BIOLOGICAL REVIEWS
Cambridge Philosophical Society
Biol. Rev. (2021), pp. 000–000.
doi: 10.1111/brv.12778

The evolution of perennially enlarged breasts in women: a critical review and a novel hypothesis

Bogusław Pawłowski and Agnieszka Żelaźniewicz*

Department of Human Biology, University of Wrocław, ul. Przybyszewskiego 63, Wrocław, 51-148, Poland

ABSTRACT

The possession of permanent, adipose breasts in women is a uniquely human trait that develops during puberty, well in advance of the first pregnancy. The adaptive role and developmental pattern of this breast morphology, unusual among primates, remains an unresolved conundrum. The evolutionary origins of this trait have been the focus of many hypotheses, which variously suggest that breasts are a product of sexual selection or of natural selection due to their putative role in assisting in nursing or as a thermoregulatory organ. Alternative hypotheses assume that permanent breasts are a by-product of other evolutionary changes. We review and evaluate these hypotheses in the light of recent literature on breast morphology, physiology, phylogeny, ontogeny, sex differences, and genetics in order to highlight their strengths and flaws and to propose a coherent perspective and a new hypothesis on the evolutionary origins of perennially enlarged breasts in women. We propose that breasts appeared as early as *Homo ergaster*, originally as a by-product of other coincident evolutionary processes of adaptive significance. These included an increase in subcutaneous fat tissue (SFT) in response to the demands of thermoregulatory and energy storage, and of the ontogenetic development of the evolving brain. An increase in SFT triggered an increase in oestradiol levels (E2). An increase in meat in the diet of early *Homo* allowed for further hormonal changes, such as greater dehydroepiandrosterone (DHEA/S) synthesis, which were crucial for brain evolution. DHEA/S is also easily converted to E2 in E2-sensitive body parts, such as breasts and gluteofemoral regions, causing fat accumulation in these regions, enabling the evolution of perennially enlarged breasts. Furthermore, it is also plausible that after enlarged breasts appeared, they were co-opted for other functions, such as attracting mates and indicating biological condition. Finally, we argue that the multifold adaptive benefits of SFT increase and hormonal changes outweighed the possible costs of perennially enlarged breasts, enabling their further development.

Key words: *Homo*, mammary glands, adaptation, by-product, co-option, subcutaneous fat, oestradiol, adrenarche, DHEA, sexual selection

CONTENTS

* Address for correspondence (Tel: +0048713752589; E-mail: agnieszka.zelazniewicz@uwr.edu.pl)

I. INTRODUCTION

The mammary gland is an organ unique to the class Mammalia, with the specific function to synthesise, secrete, and deliver milk to a newborn, thereby promoting optimal nourishment, health, and development (Howard & Gusterson, 2000). Surprisingly, women's mammary glands develop markedly during puberty, long before the first pregnancy and lactation occur, and remain perennially enlarged across the female lifespan, exceeding the reproductive period. Such enlarged, permanent breasts are described as unique to humans (Cant, 1981), not only in the Primates, but also among all 6495 recognised species of mammals (Burgin *et al.*, 2018).

The evolutionary origins of this unique morphology remain unclear, and thus the presence of breasts, which emerged only in hominine, and most likely only in the *Homo* genus, are still an unresolved conundrum, intriguing biologists and leading to a number of hypotheses attempting to explain this phenomenon. These hypotheses can be grouped according to the type of selection pressure or proposed functionality. The most popular are hypotheses that explain breast origin as a product of sexual selection, suggesting that breast morphology could be perceived as an ornament that serves to attract sexual partners [men consider larger breasts to be sexually attractive, at least in Western cultures (Dixson, Duncan & Dixson, 2015; Kościński, 2019)], as a signal of biological condition or reproductive value (Cant, 1981; Gallup Jr, 1982; Marlowe, 1998), or as a deceptive signal (Low, Alexander & Noonan, 1987). Here, 'signal' refers to a trait that evolved because it alters the behaviour of a receiver, and that is effective because the receiver's response has also evolved; its should be differentiated from a 'cue', which refers to a trait that has not evolved for the purpose of conveying information about the signaller but is correlated with some trait of interest for the receiver (Smith & Harper, 2003). The second group of hypotheses addresses women's breasts as a trait selected due to their functional role, *via* mechanisms of natural selection, such as fat storage when resources are scarce (Anderson, 1983), milk warming (Anderson, 1988), physical comfort to an infant (Smith, 1986), or as a thermoregulatory organ (Einon, 2007). The third group of hypotheses assumes that a permanent breast is a by-product of other evolutionary changes, such as the accumulation of subcutaneous fat tissue (SFT) (Pawłowski, 1999*b*), or hairlessness and bipedal locomotion (LeBlanc & Barnes, 1974). It is also plausible that if enlarged breasts appeared due to some initial selection factor, unrelated to sexual selection or to a functional role, then after the emergence of this trait, they could be later co-opted for other functions, including sexual attractiveness or as a cue of biological condition.

So far, there is no consensus around which of these hypotheses are most plausible, and indeed many of the arguments supporting one or another hypothesis have been refuted. The aim of this review is to evaluate these hypotheses in the light of recent literature on breast morphology, physiology, phylogeny, ontogeny, sex differences, and genetics in order to propose a coherent perspective of the evolutionary origins and function of perennially enlarged breasts in women. To provide a complete picture we aim to present all hypotheses (irrespective of the plausibility of the proposed selection pressure), with arguments outlining their flaws, and we also propose a new hypothesis complete with supporting evidence.

II. HUMAN PERMANENT BREAST MORPHOLOGY AND DEVELOPMENT – IN WHAT WAYS IS THIS UNIQUE?

Adult women's breasts consist of skin glands which are approximately symmetrical bilateral organs of hemispherical shape, located on the anterior thoracic wall of the chest (Howard & Gusterson, 2000). Breast morphology varies among women and inter-individual differences in breast volume are dependent mainly on genes (Li *et al.*, 2013), body adiposity (Coltman, Steele & McGhee, 2017), parity (Rauh *et al.*, 2013), age (Brown *et al.*, 2012), and hormone levels

(Jemström & Olsson, 1997). Adult breasts are composed of adipose and glandular tissue (organised into lobes, that 'invade' adipose tissue), supported by Cooper's ligaments. Although the amount and distribution of glandular tissue varies substantially among women, and lobes may differ 20- to 30-fold in size, the breast volume of non-pregnant and non-lactating women is mainly determined by adipose tissue quantity (Howard & Gusterson, 2000). Compared to women, mammary glands in all non-human female primates are more flattened and are distributed more broadly across the chest, but the histological appearance is nearly identical, and 'invasive' glandular tissue is present in all primates (Cline & Wood, 2008). Similarly, compared to other primates, the areola in women is more pigmented and distinctive in comparison to the surrounding skin, but again there are no major histological differences (Cline, 2007).

The development of mammary glands is one of the first signs of puberty in women, starting before menarche, when average sex hormone levels still do not discriminate between boys and girls (Drife, 1986). Breast growth during puberty depends on ovarian and local production of oestradiol, progesterone, prolactin, growth hormone, and insulin-like growth factor (Russo & Russo, 2004; Sternlicht *et al.*, 2006). Comparable developmental patterns are observed in non-human primates. In rhesus macaques (*Macaca mulatta*) for instance, mammary gland development is distinctive and precedes regular menstrual cycles by several months, but increases are mainly in length and width rather than thickness. During puberty, both glandular and adipose tissues develop, resulting in changes in external appearance; the most noticeable increase is in breast volume, in both humans and macaques. However, the extent of this increase, due to the accumulation of adipose tissue, is much more remarkable in women than in any other primate (Aberle, 1934; Golub *et al.*, 2003).

Changes within women's breasts related to pregnancy and lactation are similar to those observed in other primates where the development of glandular tissue also gives rise to a significant increase in breast volume (Geddes, 2007), although enlarged mammary glands in pregnant primate females do not approach the hemispherical shape of women's breasts (Cline, 2007). Breast growth at this time is stimulated by the same hormones as during puberty, and also by placental lactogen (Cox *et al.*, 1999). Following weaning, involution of glandular tissue occurs, resulting in breast volume decreases to pre-pregnancy size (Russo & Russo, 2004). During menopause, the breast regresses and glandular elements involute, resulting in a breast predominantly containing fat and stroma (Howard & Gusterson, 2000). Over time, a progressive decrease in stromal elements occurs, causing breast shrinkage and loss of contour. Cooper's ligaments relax with time, leading to breast ptosis that is positively related to age, parity, body adiposity, and breast size (Rinker, Veneracion & Walsh, 2010). In older females of non-human primates, mammary glands often become pendulous (Cline, 2007). Thus, in comparison with adolescence and youth, most older, multiparous women display a breast morphology that is not atypical for hominoids (Katch *et al.*, 1980). According to Schultz (1969, p. 186), "the glands of multiparous apes resemble more or less the sagging breasts of women though never in such extreme degrees".

Thus, the uniqueness of women's breast morphology concerns mainly the degree of adipose tissue accumulated within a breast and the moment in ontogenesis when such enlarged breasts appear. This means that any potential adaptive explanation of this trait should focus on the accumulation of fat tissue within the breast that occurs during puberty, i.e. well in advance of the first pregnancy.

III. THE BIOLOGICAL COST OF PERMANENT BREASTS

An evaluation of the probability of the various hypotheses around the evolutionary origins of perennially enlarged breasts in women requires a consideration of whether the benefits derived from the development of this trait outweigh the potential costs. It is also important to note that although breast size variability is remarkable in contemporary populations, and some women have extra-large breasts, the evolution of permanent breasts was probably a gradual process, and the first breasts that emerged in our ancestors were probably relatively small (Pawłowski, 1999*b*). Thus, the costs relevant to the evolutionary origins of this trait must be considered in relation to small or medium breast size, considering contemporary variability.

These costs may entail the necessity of investing resources in adipose breast tissue. However, in non-lactating women with a body mass index (BMI) <25, median breast weight is 1666 g (range: 418–3628 g) (Benditte-Klepetko *et al.*, 2007), and accounts for 3.5% of the total body fat (Katch *et al.*, 1980). Even considering that metabolic rate per kilogram of adipose tissue is higher in women than in men (Nookaew *et al.*, 2013), the energy cost of maintaining breast fat is small in relation to the whole adipose tissue cost.

Adipose tissue accumulated within the breast may also interfere with nursing and impede lactation, the key function of the mammary gland. Adipose tissue may inhibit epithelium growth, and so the development of glandular tissue (Howard & Gusterson, 2000), as can be observed in overweight women, who often fail to breastfeed successfully (Amir & Donath, 2007). However, these problems mainly concern women with excessive adiposity and breasts of a size that were probably not encountered in our ancestors. Babies often have problems with suckling on bulgingly hemispherical breasts and difficulties with infant breastfeeding are experienced by many women, totalling as much as 52% of primiparous women in Western populations (Wagner *et al.*, 2013). In other primates, female nipples are elongated, which makes it easier for a baby to take the nipple into its mouth and suckle. Possibly, in ancestral primiparous women breasts were more likely to be saggy instead of rounded, bearing greater similarities to other primates, and allowing greater ease of nursing (LeBlanc & Barnes, 1974).

Large breasts may impede effective movement and posture (Benditte-Klepetko *et al.*, 2007; McGhee *et al.*, 2018), and are also associated with increased muscle activation and upper shoulder muscle tension (Schinkel-Ivy & Drake, 2016). However, this burden comes with age and also mainly concerns women with breasts of a size that was probably not encountered in our evolutionary past. As an example, the corresponding probability of developing a spine disorder for a 25-year-old woman with 800 g breast weight (cup size B) is 8%; for a 25-year-old woman with 2800 g breast weight (cup size D) it is 44%. Compared to this, the risk for a 35-year-old woman with 800 g breast weight is 34% and for a 35-year-old woman with 2800 g breast weight it is 82% (Benditte-Klepetko *et al.*, 2007).

Bigger breasts have also been shown to have a negative impact on performance in long-distance running (Brown & Scurr, 2016*b*). However, this probably was not an obstacle for our ancestors, as data from contemporary hunter–gatherers suggest that adult women are mainly occupied with gathering, reproduction (i.e. being pregnant or lactating), and rearing children, activities not requiring long-distance running (Lovejoy, 1981; Marlowe, 2007). Thus, extreme (as in sport competition) long-distance running was probably not crucial for women, and relatively small breasts in our ancestors would not impact their daily movement (including running or escaping from predators).

Maintaining breast tissue is also related to a higher risk of breast cancer (Clemons & Goss, 2001). However, most breast cancer risk factors are related to a Western lifestyle, such as high oestradiol (E2) levels, low parity, not breastfeeding, or excessive adiposity and a sedentary lifestyle (Adebamowo *et al.*, 2003), factors irrelevant for breast evolutionary origins. Indeed, women from developing countries have a lower incidence of breast cancer compared to women from traditional societies (Clemons & Goss, 2001). Furthermore, most breast cancers occur only after menopause (Sineshaw *et al.*, 2014), and thus have little impact on women's reproductive success. Also, selective pressure linked to disorders occurring later in life is weaker than for those occurring in earlier life stages (Austad & Hoffman, 2018). Additionally, although breast size is positively related to breast cancer risk (Jansen, Backstein & Brown, 2014), it is rather higher breast density, not breast size, that is the risk factor (Soguel *et al.*, 2017). In fact, breast adipose tissue may have a protective role against breast cancer, as adipose tissue is thought to inhibit epithelium growth (Howard & Gusterson, 2000), thus fat that invades breast tissue may constrain glandular tissue development. In that case, an increased amount of adipose tissue within the breast could result from a selective pressure acting to protect glandular tissue from increased E2 levels.

Furthermore, the key genetic risks for breast cancer, i.e. mutations in *BRCA1* and *BRCA2* genes (Miki *et al.*, 1994; Tavtigian *et al.*, 1996), seem to be a cost of evolutionary changes, unrelated to an increase in breast fat. The perseverance of mutations in these genes is probably related to the rapid evolution of these genes in primates as a response to viral infections (Pfeffer, Ho & Singh, 2017), leading to antagonistic pleiotropy where *BRCA1/2* bearers are less susceptible to viral infection but at higher risk of developing breast cancer later in life (Lou *et al.*, 2014).

Thus, the potential costs of perennially enlarged breasts may include the resources needed to develop and maintain this trait, biomechanical costs, impeded suckling, and breast cancer risks. However, these costs are mostly related to the extra-large breast sizes resulting from the specific conditions of WEIRD (western, educated, industrialised, rich and democratic) populations, e.g. to the recent obesity pandemic or to elevated sex hormone levels. The initial cost of permanent breasts in our ancestors was presumably small, mainly related to the energy needed to develop this trait, but other costs could increase and become important with ongoing development of this trait.

IV. WOMEN'S BREASTS AS A PRODUCT OF SEXUAL SELECTION

(1) Permanent breast size and men's mate preferences

It is commonly perceived that breasts draw attention, provoke sexual arousal, and impact women's physical attractiveness, suggesting that male preferences could have played a role in the selective pressure for the emergence of permanently enlarged breasts (Swami *et al.*, 2009; Dixson *et al.*, 2011*a*,*b*, 2015; Groyecka *et al.*, 2017). Also, in Western, clothed cultures, women may choose to accentuate their breasts by wearing 'push-up' bras and tight clothes, and showing or highlighting their cleavage, all of which indicate the importance of breasts in physical attractiveness.

Although men consistently prefer symmetrical (Manning *et al.*, 1997; Dixson *et al.*, 2011*b*), firm, and non-ptotic breasts (Groyecka *et al.*, 2017; Valentova *et al.*, 2017; Kościński, 2019), preferences towards women's breast size are variable. Men have been shown to prefer medium (Horvath, 1981; Tantleff-Dunn, 2002; Guéguen, 2007; Żelaźniewicz & Pawlowski, 2011) to bigger breasts, but not very large breasts (Dixson *et al.*, 2015; Kościński, 2019). This could be explained by stabilising selection, especially as the potential costs of adipose breasts are most apparent with very large breasts. However, there are also studies that have found that men prefer smaller breasts (Kleinke & Staneski, 1980; Thornhill & Grammer, 1999; Furnham & Swami, 2007), or have no specific preference (Furnham, Swami & Shah, 2006).

What is often overlooked in the discussion about women's breasts as a product of sexual selection is the fact that they may not always have been perceived as sexually attractive in our evolutionary past, or across all cultures. Ford & Beach (1951) studied 191 societies and found that in only 13 were breasts sexually important to men. Furthermore, in these 13 societies there was no agreement on the most attractive breast appearance. In some societies, large breasts were preferred, whereas in others, preferences were apparent for

breasts that were long and pendulous (Azande and Ganda of Africa) or upright and hemispherical (Maasai of Africa and Manus of the South Pacific), but not necessarily large. Recent studies also have shown cross-cultural differences in breast size preferences (Swami *et al.*, 2009; Havlíček *et al.*, 2017; Valentova *et al.*, 2017). Furthermore, Macadam & Dettwyler (1995) described reactions of men in Mali to the information that breasts are important in sexual foreplay for Western men. They found it unnatural, perverted behaviour, and difficult to believe that men would become sexually aroused by women's breasts or that women would find such activities pleasurable. Thus, breasts are not universally perceived as sexually appealing, and breast size preference is culturally variable.

Breast size preferences covary with men's characteristics, such as their sexual strategy (men inclined for short-term relationships value bigger breasts more than men inclined for long-term relationships; Żelaźniewicz & Pawlowski, 2011), marital status (compared to unmarried men, married men prefer bigger breasts; Dixson *et al.*, 2011*b*), and resource security (men from a population with limited resources prefer bigger breasts than men from affluent populations; Swami & Tovée, 2013). However, these differences may in fact reflect differences in preferences for youthfulness and total body adiposity (Brooks *et al.*, 2015). A comparison of men's preferences for breast size in Papua New Guinea (PNG), Samoa and New Zealand showed that men from PNG preferred larger breasts than men from the other two islands (Dixson *et al.*, 2011*b*). It is likely that resource scarcity in PNG has driven the preference for padded bustlines as a side-effect of preferences for higher body adiposity, indicating sufficient energetic reserves for pregnancy and childrearing. Studies on the relative importance of various body traits in physical attractiveness perception suggest that, although breasts attract attention (Dixson *et al.*, 2011*a*), their appearance may be less important in assessing attractiveness compared to other measures of body adiposity (Furnham, Dias & McClelland, 1998; Dixson *et al.*, 2011*a*; Brooks *et al.*, 2015).

Even omitting the fact that male preferences for any 'ideal' breast size are not universal and that enlarged breasts are not regarded as a sexually attractive trait across cultures, it is puzzling why such a trait would evolve merely to attract men. The evolution of perennially enlarged breasts as a sexual ornament is probable only if we posit the existence of considerable female–female competition for men, alongside long-term pair-bonds with considerable paternal investment which would bring significant benefits to women and would outweigh the cost of perennially enlarged breasts. Although men contribute to rearing offspring and humans display mutual mate choice (Stewart-Williams & Thomas, 2013), men's mate preferences are not equivalent to female choosiness, which is particularly pronounced given the high reproductive costs that women bear (Woodward & Richards, 2005), and thus male preferences towards 'arbitrary' female ornaments are much less likely to evolve. Male choice is often based on traits that correlate with high fertility in females, or characteristics that represent a direct benefit to a male, such as readiness to mate, health, or reproductive potential, all being traits that would outweigh the costs of male choosiness (Fitzpatrick & Servedio, 2018). However, evidence that breasts convey important information about women's biological condition is equivocal, as we show in subsequent sections.

(2) Permanent breasts as a signal

Despite the fact that men's preferences towards breast size vary, breast morphology and developmental pattern in ontogenesis indicate that their function is not restricted to lactation alone, and thus breasts have been hypothesised to signal women's nutritional status (Cant, 1981), age, or sexual maturity (Gallup Jr, 1982; Marlowe, 1998). Such a signal has been hypothesised to be especially important in our species due to concealed ovulation and the decrease in women's fertility with age (ending with menopause), although both traits are not as unique to humans as was thought (Gould, Flint & Graham, 1981; Burt, 1992), which suggests the existence of some additional pressure for permanent breast evolution.

(*a*) Permanent breasts as a signal of nutritional status

As breast size in non-pregnant and non-lactating women is related to the amount of fat within the gland (Vandeweyer & Hertens, 2002), and breasts of malnourished women are often underdeveloped, it has been hypothesised that adipose breasts evolved as a signal of women's nutritional status (Cant, 1981; Caro, 1987). Adipose tissue is important for maintaining ovarian functioning and to meet the energy requirements of pregnancy and lactation (Mascia-Lees, Relethford & Sorger, 1986; Caro & Sellen, 1990; Gaskins *et al.*, 2015). As our ancestors regularly experienced transient periods of hunger, women who were capable of storing more body fat were more likely to survive not only periods of hunger, but also to maintain an energetically costly pregnancy and lactation (Cant, 1981; Gallup Jr, 1982; Caro & Sellen, 1990).

However, despite the relationship between breast volume and the quantity of adipose tissue within the gland, breast fat accounts only for 3.5% of total body fat (Katch *et al.*, 1980), thus may not be a better indicator of a woman's fat-storing ability and nutritional status than total body adiposity. Furthermore, if breasts evolved to signal energy storage, men should consistently prefer medium to bigger breasts and this trait should be preferred as much as any other trait associated with body adiposity. However, as discussed above, preference for a specific breast size is not universal (Thornhill & Grammer, 1999; Furnham & Swami, 2007; Swami *et al.*, 2009; Havlíček *et al.*, 2017), and other traits related to adipose tissue distribution [e.g. waist-to-hip ratio (WHR)] seem to be more important for body attractiveness assessment (Dixson *et al.*, 2011*b*), with a higher BMI in fact related to lower breast attractiveness (Soltanian *et al.*, 2012).

It may also be presumed that adipose tissue within the mammary gland is particularly important during pregnancy and lactation. However, although there are no reports on breast adipose tissue mobilisation during these periods, it is in fact gluteofemoral fat, not upper-body (abdominal) fat

tissue, that is preferentially used during pregnancy and lactation (Rebuffe-Scrive *et al.*, 1985; Lassek & Gaulin, 2006). In addition, fat in the gluteofemoral region has little negative impact on women's health (Manolopoulos, Karpe & Frayn, 2010), suggesting that this fat depot is more adapted for energy storage. Furthermore, the lipolytic properties of the fat tissue from the gluteofemoral region are stronger than those of the breasts (Richelsen *et al.*, 1991). Thus, breast fat is not more important than other fat depots, and given that there is no clear advantage to breast fat, it is improbable that it evolved as a signal of women's nutritional status, although in some contemporary populations it may be perceived as a cue of a woman's nutritional state, perhaps explaining preferences towards bigger breast sizes in resource-scarce populations (Dixson *et al.*, 2011*b*).

(*b*) Permanent breasts as a signal of age, sexual maturity, and residual fertility

Breast appearance changes with age and reproductive status, and thus could support a capacity to signal youth, sexual maturity, and/or residual fertility (Symons, 1979; Gallup Jr, 1982; Marlowe, 1998). Gallup Jr (1982) suggested that breast size and shape indicate what he called "probability of ovulating" reflecting both age and nutritional status. The 'nubility hypothesis' additionally proposes that perennially enlarged breasts evolved as an honest signal of residual reproductive value, that in turn is a function of a woman's age and parity, which would be advertised both by breast size and shape. The flat chests of prepubescent girls might indicate that they are still infertile, and the sagging, shrunken breasts of older and/or multiparous women might indicate they are no longer fertile, whereas protruding and firm breasts should indicate young age and greater residual reproductive value and should be perceived as the most attractive. Furthermore, as bigger breasts are more prone to ptosis and men could therefore judge the age of a woman with larger breasts better than that of a woman with smaller breasts, men should prefer larger breasts (Marlowe, 1998).

The problem is that early in the evolution of this trait, breasts would be probably have been too small to signal nubility. Marlowe (1998) argues that initially protruding breasts could have signalled puberty. We cannot be sure of the developmental sequence at the time when breasts first originated, but in contemporary human ontogenesis, thelarche precedes menarche (Drife, 1986) and optimal reproductive age by a few years, and therefore breasts would be misleading as a signal of reproductive maturity. Furthermore, even if signalling puberty/absence of menopause was needed due to loss of cues to ovulation in our ancestors, women's age may be revealed not only by breast size, but also many other traits, starting with body shape and proportions (Frisancho & Housh, 1988; Andrews *et al.*, 2017), and ending with more subtle traits, such as skin quality, or the emerging proceptive sexual behaviour of the maturing female – cues that are also present in other primates. Although adipose breasts might evolve as a redundant signal of women's age, as mentioned above, men do not necessarily find bigger breasts the most attractive (Kleinke & Staneski, 1980; Thornhill & Grammer, 1999; Furnham & Swami, 2007). However, after this trait had appeared, breast size might have become useful as a cue of a woman's age and parity, and as such may impact men's preferences (Groyecka *et al.*, 2017).

(*c*) Permanent breasts as a signal of fecundity

Differences in breast size have also been suggested to indicate differences in fecundity. As breast volume in non-pregnant and non-lactating females is primarily determined by the accumulation of adipose tissue, large nulliparous breasts could be a signal of a higher probability of conception per copulation or of other aspects of higher fecundity that are linked with adiposity (Caro, 1987).

However, there is no evidence for this hypothesis, as nulliparous breast size has not been unequivocally shown to be related to any measure of women's fecundity. Jasieńska *et al.* (2004) showed that women with larger breasts and lower WHR are characterised by higher salivary E2 and progesterone levels, which indicates a higher chance of becoming pregnant within each menstrual cycle (Lipson & Ellison, 1996). Jemström & Olsson (1997) also showed that breast volume is positively related to luteal E2 level in young women, although the study included only 20 participants. More recent studies, however, have failed to confirm the relationship between breast volume and fertility hormone levels (Grillot *et al.*, 2014; Kościński, Makarewicz & Bartoszewicz, 2020). As the relationship between breast size and E2 level shown by Jasieńska *et al.* (2004), was only observed if other measures of adiposity and fat distribution were included (WHR), this relationship may result from the relationship between total body adiposity and E2 levels (van Pelt, Gavin & Kohrt, 2015), and thus breasts may not be a better indicator of fertility compared to any other measure of SFT.

(3) Permanent breasts as a deceptive signal

Low (1979) proposed that large breasts signal women's ability to supply a large amount of high-quality milk for a period of years. As breast growth results mainly from an increase in breast adipose tissue, this signal is deceptive, and might be used by women to attract preferred males, without providing fitness benefits. Subsequently, men were selected to have a preference for bigger breasts due to glandular tissue and not due to adipose tissue, shaping preferences for women with bigger breasts but a slender figure (i.e. non-pregnant), leading to permanently enlarged breasts. However, it is hard to state unambiguously that bigger breasts are a deceptive signal of lactational capacity and not a honest signal of maternal adiposity and investment in offspring in ways other than lactation (Caro, 1987). Also, this hypothesis does not explain why men would initially be interested in women with larger breasts, when large breasts indicated pregnancy and lactation, and thus transient infertility.

(4) Permanent breasts as a sexual signal mimicking buttocks

Morris (1967) suggested that breasts appeared alongside bipedalism, as a trait that mimicked the genital display of hemispherical buttocks, shifting men's interest to the front of the body. He suggested that with an upright position the sexual signals of our rotund buttocks were not so obvious during most face-to-face social occasions and during the ventro-ventral copulatory position, and so protruding breasts began to arouse males sexually because they resembled buttocks or the anogenital swelling. However, apes do not have buttocks resembling human ones of hemispheric shape; these appeared due to the musculature necessary for the upright posture, and thus our non-bipedal ancestors probably lacked fleshy, rounded buttocks. Further, there is no evidence that the anogenital swelling was present in prehominines; in Hominoidea, swelling of the skin in the anogenital area in the proliferative and ovulatory phases of the cycle occurs only in the chimpanzee (*Pan troglodytes*) (Pawłowski, 1999*a*).

(5) Permanent breasts as a product of sexual selection: conclusions

The key problem with the hypotheses around the evolutionary origins of adipose breasts as a sexually attractive trait is that breasts appear to be especially unsuited to play this role. In our evolutionary past, similarly to contemporary apes, enlarged breasts could have been confused with temporary non-fertility, such as pregnancy and lactation, and thus it is unlikely that adipose breasts were perceived as attractive in the beginning. Smith (1984) proposed that this latter argument might have been overcome in our ancestors if females with large breasts were not as closely guarded, and thus had greater chances to engage in extra-pair copulations for social, material or genetic benefits. Such a situation could relax the selection against perennially enlarged breasts until, eventually, they had the advantage of obscuring a female's reproductive state. However, this hypothesis has been criticised on theoretical grounds, as it is not clear why other males would have attempted to copulate with a seemingly anovulatory female and why females would have benefitted from a strategy where the reduced competition between males over a seemingly anovulatory female might increase the risk of mating with a poor-quality male. Furthermore, it is not clear why males would not have guarded their females if they had or appeared to have had infants due to the possibility of infanticide by other males (van Schaik & Dunbar, 1990; Baniel, Cowlishaw & Huchard, 2016), and more importantly, regarding concealed (or not-advertised) ovulation in human female ancestors (Benshoof & Thornhill, 1979), it is not clear why selection would favour males who lowered their guard and suffered the considerable costs of cuckoldry over males who did not allow their mates such freedom (Caro, 1987).

Furthermore, as in our evolutionary past women's fertility decreased with age (Peccei, 1995) and some level of paternal investment was present (Alger *et al.*, 2020), women's biological condition and age were important factors in male mate choice. Thus, *Homo* ancestors were probably able to recognise female nubility, fertility, or health, without perennial breasts. This poses the question of why *Homo* were under a unique selection pressure for the emergence of a new morphological signal, indicating women's condition. As we discuss above, while increased paternal care, resulting in mutual mate choice, and concealed ovulation might constitute a selection pressure for a new signal, it seems unlikely that a trait related to transient infertility would be suitable for this role. It also has been proposed that bipedalism might have played a role in adipose breast evolution through sexual selection. For instance, in an upright position, protruding breasts may be more suited as a signal of women's condition, as in quadrupedal position breasts always extend straight down (Marlowe, 1998). However, although this is true for already evolved adipose breasts, bipedalism probably was not the key factor triggering adipose breast evolution, considering that breasts probably appeared much later [i.e. 1.2 million years ago (mya); see Section VIII] than habitual bipedalism, which appeared 6 mya or even earlier (Böhme *et al.*, 2019).

The high heritability [around 50% (Wade, Zhu & Martin, 2010; Eriksson *et al.*, 2012)] and variability among individuals (with a coefficient of variation of about 60%; Vandeweyer & Hertens, 2002) for breast size are typical of sexually selected traits (Pomiankowski & Møller, 1995), suggesting that, at least to some degree, adipose breast evolution has been driven by sexual selection. Given the absence of cross-cultural preferences for some specific breast size, and the fact that enlarged mammary glands represent an unequivocal signal of transient infertility (pregnancy and lactation), that in other primates would be considered an asexual trait, it seems implausible that the primary evolutionary function of permanent breasts was sexual signalling. However, it is possible that the role of breasts as a physically attractive trait could have been acquired secondarily and under the influence of factors related to cross-cultural differences in breast perception (e.g. naked *versus* clothed societies). Furthermore, it is likely that, some time after their emergence, breasts could have become a cue of factors related to women's biological condition, e.g. age, fertility (Jasieńska *et al.*, 2004), parity (Rauh *et al.*, 2013) and/or health (Kościński *et al.*, 2020). This could explain variation in men's mate preferences but not the evolutionary origins of permanent breasts in human evolution. In some societies (mostly Western) bigger breasts are preferred by men, and many studies have attempted to verify if men's preferences towards breast appearance reflect evolutionary adaptations (Manning *et al.*, 1997; Kościński, 2019; Kościński *et al.*, 2020). This may occur when a trait becomes a cue, correlated with some key information about the owner's fertility or health, that could guide mate choice. Thus, choosing a partner with a particular variant of a trait would increase an individual's reproductive success. The trait and mate preference then coevolve, driving the further development of the trait and greater interest in this trait in potential partners (Prum, 2012).

V. PERMANENT BREASTS AS A PRODUCT OF NATURAL SELECTION

(1) Breast fat and lactational capability

The primary role of mammary glands is breastfeeding, and thus it has been proposed that breast size might be related to lactational ability (Powe, Knott & Conklin-Brittain, 2010; Żelaźniewicz & Pawłowski, 2019). However, although women with bigger breasts are perceived as more nurturing (Dixson *et al.*, 2015), pre-pregnancy breast volume does not correlate with lactation (Anderson, 1983; Mascia-Lees *et al.*, 1986; Anderson, 1988). Women with small breasts are capable of feeding their babies effectively (Anderson, 1988; Pond, 1997), which probably results from the fact that non-pregnant breast size is almost entirely determined by fat rather than glandular tissue. Furthermore, apart from the condition of insufficient breast development (hypoplasia) during pregnancy (Arbour & Kessler, 2013), the increase in breast volume during pregnancy, linked to glandular tissue development, is unrelated to lactation (Cox *et al.*, 1999; Żelaźniewicz & Pawłowski, 2019). Also, perfectly functioning breast tissue may be found outside the embryonic milk streak (this 'streak' being the precursor to the mammary glands and nipples as found in the embryo), and in the absence of accompanying fatty pads. The most famous example is Therese Ventre of Marseilles in France in 1827, who suckled her children from her right breast and from an accessory gland on her right thigh (Grossl, 2000). The majority of accessory breast tissues go unnoticed unless a woman becomes pregnant, suggesting a lack of relationship between breast fat gained during puberty and effective lactation (Loukas, Clarke & Tubbs, 2007). Lactational performance seems instead to be positively related to factors such as women's general condition (Quinn *et al.*, 2012) or adiposity (Brown *et al.*, 1986; Perez-Escamilla *et al.*, 1995) – although lactation may be also impaired in obese women (Rasmussen, 2007) – and negatively related to increasing parity (Prentice, Prentice & Whitehead, 1981), but is not related to breast volume.

(2) Permanent breasts as a thermoregulatory organ

Einon (2007) suggested that enlarged breasts evolved as a "cooling fin", allowing the avoidance of foetal damage by extreme temperatures, as could have been experienced by pregnant women in our evolutionary past in Africa. Furthermore, breast fat could serve as thermal insulation and protection for glandular breast tissue (Anderson, 1988). If these hypotheses are true, one would expect that other mammals would also have evolved similar features. However, none of the contemporary African primates exhibit such a form of thermal insulation.

Furthermore, if breasts evolved as a thermoregulatory organ, breast size should be related to climatic zone, although this depends on several factors, including when and how quickly this feature evolved, and the costs associated with it. Unfortunately, comprehensive scientific data on the intra- and inter-population variation in breast volume are meagre, and there are only a few such studies. The average volume of a single breast has been shown to range from 325 ml in a Chinese study (Qiao, Zhou & Ling, 1997), 564 ml in one British study (Hussain *et al.*, 1999), to 582 ml in another British study (Scutt *et al.*, 1997). Other research shows high intra-population variability ranging from 48 to 3100 ml in an Australian study (Coltman *et al.*, 2017). The distribution of bra sizes worldwide shows a completely different trend than predicted by hypotheses on a thermoregulatory function for breast fat, with the lowest bra sizes in African and East Asian countries and the largest in North America (Anderson *et al.*, 2013). This distribution matches the worldwide distribution of obesity rather than climatic conditions (Abarca-Gómez *et al.*, 2017).

(3) Permanent breasts as a help in nursing offspring

Baker & Bellis (1995) proposed that bipedal female hominids, carrying babies, had to support their heads (because of an infant's lack of stable neck posture), and so selection favoured large breasts that could act as a cushion. Harlow & Suomi (1970) showed that soft body parts are preferred by young monkeys, which may support this hypothesis. Thus, the preference of *Homo* toddlers for a soft breast surface instead of the hard surface of the ribs and sternum might result in mammary gland enlargement (Smith, 1986). However, it is rather unlikely that such a preference could have constituted a selection pressure, influencing babies' survival rate or women's reproductive success. Similarly, Morgan (1972), elaborating on Hardy's Aquatic Ape Hypothesis, suggested that women's breasts evolved to make it easier for infants to reach the nipple while the mother nursed on land. Large, adipose, and thus buoyant, breasts would also help to breastfeed babies when immersed. However, as adipose tissue that accumulates within the breast may interfere with nursing (Howard & Gusterson, 2000; Amir & Donath, 2007) and women's breast shape is far from perfect for breastfeeding (Wagner *et al.*, 2013), it seems unlikely that breast fat evolved to support the nursing of offspring.

(4) Natural selection in permanent breast evolution: summary

Although some of the hypotheses on the possible adaptive role of breasts are intriguing, none of the proposed mechanisms explains the potential initial trigger of the evolution of perennially enlarged breasts and their early appearance in ontogeny independent from the evolution of other morphological adaptations. Most of these hypotheses argue that breasts appeared as a response to evolutionary changes which triggered other morphological adjustments, such as bipedal locomotion or relatively immature neonates (Baker & Bellis, 1995). Even more importantly, they do not answer the key question, i.e. why breasts develop during puberty and not only during pregnancy and lactation.

VI. PERMANENT BREASTS AS A BY-PRODUCT OF SUBCUTANEOUS FAT TISSUE INCREASE AND CHANGES IN HORMONAL PROFILE

Perennially enlarged breasts may be a by-product of other evolutionary processes, e.g. a pleiotropic effect of the adaptive pressure for some other trait (selected to increase an individual's survival and reproductive success in a changing environment), if the potential advantages of this change outweighed the cost of perennially enlarged breasts. Subsequently, breasts could have been co-opted and possibly developed further by sexual selection. In that case, it is not necessary to assume adaptive pressures on permanent breasts when they first appeared phylogenetically.

(1) Breast size and body adiposity

The evolutionary origins of adipose breasts may be linked to an adaptive pressure for SFT increases and a specific fat distribution in women, driven by sex hormones (Mascia-Lees *et al.*, 1986; Caro & Sellen, 1990; Pawłowski, 1999*b*). Pawłowski (1999*b*) suggested that permanent breasts evolved as a side-effect of an oestrogen-induced SFT increase at puberty and the increased sensitisation of apocrine and mammary glands to oestrogens which probably already existed in *Homo erectus* in response to environmental pressures. An additional layer of SFT evolved as an insulation against the cool night temperatures in the Pleistocene open savannah and also to provide calorie storage for the benefit of both mothers and infants. Thus, adaptive pressures might have acted on overall adiposity, and an increase in breast fat might be a by-product of these processes. This seems to be reflected in the studies on breast attractiveness, showing that breasts are a feature related to total body adiposity; breast attractiveness assessment depends on other measures of body adiposity (Furnham *et al.*, 1998) and also on the nutritional stress in a population (Dixson *et al.*, 2011*a*; Swami & Tovée, 2013).

Breast size has been consistently shown to correlate positively with body adiposity. Katch *et al.* (1980) showed a positive correlation between breast volume and body fat ($r = 0.40$), but the strength of this correlation oscillated from low to moderate due to the large variability in participants' breast volume. The strongest correlation was observed in women with lower adiposity, what might reflect the condition in our ancestors, when enlarged breasts were emerging. This was confirmed by Benditte-Klepetko *et al.* (2007), who found a moderate correlation ($r = 0.57$) between breast weight and BMI in young women with a BMI <25. Other research also identified a positive relation between body adiposity and breast size in women [$r = 0.66$ (Brown *et al.*, 2012); $r = 0.49$ (Coltman *et al.*, 2017); $r = 0.53$ (Jemström & Olsson, 1997)]. Furthermore, breast size increases observed in contemporary Western populations seem to be mainly related to the obesity pandemic (Brown & Scurr, 2016*a*), and extremely large breasts are rarely encountered in low- or normal-weight women (Coltman, Steele & McGhee, 2018). For instance, data from an Australian sample showed that women with extra-large breasts (>1200 ml) had a BMI range of 25–55 kg/m^2, whereas women with small breasts (<350 ml) had a BMI range of 19–26 kg/m^2. Reports of the volume of one breast in non-overweight women have ranged from 305–438 ml, in overweight women 608–778 ml, and in obese women 980–1153 ml (Coltman *et al.*, 2017, 2018; Steele, Coltman & McGhee, 2020). Interestingly, in contrast to obese women, those with a normal body mass have lower variation in breast size (Coltman *et al.*, 2017, 2018). Further, Anderson *et al.* (2013), mostly based on cross-country data from commercial screenings by local companies, showed that breast volume ranges in countries with a higher average bra cup were much wider compared with breast volume ranges in countries with a lower average bra cup. This might suggest that variation in breast volume in our evolutionary past was in fact smaller than in contemporary populations, which might also indicate a lesser role for sexual selection in the evolutionary origins of this trait, as sexual traits are usually more phenotypically variable than non-sexual ones (Andersson, 1994; Pomiankowski & Møller, 1995).

Breast size is determined by the amount of both adipose and glandular tissue. However, although women differ in glandular tissue amount, breast size correlates more strongly with adipose tissue than with glandular tissue amount (apart from during pregnancy or lactation) (Hytten & Leitch, 1971; Neifert, Seacat & Jobe, 1985). Thus, we presume that the initial evolutionary increase in breast volume was probably related to changes in adipose components, as a side-effect of SFT increase, and not to glandular tissue increases.

Breast size and body adiposity also have a partially in common genetic underpinning. Based on twin research, Wade *et al.* (2010) inferred that one-third of genes related to breast size were also related to BMI. Using single nucleotide polymorphisms (SNPs), Ooi *et al.* (2019) found that BMI is genetically correlated with breast size ($r = 0.50$), and that it is BMI that is causally linked to breast size and not *vice versa*. Furthermore, so far only two loci have been identified as being responsible exclusively for breast size and unrelated to the amount of SFT (locus 8p11.23 and 22q13.2) but these two SNPs only explain between 0.6 and 1% of the variance for this phenotype (Li *et al.*, 2013).

Thus, it can be concluded that breast size and body adiposity are two closely linked traits. Although sexual dimorphism in body fat quantity is also observed in some non-human primates (Zihlman & Bolter, 2015), it is not as strongly expressed as in *Homo*, and none of the living species exhibits perennially enlarged mammary glands. This suggests that there must have been some genus-specific selection pressure involved in the evolutionary origins of SFT and breast development.

(2) SFT, oestradiol and permanent breasts

Mammary gland development can be divided into embryonic, pubertal, and adult phases, each regulated differentially. Embryonic development occurs without any hormonal influences, and both female and male glands develop similarly *in*

utero (Howard & Gusterson, 2000). Breast sexual dimorphism appears during puberty, when breast growth in girls is stimulated by increasing concentrations of E2, progesterone, prolactin, growth hormone (GH), insulin-like growth factor (IGF), lactogen, and the development of the respective receptors (Sternlicht *et al.*, 2006). In boys no such development normally occurs. However, the occurrence of male gynecomastia, quite common in Western men (34%), indicates that ductal and stromal (but not lobular) development potential is also present in the male breast (Howard & Gusterson, 2000). This occurs mainly in men between the ages of 50 and 69 years when testosterone level decreases (Thiruchelvam *et al.*, 2016), or in men with higher body adiposity where it may be related to the higher conversion rates of androgens to oestrogens (Sher, Migeon & Berkovitz, 1998), as adipose tissue is an important site of extra-glandular aromatase activity (Edman *et al.*, 1978). Thus, even men have the potential to develop adipose breasts, but such growth is suppressed by a low-E2 and high-testosterone hormonal milieu.

There is plenty of evidence that the proximal mechanisms that underlie the relationship between the increases in SFT and breast size are hormonal. Sex differences in body adiposity are mainly related to E2 level (Joyner, Hutley & Cameron, 2001) and oestrogen receptor (ER) expression in fat depots (Pedersen *et al.*, 2001). E2 attenuates lipolytic responses through up-regulation of the number of antilipolytic α-2A-adreneric receptors in SFT, allowing women to 'regulate' body fat stores through different life stages (e.g. puberty, pregnancy), without altering caloric intake significantly (O'Sullivan, Hoffman & Ho, 1995; Uranga, Levine & Jensen, 2005). Furthermore, E2 is synthesised within SFT due to aromatase activity (Labrie, 1991). Thus, women with higher body adiposity also have higher levels of E2 in SFT, which in turn promotes further accumulation of SFT (Mayes & Watson, 2004), and consequently results in breast volume increase. Thus, female puberty and early pregnancy could be seen as states of efficient fat storage (in preparation for foetal development and lactation) driven by E2 levels (O'Sullivan, 2009), where increased breast size would be a side-effect of this evolutionary adaptation.

Exogenous E2 administration increases both body adiposity and breast volume in adolescent girls. Brunnhuber & Kirchengast (2002) showed that hormonal contraception resulted in weight gain in 28.8% and breast size increase in 23.7% of girls, and these two effects were strongly linked within individuals. Both body adiposity and breast volume were related to increased hormone levels despite the fact that most of the girls used low-dose ethinylestradiol. This sensitivity to even small changes in E2 is also observed in prepubertal children; due to very low levels of sex steroids, even small variations account for major changes in the total activity of E2, enabling breast increase (Aksglaede *et al.*, 2006).

The reason why increases in SFT and E2 lead to greater fat increases in the breast (and also gluteofemoral) region, compared with other body regions, is related to the different regional distribution of ERs within the body (Labrie, 1991). Higher ER density in the breast and gluteofemoral regions (Veldhuis *et al.*, 2005) was probably already present in our ancestors, as it is also observed in non-human primates. The number of ERs in chimpanzees' sexual swelling, uterus and breast is much higher than in other body regions, and higher E2 levels induce the preferential expression of ERs in these regions (Treilleux *et al.*, 1997). ER concentration is also related to the degree of engorgement in the chimpanzee sexual swelling (Ozasa & Gould, 1982). Thus, it is very likely that breast and gluteofemoral regions were especially sensitive to E2 increases in our female ancestors, and this would have led to the preferential accumulation of SFT in these regions, especially in peri-pubertal girls, characterised by high tissue sensitivity to increased E2.

The obligatory role of E2 in the growth and development of the mammary gland is well established across mammals (LaMarca & Rosen, 2008), and E2 is also responsible for sex differences in body adiposity in non-human primates (Zihlman & Bolter, 2015). Accordingly, the hormonal background that enabled eventual permanent breast enlargement was already present in the higher primates. However, only in humans did the interplay between SFT and E2 lead to perennially enlarged breasts. Since non-human primates have similar levels of E2, progesterone and gonadotropins during the menstrual cycle and pregnancy (Hobson *et al.*, 1976; Nadler *et al.*, 1985), it is not merely an increase in E2 level that could explain the evolution of adipose breasts.

(3) Adrenarche, brain evolution and the emergence of permanent breasts

In our evolutionary past, hormones other than E2 probably played a role in the proximal mechanism leading to the evolution of perennially enlarged breasts in women. We suggest that the most probable candidate is dehydroepiandrosterone (DHEA/S). Adrenarche, which sees the post-natal rise of DHEA/S (adrenal precursor of sex steroids), is unique to humans, chimpanzees (Sabbi *et al.*, 2019), bonobos (*Pan paniscus*) (Behringer *et al.*, 2012), and gorillas (*Gorilla gorilla*) (Edes, 2017), but DHEA/S is much higher in adolescent girls than in other young primates (Blevins *et al.*, 2013). DHEA/S is an important source of E2 in maturing girls during the adrenarche phase of puberty (Labrie *et al.*, 1998), and the highest rise in BMI during puberty is accompanied by an increase in DHEA/S levels (Kaplowitz, 2008). Intracrine conversion of DHEA/S in peripheral target tissues may be responsible for 75% of E2 prior to the menopause (Labrie, 1991), and circulating DHEA/S levels are 1000–10000 times higher than levels of E2 in adult women (Labrie *et al.*, 1998).

Interestingly, tissue DHEA/S levels are much higher than serum levels (Thijssen *et al.*, 1990), providing excess substrate for the local formation of oestrogens. Levels of oestrone (E1) and E2 within the breasts are also higher than in serum (James *et al.*, 1986), suggesting that high DHEA/S levels in women allow for higher local (but not serum) concentrations of E2. This may explain why, despite similar levels of serum E2 among the primates, only women have perennially enlarged breasts.

The evolutionary rationale for increasing DHEA/S levels in humans at the onset of middle childhood has been argued to be linked to brain evolution and its ontogenic development, as DHEA/S has an important impact on cortical development and cortico-limbic connectivity in children (Greaves *et al.*, 2019). Campbell (2020) suggested that higher DHEA/S levels in humans, relative to apes, might have been possible due to an increasing level of meat in the diet with the advent of the genus *Homo*, which also provided the energy to support a larger, energetically more costly brain. In girls, adrenal activity and DHEA/S levels during puberty also depends on caloric intake (Hill *et al.*, 1980), and a low-caloric and low-protein diet during thelarche is related to breast underdevelopment. Diet improvement, particularly protein consumption, and the additional use of hormonal contraceptives with oestrogens improves such atrophy (Greydanus, Parks & Farrell, 1989). Interestingly, animal protein intake has also been related to the increased production of IGF-1 (Rogers *et al.*, 2005), an important factor in the early stages of breast development.

Thus, we suggest that the emergence of perennially enlarged breasts in women was a by-product of several coinciding factors. These included increases in SFT probably related to thermoregulatory (Pawłowski, 1999*b*) or energy-storage requirements (Mascia-Lees *et al.*, 1986), and increasing energetic demands for evolving brain development (Kuzawa *et al.*, 2014). Subsequently, increased meat consumption in early *Homo* allowed for greater DHEA/S synthesis (and possibly also IGF-1), which was necessary for further brain evolution, and was also easily converted to E2 in local, E2-sensitive body parts, such as the breast and gluteofemoral regions, driving greater fat accumulation within these regions, and finally, enabling perennially enlarged breast evolution (Fig. 1). As DHEA/S in boys is preferentially converted to testosterone rather than E2 in Leydig cells (Liu *et al.*, 2015), and as ERs are much more abundant in girls compared with boys, adipose breasts evolved only in women despite DHEA/S increases in both sexes.

VII. PERMANENT BREASTS AS A TRAIT WITH CO-OPTED FUNCTIONS

The idea that breasts appeared as a by-product of other adaptive changes does not mean that adipose breasts could not have been co-opted later for some functions. Co-option is a long-recognised phenomenon in evolution (Ganfornina & Sánchez, 1999). Co-option occurs when natural selection acts on new functions for existing traits, including genes, physiological pathways, body structures and behaviours. There are many traits that have been argued to evolve based on the co-option of other traits. For example, many courtship and agonistic displays in birds are thought to be ritualised forms of locomotory intention movements (True & Carroll, 2002).

We suggest that permanent breasts later acquired functions that were not the primary evolutionary causes of their emergence. As a sexually dimorphic trait, breasts might have been co-opted as a sexual attractant or a cue of biological condition, leading to a new type of selection. This new selection could have acted on breasts independently from SFT, and thus could also have reduced the correlation between SFT and breast size. This selection could lead to a partial separation of the genes (or physiological mechanisms) responsible for breast and SFT development during ontogeny: i.e. apart from the genes commonly responsible for both SFT and breast increases, genes responsible for only one of these traits might also have emerged. This reduction in the link between SFT amount and breast volume could have health consequences, as women with bigger breast size and relatively low SFT have a higher breast cancer risk, probably due to greater amount of glandular tissue present (Markkula *et al.*, 2012).

In endorsing this hypothesis on the evolutionary origins of breasts as a by-product of selection for other traits, we need to assume that the evolutionary advantages of SFT outweighed the potential costs of increasingly adipose breasts. Most of the costs related to adipose breasts are unlikely to be relevant to the consideration of breast origins in our evolutionary past, and were probably negligible during the period when enlarged breasts appeared (see Section III). On the other hand, the benefits of SFT increase for *Homo* females were manifold: (*i*) insulation (buffering against the cold) (Pawłowski, 1998); (*ii*) acting as energy depots protecting from multiple ecological stresses (buffering against starvation); (*iii*) allowing increased maternal investment in pregnancy, particularly related to foetal brain growth (Kuzawa, 1998); and (*iv*) higher infant survival in non-thermoneutral environments and under high infection pressures due to the positive correlation between maternal and neonatal SFT (Kuzawa, 1998). Furthermore, elevated DHEA/S levels during thelarche allow enhanced brain development during the intensive synaptic pruning that takes place during this ontogenetic stage, while also resulting in higher local levels of E2 in girls (Kuzawa, 1998; Wells, 2010; Kuzawa *et al.*, 2014).

We suggest that the multifold adaptive benefits of SFT increases and hormonal changes, including levels of DHEA/S, which were probably related to encephalisation in *Homo*, easily outweighed any costs of potential side-effects, such as perennially enlarged breasts.

VIII. WHEN DID PERMANENT BREASTS APPEAR IN HUMAN EVOLUTION?

If adipose breasts are a by-product of SFT accumulation, then the first form of 'fat pads' around mammary glands in hominine evolution would have appeared alongside SFT increases in our ancestors. It is presumed that increases in SFT occurred with body hair reduction, and that hairlessness and adiposity are coupled features in a uniquely human thermoregulatory solution. This has been hypothesised to occur

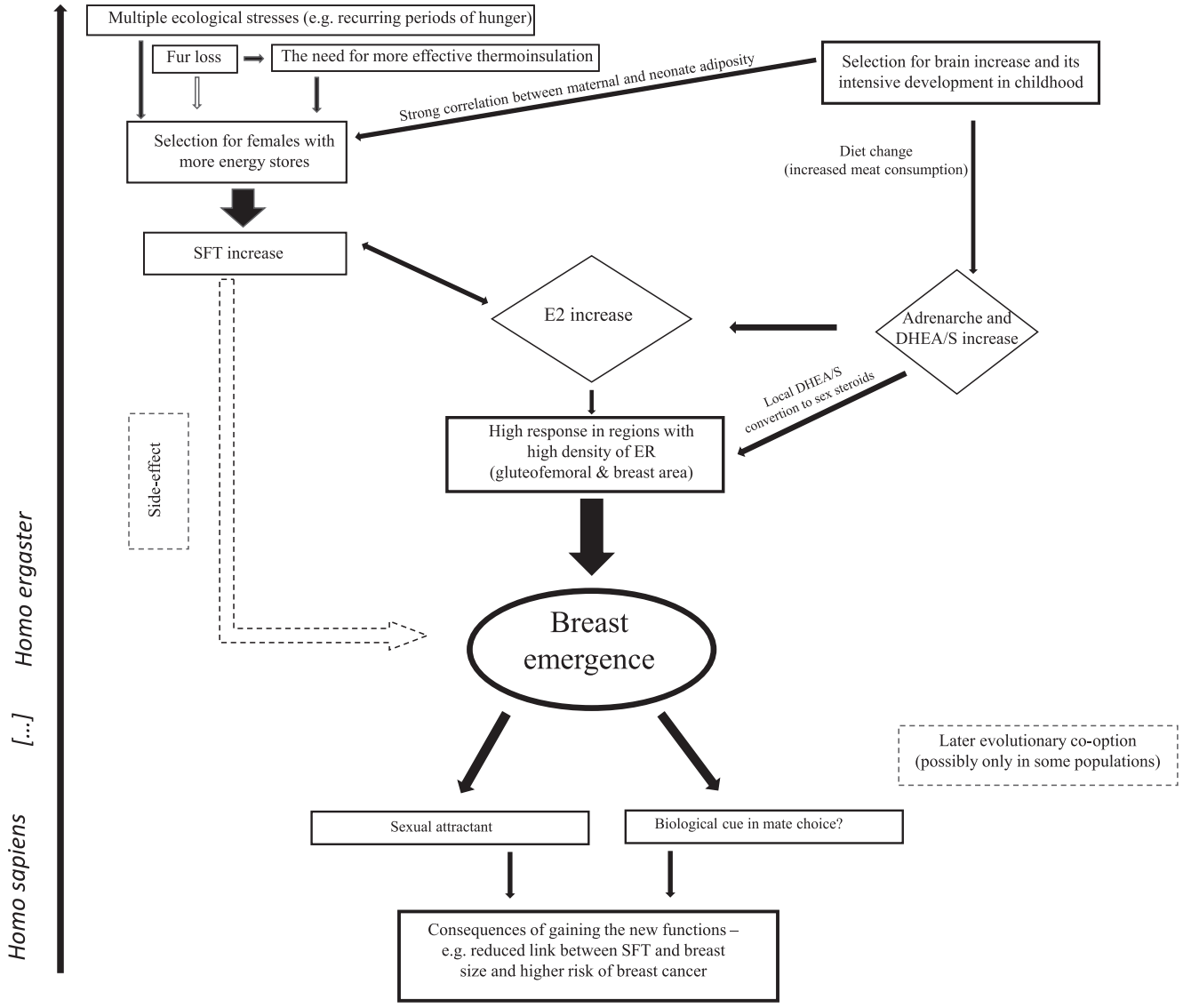

Fig 1. Scheme of the proposed model of evolution of perennially enlarged breasts in women. DHEA/S, dehydroepiandrosterone; E2, oestradiol; ER, oestradiol receptor; SFT, subcutaneous fat tissue.

as late as 1.2 mya based on molecular evidence (Rogers, Iltis & Wooding, 2004), or as early as 3–4 mya, based on analyses of the genomes of species of primate lice (Reed *et al.*, 2007), although this latter estimate requires further study as it was based on an assumption that the origin of pubic lice in humans might be an indicator of the loss of body hair. In non-human primates, skin covered by hair is unpigmented, whereas exposed skin, such as that on the face, hands and feet, is pigmented. Thus, dark skin in early African *Homo* might have been an evolutionary response to the loss of sun-shielding body hair. The sequences of the human *MC1R* gene, responsible for dark skin pigmentation, originated *ca.* 1.2 mya, so it is likely that adipose breasts appeared at a similar time (Rogers *et al.*, 2004).

Another line of reasoning is related to a decrease in sexual dimorphism in body height and increased difference in SFT that occurred in *Homo ergaster* approximately 1.9 mya. Based on estimated body composition from fossil material, it is hypothesised that fatness was slightly higher in females than in males across the entire period of hominin evolution, with a steeper increase in female body size occurring only in *H. ergaster* (Aiello & Wells, 2002).

SFT increase might be also linked with a selective pressure for increased adiposity in neonates to support brain development (Kuzawa, 1998). Maternal and neonate adiposity are strongly correlated (Yu *et al.*, 2013), and thus increases in neonate adiposity would also require an increase in maternal adiposity. This scenario suggests that brain enlargement was an important factor in the evolution of human adiposity, the former of which significantly accelerated in *Homo erectus* approximately 2 mya (Wells, 2010).

Other potential evidence that can be used to date an increased proportion of SFT, and in consequence also increased breasts in hominins, is that higher SFT amounts would make it much easier to migrate and adapt to cooler climate zones. The first *Homo* that lived outside Africa migrated to Eurasia (Dmanisi in the Georgian Caucasus) around 1.8 mya (Ferring *et al.*, 2011). Thus, we suggest that, together with the increase in SFT in females, breasts appeared in *Homo ergaster*.

IX. FUTURE RESEARCH

Understanding the evolutionary origins of permanently enlarged breasts still requires further research, as this trait has been surprisingly neglected in the scientific literature. Descriptive cross-population data on breast size and breast development during ontogeny would help us understand the ecological factors related to inter-individual variation in breast size and breast development, potentially providing support or refuting some of the proposed hypotheses (e.g. breasts as thermoregulatory organs).

As our hypothesis implies a two-step evolution of permanently enlarged breasts, initially as a side-effect of an increase in SFT and then later co-option to some other function, testing our hypothesis will require studies from different fields. Genetic studies may reveal potential shared genes related to SFT tissue increase and breast development. Furthermore, we need to know how these genes are related to DHEA/S and ER density. Future studies should also verify the relationship between DHEA/S and intra- and inter-individual breast size variation. Additionally, more data showing how breast size varies with body adiposity would be helpful. Cross-cultural studies on the relationship between breast size and women's biological condition could also allow to test its role as cue of health or fertility.

X. CONCLUSIONS

(1) There is no compelling evidence that perennially enlarged breasts evolved under direct selection for an adaptive role in the past.
(2) The evolutionary emergence of adipose breasts might be a by-product of an adaptive pressure for an increased proportion of SFT in *Homo* females, possibly driven by the same physiological mechanism that acts on SFT increases and breast development during puberty.
(3) Breasts as a side-effect of SFT increases could have been related to increased DHEA/S levels in women; DHEA/S levels are important precursors of local sex steroid production, leading to much higher local levels (in breast and gluteofemoral regions) of E2 in *Homo* females compared to other primates.
(4) The initial costs of permanent breasts were probably negligible compared to the benefits of increases in SFT and DHEA/S for *Homo* females, newborn survival, and brain development.
(5) As a sexually dimorphic trait, breasts might been co-opted as a sexual signal, which in turn possibly evoked new selection pressures on this trait, lowering the strength of the correlation between SFT and breast size. This might have resulted in some negative consequences (e.g. increased risk of breast cancer).
(6) Breasts probably evolved before 1.2 mya, and likely appeared in *Homo ergaster*.

XI. ACKNOWLEDGEMENTS

We would like to thank the reviewers for constructive suggestions and Tamsin Saxton for helpful comments and proofreading.

XII. REFERENCES

Aberle, S. B. D. (1934). Growth of mammary gland in the rhesus monkey. *Proceedings of the Society for Experimental Biology and Medicine* **32**(1), 249–251.

Abarca-Gómez, L., Abdeen, Z. A., Hamid, Z. A., Abu-Rmeileh, N. M., Acosta-Cazares, B., Acuin, C., Adams, R. J., Aekplakorn, W., Afsana, K., Aguilar-Salinas, C. A., Agyemang, C., Ahmadvand, A., Ahrens, W., Ajlouni, K., Akhtaeva, N., et al. (2017). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *The Lancet* **390**(10113), 2627–2642.

Adebamowo, C. A., Ogundiran, T. O., Adenipekun, A. A., Oyesegun, R. A., Campbell, O. B., Akang, E. E., Rotimi, C. N. & Olopade, O. I. (2003). Waist–hip ratio and breast cancer risk in urbanized Nigerian women. *Breast Cancer Research* **5**(2), R18.

Aiello, L. C. & Wells, J. C. (2002). Energetics and the evolution of the genus *Homo*. *Annual Review of Anthropology* **31**(1), 323–338.

Aksglaede, L., Juul, A., Leffers, H., Skakkebæk, N. E. & Andersson, A. M. (2006). The sensitivity of the child to sex steroids: possible impact of exogenous estrogens. *Human Reproduction Update* **12**(4), 341–349.

Alger, I., Hooper, P., Cox, D., Stieglitz, J. & Kaplan, H. S. (2020). Paternal provisioning results from ecological change. *Proceedings of the National Academy of Sciences of the United States of America* **117**(20), 10746–10754.

Amir, L. H. & Donath, S. (2007). A systematic review of maternal obesity and breastfeeding intention, initiation and duration. *BMC Pregnancy and Childbirth* **7**(1), 9.

Anderson, P. (1983). The reproductive role of the human breast. *Current Anthropology* **24**, 25–45.

Anderson, J. L. (1988). Breast, hips, and buttocks revisited: honest fatness for honest fitness. *Ethology and Sociobiology* **9**(5), 319–324.

Anderson, J. D., Chandler, S. C., Mason, M. A., Khan, C. B., Lindsay, J. E., Sandler, R. M. & Wong, L. G. (2013). Scientific analysis reveals major differences in the breast size of women in different countries. *The Journal of Female Health Sciences* **25**, 268–292.

Andersson, M. (1994). *Sexual Selection*, Edition (Volume **72**). Princeton University Press, Princeton.

Andrews, T. M., Lukaszewski, A. W., Simmons, Z. L. & Bleske-Rechek, A. (2017). Cue-based estimates of reproductive value explain women's body attractiveness. *Evolution and Human Behavior* **38**(4), 461–467.

Arbour, M. & Kessler, J. (2013). Mammary hypoplasia: not every breast can produce sufficient milk. *Journal of Midwifery & Women's Health* **58**(4), 457–461.

Austad, S. N. & Hoffman, J. M. (2018). Is antagonistic pleiotropy ubiquitous in aging biology? *Evolution, Medicine, and Public Health* **2018**(1), 287–294.

Baker, R. & Bellis, M. (1995). *Human Sperm Competition: Copulation, Masturbation and Infidelity*. Chapman & Hall, London.

Baniel, A., Cowlishaw, G. & Huchard, E. (2016). Stability and strength of male-female associations in a promiscuous primate society. *Behavioral Ecology and Sociobiology* **70**(5), 761–775.

Behringer, V., Hohmann, G., Stevens, J. M., Weltring, A. & Deschner, T. (2012). Adrenarche in bonobos (*Pan paniscus*): evidence from ontogenetic changes in urinary dehydroepiandrosterone-sulfate levels. *Journal of Endocrinology* **214**(1), 55.

Benditte-Klepetko, H., Leisser, V., Paternostro-Sluga, T., Rakos, M., Trattnig, S., Helbich, T., Schemper, M. & Deutinger, M. (2007). Hypertrophy of the breast: a problem of beauty or health? *Journal of Women's Health* **16**(7), 1062–1069.

Benshoof, L. & Thornhill, R. (1979). The evolution of monogamy and concealed ovulation in humans. *Journal of Social and Biological Structures* **2**(2), 95–106.

Blevins, J. K., Coxworth, J. E., Herndon, J. G. & Hawkes, K. (2013). Brief communication: adrenal androgens and aging: female chimpanzees (*Pan troglodytes*) compared with women. *American Journal of Physical Anthropology* **151**(4), 643–648.

Böhme, M., Spassov, N., Fuss, J., Tröscher, A., Deane, A. S., Prieto, J., Kirscher, U., Lechner, T. & Begun, D. R. (2019). A new Miocene ape and locomotion in the ancestor of great apes and humans. *Nature* **575**(7783), 489–493.

Brooks, R., Shelly, J., Jordan, L. & Dixson, B. (2015). The multivariate evolution of female body shape in an artificial digital ecosystem. *Evolution and Human Behavior* **36**(5), 351–358.

Brown, K., Akhtar, N., Robertson, A. & Ahmed, M. (1986). Lactational capacity of marginally nourished mothers: relationships between maternal nutritional status and quantity and proximate composition of milk. *Pediatrics* **78**(5), 909–919.

Brown, N. & Scurr, J. (2016a). 'Breasts are getting bigger'. Where is the evidence? *Journal of Anthropological Sciences* **94**, 1–8.

Brown, N. & Scurr, J. (2016b). Do women with smaller breasts perform better in long-distance running? *European Journal of Sport Science* **16**(8), 965–971.

Brown, N., White, J., Milligan, A., Risius, D., Ayres, B., Hedger, W. & Scurr, J. (2012). The relationship between breast size and anthropometric characteristics. *American Journal of Human Biology* **24**(2), 158–164.

Brunnhuber, S. & Kirchengast, S. (2002). Use of the oral contraceptive pill by Austrian adolescents with emphasis on the age of onset, side effects, compliance and lifestyle. *Collegium Antropologicum* **26**(2), 467–475.

Burgin, C., Colella, J., Kahn, P. & Upham, N. (2018). How many species of mammals are there? *Journal of Mammalogy* **99**(1), 1–14.

Burt, A. (1992). 'Concealed ovulation' and sexual signals in primates. *Folia Primatologica* **58**(1), 1–6.

Campbell, B. (2020). DHEAS and human development: an evolutionary perspective. *Frontiers in Endocrinology* **11**, 101.

Cant, J. (1981). Hypothesis for the evolution of human breasts and buttocks. *The American Naturalist* **117**(2), 199–204.

Caro, T. (1987). Human breasts: unsupported hypotheses reviewed. *Human Evolution* **2**(3), 271–282.

Caro, T. M. & Sellen, D. W. (1990). The reproductive advantages of fat in women. *Ethology and Sociobiology* **11**(1), 51–66.

Clemons, M. & Goss, P. (2001). Estrogen and the risk of breast cancer. *New England Journal of Medicine* **344**(4), 276–285.

Cline, J. (2007). Assessing the mammary gland of nonhuman primates: effects of endogenous hormones and exogenous hormonal agents and growth factors. *Birth Defects Research Part B* **80**(2), 126–146.

Cline, J. & Wood, C. E. (2008). The mammary glands of macaques. *Toxicologic Pathology* **36**, 130S–141S.

Coltman, C., Steele, J. & McGhee, D. (2017). Breast volume is affected by body mass index but not age. *Ergonomics* **60**(11), 1576–1585.

Coltman, C., Steele, J. & McGhee, D. (2018). Effects of age and body mass index on breast characteristics: a cluster analysis. *Ergonomics* **61**(9), 1232–1245.

Cox, D., Kent, J., Casey, T., Owens, R. & Hartmann, P. (1999). Breast growth and the urinary excretion of lactose during human pregnancy and early lactation: endocrine relationships. *Experimental Physiology* **84**(2), 421–434.

Dixson, B., Duncan, M. & Dixson, A. (2015). The role of breast size and areolar pigmentation in perceptions of women's sexual attractiveness, reproductive health, sexual maturity, maternal nurturing abilities, and age. *Archives of Sexual Behavior* **44**(6), 1685–1695.

Dixson, B., Grimshaw, G., Linklater, W. & Dixson, A. F. (2011a). Eye tracking of men's preferences for female breast size and areola pigmentation. *Archives of Sexual Behavior* **40**(1), 51–58.

Dixson, B., Vasey, P., Sagata, K., Sibanda, N., Linklater, W. & Dixson, A. (2011b). Men's preferences for women's breast morphology in New Zealand, Samoa, and Papua New Guinea. *Archives of Sexual Behavior* **40**(6), 1271–1279.

Drife, J. (1986). Breast development in puberty. *Annals of the New York Academy of Sciences* **464**(1), 58–65.

Edes, A. (2017). Dehydroepiandrosterone-sulfate (DHEA-S), sex, and age in zoo-housed western lowland gorillas (*Gorilla gorilla gorilla*). *Primates* **58**(3), 385–392.

Edman, C., Aiman, E., Porter, J. & MacDonald, P. (1978). Identification of the estrogen product of extraglandular aromatization of plasma androstenedione. *American Journal of Obstetrics & Gynecology* **130**(4), 439–447.

Einon, D. (2007). The shaping of women's bodies: men's choice of fertility or heat stress avoidance? In *The Body Beautiful*, pp. 131–158. Palgrave Macmillan, London.

Eriksson, N., Benton, G., Do, C., Kiefer, A., Mountain, J., Hinds, D., Francke, U. & Tung, J. Y. (2012). Genetic variants associated with breast size also influence breast cancer risk. *BMC Medical Genetics* **13**(1), 53.

Ferring, R., Oms, O., Agustí, J., Berna, F., Nioradze, M., Shelia, T., Tappen, M., Vekua, A., Zhvania, D. & Lordkipanidze, D. (2011). Earliest human occupations at Dmanisi (Georgian Caucasus) dated to 1.85–1.78 Ma. *Proceedings of the National Academy of Sciences of the United States of America* **108**(26), 10432–10436.

Fitzpatrick, C. & Servedio, M. (2018). The evolution of male mate choice and female ornamentation: a review of mathematical models. *Current Zoology* **64**(3), 323–333.

Ford, C. & Beach, F. (1951). *Patterns of Sexual Behaviour*. Harper & Row, New York.

Frisancho, A. & Housh, C. (1988). The relationship of maturity rate to body size and body proportions in children and adults. *Human Biology* **1988**, 759–770.

Furnham, A., Dias, M. & McClelland, A. (1998). The role of body weight, waist-to-hip ratio, and breast size in judgments of female attractiveness. *Sex Roles* **39**(3–4), 311–326.

Furnham, A. & Swami, V. (2007). Perception of female buttocks and breast size in profile. *Social Behavior and Personality* **35**(1), 1–8.

Furnham, A., Swami, V. & Shah, K. (2006). Body weight, waist-to-hip ratio and breast size correlates of ratings of attractiveness and health. *Personality and Individual Differences* **41**(3), 443–454.

Gallup, G. Jr. (1982). Permanent breast enlargement in human females: a sociobiological analysis. *Journal of Human Evolution* **11**(7), 597–601.

Ganfornina, M. & Sánchez, D. (1999). Generation of evolutionary novelty by functional shift. *BioEssays* **21**(5), 432–439.

Gaskins, A., Rich-Edwards, J., Missmer, S., Rosner, B. & Chavarro, J. (2015). Association of fecundity with changes in adult female weight. *Obstetrics and Gynecology* **126**(4), 850.

Geddes, D. (2007). Inside the lactating breast: the latest anatomy research. *Journal of Midwifery & Women's Health* **52**(6), 556–563.

Golub, M., Hogrefe, C., Germann, S., Lasley, B., Natarajan, K. & Tarantal, A. (2003). Effects of exogenous estrogenic agents on pubertal growth and reproductive system maturation in female rhesus monkeys. *Toxicological Sciences* **74**(1), 103–113.

Gould, K., Flint, M. & Graham, C. (1981). Chimpanzee reproductive senescence: a possible model for evolution of the menopause. *Maturitas* **3**(2), 157–166.

Greaves, R. F., Wudy, S. A., Badoer, E., Zacharin, M., Hirst, J. J., Quinn, T. & Walker, D. W. (2019). A tale of two steroids: the importance of the androgens DHEA and DHEAS for early neurodevelopment. *The Journal of Steroid Biochemistry and Molecular Biology* **188**, 77–85.

Greydanus, D., Parks, D. & Farrell, E. (1989). Breast disorders in children and adolescents. *Pediatric Clinics of North America* **36**(3), 601–638.

Grillot, R., Simmons, Z., Lukaszewski, A. & Roney, J. R. (2014). Hormonal and morphological predictors of women's body attractiveness. *Evolution and Human Behavior* **35**(3), 176–183.

Grossl, N. A. (2000). Supernumerary breast tissue: historical perspectives and clinical features. *Southern Medical Journal* **93**(1), 29–32.

Groyecka, A., Żelaźniewicz, A., Misiak, M., Karwowski, M. & Sorokowski, P. (2017). Breast shape (ptosis) as a marker of a woman's breast attractiveness and age: evidence from Poland and Papua. *American Journal of Human Biology* **29**(4), e22981.

Guéguen, N. (2007). Bust size and hitchhiking: a field study. *Perceptual and Motor Skills* **105**, 1294–1298.

Harlow, H. F. & Suomi, S. J. (1970). Nature of love: simplified. *American Psychologist* **25**(2), 161.

Havlíček, J., Třebický, V., Valentova, J. V., Kleisner, K., Akoko, R. M., Fialová, J., Jash, R., Kocnar, T., Pereira, K. J., Sterbova, Z., Varella, M. A., Vokurkova, J., Vunan, E. & Roberts, C. (2017). Men's preferences for women's breast size and shape in four cultures. *Evolution and Human Behavior* **38**(2), 217–226.

Hill, P., Wynder, E. L., Garbaczewski, L., Helman, P., Hill, M., Sporangisa, J. & Huskisson, J. (1980). Diet and menarche in different ethnic groups. *European Journal of Cancer* **16**(4), 519–525.

Hobson, W., Coulston, F., Faiman, C., Winter, J. S. & Reyes, F. (1976). Reproductive endocrinology of female chimpanzees: a suitable model of humans. *Journal of Toxicology and Environmental Health* **1**(4), 657–668.

Howard, B. A. & Gusterson, B. A. (2000). Human breast development. *Journal of Mammary Gland Biology and Neoplasia* **5**(2), 119–137.

Horvath, T. (1981). Physical attractiveness: the influence of selected torso parameters. *Archives of Sexual Behavior* **10**(1), 21–24.

Hussain, Z., Roberts, N., Whitehouse, G. H., Garcia-Finana, M. & Percy, D. (1999). Estimation of breast volume and its variation during the menstrual cycle using MRI and stereology. *The British Journal of Radiology* **72**(855), 236–245.

Hytten, F. E. & Leitch, I. (1971). *The Physiology of Human Pregnancy*, Second Edition. Blackwell, Oxford.

James, V., McNeill, J., Beranek, P., Bonney, R. & Reed, M. J. (1986). The role of tissue steroids in regulating aromatase and oestradiol 17β-hydroxysteroid dehydrogenase activities in breast and endometrial cancer. *Journal of Steroid Biochemistry* **25**(5), 787–790.

Jansen, L. A., Backstein, R. M. & Brown, M. H. (2014). Breast size and breast cancer: a systematic review. *Journal of Plastic, Reconstructive & Aesthetic Surgery* **67**(12), 1615–1623.

Jasieńska, G., Ziomkiewicz, A., Ellison, P. T., Lipson, S. F. & Thune, I. (2004). Large breasts and narrow waists indicate high reproductive potential in women. *Proceedings of the Royal Society of London. Series B: Biological Sciences* **271**(1545), 1213–1217.

Jemström, H. & Olsson, H. (1997). Breast size in relation to endogenous hormone levels, body constitution, and oral contraceptive use in healthy nulligravid women aged 19–25 years. *American Journal of Epidemiology* **145**(7), 571–580.

Joyner, J., Hutley, L. & Cameron, D. P. (2001). Estrogen receptors in human preadipocytes. *Endocrine* **15**(2), 225–230.

Kaplowitz, P. B. (2008). Link between body fat and the timing of puberty. *Pediatrics* **121**(3), S208–S217.

Katch, V. L., Campaigne, B., Freedson, P., Sady, S., Katch, F. I. & Behnke, A. R. (1980). Contribution of breast volume and weight to body fat distribution in females. *American Journal of Physical Anthropology* **53**(1), 93–100.

Kleinke, C. L. & Staneski, R. A. (1980). First impressions of female bust size. *The Journal of Social Psychology* **110**(1), 123–134.

Kościński, K. (2019). Breast firmness is of greater importance for women's attractiveness than breast size. *American Journal of Human Biology* **31**(5), e23287.

Kościński, K., Makarewicz, R. & Bartoszewicz, Z. (2020). Stereotypical and actual associations of breast size with mating-relevant traits. *Archives of Sexual Behavior* **49**(3), 821–836.

Kuzawa, C. W. (1998). Adipose tissue in human infancy and childhood: an evolutionary perspective. *American Journal of Physical Anthropology* **107**(S27), 177–209.

Kuzawa, C. W., Chugani, H. T., Grossman, L. I., Lipovich, L., Muzik, O., Hof, P. R., Wildman, D. E., Sherwood, C. C., Leonard, W. R. & Lange, N. (2014). Metabolic costs and evolutionary implications of human brain development. *Proceedings of the National Academy of Sciences of the United States of America* **111**(36), 13010–13015.

Labrie, F. (1991). Intracrinology. *Molecular and Cellular Endocrinology* **78**(3), C113–C118.

Labrie, F., Bélanger, A., Luu-The, V., Labrie, C., Simard, J., Cusan, L., Gomez, J.-L. & Candas, B. (1998). DHEA and the intracrine formation of androgens and estrogens in peripheral target tissues: its role during aging. *Steroids* **63**(5–6), 322–328.

LaMarca, H. & Rosen, J. (2008). Hormones and mammary cell fate-what will I become when I grow up? *Endocrinology* **149**(9), 4317–4321.

Lassek, W. D. & Gaulin, S. J. C. (2006). Changes in body fat distribution in relation to parity in American women: a covert form of maternal depletion. *American Journal of Physical Anthropology* **131**(2), 295–302.

LeBlanc, S. & Barnes, E. (1974). On the adaptive significance of the female breast. *The American Naturalist* **108**(962), 577–578.

Li, J., Foo, J., Schoof, N., Varghese, J., Fernandez-Navarro, P., Gierach, G. L., Quek, S. T., Hartman, M., Nord, S., Kristensen, V. N., Pollan, M., Figueroa, J. D., Thompson, D. J., Li, Y., Khor, C. C., et al. (2013). Large-scale genotyping identifies a new locus at 22q13. 2 associated with female breast size. *Journal of Medical Genetics* **50**(10), 666–673.

Lipson, S. F. & Ellison, P. T. (1996). Comparison of salivary steroid profiles in naturally occurring conception and non-conception cycles. *Human Reproduction* **11** (10), 2090–2096.

Liu, L., Kang, J., Ding, X., Chen, D., Zhou, Y. & Ma, H. (2015). Dehydroepiandrosterone-regulated testosterone biosynthesis via activation of the ERK1/2 signaling pathway in primary rat Leydig cells. *Cellular Physiology and Biochemistry* **36**(5), 1778–1792.

Lou, D. I., McBee, R. M., Le, U. Q., Stone, A. C., Wilkerson, G., Demogines, A. M. & Sawyer, S. L. (2014). Rapid evolution of *brca1* and *brca2* in humans and other primates. *BMC Evolutionary Biology* **14**(1), 155.

Loukas, M., Clarke, P. & Tubbs, R. S. (2007). Accessory breasts: a historical and current perspective. *The American Surgeon* **73**(5), 525–528.

Lovejoy, C. (1981). The origin of man. *Science* **211**(4480), 341–350.

Low, B. S. (1979). Sexual selection and human ornamentation. In *Evolutionary Biology and Human Social Behavior* (eds N. A. Chagnon and W. Irons), pp. 462–487. Duxbury, North Scituate.

Low, B. S., Alexander, R. D. & Noonan, K. M. (1987). Human hips, breasts and buttocks: is fat deceptive? *Ethology and Sociobiology* **8**, 249–257.

Macadam, P. & Dettwyler, K. (eds) (1995). *Breastfeeding: Biocultural Perspectives*. Transaction Publishers, New York.

Manning, J., Scutt, D., Whitehouse, G. & Leinster, S. (1997). Breast asymmetry and phenotypic quality in women. *Evolution and Human Behavior* **18**(4), 223–236.

Manolopoulos, K., Karpe, F. & Frayn, K. (2010). Gluteofemoral body fat as a determinant of metabolic health. *International Journal of Obesity* **34**(6), 949–959.

Markkula, A., Bromée, A., Henningson, M., Hietala, M., Ringberg, A., Ingvar, C., Rose, C. & Jernstrom, H. (2012). Given breast cancer, does breast size matter? Data from a prospective breast cancer cohort. *Cancer Causes & Control* **23**(8), 1307–1316.

Marlowe, F. (1998). The nubility hypothesis. *Human Nature* **9**(3), 263–271.

Marlowe, F. (2007). Hunting and gathering: the human sexual division of foraging labor. *Cross-Cultural Research* **41**(2), 170–195.

Mascia-Lees, F., Relethford, J. & Sorger, T. (1986). Evolutionary perspectives on permanent breast enlargement in human females. *American Anthropologist* **88**(2), 423–428.

Mayes, J. S. & Watson, G. H. (2004). Direct effects of sex steroid hormones on adipose tissues and obesity. *Obesity Reviews* **5**(4), 197–216.

McGhee, D., Coltman, K., Riddiford-Harland, D. & Steele, J. (2018). Upper torso pain and musculoskeletal structure and function in women with and without large breasts: a cross sectional study. *Clinical Biomechanics* **51**, 99–104.

Miki, Y., Swensen, J., Shattuck-Eidens, D., Futreal, P. A., Harshman, K., Tavtigian, S., Liu, Q., Cochran, C., Bennett, L. M. & Ding, W. (1994). A strong candidate for the breast and ovarian cancer susceptibility gene *BRCA1*. *Science* **266**(5182), 66–71.

Morgan, E. (1972). *The Descent of Woman*. Bantam Books, New York.

Morris, D. (1967). *The Naked Ape*. McGraw-Hill, New York.

Nadler, R., Graham, C., Gosselin, R. & Collins, D. (1985). Serum levels of gonadotropins and gonadal steroids, including testosterone, during the menstrual cycle of the chimpanzee (*Pan troglodytes*). *American Journal of Primatology* **9**(4), 273–284.

Neifert, M., Seacat, J. & Jobe, W. (1985). Lactation failure due to insufficient glandular development of the breast. *Pediatrics* **76**(5), 823–828.

Nookaew, I., Svensson, P., Jacobson, P., Jernås, M., Taube, M., Larsson, I., Andersson-Assarsson, J. C., Sjostrom, L., Froguel, P., Walley, A., Nielsen, J. & Carlsson, L. M. S. (2013). Adipose tissue resting energy expenditure and expression of genes involved in mitochondrial function are higher in women than in men. *The Journal of Clinical Endocrinology & Metabolism* **98**(2), E370–E378.

Ooi, B., Loh, H., Ho, P. J., Milne, R., Giles, G., Gao, C., Kraft, P., John, E. M., Swerdlow, A., Brenner, H., Wu, A. H., Haiman, C., Evans, G. D., Zheng, W., Fasching, P. A., et al. (2019). The genetic interplay between body mass index, breast size and breast cancer risk: a Mendelian randomization analysis. *International Journal of Epidemiology* **48**(3), 781–794.

O'Sullivan, A. (2009). Does oestrogen allow women to store fat more efficiently? A biological advantage for fertility and gestation. *Obesity Reviews* **10**(2), 168–177.

O'Sullivan, A., Hoffman, D. & Ho, K. (1995). Estrogen, lipid oxidation, and body fat. *New England Journal of Medicine* **333**(10), 669–670.

Ozasa, H. & Gould, K. (1982). Demonstration and characterization of estrogen receptor in chimpanzee sex skin: correlation between nuclear receptor levels and degree of swelling. *Endocrinology* **111**(1), 125–131.

Pawłowski, B. (1998). Why are human newborns so big and fat? *Human Evolution* **13**, 65–72.

Pawłowski, B. (1999a). Loss of oestrus and concealed ovulation in human evolution: the case against the sexual-selection hypothesis. *Current Anthropology* **40**(3), 257–276.

Pawłowski, B. (1999b). Permanent breasts as a side effect of subcutaneous fat tissue increase in human evolution. *Homo - Journal of Comparative Human Biology* **50**(2), 149–162.

Peccei, J. (1995). A hypothesis for the origin and evolution of menopause. *Maturitas* **21** (2), 83–89.

Pedersen, S., Bruun, J., Hube, F., Kristensen, K., Hauner, H. & Richelsen, B. (2001). Demonstration of estrogen receptor subtypes α and β in human adipose tissue: influences of adipose cell differentiation and fat depot localization. *Molecular and Cellular Endocrinology* **182**(1), 27–37.

Perez-Escamilla, R., Cohen, R., Brown, K., Rivera, L., Canahuati, J. & Dewey, K. (1995). Maternal anthropometric status and lactation performance in a low-income Honduran population: evidence for the role of infants. *The American Journal of Clinical Nutrition* **61**(3), 528–534.

Pfeffer, C. M., Ho, B. N. & Singh, A. T. (2017). The evolution, functions and applications of the breast cancer genes *BRCA1* and *BRCA2*. *Cancer Genomics - Proteomics* **14**(5), 293–298.

Pomiankowski, A. & Møller, A. (1995). A resolution of the lek paradox. *Proceedings of the Royal Society of London. Series B: Biological Sciences* **260**(1357), 21–29.

Pond, C. (1997). The biological origins of adipose tissue in humans. In *The Evolving Female: A Life History Perspective* (eds M. E. Morbeck, A. Galloway and A. Zihlman), pp. 147–162. Princeton University Press, Princeton.

Powe, C., Knott, C. & Conklin-Brittain, N. (2010). Infant sex predicts breast milk energy content. *American Journal of Human Biology* **22**(1), 50–54.

Prentice, A., Prentice, A. & Whitehead, R. (1981). Breast-milk fat concentrations of rural African women: 2. Long-term variations within a community. *British Journal of Nutrition* **45**(3), 495–503.

Prum, R. (2012). Aesthetic evolution by mate choice: Darwin's really dangerous idea. *Philosophical Transactions of the Royal Society B: Biological Sciences* **367**(1600), 2253–2265.

Qiao, Q., Zhou, G. & Ling, Y. (1997). Breast volume measurement in young Chinese women and clinical applications. *Aesthetic Plastic Surgery* **21**(5), 362–368.
Quinn, E., Largado, F., Power, M. & Kuzawa, C. (2012). Predictors of breast milk macronutrient composition in Filipino mothers. *American Journal of Human Biology* **24**(4), 533–540.
Rasmussen, K. (2007). Association of maternal obesity before conception with poor lactation performance. *Annual Review of Nutrition* **27**, 103–121.
Rauh, C., Faschingbauer, F., Haeberle, L., Jud, S., Heusinger, K., Fasching, P., Goecke, T. W., Rajakaruna, N., Voigt, F., Bani, M. R., Lux, M. P., Renner, S. P., Loehberg, C. R., Hartmann, A., Schulz-Wendtland, R., et al. (2013). Factors influencing breast changes after pregnancy. *European Journal of Cancer Prevention* **22**(3), 259–261.
Rebuffe-Scrive, M., Enk, L., Crona, N., Lönnroth, P., Abrahamsson, L., Smith, U. & Björntorp, P. (1985). Fat cell metabolism in different regions in women. Effect of menstrual cycle, pregnancy, and lactation. *The Journal of Clinical Investigation* **75**(6), 1973–1976.
Reed, D., Light, J., Allen, J. & Kirchman, J. (2007). Pair of lice lost or parasites regained: the evolutionary history of anthropoid primate lice. *BMC Biology* **5**(1), 7.
Richelsen, B., Pedersen, S., Møller-Pedersen, T. & Bak, J. (1991). Regional differences in triglyceride breakdown in human adipose tissue: effects of catecholamines, insulin, and prostaglandin E2. *Metabolism* **40**(9), 990–996.
Rinker, B., Veneracion, M. & Walsh, C. (2010). Breast ptosis: causes and cure. *Annals of Plastic Surgery* **64**(5), 579–584.
Rogers, I. S., Gunnell, D., Emmett, P. M., Glynn, L. R., Dunger, D. B. & Holly, J. M. (2005). Cross-sectional associations of diet and insulin-like growth factor levels in 7-to 8-year-old children. *Cancer Epidemiology and Prevention Biomarkers* **14**(1), 204–212.
Rogers, A., Iltis, D. & Wooding, S. (2004). Genetic variation at the MC1R locus and the time since loss of human body hair. *Current Anthropology* **45**(1), 105–108.
Russo, J. & Russo, I. (2004). Development of the human breast. *Maturitas* **49**(1), 2–15.
Sabbi, K. H., Muller, M. N., Machanda, Z. P., Otali, E., Fox, S. A., Wrangham, R. W. & Emery Thompson, M. (2019). Human-like adrenal development in wild chimpanzees: a longitudinal study of urinary dehydroepiandrosterone-sulfate and cortisol. *American Journal of Primatology* **82**(11), e23064.
Schinkel-Ivy, A. & Drake, J. (2016). Breast size impacts spine motion and postural muscle activation. *Journal of Back and Musculoskeletal Rehabilitation* **29**(4), 741–748.
Schultz, A. (1969). *The Life of the Primates*. Universe Books, New York.
Scutt, D., Manning, J., Whitehouse, G., Leinster, S. & Massey, C. (1997). The relationship between breast asymmetry, breast size and the occurrence of breast cancer. *The British Journal of Radiology* **70**(838), 1017–1021.
Sher, E., Migeon, C. & Berkovitz, G. (1998). Evaluation of boys with marked breast development at puberty. *Clinical Pediatrics* **37**(6), 367–371.
Sineshaw, H., Gaudet, M., Ward, E., Flanders, W., Desantis, C., Lin, C. & Jemal, A. (2014). Association of race/ethnicity, socioeconomic status, and breast cancer subtypes in the National Cancer Data Base (2010–2011). *Breast Cancer Research and Treatment* **145**(3), 753–763.
Smith, J. & Harper, D. (2003). *Animal Signals*. Oxford University Press, Oxford.
Smith, N. (1986). Psychology and evolution of breasts. *Human Evolution* **1**, 285–286.
Smith, R. (1984). *Sperm Competition and the Evolution of Animal Mating Systems*. Academic Press, New York.
Soguel, L., Durocher, F., Tchernof, A. & Diorio, C. (2017). Adiposity, breast density, and breast cancer risk: epidemiological and biological considerations. *European Journal of Cancer Prevention* **26**(6), 511.
Soltanian, H., Liu, M., Cash, A. & Iglesias, R. (2012). Determinants of breast appearance and aging in identical twins. *Aesthetic Surgery Journal* **32**(7), 846–860.
Steele, J., Coltman, C. & McGhee, D. (2020). Effects of obesity on breast size, thoracic spine structure and function, upper torso musculoskeletal pain and physical activity in women. *Journal of Sport and Health Science* **9**(2), 140–148.
Sternlicht, M., Kouros-Mehr, H., Lu, P. & Werb, Z. (2006). Hormonal and local control of mammary branching morphogenesis. *Differentiation* **74**(7), 365–381.
Stewart-Williams, S. & Thomas, A. (2013). The ape that thought it was a peacock: does evolutionary psychology exaggerate human sex differences? *Psychological Inquiry* **24**(3), 137–168.
Swami, V., Jones, J., Einon, D. & Furnham, A. (2009). Men's preferences for women's profile waist-to-hip ratio, breast size, and ethnic group in Britain and South Africa. *British Journal of Psychology* **100**(2), 313–325.
Swami, V. & Tovée, M. (2013). Resource security impacts men's female breast size preferences. *PLoS One* **8**(3), e57623.
Symons, D. (1979). *The Evolution of Human Sexuality*. Oxford University Press, New York.
Tantleff-Dunn, S. (2002). Biggest isn't always best: the effect of breast size on perceptions of women. *Journal of Applied Social Psychology* **32**(11), 2253–2265.
Tavtigian, S., Simard, J., Rommens, J., Couch, F., Shattuck-Eidens, D., Neuhausen, S., Merajver, S., Thorlacius, S., Offit, K., Stoppa-Lyonnet, D., Belanger, C., Bell, R., Berry, S., Bogdan, R., Chen, Q., et al. (1996). The complete *BRCA2* gene and mutations in chromosome 13q-linked kindreds. *Nature Genetics* **12**(3), 333–337.
Thiruchelvam, P., Walker, J., Rose, K., Lewis, J. & Al-Mufti, R. (2016). Gynaecomastia. *BMJ* **354**, i4833.
Thijssen, J., Blankenstein, M., Daroszewski, J. & Milewicz, A. (1990). Steroids in normal and neoplastic breast tissues. *Annals of the New York Academy of Sciences* **595**(1), 222–226.
Thornhill, R. & Grammer, K. (1999). The body and face of woman: one ornament that signals quality? *Evolution and Human Behavior* **20**(2), 105–120.
Treilleux, I., Peloux, N., Brown, M. & Sergeant, A. (1997). Human estrogen receptor (ER) gene promoter-P1: estradiol-independent activity and estradiol inducibility in ER+ and ER− cells. *Molecular Endocrinology* **11**(9), 1319–1331.
True, J. & Carroll, S. (2002). Gene co-option in physiological and morphological evolution. *Annual Review of Cell and Developmental Biology* **18**(1), 53–80.
Uranga, A., Levine, J. & Jensen, M. (2005). Isotope tracer measures of meal fatty acid metabolism: reproducibility and effects of the menstrual cycle. *American Journal of Physiology-Endocrinology and Metabolism* **288**(3), E547–E555.
Valentova, J., Bártová, K., Štěrbová, Z. & Varella, M. (2017). Influence of sexual orientation, population, homogamy, and imprinting-like effect on preferences and choices for female buttock size, breast size and shape, and WHR. *Personality and Individual Differences* **104**, 313–319.
Vandeweyer, E. & Hertens, D. (2002). Quantification of glands and fat in breast tissue: an experimental determination. *Annals of Anatomy - Anatomischer Anzeiger* **184** (2), 181–184.
van Pelt, R., Gavin, K. & Kohrt, W. (2015). Regulation of body composition and bioenergetics by estrogens. *Endocrinology and Metabolism Clinics* **44**(3), 663–676.
van Schaik, C. P. & Dunbar, R. I. (1990). The evolution of monogamy in large primates: a new hypothesis and some crucial tests. *Behaviour* **115**(1–2), 30–61.
Veldhuis, J., Roemmich, J., Richmond, E., Rogol, A., Lovejoy, J., Sheffield-Moore, M., Mauras, N. & Bowers, C. Y. (2005). Endocrine control of body composition in infancy, childhood, and puberty. *Endocrine Reviews* **26**(1), 114–146.
Wade, T., Zhu, G. & Martin, N. (2010). Body mass index and breast size in women: same or different genes? *Twin Research and Human Genetics* **13**(5), 450–454.
Wagner, E., Chantry, C., Dewey, K. & Nommsen-Rivers, L. (2013). Breastfeeding concerns at 3 and 7 days postpartum and feeding status at 2 months. *Pediatrics* **132**(4), e865–e875.
Wells, J. (2010). *The Evolutionary Biology of Human Body Fatness: Thrift and Control*, Edition (Volume **58**). Cambridge University Press, Cambridge.
Woodward, K. & Richards, M. H. (2005). The parental investment model and minimum mate choice criteria in humans. *Behavioral Ecology* **16**(1), 57–61.
Yu, Z., Han, S., Zhu, J., Sun, X., Ji, C. & Guo, X. (2013). Pre-pregnancy body mass index in relation to infant birth weight and offspring overweight/obesity: a systematic review and meta-analysis. *PLoS One* **8**(4), e61627.
Zihlman, A. L. & Bolter, D. R. (2015). Body composition in *Pan paniscus* compared with *Homo sapiens* has implications for changes during human evolution. *Proceedings of the National Academy of Sciences of the United States of America* **112**(24), 7466–7471.
Żelaźniewicz, A. & Pawlowski, B. (2011). Female breast size attractiveness for men as a function of sociosexual orientation (restricted vs. unrestricted). *Archives of Sexual Behavior* **40**(6), 1129–1135.
Żelaźniewicz, A. & Pawłowski, B. (2019). Maternal breast volume in pregnancy and lactation capacity. *American Journal of Physical Anthropology* **168**(1), 180–189.

(*Received 6 May 2020; revised and accepted 2 July 2021*)