stress index (ref.: No)							
Yes - not upsetting	-	1.01 (0.95-1.06)	-	1.01 (0.95-1.06)	-	1.11 (0.78-1.60)	-
Yes - somewhat upsetting	-	1.03 (0.98-1.07)	-	1.03 (0.98-1.07)	-	1.02 (0.79-1.30)	-
Yes - very upsetting	-	1.07 (1.03-1.13)	-	1.07 (1.03-1.13)	-	1.31 (1.01-1.69)	-
Yes - very upsetting and still upsetting	-	1.21 (1.15-1.26)	-	1.20 (1.15-1.26)	-	1.57 (1.23-2.00)	-
Stress dose	-	-	1.03 (1.02-1.03)	-	1.03 (1.02-1.03)	-	1.05 (1.02-1.07)
Support	0.98 (0.96-1.00)	-	-	0.99 (0.96-1.01)	0.99 (0.96-1.01)	1.02 (0.97-1.07)	1.00 (0.97-1.03)
Support*Stress index (ref.: No)							
Support*Yes - not upsetting	-	-	-	-	-	0.98 (0.90-1.06)	-
Support*Yes - somewhat upsetting	-	-	-	-	-	1.00 (0.95-1.06)	-

Table 5.6. *Association between support, stress, and vasomotor symptoms. Results from multilevel Poisson regression, showing the incidence rate ratio and 95% confidence interval. All models adjust for age (linear, cubic, and quadratic), marital status, smoking status, education, ethnicity, health, and menopause status.*

	Support model	**Stress model 1**	**Stress model 2**	**Full model 1**	**Full model 2**	**Interaction model 1**	**Interaction model 2**
Support*Yes - very upsetting	-	-	-	-	-	0.96 (0.90-1.01)	-
Support*Yes - very upsetting and still upsetting	-	-	-	-	-	0.94 (0.89-0.99)	-
Support*Stress dose	-	-	-	-	-	-	1.00 (0.99-1.00)

has not yet been tested. Results presented in this chapter offer little support for this idea. Rather, I find a model including a proxy of stress but not support best fits the data, and that in models that do include support, there is little relationship with menopause symptoms. Therefore, based on this data, it does not appear that increased support decreases menopause symptom severity, nor that it buffers against the negative effects of stress. This contrasts with some other research finding that various types of support (e.g. familial, emotional) are associated with less severe and more infrequent menopause symptoms (Ayers et al., 2010; Yazdkhasti et al., 2012; Zhao et al., 2019). It may be that, in this data, the support measure was not precise enough to capture its effect on menopause symptoms, and that a question that specifically refers to health or menopause symptoms is needed (e.g. "How often do you feel like you have someone to talk to about your health?") (Yazdkhasti et al., 2012). However, there is some research showing that support tailored to the menopausal transition can have detrimental effects. Many women attend support groups or 'menopause cafes' as a means of managing menopause through leaning on other women who are at the same life stage; but research has shown that in some cases support groups/increased discussion about menopause symptoms can cause

women to anticipate and expect negative symptoms, leading to a higher reporting of them (Bauld & Brown, 2009). Therefore, while support in some studies has been shown to have a beneficial effect on menopause symptoms (Ayers et al., 2010; Yazdkhasti et al., 2012; Zhao et al., 2019), as shown in this chapter it is not a universal phenomenon, and in some cases specific support around the menopausal transition can cause women to perceive their symptoms to be worse (Bauld & Brown, 2009). Hence, understanding how and why different forms of support influence menopause symptoms in different ways is important to understand, and should be targeted in future research.

While I found no relationship between support and menopause symptoms, both measures of stress I included in analyses showed a positive relationship with symptom frequency, in which more stress was associated with more frequent symptoms. Regarding the *stress index* measure specifically, it appears that it is not simply experiencing a stressful event that worsens menopause symptoms, but the degree of upset that results from the life stressor. Here, women who said they were very upset or still upset by a stressful life event reported more frequent menopause symptoms than those who had not experienced a stressful event. Further, women who had experienced a stressful event that did not upset them did not report significantly more frequent symptoms compared to women who had not experienced a stressful event. Therefore, life stress does not inevitably make menopause symptoms worse; rather, it is partially related to how the woman is affected by it. While I did not include a measure of resilience in this chapter, how one reacts to stressors is somewhat related to how resilient they are. As such, previous research has found that women with higher resilience scores also reported less severe vasomotor symptoms (Zhao et al., 2019) suggesting how women react to and deal with life stress is important when it comes to menopause symptoms. Of course, many stressors in life are unavoidable. Midlife is often accompanied by many stressful life changes, such as the death of parents, children leaving home, and the breakdown of relationships (Bove et al., 2016). In addition to this, the menopausal transition is also often seen to symbolise a change in the woman's role in society: as fecundity wanes and children move away, many women report experiencing feeling as though they are losing a large part of their identity, which can be highly distressing and upsetting (de Bruin et al., 2001; Wadsworth & Green, 2003; Duffy et al., 2011; Sergeant & Rizq, 2017). As I have shown in this chapter, the accumulation of

stressful events (the *stress dose* measure) is associated with more frequent menopause symptoms, and thus the coincidence of many major life changes can be expected to worsen symptom experience. As stated, these stressors are largely unavoidable, and it is not useful to simply advise women to 'avoid stress' to manage their menopause symptoms. Rather, providing women with the knowledge and means to deal with this stress may be beneficial. Mindfulness has already been shown to ease menopause symptoms (Sood et al., 2019), and thus teaching women these techniques may be a means of mitigating the negative impact of stress on menopause symptoms.

Changing societal perceptions of ageing in women might also contribute to easing the menopausal transition. As previously mentioned, many women find the menopausal transition to be a difficult life period as they feel like they are losing a large part of their identity, and this would be partially due to the culturally specific attitudes towards age and the ageing process. In the US, qualities associated with youth (e.g. beauty, fertility, vitality) are valued, while qualities associated with the ageing process (e.g. greying hair, wrinkles) are often actively avoided (Chonody & Teater, 2018). Hence, the negative perception of the ageing process in the US would contribute to women finding menopause to be a stressful life period. In contrast, within cultures where older members of the population are revered and respected (e.g. many East Asian cultures), menopause symptoms have been found to be less severe (Minkin et al., 2015). Similarly, when women reported feeling empowered by the menopausal transition, they were less likely to report symptoms (Deeks & McCabe, 2004). Hence, culturally specific attitudes towards ageing may require a shift to contribute to the easing of symptoms experienced during menopause.

It should be noted that currently, the mechanistic relationship between stress and vasomotor symptoms is unclear. It may be that increased levels of cortisol produced as part of the stress response mimic the neuroendocrinological changes that occur alongside vasomotor symptoms. However, research has shown that increased perceived stress does not associate with higher levels of inflammation (measured through fibrinogen), so this may not be the case (Falconi et al., 2016). Alternatively, it could be that menopause symptoms that would have otherwise been mild or unnoticeable are exacerbated by stressors, in a similar way to how pain is often exacerbated by stress (Ahmad & Zakaria, 2015). As part of tackling stress' impact on menopause symptoms, focus needs to be paid to unravelling the precise causal mechanisms.

Results from my models replicated findings from previous research relating to ethnicity. As in previous studies, women who self-identified as African American reported the most frequent symptoms, while Chinese and Japanese American women reported symptoms less often. It has been suggested that this relationship is somewhat the product of the effect of stress on symptom experience. The data used in this chapter is taken from the US, where African Americans have been found to present more markers of stress than other ethnicities (Chyu & Upchurch, 2011), possibly due to economic and systematic factors (Brown et al., 2020). However, results from this chapter suggest that increased stress does not completely mediate the relationship between ethnicity and vasomotor symptoms, as the relationship still persists after the inclusion of the stress index and stress dose within the model (Table 5.6). Further, if the ethnic differences in menopause symptoms were completely explained by stress, it would not completely account for why women from an East Asian background consistently report fewer and less severe menopause symptoms. Women of East Asian ancestry living in the US have been found to have higher levels of biomarkers associated with stress (Fang et al., 2014), and so based on this one might predict that they would experience more severe menopause symptoms like African American women, but this is not the case. Therefore, it does not appear that stress satisfactorily explains the ethnic differences in menopause symptoms, and thus further research is needed to understand this variation.

It should be noted that this chapter has important limitations. Firstly, missingness in the independent variables of interest (stress and support) is predicted by being Hispanic, being less well educated, having ever smoked, and also having less severe menopause symptoms. While the SWAN researchers made efforts to collect data from a representative sample of women, this pattern of missingness means that the results are not necessarily generalisable, and therefore possibly only applicable to a certain demographic of women. Secondly, while I show evidence of a possible causal pathway between stress and vasomotor symptoms, further research using more targeted data collection is likely required to establish the precise causal pathway and mechanisms underlying the relationship.

5.6 Conclusion

Research from this chapter contributes to the growing body of research linking stress with a worse menopausal transition. For most, stress is an unavoidable aspect of

daily life, and these results confirm an association between stressful events and worse menopause symptoms. However, results here suggest it is not necessarily the occurrence of a stressor that exacerbates menopause symptoms, but how the woman psychologically responds to it. In this sample, women who were "still upset" by a stressful event reported the most severe symptoms, whereas women who had experienced the same event but were not upset by it did not report significantly worse symptoms. This suggests that resilience may be important in the relationship between stress and menopause symptoms; however, further research is required to establish this. Little evidence was found for support reducing menopause symptoms, or acting as a buffer against the effects of stress. This may be relevant for understanding treatments and interventions for menopause symptoms, in particular, whether support-based treatments to tackle menopause symptoms are effective. Future research should focus on whether social interventions do have the desired effect on menopause symptoms, and also whether certain personality types – in regards to resilience – experience menopause in a different way.

CHAPTER SIX

Menopause status and kin care

6.1 Chapter summary

Within evolutionary sciences, care towards younger kin is well understood from an inclusive fitness framework, but why adults would care for older relatives has been less well researched. One existing model has argued that care directed towards elderly parents might be adaptive because of their benefits as carers themselves, with their help freeing up the middle generations' energy which can then be invested into direct reproduction. However, in this model, elder care is more beneficial to fitness if the carer is fecund. To offer an initial test of this hypothesis, in this chapter I look at caring behaviour relative to fecundity status in a dataset from the UK. If elder care is contingent on possible direct fitness benefits, I would expect women who are still menstruating to care more for their parents than women who can no longer reproduce. Based on this, I also predict that women who are physiologically post-reproductive would invest more in their grandchildren, through whom they can increase their inclusive fitness. After controlling for age and other relevant factors, I find that women who are still menstruating spend more time caring for their parents than those who are not, and the reverse is true when looking at time spent caring for grandchildren. These findings demonstrate that potential inclusive fitness outcomes influence how women allocate care up and down the generations.

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6.2 Introduction

Kin care is ubiquitous in human cultures, with cooperative breeding being a key component of our success as a species (Hawkes et al., 1998; Kramer, 2010). While kin structures in many WEIRD societies have shifted dramatically in recent years, there are still societal expectations in place for kin to assist in allocaring. Usually investment flows through families down the generations: younger kin are helped by older kin (Kaplan, 1994; Lee, 2000; Lee, 2013); and the amount of allocare received is generally predicted by degree of relatedness and the reproductive value of the

recipient, so that any costs incurred by helping will be offset by the possible inclusive fitness benefits (Hamilton, 1964; Crittenden & Marlowe, 2008; Page et al., 2019). However, it is common to observe younger individuals caring for older family members such as parents and grandparents. Cultural norms promoting elder care are widespread (Silverstein et al., 2006; de Valk & Schans, 2008; Mureşan & Hărăguş 2015), including in hunter-gatherer populations (Biesele & Howell, 1981; Hooper et al., 2015), and such behaviour is encoded in many religions and moral codes, with these injunctive norms being further institutionalised in some countries where there are laws making filial care (care from children to parents) a legal requirement (Pearson, 2013; Serrano et al., 2017). Despite the prevalence of elder care, it is poorly understood and seldom empirically researched from an evolutionary perspective.

Much of the evolutionary research looking at kin care has focussed on care flowing down the generations. As a result, there is an ever-growing body of empirical research demonstrating the inclusive fitness benefits associated with care directed from older family members to younger ones (Hawkes et al., 1998; Hawkes, 2003; Lahdenpera et al., 2004; Gibson & Mace, 2005; Sear & Mace, 2008; Chapman et al., 2021), with such indirect fitness gains thought to be responsible for the maintenance of the female post-reproductive lifespan (Hawkes et al., 1998). While the principles of kin selection can explain why people invest heavily in younger kin who are reproductive, why adults would invest in their older relatives is less clear from a fitness perspective. Older women are likely to be post-menopausal and therefore unable to directly reproduce, making the possible indirect fitness gains minimal; and fathers of adults may physiologically be able to reproduce in old age, but their fertility will be constrained by mate availability, as it is likely these older men will be partnered with women of a similar age who are post-menopausal making reproduction unlikely (Harris et al., 2011). Despite the apparent limited inclusive fitness gains to be had from the behaviour, caring for elderly parents is a cross-cultural norm.

At present, I am only aware of one model attempting to address elder care from a fitness maximising framework (Garay et al., 2018). Here, in a demographic model it is proposed that upwards intergenerational care would be selected for due to the possible reciprocal benefits of the behaviour. Graphically described in Figure 6.1, it is modelled that in a four-generational family structure if an adult (e.g. G3 in Figure

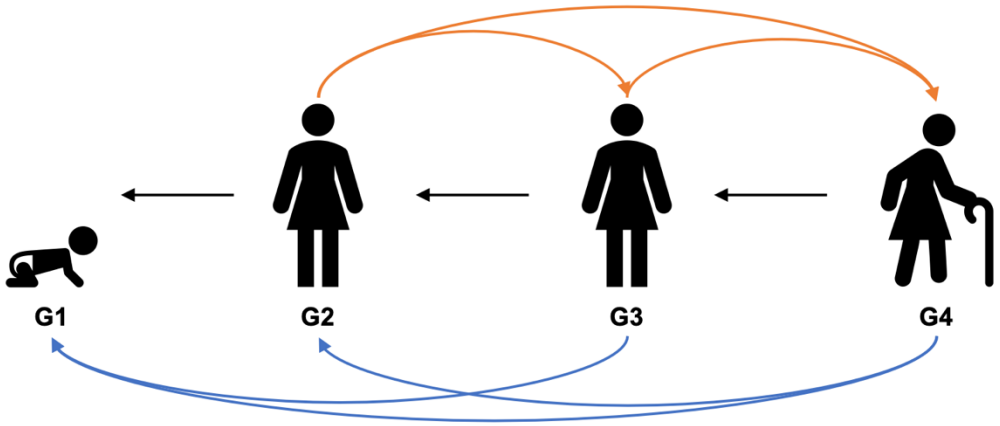

Figure 6.1. *Graphical description of the model proposed by Garay et al. suggesting that elder care would have evolved because of their benefits as carers themselves. Here, care from G3 to G4 is thought to be adaptive if G4 invests more in the younger generations (e.g. G1, G2) which frees up G3s energy that can be allocated to increasing direct fitness. Upwards intergenerational care is represented by orange arrows, downwards intergenerational care by blue arrows, and black arrows indicate parental care.*

6.1) directs care towards an older adult (e.g. G4 in Figure 6.1), it will make the latter group more able to offer downwards intergenerational care, such as to grandchildren or great-grandchildren (e.g. G4 to G1 in Figure 6.1). This additional instrumental help from elderly family members (i.e. in the form of additional childcare) decreases the burden of parental care for the middle generation adults, which frees up some of their energy that can be invested into other activities such as foraging (which they would do with higher efficacy than older kin) or reproduction (Cyrus & Lee, 2013). Therefore, care towards the elderly would be selected for due to their benefits as carers themselves. Though the elderly (G4) would already have an incentive to invest in younger kin due to the inclusive fitness benefits, it is thought that through receiving care from adults (e.g. G3), it makes the elderly *more physically able* to offer care towards younger relatives (e.g. G1). This additional help from older, post-reproductive individuals then frees up some of the fertile adults' energy, which can be allocated into other activities that they would do at higher efficacy than the older generation (such as working, foraging, and so on) or into direct reproduction (which it would not be possible for the older generation to do). Hence, the distribution of labour and activities that increase fitness are being optimally divided up between the family, and so each generation is doing what task they are 'best' at (Garay et al., 2018). Within Garay et al. (2018)'s model, it is assumed that the adults directing care towards the elderly are fecund and therefore physiologically able to increase their

fitness directly. This is because, if G3 is fecund, then through investing in G4 she may improve the health and longevity of that generation, meaning that there is another helping-hand to assist with caring for the younger generations (i.e. G1 in Figure 6.1), allowing her to invest energy into direct reproduction. If G3 is post-fecund and therefore unable to increase her direct fitness, then caring for younger family members is her only route to increasing fitness, and hence the possible reciprocal benefits to be had from helping elderly parents will wane alongside her fecundity, as after menopause she would no longer be able to translate any reciprocated help into direct reproduction. In line with this, once a woman is post-menopausal and the benefits to be had from investing in older relatives decline (though do not disappear completely), it may be better from a fitness perspective to invest energy into relatives through whom there is a way to increase inclusive fitness, such as children or grandchildren. Of course, the elderly generation would invest in grandchildren and children regardless of whether or not G3 offered upwards investment, however, this additional investment is hypothesised to make G4 *more able* to provide effective downwards care.

Currently, the hypothesis outlining the conditions for elder care to be evolutionarily adaptive has not been tested empirically. As the benefits of elder care are greater if the adult can reproduce, I test the hypothesis using a sample of women from the NCDS, some of whom are still menstruating and some of whom who are not, to look at care directed towards parents relative to fecundity status based on the predictions presented in Figure 6.1, with this chapter focussing on the behaviour of G3 from this model. As the benefits of elder care are predicted to decrease if the actor is physiologically post-reproductive, I would expect women who can no longer have children to invest less in their parents than those who can. Further, I additionally test whether there is a difference in downwards intergenerational care relative to fecundity status. If women are optimally allocating their time, then I would predict that women who are no longer menstruating would invest in younger relatives to a greater degree than women who are still fecund. Should caring behaviour relate to fitness outcomes that change relative to fecundity status, it may provide us with some information on how caring norms may have evolved.

6.3 Materials and methods

6.3.1 Data

Data were drawn from the NCDS, which is a nationally representative study that has followed a cohort of participants all born in a single week in the UK since 1958. Since birth, they have been followed up a total of 11 times at ages 7, 11, 16, 23, 33, 42, 44, 46, 50 and 55. As data on time spent caring for grandchildren is only available from the most recent interview, all analyses here are cross-sectional, with all women included in the sample being aged either 55 or 56 (depending on whether the interview was conducted in 2013 or 2014). Women in this sample are 'G3' from Figure 6.1, with both grandchildren and parents alive, meaning that I am looking at care within a four-generation family. This is partially because of data availability, with women in this dataset not being asked about how much their children care for them/how much they care for their children; and partially because the model of interest is based on a four-generation family, meaning the data in this chapter is suitable for testing the hypothesis. Though families with four generations alive are historically rare in cultures where there is a late age of first birth, due to longer life expectancies and improved health they are becoming increasingly common (Powell, 2018).

6.3.2 Variables

6.3.2.1 Hours spent helping parents per week

Information regarding parental caregiving was included as a count variable. In the most recent interviews, participants were asked whether they ever do various activities for their parents (e.g. shopping for them, helping with basic personal needs, giving them lifts, etc.), and if they do, how many hours on average per week do they spend doing so. Any women who reported not helping their parents do any of the activities were coded as helping their parents for zero hours per week.

6.3.2.2 Hours spent caring for grandchildren per month

The number of hours spent caring for grandchildren per month was also included as a count variable. Women were asked whether they ever look after their grandchildren without the grandchild's parents being present, and if they do, at what frequency and for how many hours. Women who stated that they did not care for their

grandchildren or did so less often than monthly were coded as caring for their grandchildren for zero hours per month. This measure also includes overnight stays.

6.3.2.3 Fecundity status at age 55

Fecundity status was derived from information on age, year, and reason for last menstrual period, which was collected at ages 44, 50, and 55. Based on this, a binary categorical variable was derived where women were coded as either 'Still menstruating' or 'No longer menstruating'. The latter category comprised of women who were post-menopausal or who had stopped menstruating for another reason, such as a surgical menopause. Women who had stopped menstruating due to menopause or other reasons were grouped together as the direct fitness implications of no longer menstruating are the same, regardless of the reason for it.

6.3.2.4 Control variables

Covariates included were selected based on their expected effect on the woman's ability to help other family members. As a proxy of socioeconomic status, the age at which the woman left education was included. Employment status was utilised to give an indication of the woman's time constraints (i.e. if she was employed, it can be expected she had less time to care for kin) (Ciccarelli & Van Soest, 2018), with women being coded as either employed, unemployed, or other, with the latter category including those who are doing something other than formal employment but do not classify themselves as unemployed (e.g. retired, volunteering, studying, etc.). Self-perceived health was used as a measure of how physically able the woman is to help family members (Pavalko & Woodbury, 2000), and number of grandchildren was also included to adjust for how many grandparenting responsibilities a woman had. I also included information on the mortality status of the woman's parents (i.e. whether she had both parents alive or not), which was derived from interviews at ages 7, 11, 16, 23, 42, 46, 50 and 55. The focal woman's mother's and father's age at birth (collected in the perinatal interview) were also included to control for the amount of help her parents may need, as older parents would be expected to be more in need of assistance. Finally, in models predicting hours spent caring for parents, time spent caring for grandchildren was adjusted for, and vice versa for models where hours spent caring for grandchildren was the outcome.

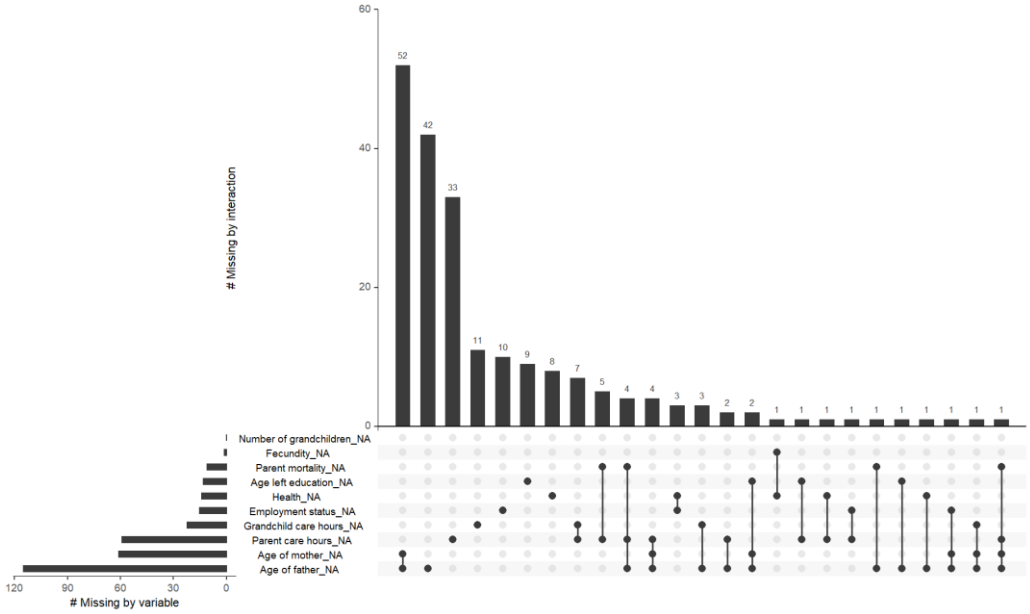

Figure 6.2. *Missing data by variable. Horizonal bar chart on the left shows the number of missing observations by variable. Main plot shows the interaction between missing variables, and the amount of missingness relating to those interactions (e.g. 52 participants had missing data for both their age of mother and age of father variables, 42 women had missing data for just the age of their father, and so on).*

6.3.3 Sample

As I am interested in looking at how women care when there are multiple generations alive to care for, the sample was limited to just women who had at least one parent alive and at least one grandchild, which left a sample of 1139 women out of the 4703 women who responded to this year of interviews. A complete case analysis was carried out, and therefore any women who had missing data were dropped, resulting in a final analytical sample of 934 women.

The distribution of missingness across the dataset is plotted in Figure 6.2, where it can be seen that the majority of missingness came from data in the woman's father's age, age of mother, and hours spent caring for parents. The dropping of women who had missing data for the father's age likely biased the sample somewhat. Parental age was collected at birth, and while in some cases the missing data just represents random missingness, in other cases it will represent the father age being unknown to the mother and possibly the father not involved in the child's life (Power & Elliott, 2006). Hence, the missing data in this variable may be a proxy of a single parent household. In the 1950s, when this variable was collected, the nuclear family household structure was very much the norm in the UK, with single mothers likely being of a lower socioeconomic position than those in a stable partnership, and

the non-response rates also being higher amongst those whose mothers were unmarried at birth (Hypponen et al., 2005). Therefore, by dropping women from the study whose father's age was not recorded at their birth, it may be biasing the sample slightly towards women who had a slightly higher socioeconomic position at birth.

6.3.4 Analyses

Time spent helping parents and caring for grandchildren were both modelled using zero-inflated negative binomial regression (ZINB). This modelling procedure was selected both due to the over-dispersed nature of the data with excess zeros, and because zero-inflated models allow for zeros to be generated through two distinct processes. Here, the model distinguishes between excess zeroes, which occur when the event could not have happened, and true zeros, which occur when there could have been an event. Therefore, the model estimates a binary outcome (does not care versus does care) and a count outcome (the number of hours spent caring). This method is theoretically appropriate, as there are many different reasons people would offer no care to kin: while some people may choose to invest less, for some people the choice is out of their control, with external factors influencing caring behaviours, such as living far away from kin (Engelhardt et al., 2019). In addition to this, ZINB was found to better fit the data than negative binomial regression (Appendix F Table F1).

Time spent helping parents was first modelled. A 'base' model was first made containing the age the woman left education, employment status, marital status, self-perceived health, number of grandchildren, parent mortality status, age of parents, and time spent caring for grandchildren. Fecundity status was subsequently added, and model fitting then carried out on these two models, utilising their AIC value to understand whether a model including fecundity better fit the data than one without. The model with the lowest AIC value is taken to best fit the data. As AIC values penalise models for complexity, it means the model with the most terms will not automatically be selected as the best. The ΔAIC was also calculated, which is the difference between the candidate models AIC and the AIC value of the best fitting candidate model. If the ΔAIC value is ≤ 2, then it indicates that there is still good evidence to support the candidate model, meaning that a candidate model with a ΔAIC of ≤ 2 is almost as good as the best fitting model. A ΔAIC value of between 4 and 7 is taken to indicate the candidate model has considerably less support, and a

Table 6.1. *Participant characteristics stratified by fecundity status, showing n and % or median and interquartile range.*

	All	**Still menstruating**	**No longer menstruating**
n	934	98	836
Hours spent caring for grandchildren per month	18.00 (0.00, 48.00)	13.50 (0.00, 32.00)	20.00 (0.00, 48.00)
Hours spent helping parents per week	2.00 (0.00, 5.00)	1.00 (0.00, 6.00)	2.00 (0.00, 5.00)
Fecundity status			
Still menstruating	98 (10.49)	98 (100.00)	0 (0.00)
No longer menstruating	836 (89.51)	0 (0.00)	836 (100.00)
Age left education	16.00 (16.00, 17.00)	16.00 (16.00, 18.00)	16.00 (16.00, 17.00)
Employment status			
Employed	717 (76.77)	80 (81.63)	637 (76.20)
Unemployed	68 (7.28)	6 (6.12)	62 (7.42)
Other	149 (15.95)	12 (12.24)	137 (16.39)
Self-perceived health			
Excellent	101 (10.81)	15 (15.31)	86 (10.29)
Very good/good	615 (65.85)	65 (66.33)	550 (65.79)
Fair	144 (15.42)	11 (11.22)	133 (15.91)
Poor/very poor	74 (7.92)	7 (7.14)	67 (8.01)
Father's age at birth	27.00 (24.00, 30.00)	27.00 (24.00, 31.75)	27.00 (24.00, 30.00)
Mother's age at birth	24.00 (21.00, 27.00)	24.00 (22.00, 28.00)	24.00 (21.00, 27.00)
Parent mortality			
Both parents alive	315 (33.73)	40 (40.82)	275 (32.89)
Mother dead, father alive	153 (16.38)	14 (14.29)	139 (16.63)

Table 6.1. *Participant characteristics stratified by fecundity status, showing n and % or median and interquartile range.*

	All	Still menstruating	No longer menstruating
Mother alive, father dead	466 (49.89)	44 (44.90)	422 (50.48)
Number of grandchildren	2.00 (1.00, 4.00)	2.00 (1.00, 4.00)	2.00 (1.00, 4.00)

ΔAIC of greater than 10 indicates there is no support for the candidate model (Burnham & Anderson, 2002). The Akaike weights (w_i) were also calculated to evaluate model fit, which give the probability that the candidate model is the best among the set of presented candidate models (Burnham & Anderson, 2002). The same procedure was then used to model time spent caring for grandchild per month: a model including just the covariates was first made, but this time adjusting for time spent helping parents rather than time caring for grandchildren, with fecundity status then being added, and model fitting was once again carried out using the methods outlined above. All analyses were carried in *R* (R Core Team, 2020) using the *zeroinfl* function with a negative binomial distribution specified (Zeileis et al., 2008), and model fitting carried out with the package *AICcmodavg* (Mazerolle, 2020). All visualisations were created using *ggplot2* (Wickham, 2016).

6.4 Results

In this sample of women from the UK (n = 934), the majority were post-menopausal or had stopped menstruating for another reason (n = 836). Women spent a median of 18 hours per month caring for grandchildren (IQR: 0, 42) and 2 hours a week caring for parents (IQR: 0, 5). Time spent caring was slightly greater towards both generations in women who were no longer menstruating, who spent 20 hours a month (IQR: 0.00, 48.00) caring for grandchildren and 2 hours per week (IQR: 0.00, 5.00) helping parents; compared to 13.5 hours a month (IQR: 0, 32) and 1 hour a week (IQR: 0, 6) caring for grandchildren and parents, respectively, in women who are still menstruating. Full participant characteristics are shown in Table 6.1.

For both outcome variables, the inclusion of fecundity within the models improved their fit to the data based on AIC value (Table 6.2). Within the negative

Table 6.2. *Results from model fitting based on Akaike Information Criterion (AIC). The lowest AIC value is deemed to best fit the data, with ΔAIC referring to the difference in AIC value from the best fitting model (shown in italics). A ΔAIC value of more than two demonstrates a significantly poorer model fit, and w_i indicates model probability. Covariates include number of grandchildren, age the woman left education, employment status, marital status, and health. Where the outcome is hours spent helping parents, hours spent caring for grandchildren is included in the covariates, and vice versa.*

Model	**K**	**AIC**	**ΔAIC**	w_i
Outcome = hours spent helping parents				
Covariates	27	4534.02	0.90	0.39
Fecundity status + covariates	*29*	*4533.12*	*0.00*	*0.61*
Outcome = hours spent caring for grandchildren				
Covariates	27	7184.35	6.59	0.04
Fecundity status + covariates	*29*	*717776*	*0.00*	*0.96*

binomial parts of the best fitting models, women who were no longer menstruating were predicted to spend significantly less time helping parents (IRR: 0.65, 95% CI: 0.43-0.97) and more time caring for grandchildren (IRR: 1.55, 95% CI: 1.19-2.02) compared to women who are still menstruating (Figure 6.3). The age a woman left

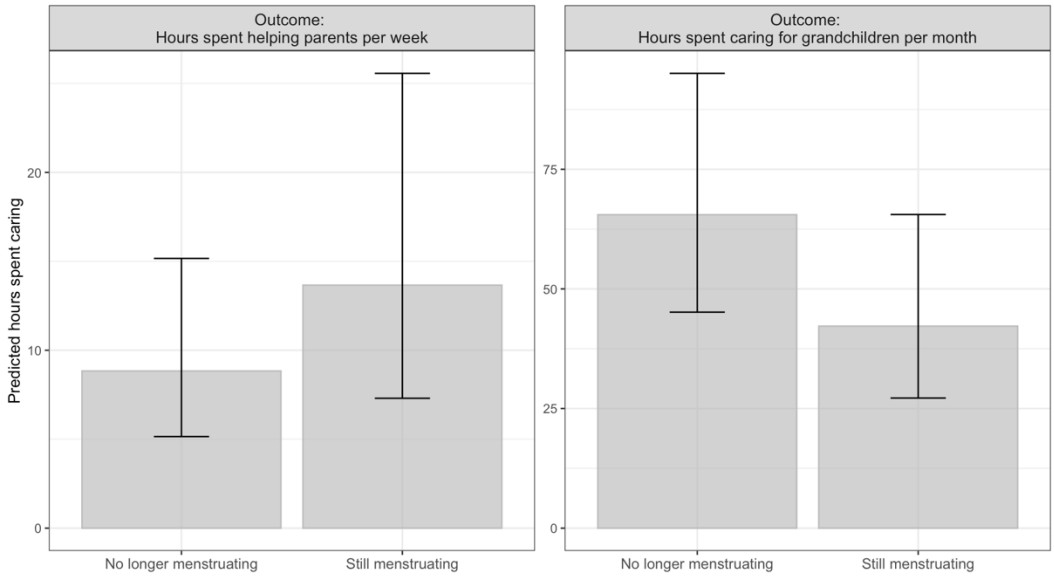

Figure 6.3. *Predicted number of hours spent helping parents and caring for grandchildren relative to fecundity status, based on the results from negative binomial parts of the best fitting models. Error bars indicate the 95% confidence intervals. Models adjust for number of grandchildren, the age the woman left education, employment status, marital status, and health. Where hours spent helping parents is the outcome, time spent caring for grandchildren is also controlled for, and vice versa where hours spent caring for grandchildren is the outcome*

Table 6.3. *Results from all models using zero-inflated negative binomial regression, reporting incidence rate ratios for the count part of the model and odds ratios for the zero-inflated part, and the 95% confidence intervals.*

	Outcome = Hours spent helping parents per week		**Outcome = Hours spent caring for grandchildren per month**	
	Covariates	**Fecundity status + Covariates**	**Covariates**	**Fecundity status + Covariates**
<u>Count model:</u>				
Fecundity status (ref.: Still menstruating)				
No longer menstruating	-	0.65 (0.43-0.97)	-	1.55 (1.19-2.02)
Age left education	0.91 (0.81-1.01)	0.89 (0.80-1.00)	0.93 (0.87-1.00)	0.94 (0.88-1.01)
Employment status (ref.: Unemployed)				
Employed	0.35 (0.19-0.63)	0.35 (0.19-0.62)	0.83 (0.60-1.15)	0.84 (0.61-1.16)
Other	0.52 (0.28-0.98)	0.49 (0.26-0.91)	1.40 (0.98-2.01)	1.37 (0.96-1.96)
Self-perceived health (ref.: Poor/very poor)				
Fair	1.04 (0.58-1.85)	1.11 (0.63-1.97)	1.09 (0.78-1.53)	1.10 (0.79-1.54)
Very good/good	1.39 (0.80-2.43)	1.42 (0.82-2.46)	1.00 (0.72-1.38)	1.00 (0.72-1.37)
Excellent	0.84 (0.43-1.64)	0.84 (0.44-1.63)	1.03 (0.69-1.53)	1.03 (0.70-1.53)
Parent mortality (ref.: Both parents alive)				
Mother alive, father dead	1.17 (0.90-1.51)	1.21 (0.94-1.57)	1.04 (0.86-1.26)	1.04 (0.86-1.25)

Table 6.3. *Results from all models using zero-inflated negative binomial regression, reporting incidence rate ratios for the count part of the model and odds ratios for the zero-inflated part, and the 95% confidence intervals.*

	Outcome = Hours spent helping parents per week		**Outcome = Hours spent caring for grandchildren per month**	
	Covariates	**Fecundity status + Covariates**	**Covariates**	**Fecundity status + Covariates**
Mother dead, father alive	2.05 (1.39-3.03)	2.14 (1.45-3.15)	0.90 (0.70-1.14)	0.88 (0.69-1.12)
Mother's age at birth	1.03 (0.99-1.06)	1.04 (1.00-1.08)	1.00 (0.97-1.02)	0.99 (0.97-1.02)
Father's age at birth	1.02 (0.99-1.05)	1.01 (0.98-1.04)	1.00 (0.98-1.02)	1.00 (0.98-1.02)
Number of grandchildren	1.00 (0.96-1.05)	1.00 (0.95-1.05)	0.99 (0.96-1.02)	0.99 (0.96-1.02)
Hours spent helping parents per week	-	-	1.00 (1.00-1.01)	1.00 (1.00-1.01)
Hours spent caring for grandchildren per month	1.00 (1.00-1.00)	1.00 (1.00-1.00)	-	-
Zero-inflated model:				
Fecundity status (ref.: Still menstruating)				
No longer menstruating	-	0.69 (0.30-1.60)	-	0.82 (0.52-1.28)
Age left education	1.09 (0.84-1.42)	1.06 (0.81-1.39)	1.11 (1.00-1.25)	1.11 (0.99-1.24)
Employment status (ref.: Unemployed)				
Employed	0.34 (0.11-1.06)	0.34 (0.11-1.04)	0.94 (0.49-1.78)	0.93 (0.49-1.77)
Other	0.50 (0.14-1.78)	0.48 (0.14-1.66)	1.09 (0.55-2.17)	1.09 (0.55-2.17)

Table 6.3. *Results from all models using zero-inflated negative binomial regression, reporting incidence rate ratios for the count part of the model and odds ratios for the zero-inflated part, and the 95% confidence intervals.*

	Outcome = Hours spent helping parents per week		**Outcome = Hours spent caring for grandchildren per month**	
	Covariates	**Fecundity status + Covariates**	**Covariates**	**Fecundity status + Covariates**
Self-perceived health (ref.: Poor/very poor)				
Fair	0.57 (0.14-2.24)	0.66 (0.18-2.39)	0.61 (0.32-1.17)	0.61 (0.32-1.18)
Very good/good	0.81 (0.26-2.51)	0.82 (0.27-2.51)	0.81 (0.44-1.50)	0.82 (0.45-1.50)
Excellent	0.69 (0.14-3.44)	0.69 (0.14-3.37)	1.11 (0.55-2.25)	1.11 (0.55-2.24)
Parent mortality (ref.: Both parents alive)				
Mother alive, father dead	0.45 (0.17-1.17)	0.47 (0.18-1.18)	0.99 (0.71-1.37)	0.99 (0.71-1.38)
Mother dead, father alive	3.10 (1.52-6.33)	3.17 (1.56-6.47)	1.05 (0.69-1.61)	1.06 (0.69-1.62)
Mother's age at birth	0.93 (0.83-1.03)	0.94 (0.84-1.04)	1.03 (0.99-1.08)	1.03 (0.99-1.08)
Father's age at birth	0.97 (0.87-1.08)	0.96 (0.86-1.07)	0.99 (0.95-1.03)	0.99 (0.95-1.03)
Number of grandchildren	0.97 (0.86-1.09)	0.96 (0.85-1.09)	0.94 (0.88-1.00)	0.94 (0.89-1.00)
Hours spent helping parents per week	-	-	0.94 (0.91-0.97)	0.94 (0.91-0.97)
Hours spent caring for grandchildren per month)	0.99 (0.97-1.00)	0.99 (0.97-1.00)	-	-

education was negatively associated with both caring behaviours, with women who were educated to an older age spending less time caring for both parents (IRR: 0.89,

95% CI: 0.80-1.00) and grandchildren (IRR: 0.94, 95% CI: 0.88-1.01). Being employed predicted significantly less help directed towards parents (IRR: 0.35, 95% CI: 0.19-0.62) but not grandchildren. Variables pertaining to parental health were also related to filial, but not grandchild, care; with having only a father alive (IRR: 2.14, 95% CI: 1.45-3.15) and an older mother (IRR: 1.04, 95% CI: 1.00-1.08) predicting more time spent helping parents. Self-perceived health did not predict either caring behaviour. Within the part of the models predicting excess zeros, no longer menstruating was not a significant predictor of care towards grandchildren (odds ratio [OR]: 0.82, 95% CI: 0.52-1.28) or parents (OR: 0.69, 95% CI: 0.30-1.60). Having just a surviving father was a predictor of parental care (OR: 3.17, 95% CI: 1.56-6.47). Full model results are shown in Table 6.3.

6.5 Discussion

Despite elder care being common cross-culturally, little empirical research from an evolutionary perspective has been conducted looking at the behaviour. In this chapter, I test a hypothesis proposing that upwards intergenerational care will be selected for as long as the actor is able to translate some of the proposed reciprocal benefits of the care into direct fitness, meaning that upwards care being adaptive is somewhat dependent upon the fecundity status of the actor. Based on this, I predicted that women who were still fecund would invest more in elder care than those who were not, as the former would be able to benefit more from any reciprocal care through increasing direct fitness. I further test the assumption that caring behaviour is contingent on the fitness outcomes through looking at whether women who are no longer able to reproduce invest more in younger relatives than women who are still menstruating. In this sample of women from the UK, support was found for this hypothesis, and it was observed that women who were still menstruating spent more time caring for their parents than women who had stopped (Figure 6.3), even after adjusting the potential 'need' their parents had for care (e.g. mortality status and parental age). This is in line with the predictions made by Garay et al. (2018): if a fertile woman helps her parents, then they reciprocate the care, therefore allowing her more time to invest in other activities, such as reproduction. As such, the benefits of investing in elderly parents would diminish as women become physiologically post-reproductive, thus explaining the difference in care directed towards parents relative to fecundity status.

I also found that post-reproductive women spent almost double the number of hours per month caring for their grandchildren compared to still menstruating women. This supports existing research suggesting that menopause may have evolved initially due to grandmothers benefits as carers (Hawkes et al., 1998; Lahdenpera et al., 2004; Sear & Mace, 2008), and also mimics findings from research by Hofer et al. (2019) where it was shown that post-menopausal women spend more time grandparenting compared to the control of volunteering. Taken together, these findings suggest that increased levels of care towards younger kin when physiologically post-reproductive might be a behavioural adaptation that was selected for to offset the costs of no longer being able to directly reproduce. This may occur at a proximate level by reallocating metabolic resources that were previously being allocated to regulating and maintaining reproductive physiology into hormonal mechanisms that affect caring behaviour towards kin (Lovejoy et al., 2008; Rilling, 2013). It should be noted that this study did not look at whether the grandchildren of women who spend more time with them are actually more successful than grandchildren to whom less time is devoted as I did not have the data to test this, and therefore I cannot say whether this behaviour was adaptive or not. However, other research has found that investing more in kin does not always translate into increased fitness down the line in modern, post-industrial societies (Goodman et al., 2012), and that low levels of familial investment in societies with a welfare state (such as the UK) might be compensated by investment from public goods, such as schooling (Downey, 2001). I did not find any relationship between fecundity and caring behaviour within the zero-inflated part of the model, but this is likely because caring for kin is often not possible due to restrictions such as proximity (Engelhardt et al., 2019), which have no bearing on whether a woman is fecund or not.

6.5.1 Limitations

This chapter looks at caring dynamics within a four-generation family, both due to the original set up of the model proposed by Garay et al. (2018) and due to the structure of the data I had available to me. Though this family structure is becoming more common in some cultures due to longer life expectancies, it was not necessarily the norm in our evolutionary history. Therefore, future research could aim to look at testing this model in a more common three-generation family, and also perhaps

looking at other older members of kin (e.g. great-aunts/uncles) to see if the findings, and apparent energetic trade-off, still hold.

It should be noted that the validity of applying this hypothesis could be brought into question based on the age of the women in this data. My sample included only women aged 55 or 56, and based on reproductive norms and oocyte degradation, even if the women were still menstruating at this age, it is unlikely they would actually reproduce (Towner et al., 2016; Gottschalk et al., 2019). Further to this, the parents of the women in this study are, on average, in their early 80s (Table 6.1), and evidence has shown that the elderly's benefits as allocarers declines with age (Chapman et al., 2019), which would suggest that the returns on filial care would also decline. If our tendency to help older generations is a behavioural adaptation that evolved for the reasons proposed by Garay et al. (2018), it could be that the adaptation is somewhat mismatched to our current environment where there are longer generations and longer lifespans, that have resulted in grandparents being older. However, investing in elderly parents might be reciprocated in ways other than allocare, such as through financial or material rewards, with more filial care possibly resulting in more money or gifts being informally given to the carer or their relatives (e.g. children or grandchildren) as a reciprocal gesture. This form of care was not factored into the original model, however, research has demonstrated that financial transfers from the elderly do not associate with help from the middle generation (Arrondel & Masson, 2006), meaning that it may not be an incentive. One could also take a more sociological stance and argue that elder care does not require an evolutionary explanation and that it can be explained proximally through various social norms and institutions (Brody, 1985). However, neither financial rewards nor social norms can explain why there is a significant difference in care-giving behaviour towards parents relative to fecundity status, as I show here.

Being limited to data from only women aged 55 or 56 also meant that few were pre-menopausal. Though the average age of menopause may be increasing (Chan et al., 2020), still being fecund at age 55/56 is relatively rare, which is reflected by the fact that only ~10% of the women included in this research have not yet stopped menstruating. While there are various reasons women may have stopped menstruating, a proportion would have gone through menopause, and a later menopause is generally associated with being of a higher socioeconomic status and better health (Gold et al., 2001; Gold, 2011). The sample may be further biased

towards women of a higher socioeconomic position, as many participants were dropped due to having missing data for their father's age. This data was collected at birth, and missingness here is indicative of the father either being absent and/or not known to the mother; with such marital status at birth being associated with a lower socioeconomic position at birth and poorer outcomes across the life course (Power & Elliott, 2006; Harkness et al., 2020). Hence, the sample would have likely already been biased towards women of a higher socioeconomic position due to choosing to use a complete case analysis, which such bias being further exacerbated by focussing exclusively on women that have not experienced menopause by age 55/56. Though I have attempted to control for these various lifestyle factors, through adjusting for health, employment, and educational attainment, it is likely that the sample of women included in this analysis do not represent the 'average' middle aged woman. Ideally, longitudinal data collected at more regular intervals would have been used; but I am restricted by the NCDS interview patterning, with there being large gaps between interviews, and questions regarding grandchildren only being asked in the 2013/14 year of interviews. It has recently been suggested that longitudinal cohort studies should be utilised in the evolutionary sciences more (Sear, 2020b), and while they are beneficial due to the scale of the data available, we are often limited by the variables provided.

Nonetheless, to my knowledge, this is the first piece of empirical research looking at elder care from an evolutionary perspective, in which I have found evidence for kin-directed care by women to be facultatively adjusted up and down the generations based on their own fecundity status.

CHAPTER SEVEN

Menopause timing and inclusive fitness

7.1 Chapter summary

Prolonging the female post-reproductive lifespan via longevity has been shown previously to be adaptive. However, in addition to variation in when the lifespan ends, there is a large amount of variation in when the physiological post-reproductive lifespan begins, with population averages in menopause timing ranging between ~45 and 55 years. In this chapter, I look at whether age of menopause and final menstruation associate with indirect fitness. Using data from the NCDS, I show that women who experience an earlier age of final menstruation – particularly those who stopped before the age of 40 – have greater fitness at age 55 compared to those who menstruate until a later age. I also show that age of menopause and final menstruation do not predict direct fitness. Given that there is little variation in direct fitness, it brings into question what the optimal age of final menstruation is in WEIRD societies, which I discuss in reference to socioeconomic confounders. The results also provide further evidence for a post-reproductive lifespan having beneficial inclusive fitness outcomes.

7.2 Introduction

Amongst mammals, it is common for reproductive senescence to occur alongside death (Croft et al., 2015); however, in humans, females are able to survive well beyond reproductive cessation, resulting in them spending a large proportion of their lives post-reproductive (Levitis & Lackey, 2011). As infertility is generally not a trait predicted to be selected for by natural selection, multiple evolutionary theories have been put forward to explain why a prolonged reproductive lifespan might have evolved in humans. The most prominent of these theories is the Grandmother Hypothesis, which proposes selection might have favoured midlife infertility and prolonged post-reproductive survival as long as these women increase their fitness through assisting close kin with survival and reproduction (Hawkes et al., 1998; Hawkes, 2004; Kim et al., 2012; Thouzeau & Raymond, 2017; Kim et al., 2019). Since the development of this model, a great deal of empirical evidence has been

found in support of its assumptions. Children with living grandmothers have been found to have a better nutritional status and survival outcomes (Sear et al., 2000; Gibson & Mace, 2005; Nenko et al., 2020; Chapman et al., 2021), and older mothers have also been shown to increase their daughter's fitness through reducing interbirth intervals (Coall & Hertwig, 2010; Chapman et al., 2021), thereby suggesting women are able to offset the costs of being post-reproductive through increasing their indirect fitness. However, as outlined in Chapter 1, this grandmother effect has been found to be predicated on a number of factors, such as the grandmother's lineage and age, her distance from her grandchildren, and also the age and sex of the grandchildren (Beise & Voland, 2002; Gibson & Mace, 2005; Sheppard & Sear, 2016; Chapman et al., 2019; Chapman et al., 2021). Grandmothering is also thought to have contributed to the evolution of longevity in humans (Kim et al., 2012), with women gaining approximately two extra grandchildren for every ten years survived beyond the age of 50 (Lahdenpera et al., 2004).

Much of the existing research looking at the adaptive nature of the female post-reproductive lifespan has focussed on women who are either presumed to be post-menopausal (i.e. over a certain age cut-off, usually 50) or behaviourally post-reproductive (timing of last birth). However, as human females experience menopause, it also allows us to measure objective post-reproductivity, that is, through looking at the timing at which menopause (or age at last menstruation) occurs. Average age of menopause varies cross-culturally, ranging between 44.6 to 54.6 depending on geographic region (Laisk et al., 2019), meaning that women experiencing a menopause on the later end of the spectrum could theoretically have an extra 10 years of potential reproduction than women who go through menopause earlier. Though it is not currently clear what causes this variation in menopause timing, it is likely the result of an interaction between lifestyle (Gold et al., 2013) and genetic factors, with between 40-80% of the variance in menopause timing thought to be the result of the latter (Peccei, 1999; de Bruin et al., 2001; van Asselt et al., 2004; Murabito et al., 2005). While some studies have found a link between a later menopause and greater longevity (Shadyab et al., 2017), these results are not consistent (Tom et al., 2012), with findings likely being confounded by many factors such as smoking uptake and parity (Oboni et al., 2016; Shirazi et al., 2020). Therefore, an earlier menopause may result in a woman spending a greater proportion of her life post-reproductive than women who experience a later

menopause. It so follows that, if menopause is an adaptive characteristic of the female human life history, that women who go through menopause earlier should have greater indirect fitness (e.g. more grandchildren, better grandoffspring survival) to offset the costs of menopause. As shown in Chapter 6, it appears that post-menopausal women's caring behaviour shifts in the direction predicted if caring is used to promote inclusive fitness outcomes, proximately possibly as a result of metabolic resources that were previously being invested in regulating and maintaining reproductive physiology (Lovejoy et al., 2008) being redirected to hormonal mechanisms that influence caring behaviours towards kin (Rilling, 2013). While this provides indirect evidence for the female post-reproductive lifespan being used to increase inclusive fitness, due to the cross-sectional nature of the data, I could not show that increased post-reproductive care led to a greater number of grandchildren. However, using data from the same study I am able to look at the relationship between the timing at which physiological post-reproductive life begins and indirect fitness. If a post-reproductive lifespan is adaptive, I would expect the timing at which it occurs to be associated with increased indirect fitness (i.e. number of grandchildren) as means of offsetting the costs of no longer being able to directly reproduce. It has been shown previously that one's lifespan beyond 50 predicts increased indirect fitness (Lahdenpera et al., 2004), but the timing at which infertility occurs relative to indirect fitness gains has not yet been measured. Therefore, here I look at the timing of reproductive cessation and number of grandchildren, with the prediction that an earlier age of final menstruation should be offset by the woman having a greater number of grandchildren.

7.3 Materials and methods

Data were drawn from the NCDS, which is a study that has followed individuals all born in the same week in March 1958. Since birth, the participants have been interviewed a number of times, with the most recent wave of interviews occurring in 2013/14 when the individuals were aged between 55 and 56 (see Chapter 2 for a full description of the data). While both males and females were recruited for the NCDS, this analysis is limited to only participants who were coded as female in the first wave of interviews. The data is also limited to those who responded to the most recent interview request, as it was only in these interviews that the participants were asked how many grandchildren they have.

In my analyses, two variables to signal the end of fertility were used: age of natural menopause and age of final menstruation. The two were differentiated because evolutionary models primarily discuss natural menopause, but in WEIRD societies it is not uncommon for women to experience a final menstruation due to reasons other than menopause (e.g. a surgical menopause or medication). As being post-fecund has the same constraints on one's ability to reproduce regardless of the reason, I would expect the two variables to present similar relationships with number of grandchildren. Age of natural menopause was derived from information regarding whether or not a woman had experienced a menstrual period in the past 12 months, in addition to the age, year, and reason for last menstrual period, which was collected in interviews carried out when the women were aged 44, 50, and 55. From this information, a categorical variable was created to indicate at what age a woman had experienced menopause (40 or younger; 41-45; 46-50; 51-55; Stopped for another reason (e.g. medication, hysterectomy); Not yet gone through menopause). This variable was treated as categorical rather than continuous to allow for the retention of women who had not yet experienced menopause in the data. A second variable was then made which included women who had stopped menstruating for another reason within the age categories, based on the self-reported date of their final menstrual period that was followed by 12 months of amenorrhea.

Number of grandchildren served as the dependent variable in all analyses, with either age of menopause or age at last menstruation being included as the predictor of interest. A number of covariates were also included, which were selected

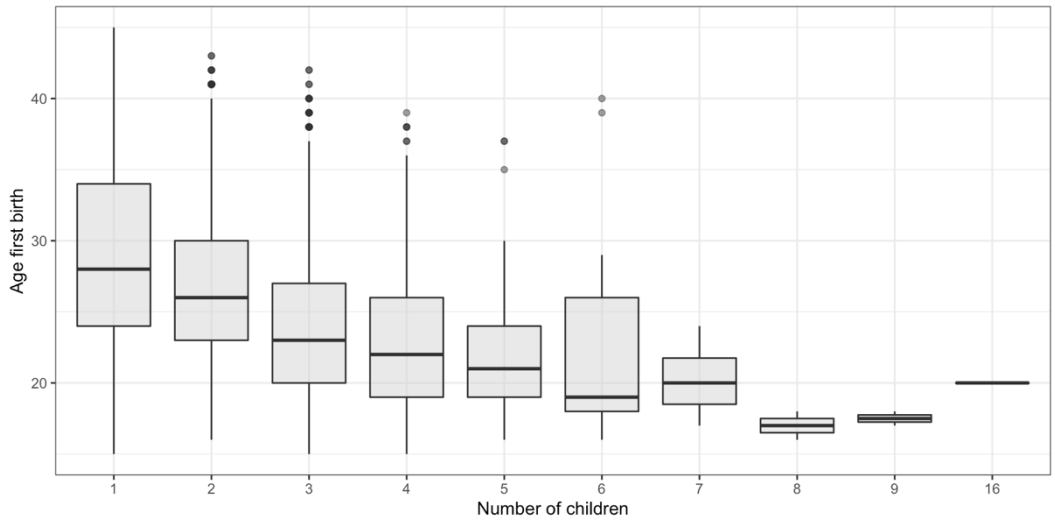

Figure 7.1. *Box and whisker plot showing the relationship between number of children and age at first birth (n = 3725).*

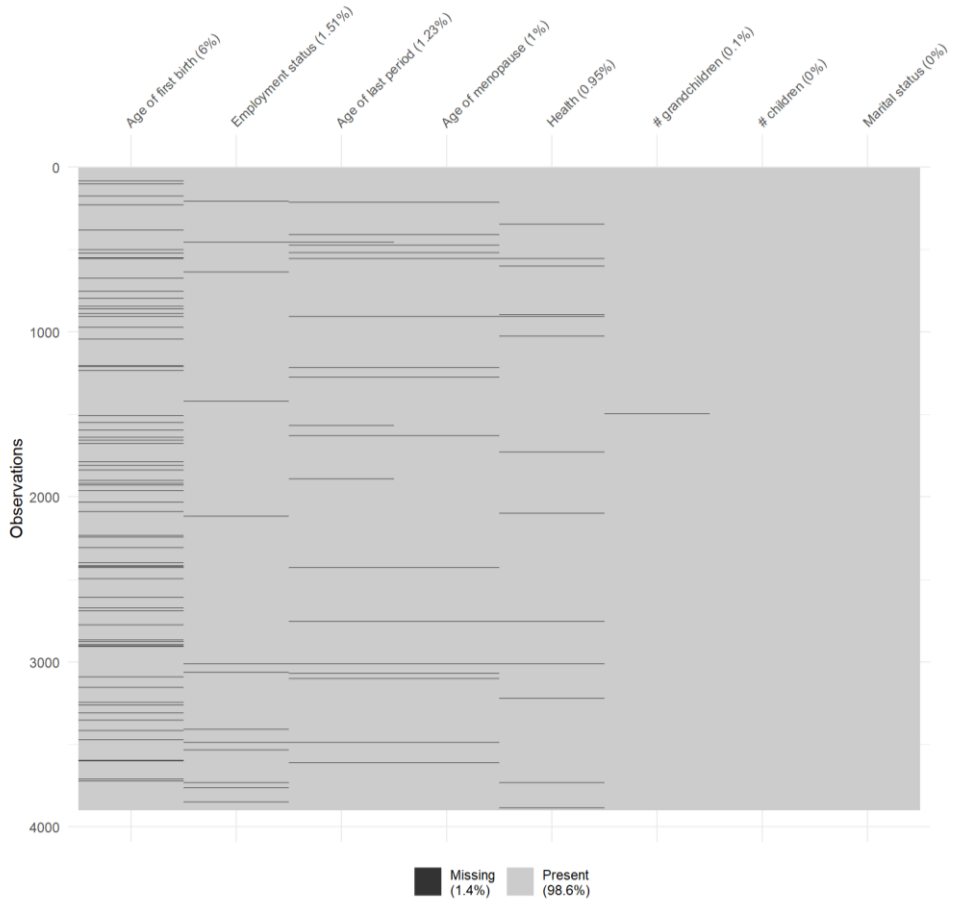

Figure 7.2. *Percentage of missing data by variable (n = 3897).*

based on existing literature pertaining to fertility. These included employment status and marital status, which have previously been linked to both direct and indirect fitness, in which being married and unemployed has been linked to higher female reproductive success (Goodman & Koupil, 2009). Health was also included as a confounder, as poorer health has been associated with lower reproductive success (Ekholm et al., 2005; Swamy et al., 2008). Further, in line with the Grandmother Hypothesis (Hawkes et al., 1998), healthier women will be more able to help with children out, which may increase the number of children they have, thus increasing her number of grandchildren. Two measures of direct reproduction were also adjusted for, which included number of children and age of first birth. These were included as number of grandchildren can be expected to strongly relate to both of them: those with more children likely have more grandchildren; likewise, those who had an earlier age of first birth will be more likely to have offspring of childbearing age, and therefore be more likely to have grandchildren than women who did not have their own children until their late 30s, for example. These measures were not included within the same models due to their high degree of collinearity (Figure 7.1).

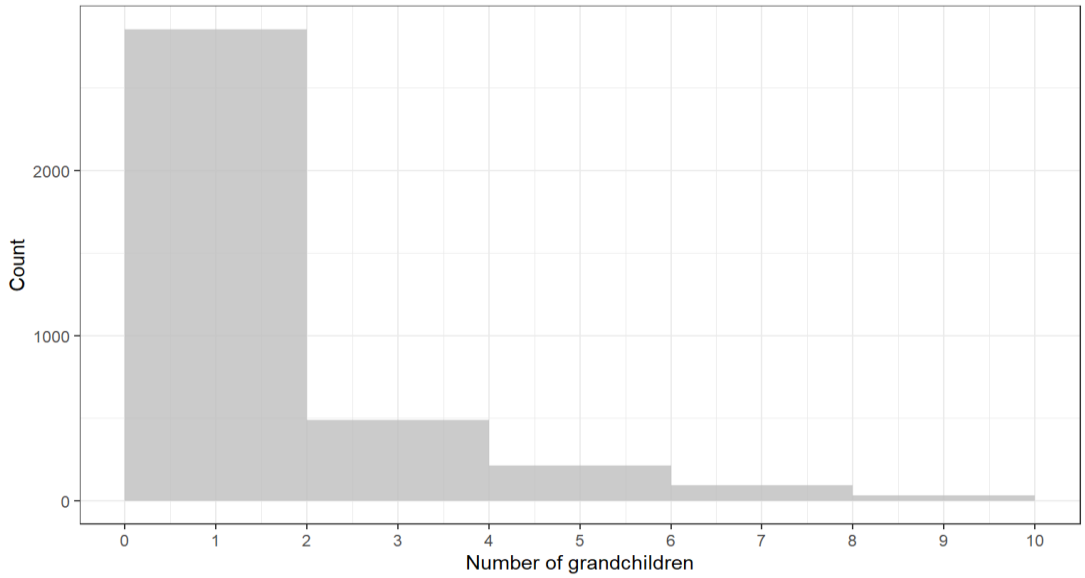

Figure 7.3. *Distribution of number of grandchildren within the sample.*

A complete case analysis including respondents who had information on the aforementioned variables was conducted. As data on grandchildren was only collected in the most recent interviews, the analysis is limited to just women who responded in that year (n = 4703), with women being dropped if they had conflicting information on fertility (e.g. reporting to having natural grandchildren, but no natural children), resulting in a sample of 4600. As I am interested in looking at the number of grandchildren, the data were further subset to include women who had more than one child (n = 3897). A complete case analysis was carried out, and so anyone with missing data was dropped. As can be seen in Figure 7.2, the amount of missingness across the variables was low (<2%), with age of first birth having the greatest amount of missing data (6%). As such, two different datasets were made for the analyses: one that did not include age at first birth to allow for a slightly larger sample size (Dataset 1; n = 3725), and one including age at first birth meaning women with missing data for this variable were also dropped resulting in a reduced sample (Dataset 2; n = 3519).

7.3.1 Analysis

General linear models were used to predict number of grandchildren at age 55/56 relative to age of menopause and final menstruation, with negative binomial regression being utilised to account for the over dispersal of the dependent variable (Figure 7.3). Models were made just including the covariates (number of children or age of first birth, employment status, marital status, self-perceived health), with age

Table 7.1. *Participant characteristics of the datasets used in Chapter 7, showing the n and % or median and interquartile range. Dataset 2 is reduced as women with no data on age at first birth are excluded.*

Variable	Dataset 1	Dataset 2
n	3725	3519
Age of menopause		
Stopped for another reason	907 (24.35)	862 (24.50)
40 or younger	48 (1.29)	44 (1.25)
41-45	255 (6.85)	243 (6.91)
46-50	1005 (26.98)	939 (26.68)
51-55	1048 (28.13)	996 (28.30)
Still menstruating	462 (12.40)	435 (12.36)
Age of last period		
40 or younger	327 (8.78)	305 (8.67)
41-45	483 (12.97)	461 (13.10)
46-50	1302 (34.95)	1225 (34.81)
51-55	1151 (30.90)	1093 (31.06)
Still menstruating	462 (12.40)	435 (12.36)
Number of grandchildren	0.00 (0.00, 2.00)	0.00 (0.00, 2.00)
Number of children	2.00 (2.00, 3.00)	2.00 (2.00, 3.00)
Age first birth	-	25.00 (22.00, 29.00)
Marital status		
Other (e.g. divorced, widowed, single)	934 (25.07)	893 (25.38)
Married/Civil partnership	2791 (74.93)	2626 (74.62)
Self-perceived health		
Poor/very poor	245 (6.58)	226 (6.42)
Fair	510 (13.69)	493 (14.01)

Table 7.1. *Participant characteristics of the datasets used in Chapter 7, showing the n and % or median and interquartile range. Dataset 2 is reduced as women with no data on age at first birth are excluded.*

Variable	Dataset 1	Dataset 2
Very good/good	2480 (66.58)	2349 (66.75)
Excellent	490 (13.15)	451 (12.82)
Employment status		
Unemployed	257 (6.90)	243 (6.91)
Employed	2959 (79.44)	2797 (79.48)
Other	509 (13.66)	479 (13.61)

of menopause and age of last menstruation subsequently being added in separate models. To see whether a model that includes a measure of reproductive cessation better fits the data than one without, the base model (that just included covariates) was independently compared to the two other models based on their AIC value (see Table 7.2 for model composition). Here, a lower AIC value suggests a better fitting model, and a ΔAIC value of greater than two indicates that a model is a significantly poorer fit when compared to the best fitting model, which would have a ΔAIC value of zero (Burnham & Anderson, 2002).

7.4 Results

In the larger dataset that did not subset based on data on age at first birth (n = 3725), the majority of women went through menopause between the ages of 46-50 (27%) and 51-55 (28%). Few women were still menstruating at age 55/56 (12%), and a number of women had ceased menstruating for another reason (24%). Only 1% of women experienced menopause at the age of 40 or younger. When looking at age of last period, this age category increases to 9%, and the majority of women experienced their final menstruation between the ages of 46 and 50 (35%). In the sample, most women were either married or in a civil partnership (75%), had very good or good health (67%), and were still employed (79%). Further, women had a median of 2 children (IQR: 2, 3), and no grandchildren (IQR: 0, 2; see Figure 7.3). Amongst women who had data on age of first birth the average first reproduction was 25 (IQR: 22, 29). Full participant characteristics are shown in Table 7.1.

Table 7.2. *Results from model fitting based on Akaike Information Criterion (AIC). The lowest AIC value is deemed to best fit the data, with ΔAIC referring to the difference in AIC value from the best fitting model (shown in italics). A ΔAIC value of more than two demonstrates a significantly poorer model fit, and w_i indicates model probability. The base model includes self-perceived health, employment status and marital status.*

Model	**K**	**AIC**	**ΔAIC**	w_i
Base model (inc. number of children)	9.00	11948.13	40.15	0.00
Age of menopause + Base model (inc. number of children)	14.00	11916.84	8.87	0.01
Age last period + Base model (inc. number of children)	*13.00*	*11907.97*	*0.00*	*0.99*
Base model (inc. age first birth)	9.00	10343.12	2.30	0.23
Age of menopause + Base model (inc. age first birth)	14.00	10346.52	5.70	0.04
Age last period + Base model (inc. age first birth)	*13.00*	*10340.82*	*0.00*	*0.73*

Using the procedures outlined in Section 7.2.1, model fitting was carried out, with the results presented in Table 7.2. Whether controlling for number of children or age at first birth, a model that included the covariates plus age of last menstruation best fit the data. The inclusion of age of menopause within the models resulted in a poorer fit to the data than the models adjusting for age of final menstruation, suggesting the latter better captures the variation in number of grandchildren.

As shown in Figure 7.4c and 7.4d, there is a near linear relationship between age of last menstruation and number of grandchildren, with an earlier final menstruation predicting a greater number of grandchildren at age 55/56 (these results are replicated in Appendix G Table G1 using the smaller dataset that is restricted based on information on age of first birth). The effect size decreases when adjusting for age of first birth rather than number of children, but the predicted relationship (earlier final menstruation = more grandchildren) is still maintained. A similar relationship is seen between age of natural menopause and number of grandchildren: later menopause predicts fewer grandchildren (Figures 7.4a-b). In models adjusting for age of menopause, those who stopped menstruating for a reason other than menopause were predicted to have a more grandchildren. Like age

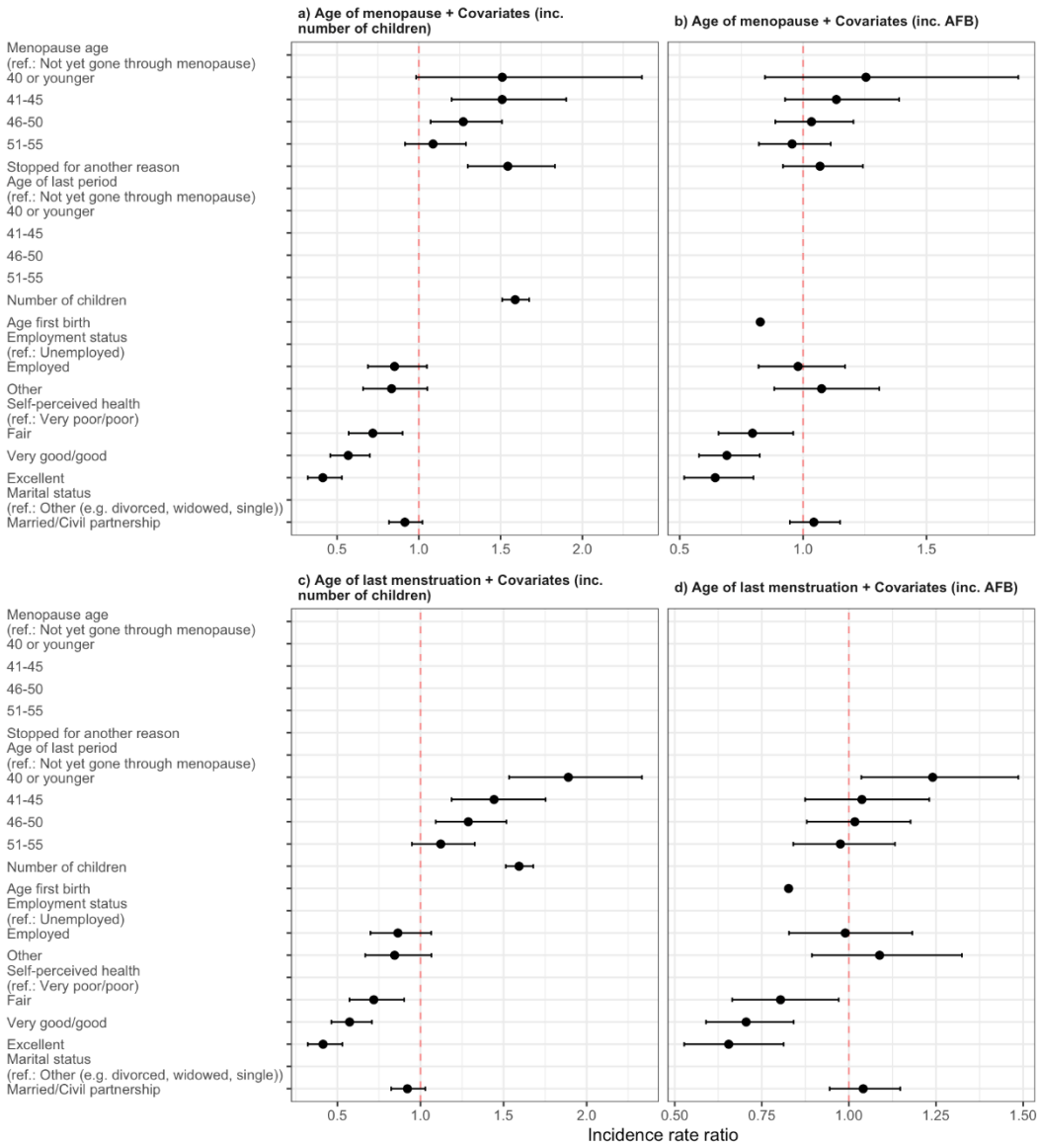

Figure 7.4. *Forest plots showing the results of models using negative binomial regression to predict number of grandchildren at age 55/56. A higher incidence rate ratio indicates a greater predicted number of grandchildren. Error bars represent a 95% confidence interval.*

at final menstruation, the effect size weakened when adjusting for age of first birth rather than number of children (Figure 7.4b).

In all models adjusting for age of first birth, an earlier first reproduction was a significant predictor of having more grandchildren, as was having more children. Health also had a linear relationship with indirect fitness, with better health predicting fewer grandchildren. Number of grandchildren had little relationship with employment or marital status. Full results are shown in Table 7.3.

Table 7.3. *Results from all models predicting number of grandchildren using Poisson regression, reporting incidence rate ratios and the 95% confidence intervals. Models including number of children use a dataset with 3725 participants, and models including age of first birth use a dataset with 3519 participants.*

	Covariate model 1	**Covariate model 2**	**Age of menopause + Covariate model 1**	**Age of last menstruation + Covariate model 1**	**Age of menopause + Covariate model 2**	**Age of last menstruation + Covariate model 2**
Menopause age (ref.: Not yet gone through menopause)						
40 or younger	-	-	1.51 (0.98-2.36)	-	1.25 (0.85-1.87)	-
41-45	-	-	1.51 (1.20-1.90)	-	1.13 (0.93-1.39)	-
46-50	-	-	1.27 (1.07-1.51)	-	1.03 (0.89-1.20)	-
51-55	-	-	1.09 (0.92-1.29)	-	0.96 (0.82-1.11)	-
Stopped for another reason	-	-	1.54 (1.30-1.83)	-	1.07 (0.92-1.24)	-
Age of last period (ref.: Not yet gone through menopause)						
40 or younger	-	-	-	1.89 (1.53-2.33)	-	1.24 (1.04-1.49)
41-45	-	-	-	1.44 (1.19-1.75)	-	1.04 (0.87-1.23)
46-50	-	-	-	1.29 (1.09-1.52)	-	1.02 (0.88-1.18)
51-55	-	-	-	1.12 (0.95-1.33)	-	0.98 (0.84-1.13)

Table 7.3. *Results from all models predicting number of grandchildren using Poisson regression, reporting incidence rate ratios and the 95% confidence intervals. Models including number of children use a dataset with 3725 participants, and models including age of first birth use a dataset with 3519 participants.*

	Covariate model 1	**Covariate model 2**	**Age of menopause + Covariate model 1**	**Age of last menstruation + Covariate model 1**	**Age of menopause + Covariate model 2**	**Age of last menstruation + Covariate model 2**
Number of children	1.58 (1.50-1.67)	-	1.59 (1.51-1.67)	1.59 (1.51-1.68)	-	-
Age first birth	-	0.83 (0.82-0.83)	-	-	0.83 (0.82-0.84)	0.83 (0.82-0.84)
Employment status (ref.: Unemployed)						
Employed	0.82 (0.66-1.01)	0.97 (0.81-1.16)	0.85 (0.69-1.05)	0.86 (0.70-1.07)	0.98 (0.82-1.17)	0.99 (0.83-1.18)
Other	0.81 (0.64-1.02)	1.07 (0.88-1.30)	0.83 (0.66-1.05)	0.84 (0.67-1.07)	1.07 (0.88-1.31)	1.09 (0.89-1.32)
Self-perceived health (ref.: Very poor/poor)						
Fair	0.72 (0.57-0.91)	0.80 (0.66-0.96)	0.72 (0.57-0.90)	0.72 (0.57-0.90)	0.80 (0.66-0.96)	0.80 (0.67-0.97)
Very good/good	0.55 (0.45-0.68)	0.69 (0.58-0.82)	0.57 (0.46-0.70)	0.57 (0.46-0.71)	0.69 (0.58-0.82)	0.71 (0.59-0.84)
Excellent	0.39 (0.30-0.50)	0.63 (0.51-0.79)	0.41 (0.32-0.53)	0.41 (0.32-0.53)	0.64 (0.52-0.80)	0.65 (0.53-0.81)
Marital status (ref.: Other (e.g. divorced, widowed, single))						
Married/Civil partnership	0.91 (0.81-1.02)	1.04 (0.94-1.14)	0.91 (0.82-1.02)	0.92 (0.82-1.03)	1.04 (0.95-1.15)	1.04 (0.94-1.15)

7.4.1 Sensitivity analysis

To determine the robustness of these results, I provide an additional analysis presented in Appendix G (Table G3) in which models are replicated not adjusting for number of children. The previously reported results still hold true, and the effect sizes are slightly larger. Compared to women who had not yet gone through the menopause, those who went through the menopause at age 40 or younger were predicted to have 3.76 as many grandchildren by the age of 55 (95% CI: 2.31-8.58); with this effect increasing to 5.63 when looking at women's age of last period (95% CI: 3.99-8.69).

7.5 Discussion

In this chapter, I look at the timing of physiological reproductive cessation and its implications o for inclusive fitness. Based on the Grandmother Hypothesis for the evolution of menopause, I predicted that age of menopause and final menstruation would be associated with number of grandoffspring, with earlier reproductive cessation associating with a greater number of grandchildren, else the fitness costs of being infertile would not be offset. Some evidence was found in favour of this: a later age of menopause and final menstruation associated with fewer grandchildren. Also using data from the NCDS, I showed in Chapter 6 that fecundity corresponds with caring behaviour, in which women who are no longer menstruating spend more time caring for their grandchildren than women who are still having periods. For completeness, in Appendix G (Figure G1) I show that hours spent caring for grandchildren is positively associated with number of grandchildren; however, a causal relationship cannot be established here due to only having cross-sectional data, and the fact that there is likely a bidirectional relationship, with a greater number of grandchildren likely requiring more care. However, the results from this chapter, in combination with those from Chapter 6, offer evidence in favour of the idea that post-reproductive women allocate their energy adaptively, which works to offset the costs of being post-reproductive through increasing inclusive fitness.

One of the strongest predictors of number of grandchildren was age at first birth. This is expected, as the women included in this analysis were aged either 55 or 56, and therefore those who had waited until later to reproduce (e.g. late 30s, early 40s) would not yet have children who are of reproductive age themselves. Further, as reproductive norms – such as age at first birth – are often thought to be somewhat

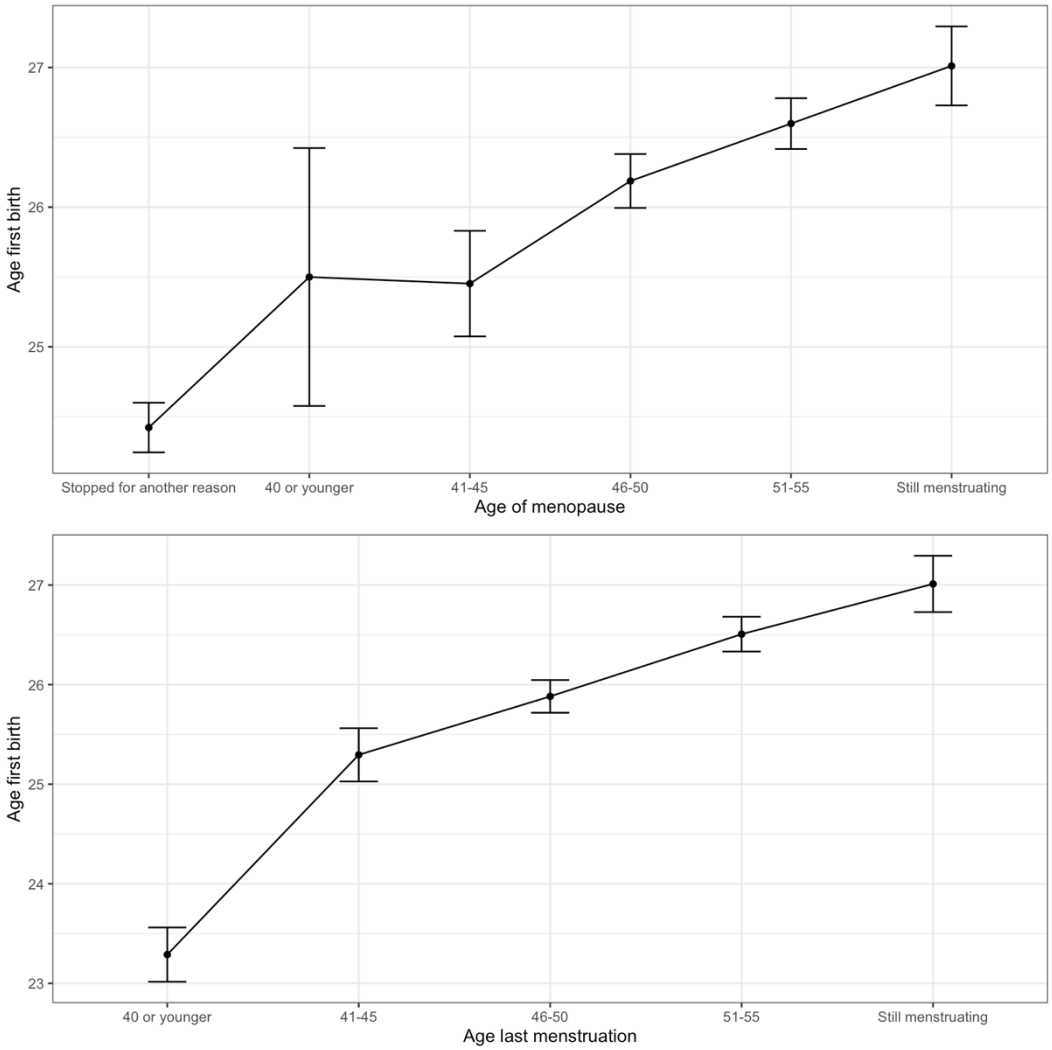

Figure 7.5*. Mean age at first birth based on age of menopause and age at last menstruation. Error bars show the standard error.*

culturally transmitted (Barber, 2001; Steenhof & Liefbroer, 2008; Colleran, 2016), it is likely mothers who were late reproducers will have children who follow similar reproductive patterns. As a result of this, a large amount of the variation in number of grandchildren is explained by reproductive timing. Age of first birth itself has a complicated relationship with menopause timing. As shown in Figure 7.5, in this data, both a later age of natural menopause and final menstrual period were associated with a later age of first birth. Similar results have been found elsewhere (Nagel et al., 2005; Abdollahi et al., 2013), and reproduction has been linked to menopause timing in other ways, with nulliparity being a strong predictor of an earlier menopause (Mishra et al., 2017), and higher parity typically being associated with a later menopause (Hardy & Kuh, 1999; Gold et al., 2013). It is thought that this is a result of higher parity reducing the number of ovulatory cycles in early life, meaning that the oocytes are preserved for longer and so menopause occurs later.

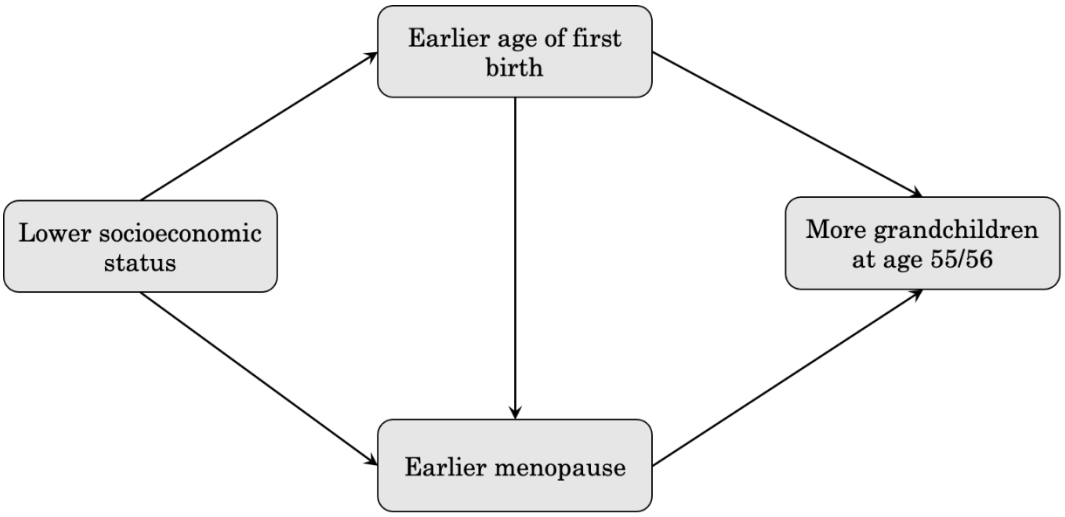

Figure 7.6. *The possible relationship between socioeconomic status, age at first birth, menopause timing, and number of grandchildren.*

However, these findings can be seen to somewhat contradict each other: an earlier first birth is associated with an earlier menopause, but high parity is commonly predictive of a later menopause, yet a younger age at first reproduction is traditionally associated with greater lifetime reproductive success (Tropf et al., 2015), as is the case in this data (Figure 7.1). Therefore, the relationship between reproduction and menopause timing might somewhat be a reflection of how socioeconomic status associates with reproductive norms in the specific culture from which the data were taken. This is because a lower socioeconomic status in WEIRD societies is commonly associated with an earlier menopause (Lawlor et al., 2003; Gold et al., 2013; Schoenaker et al., 2014; Ceylan & Ozerdogan, 2015; though findings are not consistent, see: Sheppard & Van Winkle, 2020), and a low socioeconomic position is associated with earlier reproduction (as has been found previously in the NCDS, see Nettle (2010)). However, there is lower variation in parity now in non-natural fertility societies (Stulp et al., 2016), meaning that women who had their first reproduction at a younger age will not only become grandmothers earlier by virtue of the age of their children, but they will also be at a greater risk of early menopause. Once these women are physiologically post-reproductive, it is predicted that they will invest more heavily in existing kin to increase their inclusive fitness (see Chapter 6). Hence, while age of first birth and socioeconomic status themselves confound both menopause timing and the number of grandchildren a woman has by her mid-50s (Figure 7.6), it may be that the change in behaviour following menopause does work to increase inclusive fitness, meaning that an earlier menopause associates with

indirect fitness independently of these factors. This is reflected in the fact that, even after adjustment, age of final menstruation still associates with number of grandchildren in the predicted way, with women who stopped menstruating before the age of 40 being estimated to have 1.3 times as many grandchildren than women who were still menstruating at the time of interview.

The relationship between fertility and number of grandchildren also brings into question what the optimal age of final menstruation is. Additional analyses presented in Appendix G (Table G2) show that age of menopause and final menstruation had little relationship with number of children, yet stopping menstruating earlier was found to result in increased indirect fitness at age 55/56. Therefore, if menopause timing has no bearing on number of children, but does predict number of grandchildren, perhaps it might be better to stop menstruating earlier. Existing research has shown that – even in natural fertility populations – age of last birth typically precedes age at menopause by approximately 10-11 years (te Velde & Pearson, 2002; Towner et al., 2016), but there is a large amount of variation in this number (Towner et al., 2016; Mattison et al., 2018), with the gap between age of last birth and menopause increasing the later a woman goes through menopause, meaning that women who experience a later menopause are very unlikely to reproduce close to the event (Gottschalk et al., 2019). Therefore, it does not appear that there is a direct fitness advantage to going through menopause later; and based on the observation that an earlier final menstruation might increase indirect fitness, it may be that an earlier menopause is slightly advantageous in terms of inclusive fitness outcomes. Despite the relationship between menopause timing/age last menstruation and indirect fitness, there is evidence showing moderate selection for a later age of natural menopause (Kirk et al., 2001; Byars et al., 2010; Stearns et al., 2010). This has been somewhat attributed to the "transition to modernity" (Corbett et al., 2018:421), with menopause timing being determined by various sociodemographic, reproductive, lifestyle, and genetic factors (Gold et al., 2013; Laisk et al., 2019; Arnot & Mace, 2020). There is evidence to show that various life history traits, such as number of children, menarche, menopause, and so on do not correlate consistently in the way predicted by natural selection (Sheppard & Van Winkle, 2020); therefore, it might be that the delayed age of menopause, in combination with reproductive norms present in WEIRD societies, is not optimal in

terms of inclusive fitness, but is persisting due to better health care and various sociodemographic factors.

As alluded to throughout this discussion, in addition to age of natural menopause, age of last menstruation was also included in this analysis. This was included as an additional measure because once a woman is physiologically post-reproductive, based on inclusive fitness theory, we would expect women who are no longer menstruating to experience the same trade-offs and shifts in caring behaviour regardless of whether a natural or non-natural menopause was experienced, as they would be experiencing the same constraints on their direct fitness. As such, the 'menopause timing' and 'age of last menstruation' have been used somewhat interchangeably throughout this chapter. However, there are a few demographic differences between women who go through a natural and non-natural menopause. Firstly, the primary reason for a woman to stop menstruating for reasons other than menopause is surgery, and women who do have a surgical menopause typically do so at a younger age than women would experience a natural menopause (Pokoradi et al., 2011). This means the majority of women who did stop menstruating before 40 did not go through menopause naturally. Secondly, while health problems are a major contributing factor to why a woman might experience a non-natural menopause (e.g. endometriosis, painful menstrual cramps) (Pokoradi et al., 2011), there is a degree of choice associated with the process, and having existing children has been found to be a major influence over this choice (Uskul et al., 2003; Cooper et al., 2008). An earlier age of first birth associates with an increased risk of a hysterectomy (Meilahn et al., 1989; Brett et al., 1997), likely due to the fact that a woman may be more willing to experience a surgical menopause in response to health problems if she has existing children. Therefore, there will be a bias in age amongst women who experienced a surgical menopause.

As a final note, as the women included in this study are only aged 55 and 56, it is likely that their extended families are still growing. Data from follow up interviews will allow me to further examine whether an earlier age of final menstruation does result in greater overall fitness, or whether the relationship we see here is merely an artefact of reproductive timing.

7.6 Conclusion

In conclusion, the results shown in this chapter lend additional support for the idea that women adaptively offset the costs of being post-reproductive by increasing their inclusive fitness, most likely through the additional care demonstrated in Chapter 6. My results also beg the question of what the optimal age to finish menstruating is. Neither age of menopause nor age of last period predicted number of children, but did predict number of grandchildren. This highlights that menopause is not necessarily a constraint on reproduction, as women typically stop well before menopause, and suggests that the inclusive fitness benefits associated with being post-reproductive may outweigh the benefits of menstruating until a later age.

CHAPTER EIGHT

Final remarks

8.1 Summary of findings

It has long been argued that, despite its apparent detriment to fitness, menopause and the accompanying post-reproductive lifespan have evolutionary benefits (Williams, 1957; Hawkes et al., 1998; Cant & Johnstone, 2008). The aim of this thesis was not to try and 'prove' whether menopause is a selected for trait or not, but rather to test evolutionary hypotheses relating to menopause and to understand the demographic trends in menopause timing and symptoms from an evolutionary perspective.

Overall, I have shown evidence that the current variation in menopause timing could have a facultative underpinning, but I found no support for the idea that menopause timing covaries with residence patterns in the way predicted by Cant & Johnstone (2008) and Ubeda et al. (2014). Further, I have also shown that there is no evidence for an adaptive theory of menopause symptoms, and I concluded that – even if menopause itself is adaptive – menopause symptoms do not necessarily require an evolutionary explanation. I have also demonstrated that post-reproductive caring behaviours in a recent sample of women from the United Kingdom are still in line with predictions made by an evolutionary framework, and that who it is adaptive to direct care to likely varies relative to fecundity status and the possible inclusive fitness outcomes. Finally, I have shown evidence for an earlier menopause being offset by indirect fitness gains, lending further evidence for the idea that menopause evolved due to the inclusive fitness gains to be had from being post-reproductive.

Evolutionary anthropologists have previously used current variation in age of menopause to test evolutionary hypotheses relating to the emergence of menopause, with the assumption that a current earlier menopause represents selection for reproductive cessation (e.g. Snopkowski et al., 2014). For instance, menopause timing has been used to test Cant & Johnstone (2008)'s Reproductive Conflict Hypothesis, in which an earlier age of menopause under a patrilocal residence pattern would be taken as evidence that reproductive conflict between generations resulted in selection for menopause in our evolutionary history. However, as much of the research into

the timing at which menopause occurs has been epidemiological, there is the underlying assumption in evolutionary research that menopause timing today is facultative and not just the result of lifestyle and genetic factors. In Chapter 3, I showed that menopause timing does appear to vary in an optimal way, which was interpreted as evidence for menopause timing being facultatively plastic in response to external stimuli. So, the findings from this chapter suggest that (within reason) menopause timing is somewhat malleable and varies in a way as predicted by an evolutionary model, giving some grounding for its use as a proxy of the evolution of the trait.

Despite this, it should be noted that just because a trait appears to be adaptive today, it does not mean that this is indicative of how or why it evolved. There is a large body of evidence showing that a post-reproductive lifespan *now* often has fitness benefits, and results from this thesis suggest that variation in menopause timing *now* might be facultative; but all data we are using today are from populations that already have menopause and post-reproductive lifespan. Hence, one could argue is that all these models are showing is optimal variation of a trait within its usual range and not why the trait emerged in the first place, which could have been for a completely different reason. There are many cases across the natural world where the current function of a phenotype confers a fitness advantage, but the phenotype did not evolve as a result of natural selection for *that function*. A classic example of this is bird feathers. Fossil evidence suggests that the earliest feathers did not evolve for flying purposes, but rather to assist in thermoregulation. Now many birds use their feathers for flight, and flight provides a fitness advantage in many ecologies; but feathers are not an adaption for flight (Benton et al., 2019). As such, it could be that a prolonged period of infertility in females does appear to be adaptive today because it prevents reproductive conflict and increases the survival and reproduction of younger kin, but it does not mean that this is why the phenotype was selected for. We only know that feathers evolved to regulate the temperature of birds (and their ancestors) because of physical fossil evidence. As menopause does not fossilise, based on current scientific advances it does not seem possible that we will ever know when or why it evolved in our evolutionary history. At present, all we have is data from current (or historically recent) populations, and we should make do with it the best we can. But, there is evidence that menopause timing is facultative today, and so age of menopause is still useful to use in evolutionary models as long as it is

acknowledged that we are not necessarily modelling the evolution of the trait, but whether it is facultative today in a way predicted by evolutionary theory.

The assumption that all the phenotypes we see today are the result of positive selection in our evolutionary history also led Ubeda et al. (2014) to the theoretically unsubstantiated conclusion that if menopause is adaptive, it should not be a "long and difficult process" (Ubeda et al., 2014:165). However, this statement assumes that natural selection produces 'perfect' phenotypes without flaws, which is not the case. Natural selection selects for traits that are optimal relative to the local ecology, and sometimes traits that confer a survival advantage in one domain can be slightly detrimental in another. As long as any negative side effects/by-products of selected-for traits do not negatively affect survival and/or reproduction, then there would be no selective pressure against them. Menopause symptoms do not affect fitness: there is no evidence that they affect survival, and by the time a woman begins experiencing them during peri-menopause she is likely behaviourally post-reproductive (Towner et al., 2016; Mattison et al., 2018), and therefore the menopause symptoms would be in the selection shadow. Further, there is no reason to think that a biological process shaped by natural selection should be smooth. Growth, puberty, and reproduction are all adaptive biological processes that are not 'smooth' or painless, and there is no reason to think that menopause would be any different. It is therefore unsurprising that I found little evidence in support of Ubeda et al. (2014)'s model for menopause symptoms, and more evidence in support of a biosocial model, in which lifestyle factors influence symptom severity.

In the final analytical chapter of this thesis, I also introduced the idea of whether there is an optimal age of menopause today. As there is a large amount of variation in menopause timing, it begs the question of whether is better to have an earlier or later menopause in terms of fitness. I showed that, at age 55, women who went through menopause earlier had more grandchildren than women who experienced a later menopause (though this may somewhat be a product of reproductive timing), which could mean that women who go through menopause earlier have greater inclusive fitness. Given women seldom reproduce right up until menopause (both in natural and non-natural fertility populations); that the gap between age of last reproduction and age of menopause increases as menopause gets later (Towner et al., 2016); and that women spend more time caring for their reproductive kin when they are post-menopausal than pre-menopausal, it may be that

it is better to stop menstruating slightly earlier so that energy being invested into maintaining menstrual cycles (when reproduction is unlikely) can be invested into activities that increase indirect fitness (i.e. caring for kin). Despite the possible advantages of a slightly earlier menopause, research has shown that menopause is getting later because of factors associated with "modernity" (Corbett et al., 2018) (e.g. better health care, increased use of hormonal contraceptives). Therefore, future research could aim to consider whether there is an optimal age of menopause, and whether women are moving towards or away from this optimal age.

8.2 Reflections

This thesis was motivated by a general interest in all things 'low fertility'. One of the first things students of evolutionary sciences are taught is that selection works to increase Darwinian fitness, and therefore phenotypes we see today should be optimal relative to their local ecology. Yet this of course is not always the case in humans, as we show a suite of both physical and cultural traits that mean we do not always attain what would be considered optimal fertility. I decided to focus on menopause as it seemed that, despite decades of research into the function of menopause by evolutionary biologists and anthropologists there is still no answer to the question of why we have menopause. Therefore, I saw an opportunity to contribute to the ever-growing body of evolutionary research into menopause and the post-reproductive lifespan. As previously stated, upon embarking on this research I did not set out to try and answer the question of "*why do humans have menopause and a post-reproductive lifespan?*"; rather, I planned to look at current trends in various aspects of the menopausal transition and interpret them from an evolutionary framework.

8.2.1 Data sources

The majority of this thesis used data from secondary, publicly available, longitudinal datasets, which is not traditionally the norm for evolutionary anthropologists. Rather, people from this field usually collect data from traditional societies, like hunter-gatherer groups, as a means of trying to work out why we are the way we are. I did not do this. I suspect that this thesis is a rarity in the UCL Anthropology department in that it involved no fieldwork carried out by myself, and instead relied solely on data collected by others. It was not my intention for this to be the case. As mentioned in Chapter 5, upon embarking on my PhD I had planned to organise

some of my own research in China and assist in the data collection. As such, I had planned to travel out there in the spring of 2020 to assess the feasibility of what I wanted to do, and plan that years field session with the other researchers. However – like most things in 2020 – this did not happen. I was lucky enough to have visited Lanzhou University in 2018 and 2019, and during these visits I was able to talk to the researchers who had been involved in the data collection. This allowed me a better feel of how the fieldwork had been carried out than if all conversations about the data had been carried out over email – so I am grateful for this. I am also grateful for the huge amount of data available from the UK Data Service and other online repositories, which have surely been invaluable to researchers such as myself whose work was hindered by the travel restrictions associated with the global pandemic. It seems to me a lot of this data is relatively untapped by evolutionary anthropologists. Though this kind of data is somewhat restrictive in terms of the questions that are asked (very few longitudinal studies ask questions about menopause, for example), once you do find a dataset that does suit your needs then you have access to information on thousands and thousands of participants. The sheer volume of data means that models are more powered than those generated from data on people from small-scale societies (with most anthropological research having sample sizes in the tens or hundreds). Therefore, while the data used in this thesis may not be traditional by anthropological standards, it has suited the needs of this thesis. Moving forward, while I plan to keep making use of the cohort studies, in the coming years I do intend on designing my own studies and collecting my own data.

8.2.2 Future directions

8.2.2.1 Menopause symptoms

Though it has previously been suggested that menopause symptoms have evolutionary roots (Ubeda et al., 2014; Sievert & Masley, 2015), I found more evidence for the idea they are the product of environmental and social factors. This demonstrates that menopause symptoms are not an inevitability of the menopausal transition, as many think they are, but rather the product of factors that are somewhat malleable. Many individuals going through the menopausal transition take hormone replacement therapy as a means of dealing with their symptoms; however, the knowledge that there are factors other than simply hormones causing their menopausal transition to be unpleasant may offer women the option to use methods

other than medication to deal with their menopause symptoms, and thus allow them a greater amount of autonomy over their transition. Though recently there has been a focus on getting people talking about menopause, research has shown that understanding of the menopausal transition is generally quite poor amongst the general public, with the most knowledgeable individuals being those who are post-menopausal, and have therefore learned about it from experience (Rubinstein & Foster, 2013). Hence, it might be that even though research is being conducted in this area of reproductive health, findings are not being effectively communicated to the public. Additionally, it is likely that people only go looking for information and help once the menopausal transition has started happening to them. This means menstruating individuals are unprepared for their own transition, and that those around them do not have the knowledge or understanding to support them. In 2020, menopause was added to the sex education curriculum in the UK. Prior to this, it was not taught in schools at all: girls were told that their periods would start, but not that they would stop (Department for Education, 2020). This is a step in the right direction in terms of normalising the menopausal transition, and giving people the right tools to seek more education about it. Throughout my PhD, I have tried to communicate my research with the public as much as possible and, as such, my publications have been picked up by multiple media outlets. Based on talking to the public, it is apparent that menopausal individuals are eager to share and talk about their experiences; but they also have said to me that they still feel like not enough is being done to normalise and publicise what they are going through. I hope my research and communication with the public has got people talking about menopause slightly more; however, it is still apparent that more work needs to be done in terms of providing women with the information they need.

8.2.2.2 Menopause timing

Apparent to me throughout the writing of this thesis is that evolutionary researchers are not clear about what we would expect menopause to look like today (based on evolutionary principles). As previously stated, menopause is an important life history trait, and the timing of the trait could theoretically have important implications for individual fitness. However, it is not clear whether, given various ecological conditions, an earlier or later menopause would be optimal, and it is also unclear how we would predict it to cluster with other life history traits. Some sub-fields of the

evolutionary social sciences get criticised for creating 'just-so' stories about the evolution of traits, through looking at behaviours we see today and then trying to explain how they would have been present in our evolutionary history (Gould & Lewontin, 1979). Human behavioural ecology is generally quite robust to these criticisms, as most researchers adopt the procedure in which a model predicting what the optimal strategy is first generated, with real world data then being used to see if this optimality model fits what we see happening. This is the kind of model we are lacking when it comes to menopause timing. It might be that most of the variation in menopause timing we see today is just simply the product of non-evolutionary factors; but we cannot be sure this is the case unless it is modelled and tested. If it is the case that menopause timing can be explained by an evolutionary framework, then this understanding may contribute to our knowledge of how menopause and the post-reproductive lifespan evolved in the first place. In addition to this, currently the majority of the evolutionary research into menopause has focussed on why it evolved, with current menopause timing being used in models as a proxy of the trait evolving (e.g. an earlier population average age of menopause representing selection for an earlier menopause ancestrally). But, given we do not actually know 1) whether individual-level differences in menopause timing can be explained by evolutionary theory, and 2) what an optimal menopause timing is given one's ecology; how valid it is to use current menopause timing as a model of the trait evolving is not clear. By paying more attention to understanding current variation in menopause today, and developing a formal model of how we would predict it to vary based on evolutionary trade-offs, it will hopefully make us better informed when making predictions about how the trait evolved in the first place.

8.2.2.3 Menopause evolution

Currently it seems the two most widely accepted hypotheses for the evolution of menopause and a post-reproductive lifespan are the Grandmother Hypothesis and the Reproductive Conflict Hypothesis. Often, these two hypotheses are discussed as if they are complementary of one another, with the intergenerational reproductive conflict proposed by Cant & Johnstone (2008) supplementing the kin selected benefits of a post-reproductive lifespan proposed by Hawkes et al. (1998). Despite the fact the two hypotheses are generally taken as read, there is little empirical evidence supporting the Reproductive Conflict Hypothesis (Snopkowski et al., 2014;

Yang et al., 2019). Further, the hypotheses are not as complementary as they are made out to be. The Reproductive Conflict Hypothesis requires strict female dispersal, where the only way a woman can increase her fitness is through direct reproduction. If there is still contact with her maternal relatives, then there is an avenue to increase her fitness through investing in them, meaning the conflict with the family of her husband would be less severe. However, the Grandmother Hypothesis is somewhat predicated on contact with maternal relatives. While this condition was not included in the original model of grandmothering, much of the empirical evidence finds that maternal grandmothers are beneficial for grandoffspring health and survival, whereas there are mixed findings for the benefits of paternal grandmothers, who are even sometimes detrimental for grandchild survival. Hence, based on current research on the inclusive fitness benefits of a post-reproductive lifespan, it seems that a degree of contact with maternally related kin would be required for grandmothering to contribute to the selection of menopause and a post-reproductive lifespan. However, too much contact outside of the woman's husbands' kin-group may affect the amount of effort younger women put into reproductive conflict, possibly resulting in selection not being strong enough for menopause to have evolved. This issue is seldom addressed in the evolutionary-menopause literature, and future research into the evolution of the trait should aim to reconcile the differences between the two hypotheses.

8.3 Conclusion

In this thesis, I have looked at the current trends we see surrounding aspects of the menopausal transition and analysed them from an evolutionary perspective. Throughout, I also have highlighted areas that require future research as, despite menopause happening to approximately 50% of the human population, it is still a largely under researched subject and there are still many gaps in the literature. However, I hope that the findings from this thesis have filled a few of them, and that it generates future research into the subject.

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Appendices

Appendix A. Supplementary information for Chapter 1

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Acinonyx jubatus	Cheetah	Carnivora	0.003	Wild	Ellis et al. (2018a)
Alces alces	Moose	Cervidae	0.020	Wild	Ellis et al. (2018a)
Alouatta caraya	Black howler monkey	Primate	0.113	Zoo	Levitis & Lackey (2011)
Aotus lemurinus	Grey-bellied night monkey	Primate	0.076	Zoo	Levitis & Lackey (2011)
Aotus trivirgatus	Three-striped night monkey	Primate	0.035	Zoo	Levitis & Lackey (2011)
Arctocephalus gazella	Antarctic fur seal	Carnivora	0.004	Wild	Ellis et al. (2018a)
Arctocephalus pusillus	Australian fur seal	Carnivora	0.002	Wild	Ellis et al. (2018a)
Ateles fusciceps	Black-headed spider monkey	Primate	0.195	Zoo	Levitis & Lackey (2011)
Ateles geoffroyi	Geoffroy's spider monkey	Primate	0.185	Zoo	Levitis & Lackey (2011)
Balaenoptera physalus	Fin whale	Cetacea	0.006	Wild	Ellis et al. (2018a)
Berardius bairdii	Baird's beaked whale	Cetacea	0.001	Wild	Ellis et al. (2018b)
Bison bison	American bison	Artiodactyla	0.028	Wild	Ellis et al. (2018a)
Brachyteles hypoxanthus	Northern muriqui	Primate	0.060	Wild	Croft et al. (2015)
Brachyteles hypoxanthus	Northern muriqui	Primate	0.060	Wild	Alberts et al. (2013)
Callimico goeldii	Goeldi's monkey	Primate	0.014	Zoo	Levitis & Lackey (2011)
Callithrix argentata	Silvery marmoset	Primate	0.089	Zoo	Levitis & Lackey (2011)
Callithrix geoffroyi	White-headed marmoset	Primate	0.021	Zoo	Levitis & Lackey (2011)
Callithrix jacchus	Common marmoset	Primate	0.107	Zoo	Levitis & Lackey (2011)
Callithrix kuhli	Wied's black-tufted-ear marmoset	Primate	0.120	Zoo	Levitis & Lackey (2011)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Callithrix penicillata	Black-tufted marmoset	Primate	0.060	Zoo	Levitis & Lackey (2011)
Callithrix pygmaea	Pygmy marmoset	Primate	0.083	Zoo	Levitis & Lackey (2011)
Callorhinus ursinus	Northern fur seal	Carnivora	0.002	Wild	Ellis et al. (2018a)
Callospermophilus lateralis	Golden-mantled ground squirrel	Rodentia	0.000	Wild	Ellis et al. (2018a)
Capricornis crispus	Japanese serow	Artiodactyla	0.000	Wild	Ellis et al. (2018a)
Castor canadensis	North American beaver	Rodentia	0.003	Wild	Ellis et al. (2018a)
Cebus apella	Tufted capuchin	Primate	0.176	Zoo	Levitis & Lackey (2011)
Cebus capucinus	White-headed capuchin	Primate	0.040	Wild	Alberts et al. (2013)
Cebus capucinus	White-headed capuchin	Primate	0.004	Wild	Ellis et al. (2018a)
Cercocebus torquatus	Collared mangabey	Primate	0.105	Zoo	Levitis & Lackey (2011)
Cercopithecus diana	Diana monkey	Primate	0.163	Zoo	Levitis & Lackey (2011)
Cercopithecus mitis	Blue monkey	Primate	0.020	Wild	Alberts et al. (2013)
Cercopithecus mitis	Blue monkey	Primate	0.005	Wild	Ellis et al. (2018a)
Cercopithecus neglectus	De Brazza's monkey	Primate	0.128	Zoo	Levitis & Lackey (2011)
Cervus elaphus	Red deer	Artiodactyla	0.001	Wild	Ellis et al. (2018a)
Cheirogaleus medius	Fat-tailed dwarf lemur	Primate	0.284	Zoo	Levitis & Lackey (2011)
Chlorocebus aethiops	Grivet	Primate	0.219	Zoo	Levitis & Lackey (2011)
Colobus angolensis	Angola colobus	Primate	0.046	Zoo	Levitis & Lackey (2011)
Colobus guereza	Mantled guereza	Primate	0.028	Zoo	Levitis & Lackey (2011)
Delphinapterus leucas	Beluga whale	Cetacea	0.270	Wild	Ellis et al. (2018b)
Equus quagga	Plains zebra	Perissodactyla	0.006	Wild	Ellis et al. (2018a)
Erythrocebus patas	Patas mokey	Primate	0.148	Zoo	Levitis & Lackey (2011)
Eulemur fuvus	Common brown lemur	Primate	0.156	Zoo	Levitis & Lackey (2011)
Eulemur macaco	Black lemur	Primate	0.18	Zoo	Levitis & Lackey (2011)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Eumetopias jubatus	Stellar sea lion	Carnivora	0.017	Wild	Ellis et al. (2018a)
Galago moholi	Mohol bushbaby	Primate	0.150	Zoo	Levitis & Lackey (2011)
Galago senegalensis	Senegal bushbaby	Primate	0.129	Zoo	Levitis & Lackey (2011)
Globicephala macrorhynchus	Short-finned pilot whale	Cetacea	0.150	Wild	Ellis et al. (2018b)
Globicephala macrorhynchus	Short-finned pilot whale	Cetacea	0.280	Wild	Photopoulou et al. (2017)
Globicephala macrorhynchus	Short-finned pilot whale	Cetacea	0.260	Wild	Ellis et al. (2018a)
Globicephala melas	Long-finned pilot whale	Cetacea	0.002	Wild	Ellis et al. (2018a)
Globicephala melas	Long-finned pilot whale	Cetacea	0.010	Wild	Ellis et al. (2018b)
Gorilla beringei	Eastern gorilla	Primate	0.040	Wild	Alberts et al. (2013)
Gorilla beringei	Eastern gorilla	Primate	0.022	Wild	Ellis et al. (2018a)
Gorilla gorilla	Western gorilla	Primate	0.214	Zoo	Levitis & Lackey (2011)
Hemitragus jemlahicus	Himalyan tahr	Artiodactyla	0.003	Wild	Ellis et al. (2018a)
Hippopotamus amphibius	Hippopotamous	Artiodactyla	0.009	Wild	Ellis et al. (2018a)
Homo sapien	Human	Primate	0.486	Afghanistan	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.460	Haiti	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.760	Japan	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.643	Less developed countries	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.607	Less developed regions	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.490	Niger	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.489	Papua New Guinea	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.497	Somalia	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.707	Sweden	Levitis & Lackey (2011)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Homo sapien	Human	Primate	0.477	Sweden 1751	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.668	USA	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.426	Kung	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.439	Ache	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.481	Hadza	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.315	Trinidad Slaves	Levitis & Lackey (2011)
Homo sapien	Human	Primate	0.443	Hadza	Ellis et al. (2018a)
Hylobates lar	Lar gibbon	Primate	0.182	Zoo	Levitis & Lackey (2011)
Hylobates syndactylus	Siamang	Primate	0.164	Zoo	Levitis & Lackey (2011)
Kobus leche	Lechwe	Artiodactyla	0.003	Wild	Ellis et al. (2018a)
Lagotricha lagotricha	Brown woolly monkey	Primate	0.155	Zoo	Levitis & Lackey (2011)
Lemur catta	Ring-tailed lemur	Primate	0.106	Zoo	Levitis & Lackey (2011)
Lemur catta	Ring-tailed lemur	Primate	0.001	Wild	Ellis et al. (2018a)
Leontopithecus chrysomelas	Golden-headed lion tamarin	Primate	0.213	Zoo	Levitis & Lackey (2011)
Leontopithecus rosalia	Golden lion tamarin	Primate	0.204	Zoo	Levitis & Lackey (2011)
Leptonychotes weddellii	Weddell seal	Carnivora	0.001	Wild	Ellis et al. (2018a)
Lissodelphis borealis	Northern right-whale dolphin	Cetacea	0.030	Wild	Ellis et al. (2018b)
Loris tardigradus	Red slender loris	Primate	0.175	Zoo	Levitis & Lackey (2011)
Loxodonta africana	African elephant	Proboscidea	0.035	Wild	Ellis et al. (2018a)
Macaca fascicularis	Crab-eating macaque	Primate	0.177	Zoo	Levitis & Lackey (2011)
Macaca fuscata	Japanese macaque	Primate	0.247	Zoo	Levitis & Lackey (2011)
Macaca fuscata	Japanese macaque	Primate	0.055	Semi-wild	Levitis & Lackey (2011)
Macaca fuscata	Japanese macaque	Primate	0.005	Wild	Ellis et al. (2018a)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Macaca mulatta	Rhesus macaque	Primate	0.178	Zoo	Levitis & Lackey (2011)
Macaca nigra	Celebes crested macaque	Primate	0.010	Zoo	Levitis & Lackey (2011)
Macaca silenus	Lion-tailed macaque	Primate	0.225	Zoo	Levitis & Lackey (2011)
Macaca sylvanus	Barbary macaque	Primate	0.139	Zoo	Levitis & Lackey (2011)
Mandrillus leucophaeus	The drill	Primate	0.259	Zoo	Levitis & Lackey (2011)
Mandrillus sphinx	The mandrill	Primate	0.189	Zoo	Levitis & Lackey (2011)
Marmota flaviventris	Yellow-bellied marmot	Rodentia	0.006	Wild	Ellis et al. (2018a)
Meles meles	European badger	Carnivora	0.004	Wild	Ellis et al. (2018a)
Microcebus murinus	Grey mouse lemur	Primate	0.077	Zoo	Levitis & Lackey (2011)
Monachus schauinslandi	Hawaiian monk seal	Carnivora	0.000	Wild	Ellis et al. (2018a)
Monodon monoceros	Narwhal	Cetacea	0.240	Wild	Ellis et al. (2018b)
Mungos mungo	Banded mongoose	Carnivora	0.000	Wild	Ellis et al. (2018a)
Nycticebus coucang	Sunda slow loris	Primate	0.102	Zoo	Levitis & Lackey (2011)
Nycticebus pygmaeus	Pygmy slow loris	Primate	0.219	Zoo	Levitis & Lackey (2011)
Odobenus rosmarus	Walrus	Carnivora	0.018	Wild	Ellis et al. (2018a)
Orchinus orca	Killer whale	Cetacea	0.220	Wild	Photopoulou et al. (2017)
Orchinus orca	Killer whale	Cetacea	0.309	Wild	Ellis et al. (2018a)
Otolemur crassicaudatus	Brown greater galago	Primate	0.225	Zoo	Levitis & Lackey (2011)
Otolemur garnettii	Northern greater galago	Primate	0.206	Zoo	Levitis & Lackey (2011)
Ovis aries	Soay sheep	Artiodactyla	0.001	Wild	Ellis et al. (2018a)
Ovis canadensis	Bighorn sheep	Artiodactyla	0.004	Wild	Ellis et al. (2018a)
Pan troglodytes	Chimpanzee	Primate	0.224	Zoo	Levitis & Lackey (2011)
Pan troglodytes	Chimpanzee	Primate	0.018	Synthetic wild	Levitis & Lackey (2011)
Pan troglodytes	Chimpanzee	Primate	0.020	Wild	Alberts et al. (2013)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Pan troglodytes	Chimpanzee	Primate	0.006	Wild	Ellis et al. (2018a)
Panthera leo	Lion	Carnivora	0.004	Wild	Ellis et al. (2018a)
Panthera pardus	Leopard	Carnivora	0.012	Wild	Ellis et al. (2018a)
Papio anubis	Olive baboon	Primate	0.020	Wild	Ellis et al. (2018a)
Papio cynocephalus	Yellow baboon	Primate	0.010	Wild	Alberts et al. (2013)
Papio cynocephalus	Yellow baboon	Primate	0.036	Wild	Ellis et al. (2018a)
Papio hamadryas	Hamadryas baboon	Primate	0.005	Amboseli	Levitis & Lackey (2011)
Papio hamadryas	Hamadryas baboon	Primate	0.084	Wild	Levitis & Lackey (2011)
Pecari tajacu	Collared peccary	Artiodactyla	0.005	Wild	Ellis et al. (2018a)
Physeter macrocephalus	Sperm whale	Cetacea	0.000	Wild	Ellis et al. (2018b)
Pithecia pithecia	White-faced saki	Primate	0.121	Zoo	Levitis & Lackey (2011)
Pongo abelii	Sumatran orangutan	Primate	0.231	Zoo	Levitis & Lackey (2011)
Pongo pygmaeus	Bornean orangutan	Primate	0.192	Zoo	Levitis & Lackey (2011)
Procyon lotor	Raccoon	Carnivora	0.004	Wild	Ellis et al. (2018a)
Propithecus verreauxi	Verreaux's sifaka	Primate	0.020	Wild	Alberts et al. (2013)
Propithecus verreauxi	Verreaux's sifaka	Primate	0.003	Wild	Ellis et al. (2018a)
Pseudorca crassidens	False killer whale	Cetacea	0.030	Wild	Ellis et al. (2018b)
Pseudorca crassidens	False killer whale	Cetacea	0.140	Wild	Photopoulou et al. (2017)
Rangifer tarandus	Reindeer	Cervidae	0.001	Wild	Ellis et al. (2018a)
Rupicapra pyrenaica	Pyrenean chamois	Artiodactyla	0.001	Wild	Ellis et al. (2018a)
Saguinus bicolor	Pied tamarin	Primate	0.024	Zoo	Levitis & Lackey (2011)
Saguinus fuscicollis	Brown-mantled tamarin	Primate	0.118	Zoo	Levitis & Lackey (2011)
Saguinus geoffroyi	Geoffroy's tamarin	Primate	0.030	Zoo	Levitis & Lackey (2011)
Saguinus imperator	Emperor tamarin	Primate	0.111	Zoo	Levitis & Lackey (2011)
Saguinus labiatus	White-lipped tamarin	Primate	0.029	Zoo	Levitis & Lackey (2011)

Table A1. *Data used for Figure 1.2 in Chapter 1. Where multiple values were available for the same species in the same environment, the mean was taken for the figure. The average of all human values was used. PrR = Post-reproductive representation.*

Species	Common name	Order	PrR	Environment	Reference
Saguinus midas	Red-handed tamarin	Primate	0.143	Zoo	Levitis & Lackey (2011)
Saguinus oedipus	Cotton-top tamarin	Primate	0.099	Zoo	Levitis & Lackey (2011)
Saimiri boliviensis	Black-capped squirrel monkey	Primate	0.161	Zoo	Levitis & Lackey (2011)
Saimiri sciureus	Common squirrel monkey	Primate	0.176	Zoo	Levitis & Lackey (2011)
Semnopithecus entellus	Grey langur	Primate	0.090	Zoo	Levitis & Lackey (2011)
Stenella attenuata	Pantropical spotted dolphin	Cetacea	0.020	Wild	Ellis et al. (2018b)
Stenella longirostris	Spinner dolphin	Cetacea	0.010	Wild	Ellis et al. (2018b)
Suricata suricatta	Meerkat	Carnivora	0.002	Wild	Ellis et al. (2018a)
Tamiasciurus hudsonicus	American red squirrel	Rodentia	0.000	Wild	Ellis et al. (2018a)
Therapithecus gelada	Gelada	Primate	0.153	Zoo	Levitis & Lackey (2011)
Trachypithecus auratus	Javan lutang	Primate	0.086	Zoo	Levitis & Lackey (2011)
Trachypithecus cristatus	Silvery lutang	Primate	0.065	Zoo	Levitis & Lackey (2011)
Trachypithecus obscurus	Dusky leaf monkey	Primate	0.049	Zoo	Levitis & Lackey (2011)
Trichechus manatus	West Indian manatee	Sirenia	0.009	Wild	Ellis et al. (2018a)
Urocitellus beldingi	Belging's ground squirrel	Rodentia	0.001	Wild	Ellis et al. (2018a)
Ursus arctos	Brown bear	Carnivora	0.002	Wild	Ellis et al. (2018a)
Ursus maritimus	Polar bear	Carnivora	0.013	Wild	Ellis et al. (2018a)
Varecia variegata	Black-and-white ruffed lemur	Primate	0.191	Zoo	Levitis & Lackey (2011)
Vulpes lagopus	Arctic fox	Carnivora	0.002	Wild	Ellis et al. (2018a)

Appendix B. Supplementary information for Chapter 2

Figure B1. *The Menopause Rating Scale used to collect data on menopause symptoms in China. Responses to symptoms 1, 2, 3, and 11 were used to measure somato-vegetative menopause symptoms (Heinemann et al., 2004).*

Menopause Rating Scale (MRS)

Which of the following symptoms apply to you at this time? Please, mark the appropriate box for each symptom. For symptoms that do not apply, please mark 'none'.

Symptoms:	none	mild	moderate	severe	very severe
Score =	0	1	2	3	4
1. Hot flushes, sweating (episodes of sweating)	☐	☐	☐	☐	☐
2. Heart discomfort (unusual awareness of heart beat, heart skipping, heart racing, tightness)	☐	☐	☐	☐	☐
3. Sleep problems (difficulty in falling asleep, difficulty in sleeping through, waking up early)	☐	☐	☐	☐	☐
4. Depressive mood (feeling down, sad, on the verge of tears, lack of drive, mood swings)	☐	☐	☐	☐	☐
5. Irritability (feeling nervous, inner tension, feeling aggressive)	☐	☐	☐	☐	☐
6. Anxiety (inner restlessness, feeling panicky)	☐	☐	☐	☐	☐
7. Physical and mental exhaustion (general decrease in performance, impaired memory, decrease in concentration, forgetfulness)	☐	☐	☐	☐	☐
8. Sexual problems (change in sexual desire, in sexual activity and satisfaction)	☐	☐	☐	☐	☐
9. Bladder problems (difficulty in urinating, increased need to urinate, bladder incontinence)	☐	☐	☐	☐	☐
10. Dryness of vagina (sensation of dryness or burning in the vagina, difficulty with sexual intercourse)	☐	☐	☐	☐	☐
11. Joint and muscular discomfort (pain in the joints, rheumatoid complaints)	☐	☐	☐	☐	☐

Appendix C. Supplementary information for Chapter 3

Table C1. *Percentage of observed data by wave. Note: total sample size decreases rapidly across the waves as women were dropped from the sample once they have experienced menopause*

	Wave								
	00	**01**	**02**	**03**	**04**	**05**	**06**	**08**	**10**
Sample not excluding missing data	3300	2790	2594	2471	2265	2026	1646	1062	688
Total sample included in analysis following excluding missing data	3093	2586	2367	2212	1954	1661	1304	789	521
n and % of non-missing data by variable									
Event (menopause)	3258 (98.7)	2781 (99.7)	2587 (99.7)	2465 (99.8)	2253 (99.5)	2019 (99.7)	1643 (99.8)	1058 (99.6)	682 (99.1)
Sexual frequency	3254 (98.6)	2752 (98.6)	2536 (97.8)	2388 (96.6)	2143 (94.6)	1880 (92.8)	1434 (87.1)	878 (82.7)	622 (90.4)
Male in household	3294 (99.8)	2787 (99.9)	2584 (99.6)	2467 (99.8)	2239 (98.9)	1993 (98.4)	1580 (96.0)	997 (93.9)	620 (90.1)
Romantic male in household	3294 (99.8)	2787 (99.9)	2584 (99.6)	2467 (99.8)	2239 (98.9)	1993 (98.4)	1580 (96.0)	997 (93.9)	620 (90.1)
Total # of males in household	3294 (99.8)	2787 (99.9)	2584 (99.6)	2467 (99.8)	2239 (98.9)	1993 (98.4)	1580 (96.0)	997 (93.9)	620 (90.1)
Marital status	3300 (100)	2790 (100)	2594 (100)	2471 (100)	2265 (100)	2026 (100)	1646 (100)	1062 (100)	688 (100)
Education	3269 (99.1)	2766 (99.1)	2577 (99.3)	2453 (99.3)	2246 (99.2)	2011 (99.3)	1632 (99.1)	1057 (99.5)	686 (99.7)
Body mass index	3258 (98.7)	2677 (95.9)	2455 (94.6)	2287 (92.6)	2066 (91.2)	1751 (86.4)	1421 (86.3)	877 (82.6)	578 (84.0)
Ethnicity	3300 (100)	2790 (100)	2594 (100)	2471 (100)	2265 (100)	2026 (100)	1646 (100)	1062 (100)	688 (100)
Ever smoked	3294 (99.8)	2790 (100)	2594 (100)	2471 (100)	2265 (100)	2026 (100)	1646 (100)	1062 (100)	688 (100)

Table C1. *Percentage of observed data by wave. Note: total sample size decreases rapidly across the waves as women were dropped from the sample once they have experienced menopause*

	Wave								
	00	**01**	**02**	**03**	**04**	**05**	**06**	**08**	**10**
# of live births	3300 (100)	2790 (100)	2594 (100)	2471 (100)	2265 (100)	2026 (100)	1646 (100)	1062 (100)	688 (100)
Menarche	3271 (99.1)	2766 (99.1)	2566 (98.9)	2451 (99.2)	2244 (99.1)	2010 (99.2)	1632 (99.1)	1052 (99.1)	683 (99.3)
Health	3247 (98.4)	2770 (99.3)	2563 (98.8)	2408 (97.5)	2197 (97.0)	1964 (96.9)	1602 (97.3)	1042 (98.1)	669 (97.2)

Table C2. *Results from all models predicting age of menopause using Cox regression. Hazard ratio and 95% confidence interval are reported, with a lower hazard ratio indicating a decreased risk of entering menopause.*

	Univariate	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**
Sexual frequency (ref.: Less than monthly)								
Monthly	0.82 (0.70-0.96)	-	-	0.82 (0.70-0.96)	0.79 (0.67-0.93)	-	-	-
Weekly	0.72 (0.63-0.82)	-	-	0.72 (0.63-0.82)	0.68 (0.58-0.79)	-	-	-
Marital status (ref.: Divorced/ Separated)								
Married/ In a relationship	0.93 (0.81-1.06)	-	0.97 (0.84-1.11)	-	1.14 (0.98-1.33)	-	-	-

Table C2. *Results from all models predicting age of menopause using Cox regression. Hazard ratio and 95% confidence interval are reported, with a lower hazard ratio indicating a decreased risk of entering menopause.*

	Univariate	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**
Widowed	1.10 (0.80-1.51)	-	1.14 (0.83-1.58)	-	1.11 (0.80-1.54)	-	-	-
Lives with a romantic male partner (ref.: No)								
Yes, lives with a romantic male partner	0.90 (0.80-1.01)	-	-	-	-	0.95 (0.84-1.08)	-	-
Lives with a male (ref.: No)								
Yes, lives with a male	0.99 (0.86-1.14)	-	-	-	-	-	1.05 (0.91-1.22)	-
Total number of males in household	1.02 (0.96-1.07)	-	-	-	-	-	-	1.03 (0.97-1.10)
Age at first interview	0.89 (0.87-0.91)	0.88 (0.86-0.90)	0.88 (0.86-0.90)	0.88 (0.86-0.90)	0.88 (0.86-0.90)	0.88 (0.86-0.90)	0.88 (0.86-0.90)	0.88 (0.86-0.90)
Education (ref.: Less than high school)								
High school	0.85 (0.65-1.11)	0.85 (0.64-1.14)	0.86 (0.64-1.15)	0.87 (0.65-1.17)	0.88 (0.65-1.17)	0.85 (0.64-1.14)	0.85 (0.64-1.14)	0.85 (0.64-1.14)
Some college/ technical school	0.88 (0.69-1.12)	0.82 (0.62-1.10)	0.83 (0.62-1.10)	0.85 (0.64-1.14)	0.86 (0.65-1.15)	0.82 (0.62-1.09)	0.82 (0.62-1.10)	0.83 (0.62-1.10)

Table C2. *Results from all models predicting age of menopause using Cox regression. Hazard ratio and 95% confidence interval are reported, with a lower hazard ratio indicating a decreased risk of entering menopause.*

	Univariate	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
College degree	0.79 (0.61-1.02)	0.71 (0.52-0.95)	0.71 (0.52-0.96)	0.72 (0.53-0.97)	0.73 (0.54-0.98)	0.71 (0.52-0.95)	0.71 (0.52-0.96)	0.71 (0.53-0.96)
Post-graduate education	0.77 (0.60-0.99)	0.72 (0.53-0.97)	0.72 (0.53-0.97)	0.75 (0.55-1.01)	0.76 (0.56-1.02)	0.72 (0.53-0.97)	0.71 (0.53-0.97)	0.71 (0.53-0.97)
Body mass index	1.00 (0.99-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)
Ethnicity (ref.: African American)								
Chinese	0.96 (0.77-1.18)	0.92 (0.73-1.17)	0.92 (0.73-1.17)	0.93 (0.74-1.18)	0.92 (0.72-1.16)	0.93 (0.73-1.19)	0.91 (0.72-1.16)	0.91 (0.72-1.15)
Japanese	0.87 (0.71-1.08)	0.80 (0.64-1.00)	0.81 (0.64-1.01)	0.78 (0.62-0.98)	0.76 (0.60-0.95)	0.81 (0.65-1.02)	0.79 (0.63-0.99)	0.79 (0.63-0.99)
White	0.95 (0.83-1.09)	0.93 (0.80-1.07)	0.93 (0.81-1.08)	0.95 (0.82-1.10)	0.94 (0.81-1.08)	0.94 (0.81-1.09)	0.92 (0.80-1.07)	0.92 (0.80-1.07)
Hispanic	1.17 (0.89-1.54)	1.28 (0.94-1.74)	1.28 (0.94-1.74)	1.29 (0.95-1.76)	1.30 (0.96-1.78)	1.29 (0.94-1.75)	1.28 (0.94-1.74)	1.28 (0.94-1.74)
Smoking status (ref.: Never smoked)								
Ever smoked	1.23 (1.10-1.37)	1.24 (1.11-1.40)	1.24 (1.10-1.39)	1.24 (1.10-1.39)	1.24 (1.11-1.40)	1.24 (1.10-1.39)	1.24 (1.11-1.40)	1.25 (1.11-1.40)

Table C2. *Results from all models predicting age of menopause using Cox regression. Hazard ratio and 95% confidence interval are reported, with a lower hazard ratio indicating a decreased risk of entering menopause.*

	Univariate	**Model 1**	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**
Number of live births	0.96 (0.92-1.00)	0.96 (0.92-1.00)	0.96 (0.92-1.00)	0.97 (0.92-1.01)	0.96 (0.92-1.00)	0.96 (0.92-1.00)	0.95 (0.91-1.00)	0.95 (0.91-0.99)
Menarche	0.98 (0.95-1.02)	0.98 (0.95-1.01)	0.98 (0.95-1.01)	0.98 (0.95-1.02)	0.98 (0.95-1.02)	0.98 (0.95-1.01)	0.98 (0.95-1.02)	0.98 (0.95-1.02)
Self-perceived health (ref.: Poor)								
Fair	0.78 (0.52-1.19)	0.78 (0.51-1.18)	0.78 (0.52-1.19)	0.79 (0.52-1.20)	0.79 (0.52-1.20)	0.78 (0.51-1.19)	0.78 (0.51-1.18)	0.78 (0.51-1.18)
Good	0.76 (0.51-1.13)	0.76 (0.51-1.13)	0.76 (0.51-1.14)	0.79 (0.53-1.17)	0.78 (0.53-1.17)	0.76 (0.51-1.13)	0.76 (0.51-1.13)	0.76 (0.51-1.13)
Very good	0.72 (0.48-1.07)	0.74 (0.50-1.11)	0.75 (0.50-1.12)	0.78 (0.52-1.17)	0.78 (0.52-1.17)	0.75 (0.50-1.12)	0.74 (0.50-1.11)	0.75 (0.50-1.12)
Excellent	0.71 (0.47-1.07)	0.76 (0.50-1.16)	0.77 (0.51-1.17)	0.81 (0.53-1.23)	0.81 (0.53-1.24)	0.77 (0.50-1.17)	0.77 (0.50-1.17)	0.77 (0.50-1.17)

Table C3. *Results from a lagged analysis using Cox regression, where sexual frequency was measured at the year prior to the menopause measurement, and an additional sensitivity analysis where sexual frequency was only measured at the baseline. Hazard ratios and 95% confidence intervals are reported, where a hazard ratio of > 1 indicates a great risk of menopause, or earlier menopause.*

	Model 3 with lagged sexual freq. var	**Model 4 with lagged sexual freq. var**	**Model 3 using sexual freq. from the first wave**	**Model 4 using sexual freq. from first wave**
Sexual frequency (lagged) (ref.: Less than monthly)				
Monthly	0.81 (0.68-0.97)	0.79 (0.66-0.96)	-	-
Weekly	0.77 (0.66-0.90)	0.74 (0.62-0.88)	-	-
Sexual frequency (baseline) (ref.: Less than monthly)				
Monthly	-	-	0.96 (0.82-1.12)	0.95 (0.80-1.12)
Weekly	-	-	0.96 (0.84-1.10)	0.95 (0.81-1.10)
Marital status (ref.: Divorced/Separated)				
Married/In a relationship	-	1.11 (0.94-1.30)	-	1.03 (0.88-1.19)
Widowed	-	1.16 (0.81-1.67)	-	0.95 (0.69-1.30)
Age at first interview	0.92 (0.89-0.94)	0.92 (0.89-0.94)	0.93 (0.91-0.95)	0.93 (0.91-0.95)
Education (ref.: Less than high school)				
High school	0.88 (0.64-1.21)	0.88 (0.64-1.22)	0.84 (0.62-1.12)	0.83 (0.62-1.12)

Table C3. *Results from a lagged analysis using Cox regression, where sexual frequency was measured at the year prior to the menopause measurement, and an additional sensitivity analysis where sexual frequency was only measured at the baseline. Hazard ratios and 95% confidence intervals are reported, where a hazard ratio of > 1 indicates a great risk of menopause, or earlier menopause.*

	Model 3 with lagged sexual freq. var	**Model 4 with lagged sexual freq. var**	**Model 3 using sexual freq. from the first wave**	**Model 4 using sexual freq. from first wave**
Some college/technical school	0.89 (0.65-1.22)	0.90 (0.65-1.24)	0.79 (0.60-1.06)	0.79 (0.60-1.06)
College degree	0.76 (0.54-1.05)	0.76 (0.55-1.06)	0.67 (0.50-0.90)	0.67 (0.50-0.91)
Post-graduate education	0.78 (0.56-1.08)	0.78 (0.56-1.09)	0.69 (0.51-0.93)	0.68 (0.51-0.92)
Body mass index	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.98 (0.97-0.99)	0.98 (0.97-0.99)
Race (ref.: African American)				
Chinese	0.92 (0.71-1.18)	0.91 (0.70-1.17)	0.90 (0.71-1.14)	0.89 (0.70-1.13)
Japanese	0.76 (0.60-0.97)	0.75 (0.59-0.95)	0.79 (0.63-0.99)	0.78 (0.62-0.98)
White	0.94 (0.80-1.10)	0.93 (0.80-1.09)	0.91 (0.79-1.05)	0.91 (0.78-1.05)
Hispanic	1.37 (0.98-1.92)	1.38 (0.98-1.93)	1.38 (1.01-1.89)	1.38 (1.01-1.89)
Smoking status (ref.: Never smoked)				
Ever smoked	1.19 (1.05-1.35)	1.19 (1.05-1.35)	1.27 (1.13-1.43)	1.27 (1.13-1.43)
Number of live births	0.96 (0.92-1.01)	0.96 (0.91-1.00)	0.95 (0.91-1.00)	0.95 (0.91-1.00)
Menarche	0.97 (0.94-1.01)	0.97 (0.94-1.01)	0.97 (0.94-1.01)	0.97 (0.94-1.01)

Table C3. *Results from a lagged analysis using Cox regression, where sexual frequency was measured at the year prior to the menopause measurement, and an additional sensitivity analysis where sexual frequency was only measured at the baseline. Hazard ratios and 95% confidence intervals are reported, where a hazard ratio of > 1 indicates a great risk of menopause, or earlier menopause.*

	Model 3 with lagged sexual freq. var	**Model 4 with lagged sexual freq. var**	**Model 3 using sexual freq. from the first wave**	**Model 4 using sexual freq. from first wave**
Self-perceived health (ref.: Poor)				
Fair	0.73 (0.47-1.13)	0.73 (0.47-1.13)	0.88 (0.58-1.32)	0.87 (0.58-1.31)
Good	0.70 (0.46-1.06)	0.69 (0.46-1.06)	0.85 (0.58-1.25)	0.85 (0.57-1.25)
Very good	0.69 (0.45-1.06)	0.69 (0.45-1.05)	0.92 (0.62-1.35)	0.91 (0.62-1.35)
Excellent	0.74 (0.48-1.16)	0.74 (0.48-1.16)	1.02 (0.68-1.53)	1.02 (0.67-1.53)

Appendix D. Supplementary information for Chapter 4

Table D1. *Full results from Hypothesis 1, using Poisson regression to model vasomotor symptoms. Results show the incidence rate ratio and 95% confidence interval. An incidence rate ratio of >1 indicates more severe menopause symptoms.*

	Covariates	Current and ancestral residence pattern	Ancestral residence pattern	Current residence pattern
Ancestral residence pattern (ref.: Duolocal)				
Mixed	-	1.20 (0.99-1.45)	1.18 (0.97-1.43)	-
Patrilocal	-	1.06 (0.94-1.18)	1.21 (1.09-1.33)	-
Current residence pattern (ref.: Lives with natal group)				
Lives away from natal group	-	1.26 (1.14-1.39)	-	1.28 (1.17-1.40)
Age	0.98 (0.98-0.99)	0.98 (0.98-0.99)	0.98 (0.98-0.99)	0.98 (0.98-0.99)
Menopause status (ref.: Perimenopausal)				
Post-menopausal	0.91 (0.79-1.04)	0.92 (0.80-1.06)	0.90 (0.78-1.04)	0.92 (0.80-1.07)
Body mass index	1.00 (0.99-1.01)	1.00 (0.99-1.01)	1.00 (0.99-1.01)	1.00 (0.99-1.01)
Parity	1.09 (1.07-1.12)	1.08 (1.05-1.11)	1.09 (1.06-1.11)	1.08 (1.06-1.11)
Experienced financial difficulty in the past year (ref.: Yes)				

Table D1. *Full results from Hypothesis 1, using Poisson regression to model vasomotor symptoms. Results show the incidence rate ratio and 95% confidence interval. An incidence rate ratio of >1 indicates more severe menopause symptoms.*

	Covariates	Current and ancestral residence pattern	Ancestral residence pattern	Current residence pattern
No	0.80 (0.73-0.88)	0.82 (0.75-0.90)	0.82 (0.75-0.90)	0.81 (0.74-0.89)
Smoking (ref.: Ever smoked)				
Never smoked	1.13 (0.95-1.35)	1.19 (1.00-1.43)	1.18 (0.99-1.42)	1.19 (1.00-1.43)

Table D2. *Full results from Hypothesis 2, using Cox regression to model menopause symptom duration. Results show the hazard ratio and 95% confidence interval. A hazard ratio of > 1 indicates a shorter symptom duration.*

	Covariates	Current and ancestral residence pattern	Ancestral residence pattern	Current residence pattern
Ancestral residence pattern (ref.: Duolocal)				
Mixed	-	1.93 (0.44-8.51)	1.93 (0.44-8.52)	-
Patrilocal	-	1.24 (0.59-2.63)	1.22 (0.69-2.18)	-
Current residence pattern (ref.: Lives with natal group)				
Lives away from natal group	-	0.98 (0.45-2.11)	-	1.06 (0.60-1.86)
Age	1.06 (1.02-1.10)	1.06 (1.02-1.10)	1.06 (1.02-1.10)	1.06 (1.02-1.10)

Table D2. *Full results from Hypothesis 2, using Cox regression to model menopause symptom duration. Results show the hazard ratio and 95% confidence interval. A hazard ratio of > 1 indicates a shorter symptom duration.*

	Covariates	Current and ancestral residence pattern	Ancestral residence pattern	Current residence pattern
Body mass index	1.01 (0.93-1.09)	1.02 (0.94-1.12)	1.02 (0.94-1.12)	1.01 (0.93-1.10)
Parity	0.92 (0.79-1.08)	0.89 (0.75-1.06)	0.89 (0.75-1.06)	0.92 (0.79-1.08)
Experienced financial difficulty in the past year (ref.: Yes)	1.16 (0.69-1.95)	1.26 (0.71-2.21)	1.26 (0.72-2.20)	1.18 (0.68-2.04)
No				
Smoking (ref.: Ever smoked)				
Never smoked	0.53 (0.27-1.04)	0.50 (0.25-1.00)	0.50 (0.25-1.00)	0.54 (0.27-1.06)

Table D3. *Full results from Hypothesis 3, using Cox regression to predict menopause symptom duration. Results show the hazard ratio and 95% confidence interval. A hazard ratio of >1 indicates an earlier menopause.*

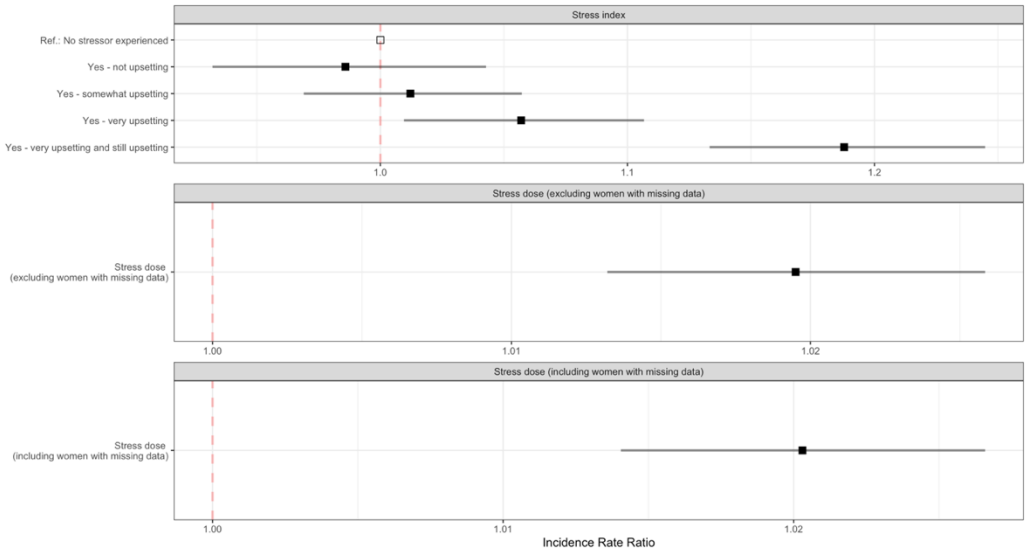
	Covariates	Current and ancestral residence pattern	Ancestral residence pattern	Current residence pattern
Ancestral residence pattern (ref.: Duolocal)				
Mixed	-	1.03 (0.66-1.62)	1.03 (0.66-1.61)	-
Patrilocal	-	1.04 (0.78-1.39)	0.99 (0.78-1.26)	-

Table D3. *Full results from Hypothesis 3, using Cox regression to predict menopause symptom duration. Results show the hazard ratio and 95% confidence interval. A hazard ratio of >1 indicates an earlier menopause.*

	Covariates	**Current and ancestral residence pattern**	**Ancestral residence pattern**	**Current residence pattern**
Current residence pattern (ref.: Lives with natal group)				
Lives away from natal group	-	0.92 (0.73-1.18)	-	0.94 (0.77-1.15)
Body mass index	0.97 (0.94-0.99)	0.97 (0.94-0.99)	0.97 (0.94-0.99)	0.97 (0.94-0.99)
Parity	1.07 (1.02-1.12)	1.07 (1.02-1.12)	1.07 (1.02-1.12)	1.07 (1.02-1.12)
Experienced financial difficulty in the past year (ref.: Yes)				
No	1.00 (0.82-1.23)	1.00 (0.82-1.23)	1.00 (0.82-1.23)	1.00 (0.82-1.23)
Smoking (ref.: Ever smoked)				
Never smoked	0.51 (0.34-0.75)	0.51 (0.34-0.76)	0.50 (0.34-0.75)	0.50 (0.34-0.74)

Appendix E. Supplementary information for Chapter 5

Figure E1. *Relationship between the different stress measures and vasomotor symptoms, modelled using multilevel Poisson regression with random effects. Models are unadjusted, with a higher Incidence Rate Ratio indicating more frequent symptoms.*



To create the stress dose variable, how many of the 18 stressful life events the woman had experienced were counted. However, some women had not responded to all 18 events. To deal with this missing data there were two options:

1) To treat the whole derived variable as missing, and therefore not include the woman in the wave where there was missing data
2) Treat the missing stressors as 'not experienced', and therefore count the stressors the woman did report experiencing

I decided to go with the second option to avoid losing participants from the analysis as, if I went with option 1 and excluded women who did not respond to a stressor I would have list 127 women. To justify this decision, I show the results generated using variables made from both of the possible options. As shown in Figure E1, the treatment of missing data in the stress dose variable does not change the results. It was therefore justified that using treating missing stressor data as not having experienced the stressor was preferable, as it prevented losing data on 127 women.

Table E1. *Percent of observed data by variable and wave. The 'n' row shows the possible amount of data that could be observed.*

	% observed by wave								
Variable	**01**	**02**	**03**	**04**	**05**	**06**	**08**	**09**	**10**
n	2881	2748	2709	2679	2617	2448	2278	2255	2245
Age	2881 (100.0)	2748 (100)	2709 (100)	2679 (100)	2617 (100)	2448 (100)	2278 (100)	2255 (100)	2245 (100)
Menopause status	2859 (99.2)	2725 (99.2)	2693 (99.4)	2658 (99.2)	2603 (99.5)	2441 (99.7)	2269 (99.6)	2246 (99.6)	2239 (99.7)
Vasomotor symptoms	2852 (99.0)	2705 (98.4)	2637 (97.3)	2558 (95.5)	2471 (94.4)	2280 (93.1)	2182 (95.8)	2122 (94.1)	2082 (92.7)
Stress index	2770 (96.1)	2623 (95.5)	2537 (93.7)	2482 (92.6)	2398 (91.6)	2173 (88.8)	2113 (92.8)	2047 (90.8)	2087 (93.0)
Stress dose	2731 (94.8)	2589 (94.2)	2498 (92.2)	2447 (91.3)	2353 (89.9)	2117 (86.5)	2081 (91.4)	2005 (88.9)	2046 (91.1)
Social support	2718 (94.3)	2582 (94.0)	2497 (92.2)	2409 (89.9)	2313 (88.4)	2095 (85.6)	2054 (90.2)	1237 (54.9)	2000 (89.1)
Marital status	2881 (100)	2748 (100)	2709 (100)	2679 (100)	2617 (100)	2448 (100)	2278 (100)	2255 (100)	2245 (100)
Smoking status	2881 (100)	2748 (100)	2709 (100)	2679 (100)	2617 (100)	2448 (100)	2278 (100)	2255 (100)	2245 (100)
Education	2857 (99.2)	2729 (99.3)	2688 (99.2)	2658 (99.2)	2596 (99.2)	2432 (99.3)	2265 (99.4)	2242 (99.4)	2231 (99.4)
Ethnicity	2881 (100)	2748 (100)	2709 (100)	2679 (100)	2617 (100)	2448 (100)	2278 (100)	2255 (100)	2245 (100)
Health	2861 (99.3)	2715 (98.8)	2637 (97.3)	2591 (96.7)	2532 (96.8)	2384 (97.4)	2249 (98.7)	2217 (98.3)	2196 (97.8)

Table E2. *Predicting missingness in stress and social support. Results from univariable binary logistic regression predicting the odds of data being observed in the stress index, stress dose, and social support. Outcome here is observed (1) or missing (0). Odds ratios and 95% confidence intervals are reported. A higher odds ratio indicates a higher probability of the data being observed.*

	Stress index	**Stress dose**	**Social support**
Vasomotor symptoms	1.13 (1.11-1.16)	1.13 (1.11-1.16)	1.03 (1.01-1.04)
Education (ref.: Less than high school)			
High school	1.27 (1.09-1.47)	1.27 (1.09-1.47)	1.05 (0.92-1.20)
Some college/technical school	1.35 (1.17-1.55)	1.35 (1.17-1.55)	1.08 (0.96-1.22)
College degree	1.47 (1.27-1.70)	1.47 (1.27-1.70)	1.15 (1.01-1.30)
Post-graduate education	1.44 (1.24-1.66)	1.44 (1.24-1.66)	1.15 (1.02-1.31)
Smoking (ref.: Never smoked)			
Ever smoked	0.93 (0.87-0.99)	0.93 (0.87-0.99)	0.93 (0.89-0.99)
Marital status (ref.: Divorced/Separated/Single)			
Married/In a relationship	0.97 (0.90-1.05)	0.97 (0.90-1.05)	1.21 (1.14-1.28)
Widowed	2.45 (1.84-3.24)	2.45 (1.84-3.24)	1.38 (1.16-1.64)
Health (ref.: Poor)			
Fair	1.09 (0.86-1.39)	1.09 (0.86-1.39)	1.06 (0.87-1.29)
Good	1.04 (0.83-1.32)	1.04 (0.83-1.32)	1.11 (0.92-1.33)
Very good	1.11 (0.88-1.40)	1.11 (0.88-1.40)	1.22 (1.01-1.48)

Table E2. *Predicting missingness in stress and social support. Results from univariable binary logistic regression predicting the odds of data being observed in the stress index, stress dose, and social support. Outcome here is observed (1) or missing (0). Odds ratios and 95% confidence intervals are reported. A higher odds ratio indicates a higher probability of the data being observed.*

	Stress index	**Stress dose**	**Social support**
Excellent	0.85 (0.67-1.08)	0.85 (0.67-1.08)	1.12 (0.92-1.36)
Ethnicity (ref.: Black/African American)			
White	1.34 (1.24-1.44)	1.34 (1.24-1.44)	1.25 (1.18-1.33)
Chinese	1.30 (1.15-1.47)	1.30 (1.15-1.47)	1.12 (1.02-1.24)
Hispanic	0.70 (0.60-0.81)	0.70 (0.60-0.81)	1.29 (1.12-1.49)
Japanese	1.73 (1.53-1.96)	1.73 (1.53-1.96)	1.44 (1.30-1.59)
Menopausal status (ref.: Early peri-menopause)			
Pre-menopausal	0.15 (0.13-0.16)	0.15 (0.13-0.16)	0.31 (0.29-0.34)
Late peri-menopause	4.01 (3.30-4.88)	4.01 (3.30-4.88)	1.30 (1.16-1.46)
Post-menopausal	4.14 (3.70-4.63)	4.14 (3.70-4.63)	0.90 (0.84-0.96)
Other	4.63 (3.90-5.50)	4.63 (3.90-5.50)	1.49 (1.34-1.64)

***Table E3**. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	Support model	Stress model 1	Stress model 2	Full model 1	Full model 2	Interaction model 1	Interaction model 2	Lagged model
Average support received	-	0.98 (0.96-1.00)	-	-	0.99 (0.96-1.01)	0.99 (0.96-1.01)	1.02 (0.97-1.07)	1.00 (0.97-1.03)	-
Stress index (ref.: No)									
Yes - not upsetting	-	-	1.01 (0.95-1.06)	-	1.01 (0.95-1.06)	-	1.11 (0.78-1.60)	-	1.02 (0.96-1.08)
Yes - somewhat upsetting	-	-	1.03 (0.98-1.07)	-	1.03 (0.98-1.07)	-	1.02 (0.79-1.30)	-	1.01 (0.97-1.06)
Yes - very upsetting	-	-	1.07 (1.03-1.13)	-	1.07 (1.03-1.13)	-	1.31 (1.01-1.69)	-	1.00 (0.95-1.05)
Yes - very upsetting and still upsetting	-	-	1.21 (1.15-1.26)	-	1.20 (1.15-1.26)	-	1.57 (1.23-2.00)	-	1.07 (1.01-1.12)
Stress dose	-	-	-	1.03 (1.02-1.03)	-	1.03 (1.02-1.03)	-	1.05 (1.02-1.07)	-
Age	1.12 (1.09-1.15)	1.12 (1.09-1.16)	1.12 (1.09-1.16)	1.12 (1.09-1.16)	1.12 (1.09-1.16)	1.13 (1.09-1.16)	1.12 (1.09-1.16)	1.12 (1.09-1.16)	1.08 (1.05-1.12)

***Table E3**. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	**Support model**	**Stress model 1**	**Stress model 2**	**Full model 1**	**Full model 2**	**Interaction model 1**	**Interaction model 2**	**Lagged model**
Age^2	0.92 (0.91-0.93)	0.92 (0.91-0.93)	0.92 (0.91-0.94)	0.92 (0.91-0.94)	0.92 (0.91-0.94)	0.92 (0.91-0.94)	0.92 (0.91-0.94)	0.92 (0.91-0.93)	0.90 (0.88-0.91)
Age^3	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	0.99 (0.98-1.00)	1.01 (1.00-1.02)
Marital status (ref.: Divorced/ Separated/ Single)									
Married/ In a relationship	1.10 (1.05-1.16)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.11 (1.06-1.17)	1.10 (1.04-1.16)
Widowed	1.07 (0.95-1.19)	1.07 (0.95-1.20)	1.05 (0.94-1.18)	1.06 (0.95-1.19)	1.06 (0.94-1.18)	1.07 (0.95-1.19)	1.06 (0.94-1.18)	1.07 (0.95-1.19)	1.04 (0.92-1.18)
Smoking (ref.: Never smoked)									
Ever smoked	1.22 (1.12-1.32)	1.22 (1.12-1.32)	1.21 (1.11-1.31)	1.21 (1.12-1.31)	1.21 (1.11-1.31)	1.21 (1.12-1.31)	1.21 (1.12-1.31)	1.21 (1.12-1.31)	1.19 (1.09-1.30)
Education (ref.: Less than high school)									

***Table E3**. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	Support model	Stress model 1	Stress model 2	Full model 1	Full model 2	Interaction model 1	Interaction model 2	Lagged model
High school	0.95 (0.78-1.15)	0.95 (0.78-1.16)	0.94 (0.77-1.15)	0.94 (0.77-1.14)	0.94 (0.78-1.15)	0.94 (0.77-1.14)	0.94 (0.77-1.15)	0.94 (0.77-1.14)	0.96 (0.77-1.18)
Some college/ technical school	0.92 (0.76-1.12)	0.92 (0.76-1.12)	0.91 (0.75-1.10)	0.90 (0.75-1.09)	0.91 (0.76-1.11)	0.91 (0.75-1.10)	0.91 (0.76-1.11)	0.91 (0.75-1.10)	0.94 (0.77-1.15)
College degree	0.70 (0.57-0.85)	0.70 (0.57-0.86)	0.69 (0.56-0.84)	0.69 (0.56-0.84)	0.69 (0.56-0.84)	0.69 (0.56-0.84)	0.69 (0.56-0.84)	0.69 (0.56-0.84)	0.73 (0.59-0.90)
Post-graduate education	0.72 (0.59-0.88)	0.72 (0.59-0.88)	0.71 (0.58-0.87)	0.71 (0.58-0.86)	0.71 (0.58-0.87)	0.71 (0.58-0.87)	0.71 (0.58-0.87)	0.71 (0.58-0.87)	0.74 (0.60-0.92)
Ethnicity (ref.: African American)									
Chinese	0.44 (0.37-0.52)	0.44 (0.37-0.52)	0.45 (0.38-0.54)	0.45 (0.38-0.53)	0.45 (0.38-0.54)	0.45 (0.38-0.53)	0.46 (0.39-0.54)	0.45 (0.38-0.53)	0.44 (0.37-0.52)
Japanese	0.41 (0.35-0.48)	0.41 (0.35-0.48)	0.42 (0.36-0.49)	0.42 (0.36-0.50)	0.42 (0.36-0.49)	0.43 (0.36-0.50)	0.42 (0.36-0.49)	0.43 (0.36-0.50)	0.43 (0.36-0.50)
White	0.70 (0.64-0.77)	0.70 (0.64-0.77)	0.70 (0.64-0.77)	0.71 (0.65-0.78)	0.70 (0.64-0.77)	0.71 (0.65-0.78)	0.70 (0.64-0.77)	0.71 (0.65-0.78)	0.70 (0.63-0.77)

***Table E3**. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	Support model	Stress model 1	Stress model 2	Full model 1	Full model 2	Interaction model 1	Interaction model 2	Lagged model
Hispanic	0.62 (0.51-0.75)	0.62 (0.51-0.75)	0.64 (0.53-0.77)	0.65 (0.54-0.79)	0.64 (0.53-0.77)	0.65 (0.54-0.79)	0.64 (0.53-0.77)	0.65 (0.54-0.79)	0.58 (0.47-0.72)
Health (ref.: Poor)									
Fair	0.96 (0.88-1.04)	0.96 (0.88-1.04)	0.98 (0.90-1.06)	0.96 (0.88-1.05)	0.98 (0.90-1.06)	0.96 (0.88-1.05)	0.98 (0.90-1.06)	0.96 (0.88-1.04)	0.95 (0.87-1.05)
Good	0.82 (0.75-0.90)	0.82 (0.75-0.90)	0.84 (0.77-0.92)	0.83 (0.76-0.90)	0.84 (0.77-0.92)	0.83 (0.76-0.90)	0.84 (0.77-0.92)	0.82 (0.75-0.90)	0.81 (0.73-0.90)
Very good	0.74 (0.67-0.81)	0.74 (0.67-0.81)	0.76 (0.70-0.84)	0.75 (0.68-0.82)	0.76 (0.70-0.84)	0.75 (0.68-0.82)	0.77 (0.70-0.84)	0.75 (0.68-0.82)	0.73 (0.66-0.82)
Excellent	0.65 (0.58-0.72)	0.65 (0.58-0.72)	0.67 (0.60-0.74)	0.66 (0.59-0.73)	0.67 (0.60-0.74)	0.66 (0.59-0.73)	0.67 (0.61-0.74)	0.66 (0.59-0.73)	0.64 (0.57-0.71)
Menopause status (ref.: Early peri-menopause)									
Pre-menopause	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.61-0.71)	0.66 (0.60-0.73)

***Table E3**. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	Support model	Stress model 1	Stress model 2	Full model 1	Full model 2	Interaction model 1	Interaction model 2	Lagged model
Late peri menopause	1.87 (1.79-1.96)	1.87 (1.79-1.96)	1.88 (1.80-1.97)	1.88 (1.80-1.97)	1.88 (1.80-1.97)	1.88 (1.80-1.97)	1.88 (1.80-1.97)	1.89 (1.80-1.97)	1.95 (1.86-2.05)
Post-menopause	1.44 (1.37-1.51)	1.44 (1.37-1.51)	1.44 (1.37-1.50)	1.45 (1.38-1.52)	1.44 (1.37-1.51)	1.45 (1.38-1.52)	1.44 (1.37-1.51)	1.45 (1.38-1.52)	1.52 (1.45-1.60)
Other	1.07 (1.02-1.13)	1.07 (1.02-1.13)	1.07 (1.02-1.13)	1.08 (1.03-1.13)	1.07 (1.02-1.13)	1.08 (1.03-1.13)	1.07 (1.02-1.13)	1.08 (1.03-1.13)	1.11 (1.05-1.18)
Support*Stress index (ref.: No)									
Average support received* Yes - not upsetting	-	-	-	-	-	-	0.98 (0.90-1.06)	-	-
Average support received* Yes - somewhat upsetting	-	-	-	-	-	-	1.00 (0.95-1.06)	-	-
Average support received* Yes - very upsetting	-	-	-	-	-	-	0.96 (0.90-1.01)	-	-

Table E3*. Full model results from Poisson regression modelling vasomotor symptoms. Incidence rate ratio and 95% confidence intervals reported here. In the Lagged model, the stress index is measured at time-1. Age is centred in all analyses.*

	Base model	Support model	Stress model 1	Stress model 2	Full model 1	Full model 2	Interaction model 1	Interaction model 2	Lagged model
Average support received* Yes - very upsetting and still upsetting	-	-	-	-	-	-	0.94 (0.89-0.99)	-	-
Average support received*Stress dose	-	-	-	-	-	-	-	1.00 (0.99-1.00)	-

Appendix F. Supplementary information for Chapter 6

Table F1. *Comparison of models with Vuong's statistic. NB = negative binomial regression, ZINB = zero-inflated negative binomial regression.*

Outcome	First model	Second model	Vuong z-statistic	p value	Better model
Parent help	NB	ZINB	-3.623649	<0.001	ZINB
Grandchild care	NB	ZINB	-12.897627	<0.001	ZINB

Appendix G. Supplementary information for Chapter 7

Table G1. *Results from Poisson regression modelling number of grandchildren, using the smaller dataset subset to exclude women with no data for age at first birth. Incidence rate ratio and 95% confidence intervals are reported.*

	Covariate model 1	**Age of menopause + Covariate model 1**	**Age of last menstruation + Covariate model 1**
Menopause age (ref.: Not yet gone through menopause)			
40 or younger	-	1.53 (0.98, 2.42)	-
41-45	-	1.48 (1.17, 1.87)	-
46-50	-	1.26 (1.06-1.50)	-
51-55	-	1.07 (0.90-1.27)	-
Stopped for another reason	-	1.50 (1.26-1.78)	-
Age of last period (ref.: Not yet gone through menopause)			
40 or younger	-	-	1.85 (1.49-2.29)
41-45	-	-	1.38 (1.13-1.68)
46-50	-	-	1.27 (1.07-1.50)
51-55	-	-	1.11 (0.93-1.31)
Number of children	1.61 (1.52-1.70)	1.61 (1.53-1.70)	1.61 (1.53-1.70)

Table G1. *Results from Poisson regression modelling number of grandchildren, using the smaller dataset subset to exclude women with no data for age at first birth. Incidence rate ratio and 95% confidence intervals are reported.*

	Covariate model 1	**Age of menopause + Covariate model 1**	**Age of last menstruation + Covariate model 1**
Employment status (ref.: Unemployed)			
Employed	0.87 (0.70-1.08)	0.91 (0.73-1.12)	0.92 (0.74-1.14)
Other	0.87 (0.69-1.11)	0.90 (0.71-1.14)	0.91 (0.72-1.15)
Self-perceived health (ref.: Very poor/poor)			
Fair	0.71 (0.56-0.90)	0.71 (0.56-0.89)	0.71 (0.56-0.89)
Very good/good	0.54 (0.43-0.67)	0.55 (0.44-0.68)	0.56 (0.45-0.69)
Excellent	0.39 (0.30-0.50)	0.41 (0.32-0.53)	0.41 (0.32-0.53)
Marital status (ref.: Other (e.g. divorced, widowed, single))			
Married/Civil partnership	0.92 (0.82-1.03)	0.93 (0.83-1.04)	0.93 (0.83-1.04)

Table G2. *Results from Poisson regression modelling number of children, with incidence rate ratio and 95% confidence intervals reported.*

	Covariate model	**Age of menopause + Covariate model**	**Age of last period + Covariate model**
Menopause age (ref.: Not yet gone through menopause)			
40 or younger	-	0.98 (0.80-1.18)	-

Table G2. *Results from Poisson regression modelling number of children, with incidence rate ratio and 95% confidence intervals reported.*

	Covariate model	**Age of menopause + Covariate model**	**Age of last period + Covariate model**
41-45	-	0.95 (0.86-1.06)	-
46-50	-	0.91 (0.85-0.98)	-
51-55	-	0.98 (0.92-1.06)	-
Stopped for another reason	-	1.03 (0.96-1.11)	-
Age of last period (ref.: Not yet gone through menopause)			
40 or younger	-	-	1.06 (0.96-1.16)
41-45	-	-	0.96 (0.88-1.04)
46-50	-	-	0.94 (0.88-1.01)
51-55	-	-	0.99 (0.92-1.06)
Employment status (ref.: Unemployed)			
Employed	0.99 (0.90-1.08)	0.99 (0.90-1.09)	0.99 (0.90-1.09)
Other	1.04 (0.94-1.16)	1.05 (0.94-1.17)	1.05 (0.94-1.16)
Self-perceived health (ref.: Very poor/poor)			
Fair	0.90 (0.81-1.00)	0.90 (0.82-1.00)	0.91 (0.82-1.01)

Table G2. *Results from Poisson regression modelling number of children, with incidence rate ratio and 95% confidence intervals reported.*

	Covariate model	**Age of menopause + Covariate model**	**Age of last period + Covariate model**
Very good/good	0.87 (0.79-0.95)	0.87 (0.79-0.96)	0.87 (0.79-0.96)
Excellent	0.86 (0.77-0.96)	0.87 (0.78-0.97)	0.86 (0.78-0.97)
Marital status (ref.: Other (e.g. divorced, widowed, single))			
Married/Civil partnership	1.28 (1.22-1.35)	1.28 (1.21-1.34)	1.28 (1.22-1.35)

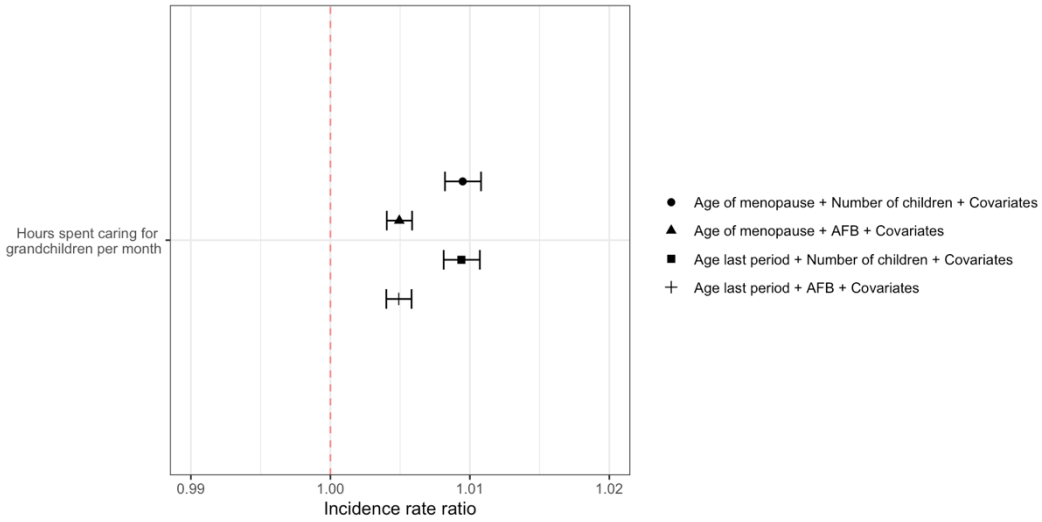
Table G3. *Sensitivity analysis, modelling number of grandchildren without controlling for variables related to direct reproduction. Results from Poisson regression models predicting number of grandchildren, showing incidence rate ratios and 95% confidence intervals.*

	Covariates	**Age of menopause + covariates**	**Age of last period + covariates**
Menopause age (ref.: Not yet gone through menopause)			
40 or younger	-	3.76 (2.31-8.58)	-
41-45	-	3.84 (2.88-5.58)	-
46-50	-	3.03 (2.52-3.76)	-
51-55	-	2.79 (2.35-3.40)	-
Stopped for another reason	-	4.16 (3.28-5.51)	-

Table G3. *Sensitivity analysis, modelling number of grandchildren without controlling for variables related to direct reproduction. Results from Poisson regression models predicting number of grandchildren, showing incidence rate ratios and 95% confidence intervals.*

	Covariates	**Age of menopause + covariates**	**Age of last period + covariates**
Age of last period (ref.: Not yet gone through menopause)			
40 or younger	-	-	5.63 (3.99-8.69)
41-45	-	-	3.60 (2.84-4.82)
46-50	-	-	3.12 (2.60-3.86)
51-55	-	-	2.89 (2.44-3.55)
Employment status (ref.: Unemployed)			
Employed	2.16 (1.85-2.63)	2.22 (1.89-2.72)	2.24 (1.90-2.74)
Other	2.42 (1.99-3.11)	2.50 (2.04-3.23)	2.51 (2.05-3.25)
Self-perceived health (ref.: Very poor/poor)			
Fair	1.96 (1.69-2.36)	1.96 (1.69-2.36)	1.97 (1.70-2.37)
Very good/good	1.61 (1.46-1.81)	1.63 (1.47-1.85)	1.64 (1.48-1.86)
Excellent	1.41 (1.30-1.56)	1.44 (1.32-1.61)	1.44 (1.32-1.61)
Marital status (ref.: Other (e.g. divorced, widowed, single))			
Married/Civil partnership	2.42 (2.19-2.70)	2.42 (2.19-2.70)	2.44 (2.20-2.72)

Figure G1. *Results from Poisson regression modelling the hours spent caring for grandchildren per month by number of grandchildren. For models that include number of children, n = 4348; for models that include age at first birth (AFB), n = 3492.*



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