We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

6,900
Open access books available

185,000
International authors and editors

200M
Downloads

Our authors are among the

154
Countries delivered to

TOP 1%
most cited scientists

12.2%
Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.
For more information visit www.intechopen.com



Chapter 2

Menopause Female Reproductive Senescence from the Viewpoint of Evolutionary Anthropology

Sylvia Kirchengast

Additional information is available at the end of the chapter

http://dx.doi.org/10.5772/intechopen.68682

Abstract

Female reproductive senescence is characterized by the so-called menopausal transition taking place between the ages of 40 and 60 years. The major event of menopausal transition is menopause itself, which is biomedically defined as the cessation of menstrual function and the irreversible termination of female reproductive capability. Recent human females experience a postreproductive period from about 30 years. Such a long postreproductive period is absolutely uncommon among animals. Consequently, human menopause is still an evolutionary puzzle and several theories to explain the evolutionary basis of menopause have been presented. Menopausal transition, however, is also seen as a period of increased somatic and psychic symptoms which make this phase of life quite uncomfortable for affected women. In the present study, menopause and climacteric complaints are discussed from the viewpoint of evolutionary anthropology.

Keywords: menopause, human evolution, climacteric symptoms, reproductive senescence

1. Introduction

Recent *Homo sapiens* has the longest lifespan of all terrestrial mammals, however, human females stop to reproduce in the middle of it [1, 2]. Every woman who lives until about 50 years and beyond experiences the irreversible cessation of reproductive function, i.e., menopause. The World Health Organization (WHO) has defined menopause as the last spontaneous menstrual bleeding or the permanent cessation of menstruation resulting from loss of ovarian follicular activity [3]. Menopause is preceded by a phase of irregular cycles and starting hormonal changes, which is commonly called perimenopause. Perimenopause continued until 12 months after the last spontaneous menstrual bleeding, because no human female

INTECH
open science | open minds

© 2017 The Author(s). Licensee InTech. This chapter is distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/3.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. CC BY

knows exactly that the actual bleeding is really the last one [4]. Therefore, the phase of postmenopause is reached when a woman had no menstrual periods over 12 months. Menopause is a universal, one-time life event, which marks the transition from reproductive to postreproductive life in females [5]. Consequently, menopause is a marker of reproductive ageing patterns typical of the female *Homo sapiens* [6]. Menopause occurs usually between 45 and 55 years, recent human females, however, routinely live for about 30 years after the cessation of reproductive function [2]. Furthermore, reproductive senescence is markedly accelerated relative to general somatic aging among female *Homo sapiens* [7]. Even in traditional foragers societies such as the Ache, Hadza, or !Kung San, that do not enjoy the benefits of modern medicine women can expect 20 and more years of active life following menopause [8–10]. From the viewpoint of evolutionary biology, this extraordinary long postreproductive phase among human females is in strict contradiction to the biogenetic imperative and the paradigm that natural selection forces organisms to maximize reproductive success [11]. According to these assumptions, reproductive senescence and in particular menopause followed by a long postreproductive period should be rare in nature. In the present review, the phenomenon of menopause and extended postreproductive life span is focused on from the viewpoint of evolutionary anthropology, in particular, human life history theory. For an appropriate analysis of menopausal transition, we have to consider two different levels of causality in biology, i.e., proximate and ultimate explanations of biological phenomena [11]. Proximate causes are immediate mechanisms, such as physiological or ontogenetic factors, whereas ultimate explanations, in contrast, tried to interpret biological phenomena in an evolutionary sense [11]. Therefore, proximate as well as ultimate causes of menopause are discussed.

2. Human life history theory

As pointed out above, according to the biogenetic imperative and natural selection theory, menopause should be rare in nature. Among *Homo sapiens*, however, menopause is an obligatory part of female life history. Before we discuss menopause from the viewpoint of evolutionary anthropology, we have considered life history theory.

Every species has evolved its own patterns of ontogeny of the individual organism from conception to death. This species typical process contains somatic growth, development, maturation, reproduction, and senescence—all of them are energetically costly events [12]. Life history theory tries to explain the evolution of these patterns of growth and reproduction by identifying trade-offs [13]. An important trade-off is between somatic growth and the maintenance on the one hand and successful reproduction on the other. For most organisms, it is not possible to provide enough energy to grow and to reproduce simultaneously. Therefore many organisms have evolved a timely separation between growth and reproduction. A period where energy is provided for growth and development is followed by a period where energy is used for successful reproduction. Human life history differs from that of other social mammals in several puzzling details. Even our next living relatives, the nonhuman primates, exhibit marked differences in certain features of life history [14]. In detail, human infants are weaned relatively early, on average by age 36 months, but after weaning human children

depend on their mothers or other older group members for food and protection much longer than do the offspring of any other mammal, usually until they age about 7 years [12, 13]. From the viewpoint of evolutionary anthropology and human life history theory, the period between weaning and age of 7 years is defined as childhood stage, which is characterized by a set of biological and behavioral traits. This life history definition of childhood differs from the commonly used term childhood which refers to any time between birth and sexual maturation. It is assumed that the life history stage of childhood evolved about 2 million years ago among *Homo erectus*. The stage of childhood is not found among nonhuman primates [15]. Furthermore, humans reach puberty, i.e., sexual maturation, later than nonhuman primates and the short-term event of puberty is followed by an extended period of adolescence [15]. Adolescence is assumed to be a quite young stage of human life history occurring among *Homo sapiens* only. Since reproduction during early adolescence is rare, the interval between sexual maturation and first reproduction is much longer among humans than among nonhuman primates. Consequently, the age at first reproduction is much older than that of nonhuman primates; nevertheless human fertility may be higher than that among our nonhuman relatives. Modern humans however differ not only in the subadult stages of life history and reproductive success markedly from nonhuman primates and all other social mammals, the most distinct features are the extraordinary long life span and the prolonged period of female reproductive senescence, i.e., a long postmenopause [16]. During the fifth decade of life, fertility declines to essentially zero in human females; although one can expect to live for about further 30 years. This marked discrepancy between ovarian ageing and general ageing is a typical feature of female *Homo sapiens*. A prolonged postreproductive phase is not found among other animals, even among great social mammals with the exception of some toothed whale species.

3. Reproductive senescence among animals

As pointed out above, human females experience the cessation of reproductive function long before they die. Some signs of reproductive ageing are found among invertebrates in particular tephritid fruit flies [17] and the nematode *Caenorhabditis elegans* [18]. A kind of reproductive ageing characterized by a decline of sex steroid levels and a reduced probability of successful reproduction is found among several free living social mammals such as toothed whales, elephants, lions, or first of all primates [19–25]. An obligatory postreproductive life stage of 30 years and more, however, is exclusively found among human females [23]. Long postreproductive periods are uncommon among animals, even among large social mammals such as nonhuman primates and elephants. Only two Cetacean species such as short-finned pilot whales (*Globicephala macrorhynchus*) and killer whales (*Orcinus orca*) exhibit postreproductive life spans comparable to those of female *Homo sapiens* [22, 26]. Female short finned pilot whales stop to reproduce by about 36 years of age but they can live up to 65 years [22]. Female killer whales stop breeding by 48 years but they can live up to 90 years [22]. Baleen whales in contrast continue to reproduce into their nineties. Among large terrestrial mammals, elephants continue to reproduce into their sixties [27]. Quite difficult is the situation among our next living relatives, the nonhuman primates, because only few studies on

reproductive ageing patterns among wild living as well as captive primates exist. Some studies suggest that with increasing age a period of reproductive instability is quite common among female primates [28]. Data from *lemurs* and *callitriche* plead for age-related decline in reproduction in some species [28]. Furthermore, hormonal changes—comparable to menopausal hormonal transition among human females—have been noted in many primate species [24]. An increasing cycle length with increasing age was reported for captive chimpanzees but not for the wild chimpanzees [24]. The main problem is that nearly all studies of chimpanzees are based on very small samples and these studies have not provided clear conclusions. According to Thompson et al., there is no evidence that menopause is an obligatory characteristic of chimpanzee life history [29]. Data concerning Orangutan and Gorilla are still rare. Some evidence for an extended postreproductive phase exists for rhesus monkeys (*Macaca mulatta*) [24]. We can summarize that menopause and a long postreproductive phase is found exclusively among humans and some toothed whales. Consequently, menopause seems to be a typical feature of *Homo sapiens* and therefore we have to ask, why do human females loose the capability of reproduction much earlier than their next living relatives and most other social mammals?

4. Physiology of menopause: proximate approach

In the first step, we have to analyze the proximate or physiological basis of female reproductive senescence. From a proximate viewpoint, menopause results from follicular atresia that starts extremely early in female ontogeny, i.e., during intrauterine phase and continues until menopause [23, 30]. In the female embryo, primordial germ cells originating from the yolk sac develop into oogonia, immature sex cells. Germ cell numbers peak at approximately 3×10^5–7×10^6 by the fifth month of fetal development [31]. Oogonia develop to oocytes. Oocyte formation, however, ceases by the time a female fetus is 5 months old. Consequently, human females are unable to continue to produce oocytes past their fifth month in utero. At this time process of follicular degeneration and resorption from 3.4 to 7 million germ cells to less than 1000 remaining follicles at the time of menopausal transition, starts. The exorbitantly high number of 7 million oogonia declines to about 2 million oocytes at the time of birth and to about 400000 at pubertal onset. Oocytes are embedded in follicular cells. The vast majority of follicles are nonproliferating, produces steroids and succumb to atresia by apoptosis [23]. Only few follicles develop to preovulatory follicles with a thick layer of granulosa and theca cells, consequently only few oocytes undergo ovulation. The majority of follicles and oocytes, which are developmental units, degenerates before ovulation. Fertility declines in human females before total depletion of oocytes. A gradual decline in fertility is observable between the ages 35 and 40 years, after this period the decline accelerates. This reduced fertility from about 35 years onward is mainly due to defects in oocytes [31]. Oocyte or follicular depletion accelerates as menopause got closer. At the time of menopause, the activity of the few remaining follicles declines drastically [23].

The follicular decline results in marked hormonal disturbances typical of perimenopause and postmenopause [4]. The main feature of menopausal transition is the dramatic decline in

estrogen levels [32, 33]. These hormonal disturbances are caused by the depletion of follicular cells. The theca and the granulosa cells of the follicle, however, are essential for estrogen synthesis in the ovary. Consequently, estrogens are no longer converted from androgen in the granulosa cells during menopausal transition [23]. The decrease of estrogen secretion resulted in consecutive disturbances of the hypothalamus-pituitary-gonad axis (HPO-axis). During reproductive phase, menstrual cycle patterns are regulated by this hormonal axis. The hypothalamus secretes gonadotropin releasing hormone (GnRh) directly to the anterior pituitary. The secretion patterns of GnRh are modified by neurotransmitters such as dopamine, serotonin, epinephrine, or endorphin. Receptors in the anterior pituitary sense the pulse frequency and amplitude of GnRh and direct the production of the gonadotropins, follicle stimulating hormone (FSH), and lutenizing hormone (LH), which are essential for reproduction. FSH stimulates follicle development, LH the estrogen synthesis in the ovaries. Both stimulate ovulation and LH induces corpus luteum development and in this way progesterone synthesis. FSH binds to specific hormone receptors on the membrane of the granulosa cells, whereas LH binds to receptors of the granulose and theca cells. Androgens are secreted under LH stimulation from the theca cells, in the granulosa cells these androgens are converted into estradiol. The hormone secretion of the HPO-axis is regulated by a negative feedback mechanism. During reproductive phase, female sex hormone secretion underlies dramatic cyclic fluctuations [32, 33].

Menopausal transition is characterized by marked endocrine changes which are mainly induced by changes within the ovary but also central neuroendocrine changes. The reduction of ovarian follicles during perimenopause results in declining levels of inhibin B, a dimeric protein, and a rise of follicle stimulating hormone (FSH) and lutenizing hormone (LH) levels. During perimenopause, estradiol levels remain relatively unchanged presumably in response to the elevated FSH levels [32, 33]. As the follicular supply is exhausted, estradiol (E_2) and estrone (E) decrease dramatically; FSH and LH, however, remain elevated. Estradiol, the most physiologically active estrogen, declines most markedly, whereas estrone continues to be produced through the conversion of androstenedione to estrone in muscle, adipose, and other tissues. Consequently, the hypothalamus-pituitary gonad axis (HPG-axis) is irreversible disturbed. Beside the decline in estrogens and progesterone (P), a decrease of testosterone (T), androstenedione (A), dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEA-S), and sex hormone binding globulin (SHBG) levels after menopausal transition was observed [32, 33]. Additionally, thyroxine (t_4) and triiodothyronine (t_3) levels as well as growth hormone (GH) decrease as a result of the general ageing process. This dramatic hormonal transition is often associated with weight gain and changes in body composition, in particular fat distribution patterns [34–41]. We can summarize from a proximate viewpoint that menopausal transition is caused by the final depletion of germ cells and it is characterized by marked hormonal disturbances which are associated with significant changes in body composition and fat patterning.

5. Evolutionary explanations of menopause: ultimate approach

According to Theodosius Dobzhansky, "nothing in biology makes sense except in the light of evolution" [42]. Consequently, we have to analyze menopausal transition from an ultimate

or evolutionary viewpoint. There is no doubt that menopause is clearly a biological phenomenon and consequently, we can assume that menopause has an evolutionary origin [43–45]. The majority of women in First world countries experience menopause usually between 47 and 55 years of life [46]. Considering an average life expectancy of about 80 years among females in First world countries, female postreproductive phase thus lasts on the average of 30 years. The potential maximum life span of recent *Homo sapiens* is even longer and is thought to be about 120 years. Considering this extraordinary long lifespan, we have to be aware that human females can spend more than half of their maximum life span potential with postreproductive period [23]. Such an extremely long postreproductive phase of life is unique in the animal kingdom and makes menopause to an extremely interesting event from an evolutionary point of view. If maximization of reproductive success is the ultimate goal of life, how can such a long postreproductive period be explained in evolutionary terms?

Since 1970s, several evolutionary scenarios of human menopause were proposed to explain the phenomenon of female reproductive senescence and in particular menopause; however, there is still no consensus about which of these hypotheses should be preferred [31, 45, 47, 48]. In general, two different types of evolutionary explanations of menopause can be distinguished: nonadaptive or by-product hypotheses and adaptive hypotheses [23]. Consequently, we have to ask that whether menopause is an adaption or an epiphenomenon [49].

At a first glimpse, the so-called by-product hypothesis seems comprehensible. The by-product hypothesis is based on the assumption that life expectancy increased dramatically during the last few centuries. In former times, however, the life expectancy was much shorter. Consequently, women did simply not live long enough to experience menopause for most time of our evolution and history. From a physiological point of view, menopause occurs when all oocytes are depleted. The maximum number of germ cells which is produced until fifth month in utero is adapted to a life expectancy of less than 50 years. According to the by-product hypothesis, it was assumed that in our past only few women lived until 50 years and beyond. Therefore, postmenopausal women did not exist. Consequently, menopause is not an adaptation, it is nothing else than a by-product of increased life span and therefore a very recent phenomenon [50, 51].

In contrast to by-product hypotheses, the adaptive hypotheses consider menopause itself as a fitness advantage [52]. The most important question is, how natural selection came to favor prolonged postreproductive phase in human life history? The antagonistic pleiotropy hypothesis—first proposed by Williams in 1957—suggests that if a gene caused both increased reproduction in early life and aging in later life, then reproductive senescence can be interpreted as adaptive. In case of menopause, it was assumed that follicular depletion may cause both more regular cycles in early life and loss of fertility in later life through menopause. Consequently, its early benefits may outweigh its late costs [53].

From the perspective of life history, the main question is, when in our evolution an extended postreproductive period occurred for the first time? At the moment, it seems that life circumstances of our ancestors changed dramatically about 2 or 1.8 million years ago at the time when *Homo erectus* lived [54]. At this stage of hominid evolution, new growth patterns and encephalization made a long dependency of offspring necessary and lead to life history patterns similar

to those of recent *Homo sapiens,* such as introduction of childhood and also menopause and an extended period of postmenopause [54]. Consequently, it is assumed that menopause and prolonged reproductive senescence occurred first among *Homo erectus* about 2 million years ago [55]. Significantly associated with the existence of an extended postreproductive phase is the introduction with the life history stage of childhood between weaning and about 6 or 7 years of life. While weaned chimpanzees must forage for their own food, human children depend after weaning on older individuals for food and protection. This prolonged period of dependence during subadult phase of life makes an especially high parental investment necessary. Weaned children need extensive care because they are not able to use resources like adolescents and adults. Consequently, mothers have to provide a substantial fraction of their weaned children's diet [55]. Parents are often supported by the grandparents, first of all grandmothers, who provide a substantial investment in their grandchildren [56–58]. This is especially true for postmenopausal women with no young children of their own who help to feed and to take care of the offspring of their daughters and near relatives [55, 59]. This successful investment of postmenopausal women in related offspring, first of all grandchildren, suggests a solution to the riddle of prolonged postmenopausal period in humans. The so-called grandmother hypothesis as an evolutionary explanation of female menopause was a consequence of such ideas [60–62].

Human females are unable to reproduce after menopause; however, they invest in the offspring of their daughters and sons. In this way, a prolonged postreproductive span may have increased inclusive fitness of postmenopausal women. This point of view resulted in the introduction of the so-called grandmother hypothesis, which suggested increased fitness of women who stop reproduction and invest in their grandchildren [55, 61, 63–65]. The grandmother hypothesis is mainly based on the results of Kirsten Hawkes extensive fieldwork among Hadza hunter-gatherers northern Tanzania in eastern Africa [55, 60]. Nevertheless, the grandmother hypotheses have been criticized by several authors [66].

Beside the grandmother hypothesis, the so-called good mother hypothesis tries to explain the evolutionary benefit of an extended postreproductive phase in female *Homo sapiens*. The termination of reproductive capability independent of general senescence ensures that human females have a real chance to be young enough at their last birth to survive until their last offspring is able to survive without a biological mother [43, 67]. It is well documented that the survival of the mother during the rearing period is a major determinant of their children's survival. Considering subadult dependency in *Homo sapiens* mothers should survive until the last offspring reaches age of 7 years. According to Pavard et al. [52], menopause and subsequent postreproductive life are significantly advantageous when two conditions are satisfied: a marked increase in stillbirth and risk of birth defects as well as in maternal mortality with mother's age. Both the grandmother hypothesis and the "mother" hypothesis are the main adaptive explanations of human menopause. Female reproductive cessation seems to be a strategy that has been selected for during human evolution because women at older ages might maximize their fitness by investing resources in the survival and reproduction of their living offspring rather than by continuing to reproduce.

Recently, some new approaches to solve the evolutionary puzzle of menopause have been provided. According to the mate-choice hypothesis, male mating preference for younger females

may lead to the accumulation of mutations deleterious to female fertility and thus lead to the evolution of an extended postreproductive period in human females [68]. Takahashi et al. [31] tried to explain the origin and evolution of menopause by combining a genetic basis, behavioral factors such as mating behavior, a life history perspective, and social changes in human evolution.

Although many different theories to explain the origin and evolution of menopause have been presented, human menopause remains as an unsolved evolutionary puzzle.

6. Climacteric syndrome from the viewpoint of evolutionary anthropology

As pointed out above, menopause is a common experience of all human females who lived until about 50 years of age and beyond [23, 46, 69]. From a biological viewpoint, menopause simply reflects reproductive senescence, the end of childbearing phase and is therefore a natural part of female life history [5]. Consequently, menopause is not pathology because all human females who live long enough experience menopausal transition and the cessation of reproductive capability. Despite this fact, menopause was increasingly interpreted as a pathological condition since early nineteenth century. This medicalization of menopause within biomedical practice has affected the way menopause is viewed within society until today. Of special importance in this case is the work of the British gynecologist E.J. Tilt, who introduced the phenomenon of menopause in British Gynecology in 1857 [70]. In continental Europe and North America, biomedicine practitioners began to think of menopause as a disease-like state by the 1930s. As endocrinology improved and as synthetic estrogens became readily available in the 1960s, menopause was treated as a hormone deficiency disease, comparably to diabetes [71]. As a consequence, the medical (pathological) viewpoint dominates menopause research for a long time.

Two different approaches to this medical viewpoint can be distinguished: on the one hand, menopause is interpreted as an own deficiency disease or endocrinopathy. According to this view, hormonal deficiency results in symptoms of the so called climacteric syndrome [6, 71, 72]. This medicalization of menopause is mainly due to the fact that many women experience a large variety of symptoms, such as hot flushes and night sweats, and also psychic problems such as depression, irritability, or insomnia during peri- and postmenopause. This symptom complex is commonly called climacteric syndrome, which make peri- and postmenopause very uncomfortable for many women. In western societies, 60–70% of menopausal women reported hot flushes and night sweats [46, 69]. Climacteric symptoms seem to be strongly related to the menopause-specific decline of estrogen levels [32, 33]. However, not all climacteric women suffer from climacteric symptoms and the interpretation of the individual symptoms varies between individuals according to culture and society [46, 73–76]. Quite different is the alternative approach: menopause is not seen as a disease by itself but menopause is interpreted as a major risk factor for the development of other diseases such as osteoporosis, cardiovascular disease, some cancers such as breast cancer, and also Alzheimer disease [4, 77]. Additionally, the decline of estrogen levels after menopause enhances the risk of cardiovascular disease such as hypertension [78].

Consequently, the risk of stroke, myocard infarct, and heart failure increases after menopause. Furthermore, menopause also seems to increase the risk of the development of certain cancers such as breast cancer [79, 80].

From the viewpoint of evolutionary anthropology, the so-called climacteric syndrome can be interpreted in an evolutionary sense. Of course, potential climacteric complaints cannot be reconstructed from fossil bones and it is not possible to search for climacteric complaints among nonhuman primates or other social mammals. However, different attitudes toward menopausal transition and climacteric symptoms are found in different cultural settings [73, 75, 76].

The climacteric syndrome, however, can also be interpreted from the viewpoint of evolutionary medicine [81, 82]. Evolutionary medicine was formalized in early 1990s, most notably by the evolutionary biologist George C. Williams and psychiatrist Randolph Nesse who tried to understand why natural selection has left the human body so vulnerable to diseases [83]. According to their concept, many medical conditions that are clearly pathological today have been adaptive in the ancestral environment in which *Homo sapiens* evolved. Evolutionary medicine is concerned with identifying and understanding these conditions in our environment of evolutionary adaptedness (EEA) [84]. Especially, the impact of changing living conditions during our evolution and also of more recent processes such as modernization and acculturation on health and disease is focused.

As pointed out above, we can assume that menopause and prolonged postmenopausal phase occurred first among members of *Homo erectus* about 2 million years ago [85]. In the first step, we have to look at the natural and social environment of our ancestors from about 2 million years ago up to 10,000 years ago when Neolithic transitions started. Ethnographic analyses of the few remaining contemporary forager populations such as the Hadza in Tanzania, the !Kung of Namibia and Botswana, Ache of Paraguay, or Efe of Central Africa provided information about diet and life style in recent foraging economy [8, 10]. The typical life style of foragers is highly mobile because high levels in daily activity in search of food, water, and sleeping sites are necessary. The diet consists mainly of lean meat, wild vegetables, tubers, berries, fruits, nuts, and roots, while excluding foods such as dairy products, grains, sugar, legumes, and fats. From a medical point of view, typical noncommunicable diseases such as hypertension, heart disease, cancer, type II diabetes, or obesity are rather unknown [86]. *Homo sapiens* is clearly adapted to an environment like this. With the emergence of agriculture in the area of the fertile crescent about 10,000 years ago, subsistence economy and life circumstances changed dramatically [87, 88]. Domestication of animals and plants allowed the production of a surplus of food, which resulted in population growth, and dietary changes [88]. Analyses of Neolithic skeletal remains indicated protein deficiencies and periodic food shortages, skeletal conditions which can clearly be interpreted as results of famine and starvation. Furthermore, the close proximity to domesticated animals exposed humans to a variety of new pathogens resulting in an increased frequency of infectious diseases [89]. Therefore, Neolithic transition has led to the so-called first epidemiologic transition [90]. A second epidemiologic transition occurred about 200 years ago during industrial revolution when a shift toward manmade diseases is observable.

During the twentieth and twenty-first century lifestyle changed again dramatically. Urbanization, technical advances, and general modernization resulted in a marked transition in human life style. Advances in medicine reduced human morbidity and mortality and lead to increased life expectancy. The daily energy effort to gather and prepare food is reduced nearly to zero, since only few individuals are working on food production. Mechanized transportation, sedentary jobs, and labor-saving household technologies reduce physical activity too. On the other hand, more than enough energy providing food, mainly consisting of sugar and fat is easily available [91, 92]. Consequently, a dramatic mismatch between current environment and human body evolved in the environment of our evolutionary adaptedness can be observed. In 99% of our evolutionary history, we have survived as foragers following a highly mobile life style in small groups. Obesity and noncommunicable diseases were quite unknown. Our gene pool was shaped by natural and sexual selection toward an optimal adaptation to these environments and life circumstances. Our recent environments, however, differ dramatically from that in which our ancestors evolved. There is no doubt that also some genetic changes had occurred since the Neolithic transition; however natural selection works slowly and our genome changed to a certain degree only. Therefore, we are still often adapted to a habitat that since more than 10,000 years no longer exists [83, 93–95].

Recent health problems such as climacteric complaints, cardiovascular disease, osteoporosis, or even postmenopausal breast cancer can be interpreted primarily as the results of a dramatic change in life style of women in contemporary societies. As pointed out above, the rapid decline in estrogen levels associated with menopause experienced by recent women in industrialized societies enhances climacteric symptoms such as hot flushes [23, 32, 33, 96]. These hormonal disturbances may be the result of our recent life style patterns. We have to be aware that life history patterns of contemporary women are unique within human evolution [65]. We can assume that female life history patterns in our environment of evolutionary adaptedness resemble those of contemporary hunter gatherer societies [65]. Recent female hunter gatherers reach sexual maturation quite late. Their reproductive span is characterized by many cycles of pregnancy, long periods of lactation, and early menopause. Consequently, the number of ovulatory cycles is quite low and about 100 ovulatory cycles are assumed during reproductive span. Therefore life-time estrogen levels were quite low [97]. These low levels of estrogens during adult life are caused by high levels of physical activity, a diet characterized by low fat contents, a low amount of body fat, and low body weight [79, 97]. Consequently, lifetime estrogen exposition was quite low in the environment of evolutionary adaptedness. Life history patterns of women in recent developed countries are quite different. Menarche occurs early and first pregnancy becomes late. In Austria, for example, first menstrual bleeding occurs at the age of 12 years on average, first birth, however, occurs at the age of 29.7 years [98]. This means a period of nearly 18 years between sexual maturation and first reproduction. Reproductive span of contemporary women living in First world countries is characterized by extremely few pregnancies, few births, and short periods of lactation. It can be assumed that a woman experiences about 400 cycles on the average. Menopause occurs late and hormone supply via hormonal contraceptives or hormone replacement therapy is usual. Consequently, life estrogen exposition is long and estrogen levels are high [99]. During menopausal transition, estrogen levels drop down very fast resulting in rapid hormone deficiency, which may lead to climacteric symptomatology [32, 33, 69].

Among recent traditional societies, following quite different life style patterns such as women in rural India or Maya women in Yucatan estrogen levels are very low during menopausal transition; nevertheless, climacteric symptoms are rarely reported. Traditional lifestyle is characterized by low life time estrogen levels. The decline of estrogen secretion during menopausal transition is therefore not as dramatic as among women in western societies. Sometimes, last lactational amenorrhea—characterized by low estrogen levels—switches to menopause [23]. Consequently, climacteric complaints as a reaction of a sudden drastic estrogen decline are prevented. The high prevalence of climacteric symptoms in western societies may therefore be interpreted as a result our recent life style.

Additionally, a high rate of physical activity and a traditional diet poor in fat reduce estrogen levels through reproductive phase and even during and shortly after menopausal transition. Estrogens are converted from androgens in adipose tissue. Consequently, a higher amount of body fat increase the estrogen levels during reproductive phase and even during menopausal transition. This positive association between body fat and estrogen levels increases also the probability to develop breast cancer during peri- and postmenopause. A sedentary life style, high fat contents in diet, high life time estrogen levels, and high rates of overweight and obesity during middle age increase the risk of several diseases associated with menopausal transition such as breast cancer, cardiovascular disease, and also osteoporosis [100].

7. Conclusion

From the viewpoint of evolutionary anthropology, menopause is a natural part of female life history and therefore not a pathology. Several theories have been proposed to explain the evolutionary basis of menopausal transition, although there is still no consensus. The climacteric syndrome—mainly caused by estrogen deficiency—may be interpreted as the result of a mismatch between recent life style and reproductive patterns and life circumstances in the environment in which our ancestors evolved.

Author details

Sylvia Kirchengast

Address all correspondence to: sylvia.kirchengast@univie.ac.at

Department of Anthropology, University of Vienna, Vienna, Austria

References

[1] Fernell RJ, Sowers MF. Longitudinal, epidemiologic studies of female reproductive aging. Annals of the New York Academy of Sciences 2010, **1204**:188-197

[2] Gold EB. The timing of the age at which natural menopause occurs. Obstetrics and Gynecology Clinics of North America. 2011;**38**:425-440

[3] World Health Organization (WHO). Research on Menopause in the 1990s. WHO Technical Reports Series No. 866. Geneva: WHO 1996

[4] Huber JC. Klimakterium – Diagnose und Therapie. Berlin: Berliner Medizinische Verlagsanstalt; 1995

[5] Melby MK, Lampl M. Menopause a biocultural perspective. Annual Review of Anthropology. 2011;**40**:53-70

[6] Utian WH. Overview of menopause. American Journal of Obstetrics & Gynecology. 1987;**156**:1280-1283

[7] Pavelka MSM, Fedigan LM. Menopause: A comparative life history perspective. Yearbook of Physical Anthropology. 1991;**34**:13-38

[8] Howell N. Life Histories of the Dobe !Kung. Food, Fatness and Well Being Over the Lifespan. Los Angeles: University of California Press; 2010

[9] Hill K, Hurtado AM. Ache Life History. The Ecology and Demography of a Foraging People. New York: de Gruyter; 1995

[10] Marlowe FW. The Hadza Hunter Gatherers of Tanzania. Los Angeles: University of California Press; 2010

[11] Voland E. Grundriss der Soziobiologie. Stuttgart: Fischer Verlag; 1993

[12] Hill K, Kaplan H. Life history traits in humans. Theory and empirical studies. Annual Review of Anthropology. 1999;**28**:397-430

[13] Bogin B, Smith H. Evolution of the human life cycle. American Journal of Human Biology. 1996;**8**:703-716

[14] Holland Jones J. Primates and the evolution of long-slow life histories. Current Biology. 2011;**21**:708-717

[15] Bogin B. Evolutionary hypotheses for human childhood. Yearbook of Physical Anthropology. 1997;**40**:63-89

[16] Kuzawa CW, Bragg JM. Plasticity in human life history strategy: Implications for contemporary human variation and the evolution of Genus *Homo*. Current Anthropology. 2012;**53**:S369-S382

[17] Carey JR, Molleman F. Reproductive ageing in tephritid fruit flies. Annals of the New York Academy of Sciences. 2010;**1204**:139-148

[18] Kenyon C. A pathway that links reproductive status to lifespan in Caenorhabditis elegans. Annals of the New York Academy of Sciences. 2010;**1204**:15-162

[19] Austad SN. Animal models of reproductive aging: What can they tell us? Annals of the New York Academy of Sciences 2010, **1204**:123-128

[20] Altmann I, Gesqiere L, Galbany J, Onyango PO, Alberts SC. Life history context of reproductive aging in a wild primate model. Annals of the New York Academy of Sciences. 2010;**1204**:127-138

[21] Hawkes K, Smith KR. Do women stop early? Similarities in fertility decline in humans and chimpanzees. Annals of the New York Academy of Sciences. 2010;**1204**:43-53

[22] Johnston RA, Cant MA. The evolution of menopause in cetaceans and humans: The role of demography. Proceedings. Biological Sciences. 2010;**22**:3765-3771

[23] Leidy-Sievert L. Menopause. A Biocultural Perspective. London: Rutgers University Press; 2006

[24] Walker MI, Herndon JG. Menopause in non human primates. Biology of Reproduction. 2008;**79**:398-406

[25] Ward EJ, Parson K, Holmes EE, Balcom KC, Ford JLB. The role of menopause and reproductive senescence in a long lived social mammal. Frontiers in Zoology. 2009;**6**:4-14

[26] Brent LJN, Franks DW, Foster EA, Balcomb KC, Cant MA, Croft DP. Ecological knowledge, leadership, and the evolution of menopause in Killer whales. Current Biology. 2015;**25**:746-750

[27] Moss CJ. The demography of an African elephant (*Loxodonta Africana*) population in Amboseli Kenya. Journal of Zoology. 2001;**255**:145-156

[28] Atsalis S, Margulis S. Primate reproductive ageing: From lemurs to humans. Interdisciplinary Topics in Gerontology 2008;**36**:186-194

[29] Thompson ME, Jones JH, Pusey AE, Brewer-Marsden S, Goodall J, Marsden D, Matsuzawa T, Nishida T, Reynolds V, Sugiyama Y, Wrangham RW. Ageing and fertility patterns in wild chimpanzees provide insights into the evolution of menopause. Current Biology. 2007;**17**:2150-2156

[30] Finch Ce, Homes DJ. Ovarian aging in developmental and evolutionary contexts. Annals of the New York Academy of Sciences 2010, **1204**:82-94

[31] Takahashi M, Singh RS, Stone J. A theory for the origin of human menopause. Frontiers in Genetics.2016, **7**:1-12

[32] Burger HG, Dudley EC, Robertson DM, Dennerstein L. Hormonal changes in the menopause transition. Recent Progress in Hormone Research. 2002;**57**:257-275

[33] Burger HG, Hale GE, Robertson DM, Dennerstein L. A review of hormonal changes during the menopausal transition: Focus on findings from the Melbourne Women's Midlife Health project. Human Reproduction Update. 2007;**13**:559-565

[34] Astrup A. Physical activity and weight gain and fat distribution changes with menopause: Current evidence and research issues. Medicine & Science in Sports & Exercise. 1999;**31**:S564-S567

[35] Douchi T, Yamamoto S, Yoshimitsu N, Andoh T, Matsuo T, Nagata Y. Relative contribution of ageing and menopause to changes in lean and fat mass in segmental regions. Maturitas. 2002;**42**:301-306

[36] Douchi T, Yamamoto S, Nakamura S, Ijuin T, Oki T, Maruta K, Nagata Y. The effect of menopause on regional and total body lean mass. Maturitas. 1998;**29**:247-252

[37] Tchernof A, Poehlmann ET. Effects of the menopause transition on body fatness and body fat distribution. Obesity Research. 1998;**6**:246-254

[38] Panotopoulus G, Ruiz JC, Raison J, Guy-Grand B, Basdevant A. Menopause, fat and lean distribution in obese women. Maturitas. 1996;**25**:11-19

[39] Messier V, Rabasa-Lhoret R, Barat-Artigar S, Elisha B, Karelis AD, Aubertin-Leheudre M. Menopause and sarcopenia: A potential role for sex hormones Maturitas. 2011;**68**:331-336

[40] Piche ME, Lapointe A, Weisnagel SJ, Corneau L, Nadeau A, Bergeron J, Lemieux S. Regional body fat distribution and metabolic profile in postmenopausal women. Metabolism – Clinical and Experimental. 2008;**57**:1101-1107

[41] Kirchengast S, Gruber D, Sator M, Hartmann B, Knogler W, Huber J. menopause associated differences in female fat patterning estimated by dual-energy-x-ray absorptiometry. Annals of Human Biology. 1997;**24**:45-54

[42] Dobzhansky T. Nothing in Biology makes sense except in the light of evolution. The American Teacher. 1973;**35**:125-129

[43] Sherman PW. The evolution of menopause. Nature. 1998;**392**:759-760

[44] Shanley DP, Sear R, Mace R, Kirkwood TBL. Testing evolutionary theories of menopause. Proceedings of the Royal Society B: Biological Sciences. 2007;**274**:2943-2949

[45] Pollycove R, Naftolin F, Simon JA, The evolutionary origin and significance of menopause. Menopause. 2011;**18**:336-342

[46] Palacios S, Henderson VW, Siseles N, Viaseca P. Age of menopause and impact of climacteric symptoms by geographical region. Climacteric. 2010;**13**:419-428

[47] Peccei JS. The origin and the evolution of menopause: The altriciality-lifespan hypothesis. Ethology and Sociobiology. 1995;**16**:425-449

[48] Wu JM, Zelinski MB, Ingram DK, Ottinger MA. Ovarian Ageing and menopause: Current theories, hypotheses and research models. Experimental Biology and Medicine. 2005;**230**:818-828

[49] Peccei JS. Menopause: Adaptation or epiphenomenon? Evolutionary Anthropology. 2001;**12**:43-57

[50] Washburn S. Longevity in primates. In: McGaugh JMJ, editors. Aging, Biology and Behaviour. New York: Academic Press; 1981. pp. 11-29

[51] Weiss KM. Evolutionary perspectives on human aging. In: Amoss PT, Harell S, editors. Other Ways of Growing Old. Stanford: Stanford University Press; 1981. pp. 25-58

[52] Pavard S, Metcalf CIF, Heyer E, Senescence of reproduction may explain adaptive menopause in humans. American Journal of Physical Anthropology. 2008;**136**:194-203

[53] Wood JW, O'Conner KA, Holman DJ, Bringle E, Barsom SH, Grimes MA. The evolution of menopause by antagonistic pleiotropy. Center for Demography and Ecology; 2001 (Working Paper)

[54] Bogin B. Patterns of Human Growth. Cambridge: Cambridge University Press; 1999

[55] Hawkes K, O'Connell JF, Blurton Jones NG, Alvarez H, Charnov AL. Grandmothering, menopause and the evolution of human life histories. Proceedings of the National Academy of Sciences. 1998;**95**:1336-1339

[56] Sear R, Mace R. Who keeps children alive? A review of the effects of kin on child survival. Evolution and Human Behavior. 2008;**29**:1-18

[57] Sear R. Kin and child survival in rural Malawi. Human Nature. 2008;**19**:277-293

[58] Coall DA, Hertwig R. Grandparental investment: Past, present, and future. Behavioral and Brain Sciences. 2010;**33**:1-59

[59] Sear R, Mace R, McGregor I A. Maternal grandmothers improve nutritional status and survival of children in rural Gambia. Proceedings of the Royal Society. 2000;**267**:1641-1647

[60] Hawkes K, O'Connell JF, Blurton Jones NG. Hadza women's time allocation, offspring provisioning and the evolution of long postmenopausal lifespans. Current Anthropology. 1997;**38**:551-577

[61] Hawkes K. Grandmothering of human longevity. American Journal of Human Biology. 2003;**15**:380-400

[62] Lahdenperä M, Lummaa V, Helle S, Tremblay M, Russell AF. Fitness benefits of prolonged post-reproductive lifespan in women. Nature. 2004;**428**:178-181

[63] Alvarez HP. Grandmother hypothesis and primate life histories. American Journal of Physical Anthropology. 2000;**113**:435-450

[64] Voland E, Chasiotis A, Schiefenhövel W. Grandmotherhood. The Evolutionary Significance of the Second Half of Female Life. New York: Rutgers University Press; 2005

[65] Blurton Jones NG, Hawkes K, O'Connell JF. Antiquity of postreproductive life. Are there modern impacts on hunter gatherer postreproductive life span. American Journal of Human Biology. 2002;**14**:184-202

[66] Peccei JS. A critique of the grandmother hypotheses: Old and new. American Journal of Human Biology. 2001;**13**:434-452

[67] Alexander RD. The evolution of social behaviour. Annual Review of Ecology and Systematics. 1974;**5**:325-383

[68] Morton RA, Stone JR, Singh RS. Mate choice and the origin of the menopause. PLoS One. 2013;**9**:e1003092

[69] Hunter M, Rendall M. Bio-psycho-socio-cultural perspectives on menopause. Best Practice & Research: Clinical Obstetrics & Gynaecology. 2007;**21**:261-274

[70] Van de Wiel L. The time of the change: Menopause's medicalization and the gender politics of aging. International Journal of Feminist Approaches to Bioethics. 2014;**7**:74-98

[71] McCrea FB. The politics of menopause: The discovery of a deficiency disease. Social Problems. 1983;**31**:111-123

[72] Bell SE. Changing ideas: The medicalization of menopause. Social Science & Medicine. 1987;**24**:535-542

[73] Beyene Y, Cultural significance and physiological manifestations of menopause: A biocultural analysis. Culture Medicine and Psychiatry. 1986;**10**:47-71

[74] Lock M. Encounters with Aging: Mythologies of Menopause in Japan and North America. Berkeley: University of California Press; 1993

[75] Kilaf E, Kirchengast S. Menopause between nature and culture: Menopausal age and climacteric symptoms among Turkish immigrant women in Vienna, Austria. Acta Medica Lituanica. 2008;**15**:313-329

[76] Pitkin J. Cultural issues and the menopause. Menopause International. 2010;**16**:156-161

[77] Worcester N, Whatley MH. The selling of HRT: Playing on the fear factor. Feminist Review. 1992;**41**:1-26

[78] Mayo JL. A natural approach to menopause. Applied Nutritional Science Reports. 1997;**5**:1-8

[79] Turner LB, A meta-analysis of fat intake, reproduction and breast cancer risk: An evolutionary perspective. American Journal of Human Biology. 2011;**23**:601-608

[80] Wu AH, Pike MC, Stram DO. Meta-analysis: Dietary fat intake, serum estrogen levels and the risk of breast cancer. Journal of the National Cancer Institute. 1999;**91**:529-534

[81] Froehle AW. Postmenopausal health and disease from the perspective of evolutionary medicine. Anthropology &Aging Quarterly. 2013;**34**:61-86

[82] Kirchengast S, Rühli F. Evolutionary medicine and its implications for endocrinological issues (e.g. menopause). Comparative Endocrinology 2013, **186**:145-149

[83] Nesse RM, Stearns SC. The great opportunity: Evolutionary applications to medicine and public health. Evolutionary Applications. 2008;**1**:28-48

[84] Bowlby J. Attachment and Loss. New York: Basic Books; 1969

[85] Henke W, Hardt T. The genus *Homo*: Origin, speciation and dispersal. In: Condemi S, Weniger G-C, editors. Continuity and Discontinuity in the peopling of Europe: One hundred fifty years of Neanderthal study, Vertebrate Paleobiology and Paleoanthropology. New York: Springer Press; 2011. pp. 17-43

[86] Eaton SB, Konner M. Paleolithic nutrition: A consideration of its nature and current implications. New England Journal of Medicine. 1985;**312**:283-289

[87] Maher LA, Richter T, Macdonald D, Jones MD, Martin L, Stock IT. Twenty thousand year old huts at a hunter gatherer settlement in eastern Jordan. PLoS One 2012;**7**:e31447

[88] Larsen C. Biological changes in human populations with agriculture. Annual Review of Anthropology. 1995;**24**:185-213

[89] Armelagos GI, Goodman AH,.Jacobs KH. The origins of agriculture: Population growth during a period of declining health. Population and Environment. 1991;**13**:9-22

[90] Barrett R, Kuzawa CW, McDade T,.Armelagos GI. Emerging and re-emerging infectious diseases: The third epidemiologic transition. Annual Review of Anthropology. 1998;**27**:247-271

[91] Popkin BM, Gordon-Larsen P. The nutrition transition: Worldwide obesity dynamics and their determinants. International Journal of Obesity. 2004;**28**:52-59

[92] Lieberman LS. Evolutionary and anthropological perspectives on optimal foraging in obesogenic environments. Appetite. 2006;**47**:3-9

[93] Trevathan WR. Evolutionary medicine. Annual Review of Anthropology. 2007;**36**: 139-154

[94] Vitzthum VI, Evolutionary models of women's reproductive functioning. Annual Review of Anthropology. 2008;**37**:53-73

[95] Wick G, Berger P, Jansen-Dürr P, Gerbeck-Loebenstein B. A Darwinian-evolutionary concept of age-related diseases. Experimental Gerontology. 2003;**38**:13-25

[96] Santoro N. The menopause transition: An update. Human Reproduction Update. 2002;**8**: 155-160

[97] Eaton SB, Pike MC, Short RV, Lee NC, Trussell J, Hatcher RA, Wood JW, Worthman CM, Nicolas GBI, Konner MJ, Bailey R, Hurtado AM, Women's reproductive cancers in evolutionary context. The Quarterly Review of Biology. 1994;**69**:353-3657

[98] Statistik Austria. Demographic Yearbook. Vienna, Austria. 2016

[99] Shaw LAM, Shaw SLJ, Menopause, evolution and changing cultures. Menopause International. 2009;**15**:175-179

[100] Strassmann BI. Menstrual cycling and breast cancer: An evolutionary perspective. Journal of Women's Health. 1999;**8**:193-202

IntechOpen

IntechOpen