

Chapter 2

Body Composition and Menopausal Transition: A Bioanthropological Perspective

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Key Points

- Menopause is defined as the permanent cessation of menstruation due to depletion of the follicle pool.
- The menstrual cycle and changes in the cyclic pattern until a complete stop are orchestrated by gonadotrophins, steroids, and inhibins.
- The median age at natural menopause is around 50–51, for centuries and across populations.
- Menopause is associated with vasomotor menopausal symptoms; of other symptoms such as incontinence, depressed feelings, and vaginal dryness it is not clear whether it is the menopause per se that causes these symptoms and complaints, or whether aging also plays a major role.
- Early menopause is associated with increased risk of cardiovascular disease and osteoporosis and a decreased risk of breast cancer.
- These effects are generally ascribed to estrogens, but for osteoporosis and breast cancer this is much more clear than for cardiovascular disease.

Keywords Age at menopause • Body composition • Lean mass • Fat mass • Fat distribution • Bone mass • Menopausal transition • Evolution

Introduction

Menopause, the cessation of menstrual function and the irreversible termination of female reproductive capability, is an event experienced by all human females who live beyond 55 years of life [1]. Understanding and interpretation of menopause differs between scientific disciplines. In Western societies menopause is mainly seen as visible sign of female ageing and it is often interpreted as a kind endocrine disease, which can be treated effectively with hormone replacement therapy. As a consequence the medical viewpoint dominates menopause research since a long time. Changes in hormone secretion and menstrual cycle patterns but first of all the occurrence of climacteric complaints were recorded and efficient treatments were tested. Nevertheless, menopause is not a disease per se it is a common experience of all human females who live beyond 55 years of life. Although all menopausal women lost reproductive capability and menstrual cycle stops irreversible, menopause is experienced quite different under

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different sociocultural conditions. As a consequence from a biocultural viewpoint menopause is not a common disease it reflects simply reproductive ageing and the end of childbearing phase in female life. Numerous studies carried out among menopausal women of different sociocultural background and among women in traditional societies demonstrated that menopause is the product of decades of physiological responses to an environment composed of cultural and biological factors [2].

Menopause from a Bioanthropological Viewpoint

Menopause is not only of medical and biocultural interest, it is also a main focus of bioanthropological research. From an evolutionary life history perspective, menopause is a universal one-time life event which marks the transition from reproductive to postreproductive life; consequently menopause is a marker of reproductive ageing patterns typical of female *Homo sapiens*. 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 cetaceans, elephants, lions, or first of all primates and captive animals, an obligatory postreproductive life stage of 30 years and more, however, is exclusively found among human females [1]. The majority of women in developed countries experience menopause between 47 and 55 year of life. This seems quite early because the average life expectancy of females in these countries is about 80 years. Consequently postreproductive phase of the human female lasts on the average 30 years in industrialized countries. The maximum life span of recent *Homo sapiens* is even longer and is thought to be about 120 years. Thus human females can spend more than half of their maximum life span potential in postreproductive life. This extremely long postreproductive phase of life among human females is unique in the animal kingdom and makes menopause to an extremely interesting event from an evolutionary point of view [3]. If maximization of reproductive success is the ultimate goal of life, how can such a long postreproductive period be explained in evolutionary terms? Since the 1970s several evolutionary scenarios of human menopause were provided. On the one hand, menopause ensures that old or abnormal eggs are not fertilized. Furthermore, the termination of reproductive capability ensures that mothers 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 [1]. These arguments, however, are not able to explain the extreme length of postreproductive phase in human females. Another possibility of an evolutionary benefit of menopause is grandparenting [4]. This point of view resulted in the introduction of the so-called grandmother hypothesis, which suggested increased fitness of women who stops reproduction and invest in their grandchildren. Seeking explanations for a long postreproductive life span resulted in publication of numerous evolutionary explanations of the menopause up to now; however, there is no consensus which scenario is the most likely one. The development of evolutionary scenarios of human menopause is therefore still a main focus of bioanthropological menopause research (see Fig. 2.1).

Additionally to an evolutionary viewpoint, bioanthropological menopause research focus on somatic changes which occur during menopausal transition. These changes, however, have also to be interpreted in an evolutionary sense. The aim of this review is to discuss somatic in particular body composition changes during menopause and to provide beside physiological explanations of these somatic alterations an evolutionary one.

Biological Basis of Menopause

The World Health Organization (WHO) has defined menopause as the permanent cessation of menstruation resulting from loss of ovarian follicular activity [5]. The phase of irregular cycles and starting hormonal changes, which precede menopause, is commonly called perimenopause. From a

Fig. 2.1 Menopause from a bioanthropological viewpoint

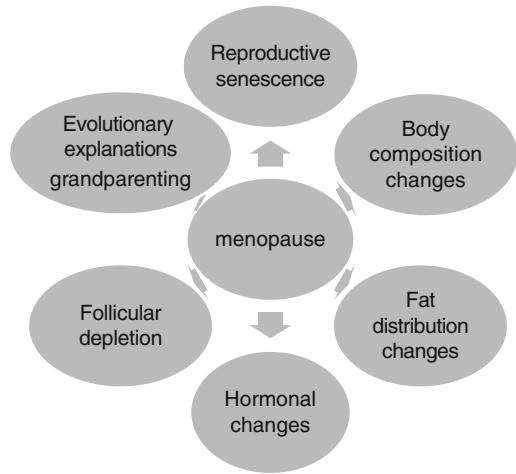
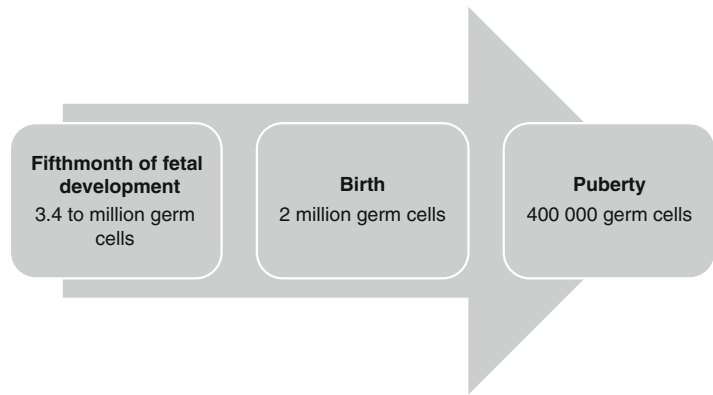


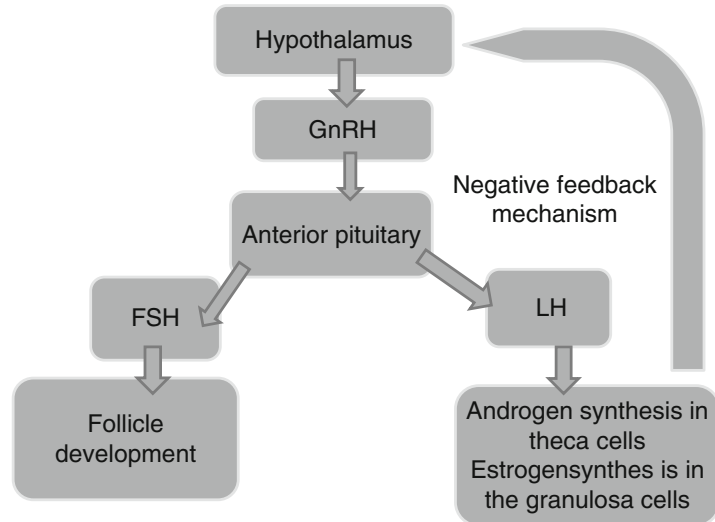
Fig. 2.2 The number of female germ cells decreases dramatically



biomedical viewpoint menopause is widely defined as the last spontaneous menstrual bleeding; however, no human female knows exactly that the actual bleeding is really the last one. Therefore, post-menopause is reached when a woman had no menstrual periods over 12 months.

On cellular level menopause is seen as a result from life long process of follicular atresia that starts during intrauterine phase and continues until menopause [1]. In the female embryo primordial germ cells originating from the yolk sac, develop into oogonia, immature sex cells. Approximately seven million oogonia are formed by the 5th month of fetal development. Oogonia develop to oocytes, almost fully developed sex cells. Oocyte formation, however, ceases by the time a female fetus is 5 months old. Human females are unable to continue to produce oocytes past their fifth month in utero. At this time the process of follicular degeneration and resorption from 3.4 to 7 million germ cells at their peak to less than 1,000 remaining follicles at the time when menopausal transition starts. The exorbitantly high number of seven million oogonia declines to about two million oocytes at the time of birth and to about 400,000 at pubertal onset. Oocytes are embedded in follicular cells, the vast majority of follicles are non-proliferating, produce steroids, and succumb to atresia by apoptosis [1]. 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. Oocyte or follicular depletion accelerates as menopause got closer. At the time of menopause the activity of the few remaining follicles decline drastically [1]. This follicular decline results in the hormonal transition typical of menopause (see Fig. 2.2).

Fig. 2.3 Hypothalamus–pituitary–gonad (HPG) axis



Hormonal Menopausal Transition

During reproductive phase menstrual cycle patterns are regulated by the hypothalamus–pituitary–ovary axis (HPO 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, FSH and 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 LH binds to receptors of the granulosa and theca cells. Androgens are secreted under LH stimulation from the theca cells, in the granulosa cells these androgens are converted to estradiol. The hormone secretion of the HPO axis is regulated by a negative feedback mechanism (see Fig. 2.3). During reproductive phase female sex hormone secretion underlies dramatic cyclic fluctuations.

Menopausal transition is characterized by marked endocrine changes which are mainly induced by central neuroendocrine changes and changes within the ovary. The reduction of ovarian follicles during perimenopause results in declining levels of inhibin B, a dimeric protein, and a rise of FSH and LH levels. During perimenopause estradiol levels remain relatively unchanged presumably in response to the elevated FSH levels [6, 7]. 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, while 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), androstendione (A), dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEA-S), and sex hormone binding globulin (SHBG) levels after menopausal transition was observed [6, 7]. Additionally

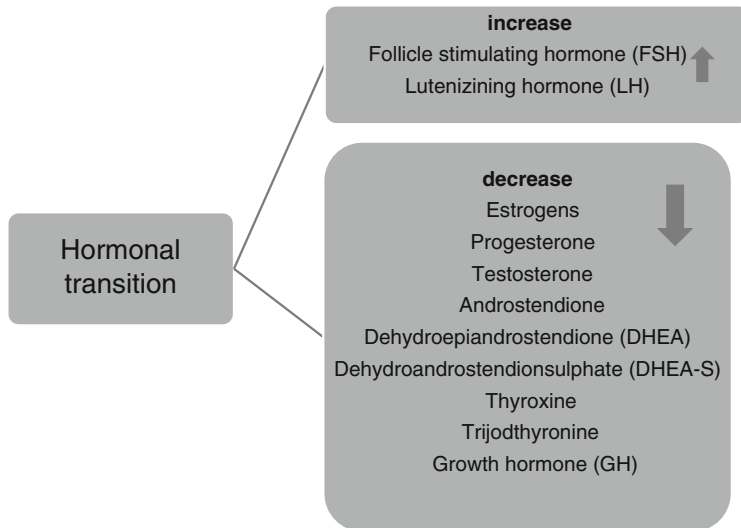


Fig. 2.4 Menopausal transition is characterized by specific hormonal changes

thyroxine (t_4) and triiodothyronine (t_3) levels as well as growth hormone (GH) decrease as results of the general ageing process (see Fig. 2.4).

Menopausal Transition and Body Composition

As pointed out above from a bioanthropological viewpoint menopause is not a disease it is a typical event of biological ageing of human females and ageing per se is not a disease. Biological ageing in general and in both sexes is associated with various changes in body build, body weight, and body composition.

Stature, Body Weight, and Weight Status

With increasing chronological age stature height decreases, on the other hand, body weight increases. Decreasing stature height is mainly due to the age related compression of intervertebral disks, microfractures of vertebral bodies, and an increased curvature of the spine [8]. Contrary to stature height body weight increases with age. Body weight starts to increase slightly since early adulthood (averaging 250 g per year) because of a decrease in lean body mass and metabolic rate. This increase of body weight accelerates during middle adulthood [9]. It is well documented that menopause is associated with weight gain and women exhibited a sharp increase in obesity rates between the ages 45 and 55 [10]. At the onset of menopause a woman's body weight reaches its maximum [11], caused mainly by the increase of fat tissue. Decreasing stature height and increasing body weight results in increased weight status determined by means of body mass index (BMI) (kg/m^2). Consequently the prevalence of overweight and obesity is higher among postmenopausal women compared to premenopausal ones

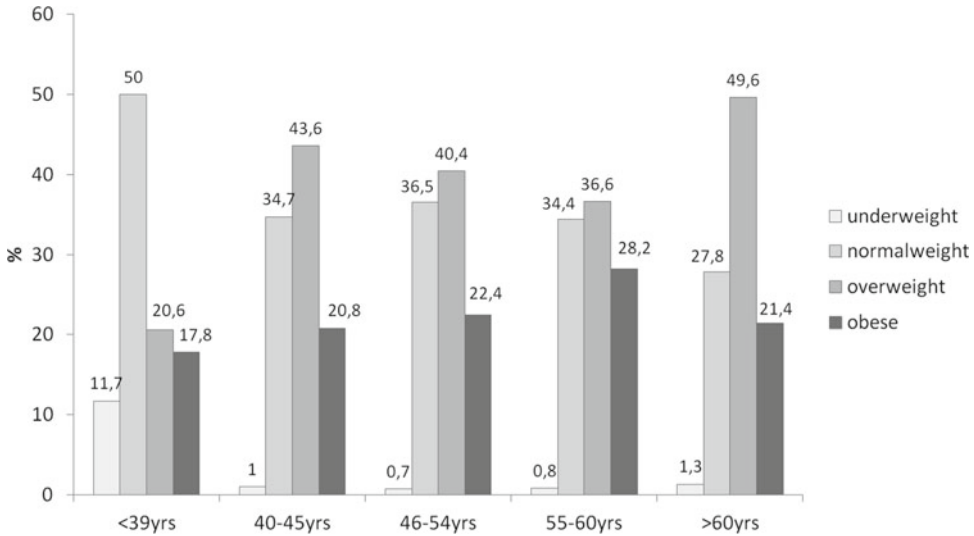


Fig. 2.5 Weight status changes with increasing age (sample of 940 Austrian women). Data source: Viennese body composition project by S. Kirchengast

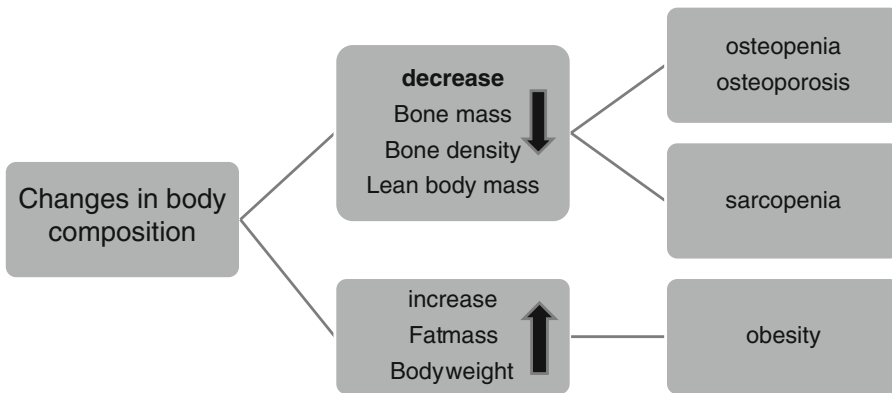


Fig. 2.6 Menopausal transition is characterized by marked changes in body composition

[12]. Women who have never suffered from weight problems experience an undesirable increase of body weight and body mass index [13] and marked alterations in body proportions. During the seventh decade of life (>60 years) body weight begins to decline and this decline accelerates during eighth decade of life (see Fig. 2.5).

Body Composition

Independent of general ageing and weight changes, during menopausal transition dramatic modifications in body composition occur [12, 14–16]. Body composition is mainly constituted by three components: lean soft tissue mass, i.e., muscle mass, bone mass, and fat mass [17]. All three components of body composition undergo certain changes in course of general ageing process and menopausal transition in particular (see Fig. 2.6).

Bone Mass and Bone Density

Age related changes in body composition include a progressive depletion of bone mass and bone density. Adult bone mass is equal to the peak bone mass achieved during early adulthood minus the amount of bone loss afterwards. Bone mass and bone density decline with increasing age, in women an accelerated rate bone loss occurs during menopausal transition and postmenopause. This menopause associated decline in bone mass has deleterious effects and may lead to the development of osteopenia or osteoporosis, which is clinically defined as areal bone mineral density (g/m^2) more than 2.5 SD below the young adult average [18].

Lean Body Mass and Sarcopenia

Ageing is generally associated with a reduction of lean body mass in particular muscle mass. Beside general ageing, menopause induces lean body mass loss, independent of ageing and stature height [16, 19]. This decrease in skeletal muscle mass has dramatic consequences. Skeletal muscle represents the largest component at the tissue-organ level of body composition in healthy adults and it is essential for locomotion and mobility. The state of pathologically reduced skeletal muscle mass is commonly called sarcopenia, from the Greek “poverty of flesh” [20, 21]. Sarcopenia, caused by reduced physical activity and hormonal factors is frequently found among postmenopausal women [22].

Body Fat and Fat Distribution Patterns

Similar to body weight the total amount of body fat as well as the fat percentage increase during middle adulthood and decreases during old age [9]. In course of menopausal transition the increase of fat mass accelerates; however, not only absolute and relative fat mass increase, fat distribution patterns change during menopausal transition too.

Body fat distribution is a typical sign of secondary sexual dimorphism in humans [23]. 65 years ago Vague [24] described differences in fat distribution patterns between men and women. During infancy and childhood fat distribution patterns are quite similar in girls and boys; during pubertal transition, however, marked differences in fat distribution patterns develop. While healthy normal weight boys develop the typical masculine or kind of fat distribution with extremely less subcutaneous fat tissue at the lower body region, i.e., buttocks, thighs, and hips, girls develop the typical gynoid kind of fat patterning with increased fat deposits at the lower body region. With the onset of reproductive maturation these sex specific fat distribution patterns are clearly visible. During adulthood and reproductive phase striking sex differences in body fat distribution intensify. Female waist to hip ratio is significantly lower than the waist to hip ratio of males. While the amount subcutaneous fat tissue is much higher in even slender women compared with weight status and age matched men, men show higher amounts of visceral fat tissue. This is mainly due to the fact that men tend to accumulate adipose tissue in the abdominal region, while healthy women tend to accumulate fat tissue in the gluteal–femoral region. For a similar fat mass, men have on average a twofold higher visceral adipose tissue accumulation compared to women [23]. Android and gynoid fat distribution patterns allow observers to distinguish between male and female body shapes which are commonly called apple shape or pear shape. Additionally in men abdominal fat tissue tends to accumulate in the visceral area to a greater extent than in women [25, 26]. While in men this kind of fat distribution pattern remain stabile through adult life and senescence, in women marked changes in fat distribution occur associated with the end of reproductive phase of life. Menopausal transition is associated with a body fat redistribution towards a dramatic increase in the accumulation of abdominal adipose tissue. Abdominal fat comprises three distinct fat stores: a superficial subcutaneous a deep subcutaneous and a visceral compartment. All three compartments, in particular visceral fat mass increase through menopausal transition [12, 27, 28]. Changes in fat patterning start during late premenopausal phase.

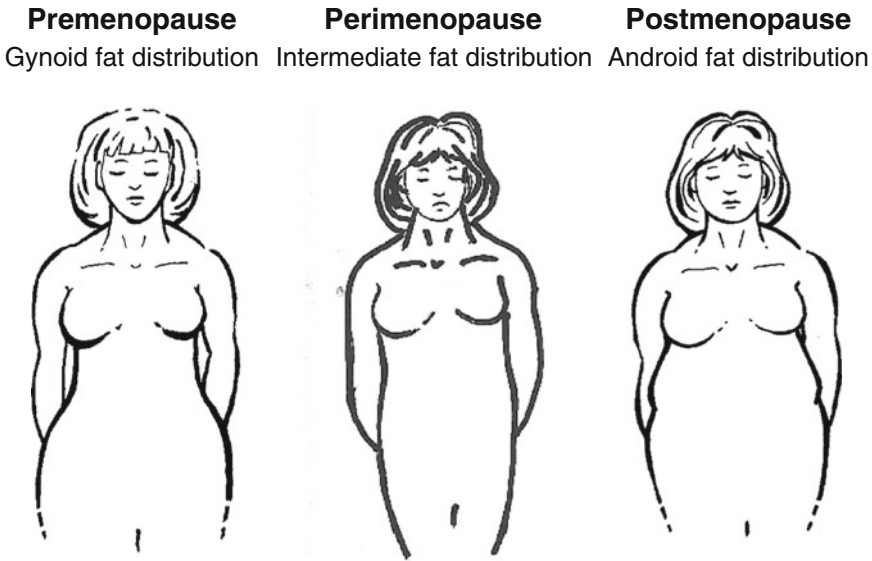


Fig. 2.7 Changes in fat distribution patterns during menopausal transition

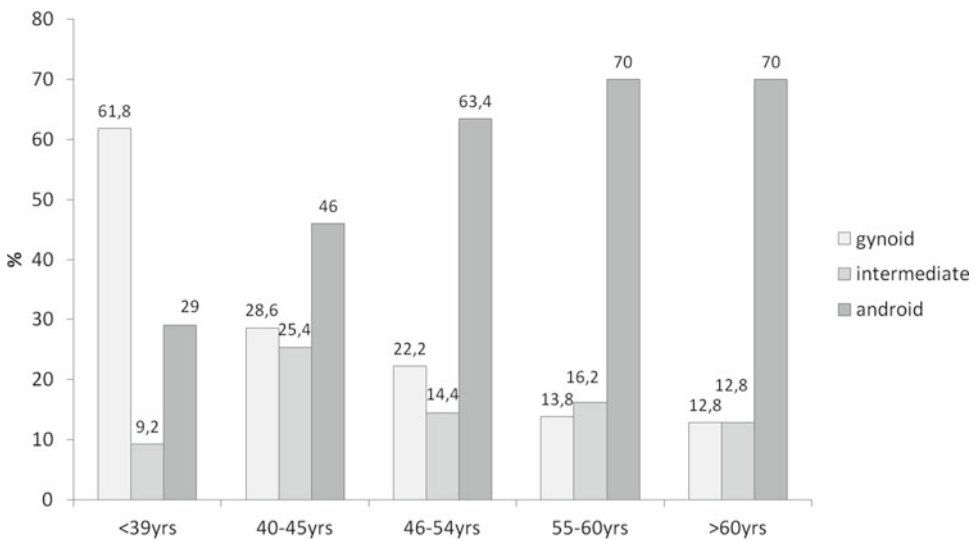


Fig. 2.8 Fat distribution with increasing age (sample of 940 Austrian women). Data source: Viennese body composition project by S. Kirchengast

At the late phase of premenopause and during perimenopause the gynoid fat patterning changes independent of age and weight status to an intermediate stadium of fat distribution between the gynoid and the android type. The amounts of abdominal fat tissue and lower body fat tissue are more or less equal during perimenopause. During the postmenopausal phase of life the intermediate type of fat patterning changes into the typical android fat patterning in the majority of women [28] (Figs. 2.7 and 2.8). With other words, menopausal transition results in a masculinization of female body shape.

Body Composition and Age at Menopause

Age at menopause is determined by the number of oocytes that the woman is born with and the rate at which those oocytes and their follicles are lost through the process of atresia [1]. Consequently age at menopause appears to be highly heritable, although it is also influenced by environmental factors. One important factor seems to be nutritional status. Elias et al. [29] analyzed the impact of Dutch famine during World War II on age at menopause. It could be demonstrated that women who were severely exposed to famine conditions experienced age at menopause on average 0.37 years earlier than women who were not exposed. The influence of famine on age at menopause was much higher when the famine was experienced between the ages 2 and 6. Those women who had suffered from malnutrition at this age reached menopause 1.83 years earlier than women who were not exposed to famine. Inconsistent results exist concerning the association between adult body mass index or body composition and age at menopause [30]. While several studies found no significant relation between adult body composition and menopausal age, others demonstrated a significant positive association between weight status as well as the amount of fat tissue and age at menopause (see Figs. 2.9 and 2.10). Body size and fat distribution have been considered in relation to age at menopause as it is hypothesized that increased peripheral conversion of androgens to estrogens might contribute to a delay of age at menopause. Consequently it was hypothesized that the higher the amount of subcutaneous fat tissue the later menopause occurs [31, 32]. On the other hand, menopause transition promotes somatic changes—as mentioned above—and therefore, a significant association between age at menopause and postmenopausal body composition may be assumed. It could be shown that age at menopause was positively associated with absolute and relative fat mass as well as lean body mass during postmenopause. Additionally a late menopause was associated with a significantly higher bone mass and bone density during postmenopause [33].

Beside natural menopause, artificial menopause as a consequence of hysterectomy is significantly associated with postmenopausal body composition characteristics. Hysterectomy due to nonma-

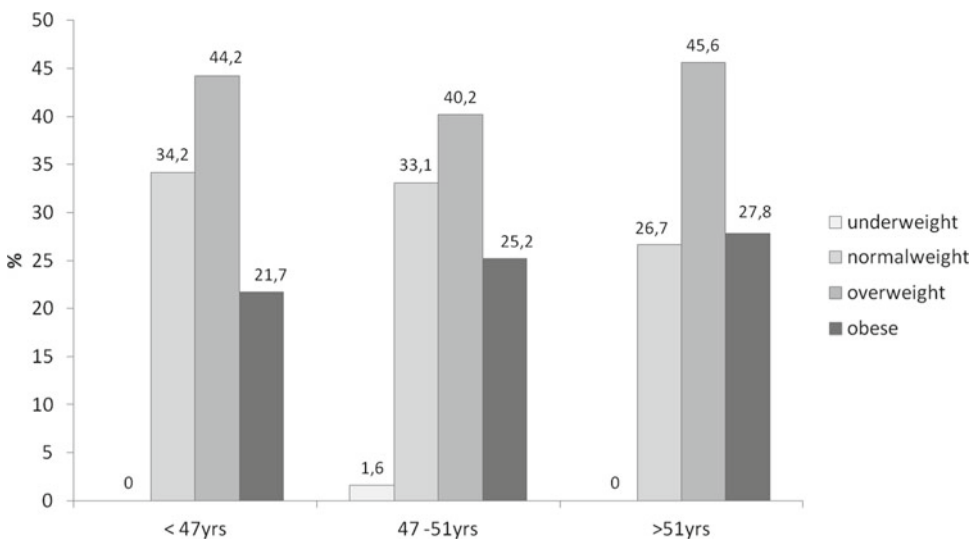


Fig. 2.9 Weight status and age at menopause (sample of 940 Austrian women). Data source: Viennese body composition project by S. Kirchengast

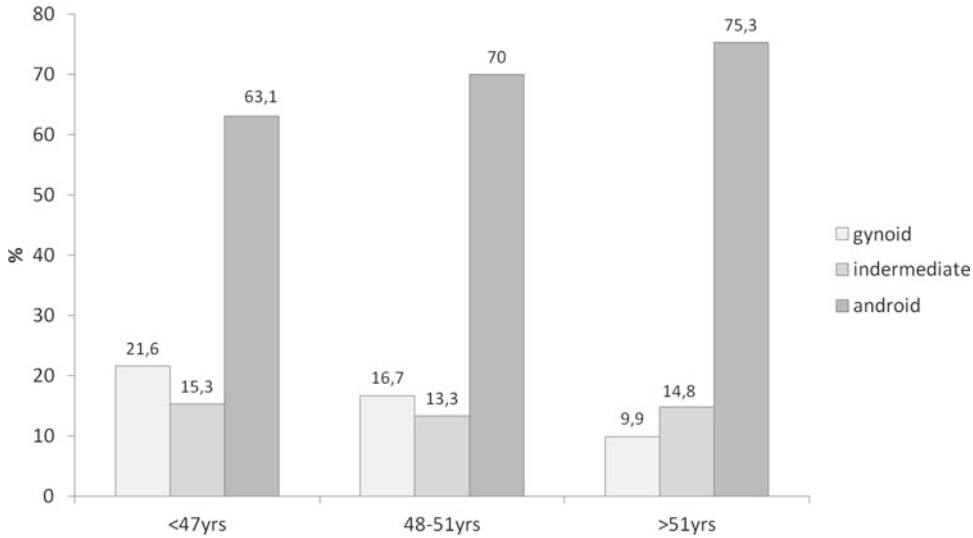


Fig. 2.10 Fat distribution and age at menopause (sample of 940 Austrian women). Data source: Viennese body composition project by S. Kirchengast

lignant cause is among the most common surgical procedures in women aged 40–60 years worldwide, although marked differences in the frequency of hysterectomies are observable between different countries and also within a population. Hysterectomy performed during pre- or perimenopause represents not only an artificial end of reproductive function, it also has an impact on weight status and body composition. Carlson et al. [34] reported a significant weight gain in 12 % of women after hysterectomy, according to Ravn et al. [35] hysterectomized women exhibited 2–11 % more body fat than women who experienced natural menopause. A Viennese study documented a significantly higher weight gain after menopause in hysterectomized women than in those who had a spontaneous menopause (9.1 kg vs. 6.0 kg). Furthermore, the percentage of obese women (BMI < 30.00) was significantly higher among hysterectomized women (34.0 % vs. 17.7 %) [36]. Furthermore, hysterectomized women showed a significantly higher amount of abdominal fat mass. Consequently hysterectomy was associated with a higher risk for the development of the centralized or android fat distribution.

Body Composition and Climacteric Complaints

Menopausal transition is accompanied by several somatic and psychic symptoms commonly called climacteric syndrome. Body composition characteristics and changes in fat distribution patterns through menopausal transition have also an important impact on the course of climacteric or on the degree of severity of climacteric symptoms. With an increasing amount of fat tissue the degree of severity of somatic and psychic symptoms increased significantly [37]. Furthermore, a significant decrease of sexual interest with increasing weight status and fat mass was observed [38]. The majority of symptoms are explained as somatic reactions of the postmenopausal estrogen deficiency. However, during climacteric the subcutaneous fat tissue has a positive impact on the endogenous estrogen levels because the extraovarian estrogen synthesis by aromatization of androgens to weak estrogens is taking place there. Therefore, the increased climacteric symptomatology and the reduced sexual interest

associated with increased fat mass may be explained by the adverse effects of psychosocial stress to which women are exposed in our society if their bodies do not correspond to our culture specific beauty ideal [37, 38]. In Western industrialized societies many women interpret weight gain and changes fat distribution patterns as visible signs of ageing and every sign of ageing is interpreted exclusively negatively in the youth-oriented culture of Western societies.

Reasons for Body Composition Changes During Menopausal Transition

But what are the reasons for body composition changes during menopausal transition and postmenopause? From a bioanthropological viewpoint we have to distinguish between proximate or physiological causes and ultimate or evolutionary reasons.

Physiological or Proximate Reasons

Hormonal Factors

Hormonal factors contribute mainly to somatic changes taking place during menopausal transition and postmenopause [39–44]. First of all the decline of estrogen levels, thyroid hormone levels, GH level, and the estrogen–androgen ratio, typical of menopausal transition and postmenopause, are discussed as responsible factors for body composition changes and weight gain. It is well documented weight gain and fat accumulation, especially visceral fat accumulation in women accelerates when estrogen levels decline. This is possibly due to direct effects of estrogen on adipose tissue estrogen, progesterone and androgen receptors which are expressed in adipose tissues [23]. Furthermore, beside the decline in estrogen levels the changes in levels of certain energy homeostasis peptides that also occur with menopause are discussed to be promoters of weight gain during menopausal transition [45]. Furthermore, the weight gain during middle age may be enhanced by the decrease of the lipolytic acting thyroid hormones and GH indicating the decrease of the basal metabolic rate (BMR) [46]. Especially GH and its mediator insulin-like growth factors I (IGF-I) decrease in as a result of estrogen deficiency. The reduction of GH levels is typical of menopausal women and may enhance visceral fat accumulation and weight gain. Hormonal factors are not only responsible for the general weight gain, they are also essential for fat redistribution through menopausal transition. Centralized fat patterning characterized by increased visceral fat mass is in men associated with low testosterone levels [26, 42]. On the other hand, the reduction of dehydroepiandrosterone (DHEA) and its sulfated prohormone dehydroepiandrosterone-sulfate (DHEA-S) have been discussed to be responsible for weight gain and increased visceral fat mass among menopausal women [46]. Beside androgens the decline in estrogen levels is discussed to be responsible for redistribution of body fat through menopausal transition. Gonadal steroid hormones have been proposed to be associated with the increase of the waist to hip ratio in menopausal women and the development of android fat patterning [46]. During reproductive phase in the lower body adipocytes at the gluteal–femoral region an increased lipoprotein lipase activity and a blunted lipolytic response can be observed in comparison with the upper body (abdominal) adipocytes. In the abdominal region the lipolysis is induced by estradiol [47, 48]. The decrease of estrogen levels during the menopausal transition induces marked metabolic changes: the lower body adipocytes no longer show an increased lipoprotein lipase activity and a further increase of lower body fat mass does not take place. On the other hand, the diminished estrogen levels reduce the lipolytic metabolism at the abdominal region and result in an increase of adipose tissue at this region.

Therefore, the menopausal hormonal transition with reduced estrogen secretion may enhance changes in fat patterning, the conversion from more gynoid to more android fat patterning.

Lifestyle Factors

Other proximate causes for body composition changes during menopausal transition are lifestyle factors. The most important component of total daily energy expenditure, the resting metabolic rate, is reduced by ageing and also by menopause independent of the effects of the normal ageing process [12]. Unfortunately at the same time marked behavioral changes occur. The energy expenditure decreases dramatically and a more sedentary lifestyle is typical of middle age, while no changes in eating habits take place. One of the most important somatic consequences of these metabolic alterations and behavioral factors is the increase of body weight, especially an increase of adipose tissue, as a result of a long term positive energy balance. On the other hand, lean body mass, especially muscle mass reduces dramatically as a result of an increased sedentary lifestyle, may be resulting in a pathological state of sarcopenia.

Reproductive History Patterns

Menstrual and reproductive history is considered to be of special importance in explaining somatic changes at the end of the reproductive phase of life [49]. Several studies plead for significant associations between age at menarche and postmenopausal body composition [50, 51]. Other studies yielded significant associations between weight status, body composition as well as fat patterning and parameters of reproductive history, while no menstrual history factors were significantly related to somatic characteristics [52]. Of special importance appear to be the amount of weight gain during pregnancies and the number of births: Obese postmenopausal women reported the significantly highest average weight gain during their pregnancies while normal weight postmenopausal women reported the significantly lowest average pregnancy weight gain. Pregnancy weight gain can in retrospect be identified as the most important triggering life event for the development of obesity and a high amount of body weight. Furthermore, postmenopausal weight status was significantly negatively associated with the age at first birth. Regarding fat distribution, only the number of births seems to be associated significantly with the fat distribution patterns. A gynoid fat patterning seems to be associated significantly positively with the number of births [52]. Concerning body composition, a significant increase of lean soft tissue mass and fat mass during postmenopause with increasing pregnancy weight gain was described [52]. Bone mineral content (BMC) and bone mineral density (BMD) increased significantly with increasing number of births (see Fig. 2.11).

Body Composition Changes During Menopausal Transition from an Evolutionary Point of View

According to Theodosius Dobzhansky “Nothing in Biology Makes Sense Except in the Light of Evolution”. As pointed out in the introduction section several theories have been formulated to find an evolutionary explanation for the phenomenon of human menopause [4]. But what about the body composition alterations? As mentioned above the main characteristics of somatic changes during menopausal transition are the increase of body fat mass and the visible changes in fat dis-

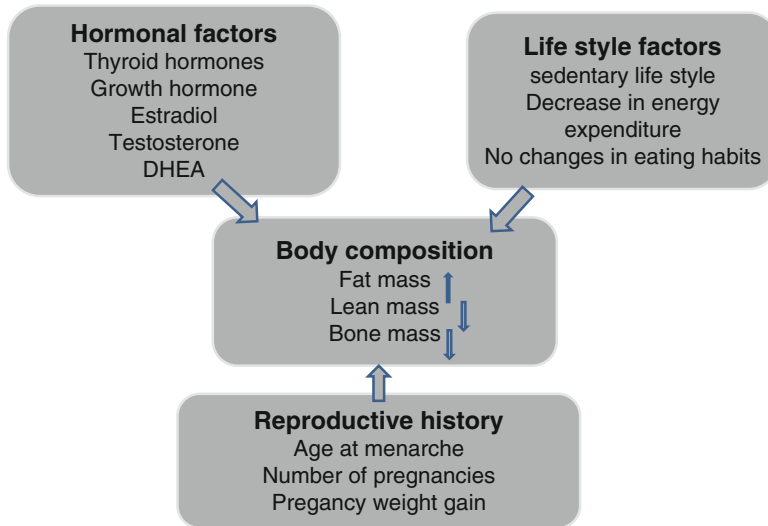


Fig. 2.11 Proximate factors influencing body composition during menopausal transition

tribution patterns. The typical fat distribution of fertile phase of life is the gynoid kind of fat patterning with a quantitatively higher amount of lower body fat, i.e., fat at the hips, buttocks, and thighs, than at the upper body. Cross-cultural analyses using the Human relation area files as data source reveal that in 90 % of investigated cultures a gynoid fat distribution is associated with female attractiveness [53] presumably because gynoid fat patterning is interpreted as an indicator for potential fertility and reproductive success of a female [54, 55]. Body fat at the lower body region is an excellent energy store for phases of increased energetic requirements [23]. The capacity to store lipids within subcutaneous fat depots especially at the lower body region is the key to facing famine and limited caloric supply especially among females. Human females are able to mobilize these energy stores to augment the caloric demands placed on the body during phases of gestation, and lactation. It is no problem for recent female *Homo sapiens* in developed countries to meet the increased energetic requirements of successful reproduction; however, our ancestors did not live in the garden of Eden. They were frequently faced with the problems of malnutrition and starvation. During pregnancy and lactation longer phases food shortages and a lack of sufficient energy had deleterious effects on reproductive outcome [49]. Sufficient energy stores in subcutaneous fat depots were visible indicators that reproductive success is possible even under worse energetic conditions (see Fig. 2.12). Lower body fat stores remain stable even during phases of starvation and malnutrition indicating the potential fertility of young women [55]. In contrast, an android fat patterning, typical for males throughout adult life, is found only among obese young females and among young females suffering from Polycystic Ovary Syndrome (PCOS), the most common endocrine cause of female infertility [56]. An android fat patterning or an android body silhouette is also found during pregnancy when a new conception is impossible. After menopausal transition nearly all postmenopausal women exhibit an android kind of fat patterning independent of their weight status (see Fig. 2.13). Android fat distribution patterns seem therefore to be excellent indicators of infertility or physiological sterility as in case of postmenopause. Therefore, a suggestion for an ultimate or evolutionary explanation of the body composition and fat distribution changes taking place during menopausal transition may be that android fat patterning could serve as an indicator for the irreversible end of female reproductive capability; however, several other evolutionary explanations are possible.

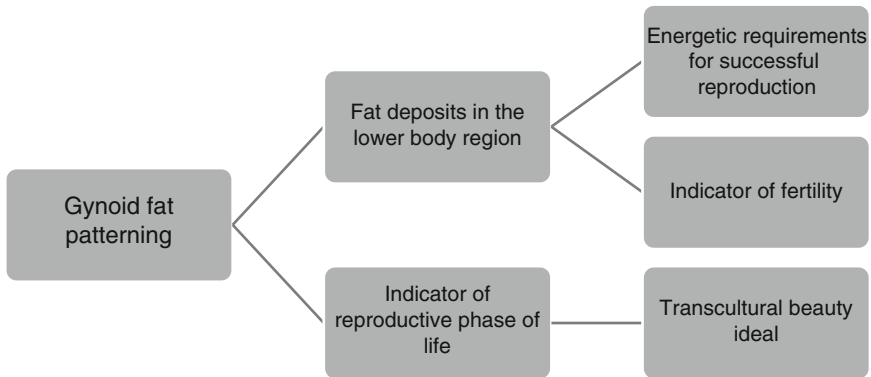


Fig. 2.12 Gynoid fat patterning as an indicator of reproductive phase

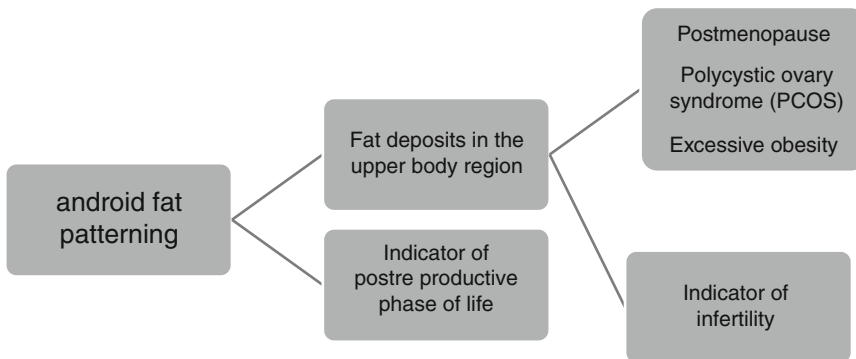


Fig. 2.13 Android fat patterning as an indicator of reduced fertility and sterility

Conclusion

Menopausal transition is associated with weight gain and dramatic changes in body composition. Although these alterations in body composition may increase the risk of various diseases such as metabolic syndrome or osteoporosis, menopause per se is not a disease, it is a natural part of female life history. The redistribution of fat tissue from a gynoid kind of fat distribution, typical of reproductive phase of life towards android fat patterning typical of postmenopause but also hyperandrogenemia in females may be interpreted as a visible marker of physiological sterility of postmenopausal women.

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