Introduction

Humans do many things anthropologists characterize as unique, when really what we mean is that we do a more extreme version of what other primates, mammals, or social animals are also doing. Similarly, we are incentivized to have unique hypotheses. Much ado has been made about which hypothesis for bipedality, big brains, or other elements of the hominin lineage is the “prime mover,” meaning it is the main hypothesis explaining a particular adaptation. The idea of a prime mover hypothesis itself derives from colonial interpretations of science and coloniality: That scientists are discoverers and pioneers who overpower nature to reveal its mystery, knowledge is hierarchical, and there can only be one right answer (Quijano 2000; Smith 2012). The desire for a prime mover hypothesis has pervaded the study of the evolution of human menstruation: Though we are not the only animals to menstruate, we must somehow be unique and rank-ordered above other animals, and there must be only one way to understand why we menstruate.

Part of the coloniality of scientific discourse rank orders women below men (Quijano 2000) which complicates our ability to produce prime mover hypotheses that involve physiological processes experienced more by women. For many years, the prime mover hypothesis for why human menstruation evolved has been that it did not evolve. Or rather, menstruation was seen as a “non-adaptive consequence” of the adaptations endemic to endometrial cycling. Functional, non-adaptive hypotheses for phenomena that are gendered female may gain prominence because they strip agency from the bearers of these traits. As an example, the grandmother hypothesis situates grandmothers as agents of their reproductive success, providing knowledge, provisioning with food, or providing other means of care to their offspring’s (usually daughter’s) children (Hawkes 2003; Gibson and Mace 2005; Sear, Mace, and McGregor 2003; Scelza and Hinde 2019; see also Block in this volume). Other hypotheses provide functional counternarratives (e.g., grandmothering is a non-adaptive consequence of ovarian aging [Peccei 2001]) or counterexamples of bad grandmothers (e.g., correlative demographic research on infant mortality and maternal/paternal grandmother presence [Johow et al. 2011; Voland and Beise 2002]).

A growing number of hypotheses have been proposed for why human menstruation evolved, and several of them offer biologically coherent narratives of agency. In this chapter, we will describe the biological traits we share with other mammals and, taken in combination within
our particular phylogenetic history, also make human menstruation a more copious version than any other in the animal record. We will describe the history of the study of human menstruation, to help us understand why the prime mover hypothesis favorable to so many was that it is a functionless byproduct. Newer research in the past decade shows us how crucial menstruation is to the suite of processes that make up endometrial cycling. Then, we compare the rising prominence of our understanding of the adaptive nature of menstruation, to the waning prominence of our understanding of the adaptive nature of the timing of menarche (see Table 11.1 for a glossary of terms appearing in *italics* in this chapter). Our shifting understanding of menstruation across many axes demonstrates the value of inclusive and justice-minded science with diverse viewpoints that acknowledges and interrogates historical and current colonial framings.

### The complex of traits that make human menstruation interesting

Humans are quite similar to their mammalian relatives when it comes to reproduction. Many reproductive traits are shared, including lactation, internal fertilization, and birthing live young. However, humans have a unique, and to some extent extreme, manifestation of certain other elements of mammalian reproduction: We have spontaneous ovulation, an especially invasive trophoblast, and overlapping dependent offspring requiring cooperative breeding. Additionally, where most mammals do not menstruate, humans do.

Menstruation is defined by the shedding of blood and tissue from the endometrium through the vaginal canal, and it varies in terms of frequency, duration, and experience within and between humans and with other menstruating species. Non-menstruating mammals often do grow an endometrial lining, but this lining is resorbed rather than shed. In comparison, menstruating mammals, which include humans, a few non-human primates, the elephant shrew, and some species of bats and rodents, cyclically shed and excrete this endometrial lining (Bellofiore et al. 2017; Carter 2018). The thinking has been that the mechanism of endometrial repair

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**Table 11.1 Definitions of key terms used in the chapter**

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Adrenarche</td>
<td>Reactivation of the adrenal cortex</td>
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<tr>
<td>Anovulation</td>
<td>When no oocyte (egg) is released in a given menstrual cycle</td>
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<tr>
<td>Hemochorial placentation</td>
<td>A placentation type where the placenta is in direct contact with the fetus</td>
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<td>Interhemal barrier</td>
<td>Layer of trophoblast cells</td>
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<tr>
<td>Life history</td>
<td>Evolutionary framework to understand variation in life history traits like growth and development, reproductive maturation, reproductive events, and lifespan</td>
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<tr>
<td>Menarche; menarcheal age</td>
<td>Pubertal process: First menstruation; age at first menstruation</td>
</tr>
<tr>
<td>Menses; menstrual effluent</td>
<td>All the material secreted in menstruation, which includes blood, endometrial tissue, and other fluids</td>
</tr>
<tr>
<td>Ovulation</td>
<td>Release of oocyte (egg) from follicle</td>
</tr>
<tr>
<td>Pubarche</td>
<td>Pubertal process: Appearance of pubic and auxiliary hairs</td>
</tr>
<tr>
<td>Pubertal tempo</td>
<td>Rate at which one matures from adrenarche to menarche</td>
</tr>
<tr>
<td>Thelarche</td>
<td>Pubertal process: Onset of breast development</td>
</tr>
<tr>
<td>Trophoblast</td>
<td>Cells on the outer layer of the four-day-old blastocyst that become the placenta</td>
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Menstrual effluent

and regrowth is similar, but menstruating mammals produce more *menstrual effluent*. Why some mammals produce enough effluent that it needs to be excreted is a source of debate. Humans are by far the most copious of the menstruators, even when considering our relative body size, and so it is instructive to explore the ways in which menstruation appears to relate to some of the unique features of human reproduction.

One reason for our copious menstrual effluent may be related to spontaneous ovulation. Spontaneous ovulators ovulate no matter what: That is, their estrus or menstrual cycle is not driven by coitus or vaginal stimulation. At the same time, the term is also a bit of a misnomer as menstrual cycle characteristics are heavily determined by external energetic, immunological, and psychosocial stressors (e.g., Schliep et al. 2015; Jasienska and Ellison 2004; Clancy et al. 2013). Given adequate resources, ovulation will happen with some regularity within a healthy menstruator's lifetime distribution of menstrual cycles. Yet, up to a third of healthy, average-length menstrual cycles are anovulatory and still involve menstruation (Prior et al. 2015). Given this variation in *anovulation* and the effect of external stressors, both ovulation and endometrial function are processes bound by resource availability and external stressors, rather than vaginal stimulation and fertilization.

This means, provided adequate resources, menstruators will grow an endometrium lining and shed it. And we grow—and shed—a lot of endometrial tissue. Humans secrete more endometrial tissue compared to other menstruating non-human primates. Approximately 35–50 milliliters (mL) of blood and tissue is secreted each menstrual cycle, and about 10% of women secrete more than 80 mL (Warrilow et al. 2004). The reason for this thick lining appears to be, at least in part, our invasive trophoblast and therefore placenta. Placenta is deep across most menstruating primates (Brosens et al. 2009). Our particular brand of *hemochorial placentation* involves an invasive and temporary placental organ that develops during pregnancy and connects the developing fetus directly with maternal blood nutrients via the umbilical cord (Brosens et al. 2009). Hemochorial placentation is not unusual among mammals or even primates, but the particular invasiveness of human placentation is unique. The uniqueness of the human system comes from the reduction of the *interhemal barrier* between mother and fetus: By the third trimester, only a single-cell layer remains (Clancy 2009). Human gestation requires significant nutrient transfer; in the first trimester, this nutrient transfer comes from the endometrium, a conducive place to invade and create placental structures necessary for this extreme maternal–fetal contact. This quantity of necessary endometrium seems to simply be too much to resorb into the body, requiring that it be expressed as menstrual effluent after a non-conceptive cycle.

Menstruation manifests more extremely in humans compared even to other menstruating primates. We have costly singleton offspring we care for and support for several decades, which relies on cooperative breeding practices across nearly all human populations (Kramer 2005). Our processes for fecundity are sensitive to the amount of stress the body is negotiating, and we invest in expensive offspring with long gestations, big brains, and long periods of breastfeeding when we are able in the context of our environments. Therefore, menstruation is a mechanism of the menstrual cycling process that prioritizes context-dependent fecundity opportunities at times when costly reproduction may be more likely to be successful.

While there is largely consensus surrounding the mechanistic meaning of menstruation—that it is a process enabling the endometrium to regrow and repair tissue for a subsequent cycle—there is recent divergence in scholars’ thinking about its adaptive meaning. This divergence, as well as recent divergences about the meaning of first menstruation or *menarche*, show the ways that changing cultural conceptions of menstruation are allowing us to ask new questions about its causes, consequences, and contexts. Questioning its colonial origins and taking an explicit feminist biological stance for how we interpret the evolution of menstruation is a part
of how we observe, understand, and navigate issues of power in the broader field of the anthropology of reproduction (Ginsburg and Rapp 1991; Frost 2014). Therefore, next, we describe the historic and current shifts in how scientists make evolutionary meaning about menstruation, and the broader implications of these shifts. These shifts point to related changes in the way we ask questions, design studies, and interpret evidence about menstruation.

The evolutionary meaning of menstruation: Byproduct, or adaptive?

Early Western ideas about the meaning of menstruation theorized about its origins from a perspective tied to Christianity and medieval medicine. One view that persisted through the mid-twentieth century was that women contained toxins that must be cyclically purged; menstruation was the mechanism by which these toxins could be excreted (Macht and Lubin 1923). “Menotoxins” were secreted not only in *menses*, but also in saliva, sweat, and breastmilk and its release had additional consequences. For example, mothers’ menotoxins were hypothesized to cause children’s asthma and colic (Ashley-Montagu 1940; Perlstein and Matheson 1936). Menotoxin science reinforced cultural ideas about the uncleanness of the menstrual body and centered mothers’ bodies as places to lay blame for childhood illness. The idea of menotoxins finally fell out of favor in the late 1970s, yet menses continued to be described as dirt, debris, and waste (Martin 1989). These ideas had a lasting cultural legacy by nourishing the idea that menstruation is unclean, impure, or even dangerous.

A few decades later, several biologists proposed new hypotheses indicating menstruation serves a specific immunological purpose. Profet (1993) hypothesized it may function to remove sperm-borne pathogens, a reversal of the idea that menses is purging toxins endemic to the menstruator. Clarke (1994) further hypothesized it may expel inviable or abnormal embryos. Both hypotheses viewed menstruation as having a possible functional and adaptive feature. They helped shift views on the possible meanings of menstruation, but have not been supported over time. The timing of menstruation does not coincide with any increased risk for pathogenic exposure, making it unlikely to serve a unique function dispelling sperm-borne pathogens (Strassmann 1996). Further, each part of the reproductive tract has evolved its own unique immunological profile, which responds to changes in ovarian hormones (Wira et al. 2010), as well as protects against pathogens while allowing sperm to enter without being attacked by the immune system.

Another explanation hypothesizes that the growth and shedding cycle is more energetically economical compared to a hypothetical model where the endometrial lining is maintained indefinitely (Strassmann 1996). The estimation of energetic expenditure for reproductive traits like menstruation, pregnancy, and lactation is particularly useful for understanding the costs and benefits of reproductive investments within *life history* models (see also Kramer, Veile, and Henry in this volume). However, estimating the costs of menstruation for our ancestors can be difficult as we likely menstruate less frequently today than in our evolutionary history (Strassmann 1999). The lifetime number of menstrual cycles for a menstruator in our evolutionary past may be less than one-third of the number experienced by the modern menstruator in the United States (Strassmann 1999, 1997).

The potential energetic savings of menstruation is proposed to come via the comparative expense of maintaining a thick endometrial lining prepared for our especially invasive implantation. Throughout the follicular phase (from *menses* to ovulation), estrogen helps grow the functional layer of the endometrium into a thick lining. In the luteal phase (ovulation to *menses*), progesterone decidualizes (differentiates) the endometrium into a tissue with the kinds of nooks and crannies that allow for invasive implantation and nourishment of the trophoblast and then
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embryo before placentation is complete. Rather than maintain this tissue indefinitely, we slough, resorb, and excrete it, indicating this process of periodic endometrial renewal may be necessary to successful reproduction.

Energetics and energetic resources are of course directly linked to resource allocation, and thus the likelihood of conception and reproductive success. With greater energetic resources, women may produce a thicker endometrial lining, or perhaps maintain it for longer during the implantation window (the six to 12 days after ovulation during which embryo implantation can occur), compared to previous cycles (Clancy 2009; Clancy et al. 2009). In one study, women living in the Bolivian highlands were more likely to become pregnant in the cycle following a longer menstrual phase, indicating greater resources may have been devoted to reproduction generally and endometrial function specifically (Vitzthum et al. 2001). Similarly, in a Polish mountainous agrarian area, higher energetic expenditure in harvest seasons has been linked to lower concentrations of progesterone, the hormone maintaining the endometrium through the implantation window and, if conception occurs, through pregnancy (Jasienska and Ellison 2004).

Yet another hypothesis proposed that menstruation is not an adaptive process, but rather a byproduct of the fact that once endometrial cells terminally differentiate, they cannot survive as such for long and must be removed to start again for the next cycle (Finn 1996, 1998). That is, endometrial cycling has an adaptive purpose ensuring the endometrium is receptive to implantation at the right time, but the process of menstruation is functionless as it is simply the outflow of that cycling process. The functional layer of the endometrium proliferates and then differentiates as it is stimulated for growth and possible implantation. Differentiation of cells puts them on a path to impending apoptosis (cell death). Thus, differentiated cells cannot be sustained across multiple cycles. In this evolutionary telling, menstruation is a simple and non-adaptive consequence of extra tissue needing somewhere to go.

The idea that menstruation is both functionless and non-adaptive has persisted for several decades. Recently, evidence and interpretation contradict both of these ideas, suggesting instead that menstruation serves as a functional priming mechanism, useful for preconditioning the uterus for future reproductive success in mammals with invasive hemochorial placentas (Brosens et al. 2009). Competing interests, including paternal and maternal reproductive success, are thought to play a role in the evolution of human hemochorial placentas where the developing fetus connects through the placenta, taking nutrients directly from it, and sending hormonal signals to maintain the pregnancy (Figure 11.1). Pregnancies with less deep placentation and unformed spiral arteries (which supply blood to the endometrium in the luteal phase and early pregnancy) characterize preeclampsia, a hypertensive disease of pregnancy and a leading cause of global maternal mortality. Comparatively, deeper placentation, combined with formed spiral arteries, tends to predict less risky pregnancy. Both endometrial cycles and menstruation itself are seen as necessary steps to precondition the uterus to reduce the likelihood of less effective placentation and spiral artery development.

Thus, endometrial cycles and menstruation may have the adaptive function of preconditioning the uterus (Lucas, Salker, and Brosens 2013; Brosens et al. 2009). Preeclampsia likely occurred throughout human evolutionary history and its cost is high, as, without treatment, preeclampsia leads to eclampsia and seizures, and then the death of the birthing parent if not also the fetus (Rosenberg and Trevathan 2007). Those pregnancies most at risk for preeclampsia include birthing parents who are very young and for whom it is their first. Those who are young have had fewer menstrual periods, and thus possibly not enough endometrial preconditioning for deep placentation. Those who have never been pregnant may similarly not have had enough priming for deep placentation; former pregnancies do not need to be viable or carried to term to decrease the risk of preeclampsia. Further, menstruating species have unique processes specific
Figure 11.1 Illustration of patterns in follicular maturation, hormonal concentrations, and endometrial lining across each phase of an ovulatory, non-conceptive menstrual cycle. The cycles are centered where ovulation is day 0. There is more natural variation in follicular dynamics, hormonal concentrations and patterns, and endometrial variation than is represented here. For example, some experience one, two, or three follicular waves (Clancy, Baerwald, and Pierson 2013), spiral arteries may be unformed (Brosens et al. 2009), the endometrial lining can regress earlier or later (Clancy 2009), and there are differences in lengths of each menstrual cycle phase. In showcasing hormonal variation, estrogen and progesterone are displayed here using urinary metabolites in 83 menstrual cycles from Polish and Polish American women. The data is averaged for each day of the menstrual cycle forming the characteristic pattern showcased in textbooks, yet the standard deviation showcases the large amount of normal variation between individuals. Source: This figure was developed using data collected in 2014, 2015, and 2017 at the Mogielica Human Ecology Study Site, which is directed by Dr. Grazyna Jasienska (see Jasienska 2013; Rogers et al. 2019; Lee et al. 2019; Lee et al. 2020) and in 2016–2017 under the Clancy Lab Sto Lat: Health and Hormones Polish American Project (see Rogers 2018).
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to maintaining the endometrial lining for future pregnancies; for example, specific proteins, released by the endometrium and found in menstrual effluent, are necessary for rapid endometrial repair and regrowth (Evans et al. 2019). These ideas of priming and preconditioning suggest menstruation is more than a non-adaptive consequence, and rather a physiologically relevant process included in endometrial cycling and important for reproductive success.

Some might argue that the intention of the non-adaptive consequence hypothesis for human menstruation was to only name the process of excreting excess menstrual effluent as the non-adaptive component of endometrial cycling. However, the work we described above positions the processes of endometrial regrowth and repair as central to our understanding of fecundity, and menstrual effluent as necessary to that process. Making the point that the excretion of additional tissue is not adaptive only reaffirms the broader narrative, as with clitorises, orgasms, and pendulous breasts, that processes and phenomena affecting assigned female bodies are inconsequential compared to those affecting assigned male bodies. It seems like both a strange and unnecessary parsing to say menstruation is non-adaptive when it is physiologically necessary to the whole. That is, not only are endometrial cycles adaptive, we would argue that given the biomarkers and healing properties found in menstrual effluent (Salamonsen and Evans 2018; Evans et al. 2019), menses is crucial to that adaptive complex.

However, rather than take a stand that only one of these hypotheses is correct or is the prime mover, we acknowledge the energetic costs of menstruation, the physiological inevitability of terminal differentiation of endometrial tissue, the necessity of menstrual priming to support adequate placentation and spiral artery development, and the necessity of menstrual effluent for supporting endometrial repair and regrowth. Human menstruation is a product of our phylogeny, as a species with spontaneous ovulation, hemochorial placentation, and spiral arteries; of basic physiology, as cells that differentiate must eventually expire; of our more recently evolved maternal–fetal closeness that likely stems from supporting large-brained singletons. Menstruation is a mechanism within a suite of processes necessary to human reproduction and linked to adaptive changes in our lineage.

While the process of menstruation is experiencing renewed attention, first menstruation, or menarche, has been in the limelight for some time. Menarcheal age has long been considered to be important to understanding life-history trade-offs, where earlier or later menarche indicates childhood stressors and consequences for adult reproduction. Whereas we argue processes of menstruation have been under-investigated relative to their importance to fecundity and reproductive success, menarcheal age has been over-investigated for presumptions that being “early” or “late” predicts pathology. Therefore, next, we describe the ways in which menarcheal age has been pathologized and demonstrate how less visible components of pubertal maturation may be more important to understanding developmental effects on health.

The meaning of menstruation is historically questioned, so why is menarche so meaningful?

In contrast to the physiology of menstruation, variation in the timing of first menstruation has largely been understood through an evolutionary, life history lens. This theoretical perspective helps reveal how the biological variation in age at menarche is in part attributed to the lived environment. The emphasis on variation is useful as age at menarche varies with environmental stressors such as childhood energetic and psychosocial stress. However, due to these associations, there has been an overemphasis on early ages at menarche as a sign of childhood stress and as an increasing risk for breast cancer and cardiovascular disease, despite evidence suggesting age at menarche may be an external sign of other underlying causes for these increased risks. Further,
traits associated with earlier ages at menarche are not universal, and thus variation in this age needs to be interpreted contextually. Finally, this emphasis on age at menarche may ignore other aspects of pubertal timing, such as *pubertal tempo*, that are equally if not more important. Thus, we argue the significance of age at menarche must be interpreted with a better understanding of lived contexts and recognition of other pubertal milestones.

**Physiology of menarche and menstruation**

During puberty, the hypothalamic-pituitary-ovarian (HPO) axis is maturing; this process and the onset of regular menstrual cycles are sensitive to environmental stressors, similar to the variation in menstruation (e.g., Núñez-De La Mora and Bentley 2007; Reiches et al. 2013; Ellis 2004). First menstruation, or menarche, is a late pubertal milestone. While skeletal growth, pubarche, and thelarche are also major processes of puberty, menarche is the milestone where the individual can clearly mark the day it occurs. Age at menarche is often memorable as this age often culturally signals reproductive maturation and is experienced differently depending on the particular sociocultural context (Uskul 2004; Freidenfelds 2009). Menarche is one of many changes occurring through puberty: tempo of puberty and the timing and order of pubarche and thelarche are all additional sources of variation in pubertal development worthy of further research (Mendle 2014; Houghton et al. 2014).

To help understand what occurs around and after menarche, we will briefly describe the physiology of the menstrual cycle in the context of pubertal timing. Figure 11.1 displays a representation of the menstrual cycle for an ovulatory and non-conceptive cycle. Figure 11.1 is not unlike those in medical textbooks, where each depiction of follicular maturation, hormonal concentrations, and endometrial lining is averaged across the cycle. However, throughout this chapter, we’ve highlighted the large range of normal variation, context-dependencies, and changes over time or across cycles. Thus, as with many averages (Wachter-Boettcher 2017; Rose 2016), Figure 11.1, in attempting to describe everyone, may result in describing no one. To illustrate this point, we showcase reproductive hormonal variation in Figure 11.2, highlighting the daily variation averaged patterns do not capture.

Puberty begins after *adrenarche* and is characterized by a resumption of the gonadotropin-releasing hormone (GnRH) pulse. Pubarche and thelarche follow, with menarche as the final pubertal milestone. While menarche is seen as a one-time event, it is part of the pubertal process (see similar discussion on menopause: Sievert 2006 and Sievert and Roy in this volume). Premenarcheal pubertal development includes ovarian maturation and likely low-concentration hormonal cycling (Zhang et al. 2008). In mature and ovulatory cycles, GnRH stimulates follicle-stimulating hormone (FSH) and luteinizing hormone (LH), which in turn stimulate maturation of ovarian follicles in waves. These ovarian follicles are surrounded by cells releasing ovarian hormones, increasing the estrogen hormone estradiol. Estradiol provides a negative feedback loop slowing GnRH, increases sensitivity to GnRH, and helps stimulate the proliferation of the endometrial lining and the emergence of spiral arteries. Menarche is the first time the endometrial lining grows thick enough to be shed, rather than resorbed (Reiches et al. 2013).

Increasing estradiol triggers a surge in LH and FSH, stimulating ovulation. Anovulation is often pathologized, but few ovulate before menarche (Gray et al. 2010); it can take months or years before menstrual cycles are regularly ovulatory (Vihko and Apter 1984), and even the cycles of healthy adults are regularly anovulatory (Prior et al. 2015). After ovulation, the cells that had surrounded the developing follicle are now called the corpus luteum and continue to secrete hormones for several days. This phase of the menstrual cycle is termed the luteal phase. The corpus luteum contributes to the increased production of progesterone, stimulating the
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Decidualization of the endometrial lining, and maintaining the endometrial lining. As the corpus luteum slowly stops secreting hormones, progesterone concentrations fall, the endometrial lining is no longer maintained, and it is shed (menstruation occurs).

Age at menarche has been used as reflective of early environments, as predictive of characteristics of the menstrual cycle like frequency of ovulation and hormonal concentrations levels, and as predictive of future health risks. However, in comparison to other sources of pubertal timing...

Figure 11.2 Averaged data showcase little of the normal variation seen in reproductive hormones across days, individuals, between each cycle, and over the reproductive lifespan. Here we showcase A) the mean (SD) of six anovulatory cycles of regular cycle length; B) mean (SD) of four cycles with possible fertilization detected with beta hCG; and C) daily reproductive hormone concentrations in four ovulatory menstrual cycles. The variation showcased in A, B, and C highlight the ways averaged data in Figure 11.1 and common menstrual cycle illustrations hide the large range of normal variation. Source: These figures were developed using data collected in 2014, 2015, and 2017 at the Mogielica Human Ecology Study Site, which is directed by Dr. Grazyna Jasienska (see Jasienska 2013; Rogers et al. 2019; Lee et al. 2019; Lee et al. 2020) and in 2016–2017 under the Clancy Lab Sto Lat: Health and Hormones Polish American Project (see Rogers 2018).
and variation, menarche may not be as informative. For example, the rate at which one matures from andrenarche to menarche, the order of pubarche and thelarche, and the timing between thelarche and menarche are all hypothesized or evidenced as related to reproductive and health outcomes (Terry et al. 2017; Houghton et al. 2014; Mendle 2014). Therefore, while it may be the best proxy in retrospective research, its visibility has led to its broader biological meaning being overemphasized. Menarche is an important pubertal milestone, but it warrants understanding why there is such a focus on this age and what we may miss by prioritizing age at menarche as our proxy for pubertal timing.

**The evolutionary meaning of menarche: What are we missing by focusing primarily on menarche?**

So why has age at menarche been emphasized over these other pubertal developmental makers? One reason is that age at menarche is a clear, visible, and memorable life-history trait. Life history theory provides an evolutionary framework to investigate how different types of evolutionary mechanisms have shaped reproductive success, particularly in the face of limited resource availability (Charnov 1993; Stearns 1989; Roff and Fairbairn 2007). A life history perspective recognizes the social and biological causes of variation in menarche and posits this variation can be adaptive in given contexts. While differences in the menstrual cycle are increasingly understood as normal variation, variation in age at menarche is often classified as indicative of a “fast” or “slow” life history strategy. This strategy of maturing earlier or later is interpreted as a response to developmental environments and has an effect on future reproductive traits. However, the definition of earlier and later ages at menarche is rarely offered in a population-specific way, the effects of seemingly similar early environments are not universal, and traits that are associated with earlier ages at menarche in one community may not be associated with earlier ages at menarche in another.

For example, energetic and psychosocial stress respectively tend to delay and accelerate pubertal timing. One common example of psychosocial stress is father absence in childhood, which has been associated with earlier menarche (Ellis and Garber 2000; Boothroyd et al. 2013; Jones et al. 1972). Father absence is often interpreted in a particular biased cultural lens that chooses to see single parenthood as not only a moral failure, but a major psychosocial stressor. Across many Western samples where this idea persists, it is perhaps unsurprising that father absence is associated with earlier menarche and a faster life history strategy towards earlier reproduction. The idea is that when extrinsic mortality is high, the life history trade-offs between survival, growth, and reproduction shift toward a faster life history track in order to mitigate the high risk of not surviving to reproductive success (Walker et al. 2006). Due to this shift, high psychosocial stress may provide a signal of a risky environment and lead to earlier ages at menarche (Ellis 2004; Coall and Chisholm 2003; Belsky et al. 1991). However, the specific relationship between father absence and earlier ages at menarche is confined mostly to white, Western samples (Sear et al. 2019). The lack of cross-cultural support for a relationship between father absence and early menarche, complemented by a lack of relationship between mother absence and menarcheal age, instead highlights nuanced cultural stressors such as the differential societal treatment of single mothers compared to single fathers (Sheppard et al. 2014; Sear et al. 2019). The dominance of white scientists and colonial scientific discourse, and our unwillingness to name and interrogate that whiteness and historical context, has reified our understanding of variables we name as psychosocial stressors because we don’t notice that we have culturally assigned these variables negatively (Clancy and Davis 2019).

The other major stressor, energetic stress, may lead to a shortage of resources and their more careful allocation, leading to slower growth trajectories and later ages at menarche (Huss-
Ashmore and Johnston 1985). Under energetic constraint, extending the period of growth ensures enough investment in growth to successfully reproduce in the future, and thus age at menarche tends to be later in populations or groups exposed to low energy status, negative energy balance, or high energy flux (Ellison 2001; Reiches et al. 2013). In the United States, participation in intensive sports like gymnastics is associated with later ages at menarche (Georgopoulos et al. 2010). This had been attributed to high physical activity levels, but intensive sports are not all associated with delayed menarche, suggesting other factors (Baxter-Jones et al. 1994). In highly competitive gymnastics, the culture surrounding the sport selects for certain body types for elite performances and encourages athletes to maintain these body types (Georgopoulos et al. 2010). Sports that have a culture where certain body types are attributed to higher success (e.g., gymnastics, ice skating, long-distance running) are more likely to have or encourage participants with disordered eating patterns where energetic intake is not high enough to compensate for their expenditure. What’s more, the psychosocial stress of maintaining a particular body composition or shape, especially through puberty where the only constant is change, may exert additional effects on the HPO and HPA axes. Thus, the cultural context of a sport and its body composition and eating norms are at least as important to understanding their role in influencing menarcheal age as the increased energy expenditure that results from participation in them.

There is also a cultural context to science in terms of what questions get asked, which studies get funded, how studies are conducted, and who is included as participants (Clancy and Davis 2019). In this colonial scientific context, variation in age at menarche has traditionally been approached in the context of pathologizing early and late ages at menarche; this has led to a large body of research focusing on the associations between negative traits and age at menarche. However, new research finds that positive traits like social support also affect HPA and HPO axis activity (Uchino 2006; Turner-Cobb et al. 2000; Gunnar 2017; Hennessy et al. 2009), and it is reasonable to hypothesize that social support may also be associated with menarcheal timing. In our own work with Midwestern American adolescents, we have seen that openness in parent–adolescent communication is associated with age at menarche (Rogers-LaVanne et al. in prep). Similarly, Sung et al. (2016) found that maternal–infant relationship buffered the effect of risky environments on age at menarche. These positive relationships indicate a need to critically examine our science and look beyond stress-based models.

Some of the focus on the relationship between negative traits and menarcheal timing seems to stem from misplacing causality on a trait that may not actually exert strong effects on adult functioning. More specifically, age at menarche is a proximate measure, rather than the prime variable predicting negative health outcomes. For example, earlier ages at menarche are associated with increased risk for diseases like depression (Allison and Hyde 2013), breast cancer (Valaoras et al. 1969), and cardiovascular disease (Remsberg et al. 2005). In each of these cases, increased risk can be better predicted by what age at menarche uniquely represents for each condition. The link between age at menarche and depression is mediated by sociocultural context including increased bullying, body shaming, and supervision/monitoring surrounding pubertal development (Allison and Hyde 2013). Similarly, the increased risk for breast cancer proxies the relationship between higher lifetime estrogen exposure and increased risk for breast cancer (Pettersson et al. 1986; Hoyt and Falconi 2015). Finally, adult cardiovascular health is better explained by pre-existing conditions like higher childhood BMI that associate with both earlier ages at menarche and adult cardiovascular disease risks (Hoyt and Falconi 2015).

There are times, including in our own research, where retrospective age at menarche is the closest we can get to knowing a participant’s pubertal timing, and we are not arguing in favor of discarding this methodology. Instead, the goals are to highlight the physiological meaning of
menarche and the importance of interpreting age at menarche within context, understand that age at menarche is not the only predictor of future reproductive success, and challenge the causal relationship between early ages at menarche and future health risks. Putting age at menarche in that context softens the biological meaning of some of our results, and, we hope, encourages scholars to try to find new ways to measure pubertal timing retrospectively and holistically.

Age at menarche is reflective of prior environmental stressors, and may serve to indicate physiological life-history strategies. In some respects, we may be overestimating the power of age at menarche alone to inform our understanding of reproductive functioning and life history. Rather, similar to menstruation, this variation in age at menarche must be interpreted within context and as part of an active response to cues in our environment, informative to life-history trade-offs of when to start or postpone investment in reproduction.

Conclusion

Menstruation is a culturally fraught phenomenon, providing a significant source of stigma for menstruators (Delaney et al. 1988; Thomas 2007). It is no surprise that the cultural heritage of predominantly white scientists working within a colonial science framework has influenced our biological theorizing about the processes of menstruation and menarcheal timing. Where scientists have sought to characterize the purpose of menstruation as purging the unclean or render it entirely inconsequential, new research offers processes of menstruation a place of higher prominence in our understanding of endometrial regeneration, endometrial practice, and successful trophoblast invasion.

Conversely, because of its negative perceptions, the onset of menstruation has developed an outsized reputation for delivering pathology to unwitting young people. Early timing of first menstruation is deemed worrisome, if not causal of breast cancer and other health conditions. However, the effects of early environment on menarcheal age are far more culturally dependent than previously thought, and menarcheal age itself seems to be a weaker and less meaningful predictor of adult health than many other pubertal factors. Therefore, we argue that menstruation has a stronger evolutionary and adaptive meaning than what corresponds to prevailing thought, while the timing of first menstruation does not deserve its designation as a harbinger of health concerns.

In this chapter, we underscored the impact of colonial roots and historical biases on the meaning of menstruation, menses, and menarche, and we highlighted new perspectives advanced through a feminist biology framework. Rather than identify a prime mover for the evolution of menstruation, a process that effectively reinforces colonial, hierarchical perspectives, we instead recognize the multiple, non-mutually exclusive motivators of reproductive variation and function. Building on this, we highlight the large range in normal menstrual cycle variation, such as in daily hormonal concentrations and spontaneously anovulatory patterns, that have been averaged out of medical texts and illustrations. Similarly, we contend that pathologizing early pubertal development removes agency from young individuals and clarify that the relationships between early age at menarche and health outcomes are often better explained by other factors.

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