RESEARCH ARTICLE SUMMARY

ANTHROPOLOGY

Demographic and hormonal evidence for menopause in wild chimpanzees

Brian M. Wood*, Jacob D. Negrey, Janine L. Brown, Tobias Deschner, Melissa Emery Thompson, Sholly Gunter, John C. Mitani, David P. Watts, Kevin E. Langergraber*

INTRODUCTION: It is not obvious why selection should favor menopause or the continued survival of individuals that can no longer reproduce. Among mammals, substantial numbers of post-reproductive females living under natural conditions in the wild have only been observed in humans and a few whale species. The rarity of this trait makes it both interesting and difficult to study. Data from our close primate relatives are especially valuable for the reconstruction and causal modeling of human life history evolution. In this study, we combined demographic and hormonal data to investigate post-reproductive life spans and their underlying physiological mechanisms in chimpanzees (*Pan troglodytes schweinfurthii*), who, along with bonobos, are humans' closest living relatives.

RATIONALE: We examined the mortality and fertility rates of 185 female chimpanzees in the Ngogo community of wild chimpanzees in Kibale National Park, Uganda, from 21 years of observation (1995–2016). We calculated the demographic measure PrR (post-reproductive representation), representing the fraction of adult life spent in a post-reproductive state. Human menopause, the nonpathological and permanent cessation of ovarian function resulting from the depletion of ovarian follicles, is reflected in increasing levels of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) and decreasing levels of ovarian steroid hormones (estrogens and progestins). To assess whether Ngogo females undergo humanlike menopause, we analyzed age-associated trends in five hormones measured in 560 urine samples from 66 females of varying reproductive status and age (range: 14 to 67 years).

RESULTS: As in other chimpanzee populations and humans, fertility declined after age 30, and no births were observed after age 50. Unlike other chimpanzee populations, but as in humans, it was not unusual for Ngogo females to live past the age of 50 (N = 16 females). The observed PrR value was 0.195, indicating that a female who reached adulthood (age 14) was post-reproductive for about one-fifth of her adult life, around half as long as human hunter-gatherers. Hormonal measures show that Ngogo females experience a reproductive transition similar to that of humans, characterized by increasing levels of FSH and LH and declining levels of estrogens and progestins as they undergo menopause.

CONCLUSION: Menopause ends reproduction around the age of 50 in both humans and wild chimpanzees. Substantial PrR has not been previously observed in any wild primate population, chimpanzees included. One explanation for this discrepancy is that substantial PrR could be a temporary response to unusually favorable ecological conditions at Ngogo, including low levels of predation, high food availability, and successful between-group competition. A second possibility is that substantial PrR is an evolved, species-typical trait in chimpanzees, which has not been observed elsewhere owing to recent negative human impacts, especially disease epidemics. The grandmother hypothesis suggests that older females could evolve to live past their reproductive years to help increase their daughters' fertility or their grandoffsprings' survival. This is unlikely to apply to chimpanzees, whose aged females generally live apart from their daughters, as daughters leave their natal groups at adulthood. In the context of female-biased dispersal, a more relevant theory may be the reproductive conflict hypothesis, which highlights the fact that after migrating into a new group, females become increasingly related to other group members as they age and face competition with younger females for limited breeding opportunities. The oldest females might stop reproducing in order to limit the inclusive fitness costs of that competition. The grandmother and reproductive conflict hypotheses are not mutually exclusive alternatives, and both may be required to explain why all human societies have higher PrR than documented here for chimpanzees. ■

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Cite this article as B. M. Wood *et al.*, *Science* **382**, eadd5473 (2023). DOI: 10.1126/science.add5473

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https://doi.org/10.1126/science.add5473

PHOTOS: (LEFT TO RIGHT) DAVID P. WATTS, KEVIN E. LANGERGRABER, AND JEFFREY T. KERBY

Three post-reproductive female chimpanzees. From left to right are MARL (died at age 69), MAR (died at 64), and Sutherland (still living at age 61).

RESEARCH ARTICLE

ANTHROPOLOGY

Demographic and hormonal evidence for menopause in wild chimpanzees

Brian M. Wood[1,2]*, Jacob D. Negrey[3], Janine L. Brown[4], Tobias Deschner[5,6], Melissa Emery Thompson[7], Sholly Gunter[8,9], John C. Mitani[10], David P. Watts[9], Kevin E. Langergraber[11]*

Among mammals, post-reproductive life spans are currently documented only in humans and a few species of toothed whales. Here we show that a post-reproductive life span exists among wild chimpanzees in the Ngogo community of Kibale National Park, Uganda. Post-reproductive representation was 0.195, indicating that a female who reached adulthood could expect to live about one-fifth of her adult life in a post-reproductive state, around half as long as human hunter-gatherers. Post-reproductive females exhibited hormonal signatures of menopause, including sharply increasing gonadotropins after age 50. We discuss whether post-reproductive life spans in wild chimpanzees occur only rarely, as a short-term response to favorable ecological conditions, or instead are an evolved species-typical trait as well as the implications of these alternatives for our understanding of the evolution of post-reproductive life spans.

In most wild vertebrates, the period of survival past the age of last reproduction is short, but the evolution of long life spans in humans is associated with a substantial post-reproductive period. In this study, we investigated whether a population of wild chimpanzees with long life expectancy experience menopause and exhibit significant post-reproductive survival. Demographic and behavioral data have been collected from the Ngogo community of wild chimpanzees in Kibale National Park, Uganda, since 1995. We examined demographic data covering the years 1995–2016, including 1611 chimpanzee risk years (i.e., the cumulative time that individuals in this study were demographically monitored) for measures of female mortality and fertility (n = 185 females). To assess post-reproductive survival at Ngogo, we calculated rates of fertility and survivorship across all ages, identified females that lived well beyond their last births, and calculated the post-reproductive representation (PrR) statistic (*1*), which represents the fraction of female adult years lived in a post-reproductive state.

In humans, reproductive cessation occurs by means of menopause, the permanent non-pathological age-associated cessation of ovarian function resulting from depletion of the lifetime supply of ovarian follicles (*2*). However, there are many other potential causes of sterility that can manifest with age, such as fetal loss, endometriosis, or infections in the reproductive tract (*3*). Studies of captive chimpanzees have produced conflicting evidence about the timing or existence of menopause, probably because researchers have used different measures of follicular depletion (e.g., postmortem counts of ovarian sections, observations of sexual swellings) and have included very few individuals in the age range where menopause is expected to occur (*4*–*7*). To assess whether Ngogo female chimpanzees experience menopause, we analyzed variation in the concentration of five hormones diagnostic of menopause. These were measured in 560 urine samples from 66 females that differed in reproductive status and age (range: 14 to 67 years). We compared the patterns found in our sample of chimpanzees with those from previous endocrinological studies of human females.

Results

Post-reproductive representation

The pattern of fertility at Ngogo resembles that in other chimpanzee communities (Fig. 1A). Probabilities of giving birth per year decline after around age 30, and reproduction ends near age 50, as in humans (*8*, *9*). However, whereas females in other chimpanzee communities rarely live past age 50, 16 females at Ngogo have survived past this age (Fig. 1B).

We categorized females as post-reproductive if they were at least 40 years old and lived a long time after their last birth, using the definition provided by Caro and colleagues (*10*). This definition identifies post-reproductive females as those who lived past the age of their last reproduction for longer than the mean plus two standard deviations of successful, closed interbirth intervals for their population. We calculated this value as 7.9 years at Ngogo [5.5 + (2 × 1.2)]. Eleven of the 34 Ngogo females who survived to age 40 (32.4%) were post-reproductive by this criterion (Fig. 2). Nine of these 11 females lived at least 10 years past their age of last reproduction, and, on average, they were observed for 14.1 years without giving birth (SD = 4.6) (Fig. 2). Six of the 11 post-reproductive females lived past the end of our formal observation window in 2016, including three who are still alive now (as of 12 October 2023).

This demographic approach for assaying post-reproductive life spans has several shortcomings (*1*). The criterion of living 7.9 years

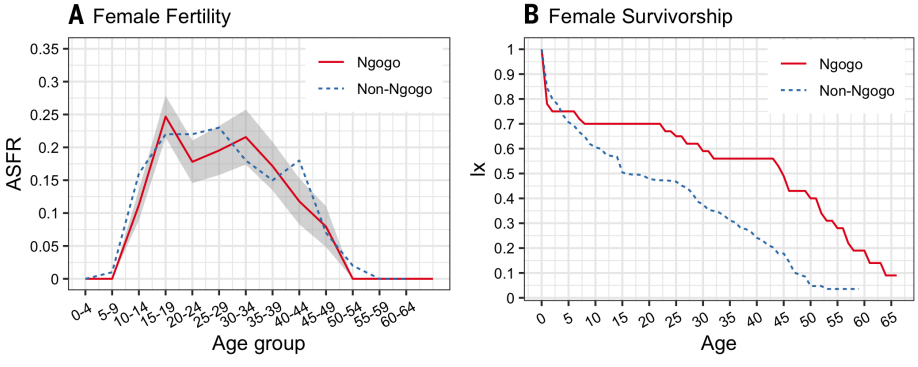

Fig. 1. Ngogo female fertility and survivorship. Female age-specific fertility rates, ASFR, were calculated for each 5-year age group by dividing the total number of births by the years of life observed in females of each age group. (**A**) Ngogo ASFR (±SE, in gray) and a composite sample of six other wild chimpanzee communities reported by Emery Thompson *et al*. (*8*). (**B**) Plot of the probability of female survival to each age (l_x) [updated from Wood *et al*. (*65*)] and a composite sample of five other wild chimpanzee communities (*8*).

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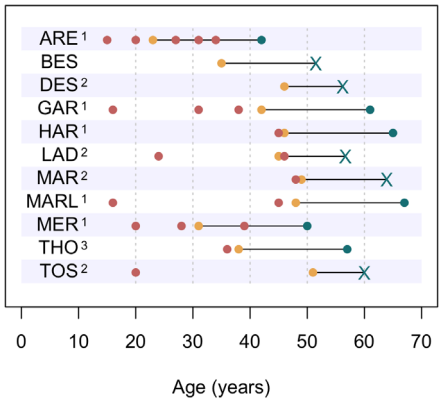

Fig. 2. Ngogo post-reproductive females (1995–2016). Their age at entry to the study is shown by yellow points, and age when giving birth (observed or inferred) is shown by red points. Age when exiting the study is represented by blue points if they were alive at the end of the study period, or by blue crosses if they died during the study period. Numbers plotted next to chimpanzee IDs describe the urine samples collected for hormonal analyses for that individual. Group 1 contributed samples to the analysis of FSH, LH, and ovarian steroid hormones; group 2, only FSH; and group 3, only ovarian steroid hormones. No urine samples were collected from individual BES.

beyond their last birth is likely to miss identifying truly post-reproductive females who died before reaching that milestone (i.e., false negatives). Furthermore, if interbirth intervals increase with age, some "post-reproductive" females may yet reproduce in the future (i.e., false positives), although none of the three living females in the sample have done so at the time of this writing. Finally, even if senescence of the reproductive system typically occurs at the same rate as senescence of the rest of the body, some individuals will deviate from this pattern and stop reproducing well before they die. These considerations indicate the need for alternative methods to determine whether the frequency and duration of post-reproductive life spans are sufficiently large to reject the null hypothesis that somatic and reproductive senescence occur in parallel.

A more informative demographic approach is to calculate the post-reproductive representation (PrR) statistic, which represents the fraction of female adult years lived in a post-reproductive state. This statistic can be validly compared across populations that vary in longevity. The calculation uses the equation

$$\mathrm{PrR} = T_M / T_B$$

where T_M is the expected life years lived beyond age M, T_B is the expected life years lived beyond age B, M is the age at which 95% of lifetime fertility has been realized, B is the age at which 5% lifetime fertility has been realized, and T_M is calculated from life tables using the formula $T_x = l_x \times e_x$, where l_x is the proportion of individuals surviving to exact age x, and e_x is future life expectancy at x.

When survival and fertility decline in parallel, PrR will be near 0. The statistical significance of PrR values greater than 0 can be determined through demographic simulations based on the null hypothesis that declines in survivorship and age-specific fertility occur simultaneously and at the same rate (*1*). Applying these formulas yields values of 14 years for B and 47 years for M at Ngogo, which results in values of T_M = 4.88, T_B = 24.97, and PrR = 0.195. This PrR value of 0.195 indicates that an Ngogo female who reaches adulthood can expect to live about one-fifth her life in a post-reproductive state. This PrR value is significantly different from 0 (see materials and methods), leading us to reject the null hypothesis of parallel declines of survivorship and fertility.

In a study of 52 wild mammal species, including nine nonhuman primates, 49 had PrR values close to 0 (range: 0 to 0.036) and not statistically different from chance under the null hypothesis of parallel declines of survival and fertility (*11*). Similarly, in a study of seven wild nonhuman primate species, including one chimpanzee population with PrR = 0.022, PrR ranged from 0.02 to 0.06 (*12*). Results of prior studies on primates specifically are summarized in Table 1. Only in humans (e.g., Hadza hunter-gatherers, PrR = 0.443), killer whales (0.309), and short-finned pilot whales (0.260) has PrR been shown to be sufficiently large to reject the null hypothesis of concurrent reproductive and somatic senescence under conditions of natural fertility and mortality. Our results are thus the first documentation of a wild nonhuman primate population that exhibits substantial and statistically significant post-reproductive representation.

Hormonal indicators of menopause

In humans, formal diagnosis of menopause typically occurs retrospectively, after a woman has experienced 12 months of amenorrhea; this method of diagnosis is difficult to implement given the observational methods used to study wild chimpanzees. However, menopause also results in a clear endocrinological signature in humans. As follicular stocks near depletion, the ovaries cannot sustain the levels of estrogen and progesterone production necessary for ovulatory cycles. The pituitary gonadotropins luteinizing hormone (LH) and follicle-stimulating hormone (FSH) fail to elicit the expected steroid response, and secretion of these hormones consequently increases via negative feedback.

Endocrinological analyses confirm that Ngogo chimpanzees experience menopause (Figs. 3 and 4). As observed in humans, concentrations of ovarian steroid hormones (estradiol, estrone, and pregnanediol, a metabolic product of progesterone) were lower in post-reproductive females (defined as above: ≥7.9 years since last birth at the time of sample collection) than in reproductive females who were regularly experiencing sexual swellings and thus experiencing ovarian cycles at the time of sample collection (2016–2018) (Fig. 3, D to F, and table S1). In another similarity to humans, post-reproductive female chimpanzees showed ovarian steroid hormone concentrations comparable to those of lactating females, indicating the absence of ovarian cycles in both groups (Fig. 3, D to F, and tables S1 and S2).

In humans, the largest change in reproductive hormones across the menopause transition involves gonadotropins. LH and FSH levels spike briefly before ovulation in normally cycling women but are otherwise low, whereas both of these hormones increase and remain elevated from perimenopause into the post-menopausal period. In serum, mean LH levels are two to five times higher and FSH levels are 3 to 15 times higher in post-reproductive women than in reproductive women, although these differences are also influenced by variation in age and breastfeeding status (*13*–*15*). Limited data suggest that the differences may be smaller for urine, with one recent study reporting mean urinary FSH levels approximately two times higher in post-reproductive individuals than in reproductive individuals (*16*). Although methodological differences limit the utility of precise quantitative comparisons, the differences in gonadotropin levels observed between reproductive and post-reproductive Ngogo chimpanzees fell within the range seen in human females. Post-reproductive Ngogo chimpanzees had median LH levels 5.6 times higher and FSH levels 5.4 times higher than those of reproductive females (samples collected from 2016 to 2018) (Fig. 3, A and B, and table S2). In a smaller set of samples collected 10 years earlier using a different assay and including some individuals not available in the later set of samples, FSH levels were 1.8 times higher in post-reproductive than in reproductive individuals (2006–2007 samples) (Figs. 3C and 4C and table S2). Individual-level patterns in the 2016–2018 data (fig. S1) show that all post-reproductive females had FSH and LH values that were higher than the median for reproductive females, except for the post-reproductive female with the earliest age of reproductive cessation (individual ARE, 34 years) (Fig. 2). ARE's reproductive cessation may have therefore been due to a nonmenopausal form of secondary sterility, or she may have been in the early stages of perimenopause, and we underestimated her age (which in our study is generally more likely than age overestimation; see the Materials and methods section).

To verify that reproductive cessation generally occurred as a result of menopause rather than other pathological causes, we also examined the

Table 1. Published estimates of post-reproductive representation for nonhuman primates and selected human populations. The statistical test for PrR is based on simulations of a null model of parallel declines in fertility and mortality, following the procedure described in (*1*). An asterisk in the "Source for demographic measures" column indicates that age-specific measures of mortality and fertility were calculated from a proprietary International Species Inventory System database (*23*) and used to calculate PrR, but user restrictions prevented the authors from publishing or otherwise sharing the underlying data, life tables, or fertility measures.

Species	Community	PrR	PrR source	PrR statistical significance	Source for demographic measures
		Wild nonhuman primates			
Chimpanzee (*Pan troglodytes*)	Ngogo, Kibale, Uganda	0.195	This study	Significant	This study
Chimpanzee (*P. troglodytes*)	Gombe, Tanzania	0.006	(*11*)	Not significant	(*40*)
Chimpanzee (*P. troglodytes*)	Gombe, Tanzania	0.02	(*12*)	Not tested	(*12*)
Chimpanzee (*P. troglodytes*)	Composite of Gombe, Tai, Kanyawara, Mahale, Bossou	0.018	(*29*)	Not significant	(*8*, *66*)
Mountain gorilla (*Gorilla beringei*)	Rwanda	0.022	(*11*)	Not significant	(*11*)
Mountain gorilla (*G. beringei*)	Rwanda	0.04	(*12*)	Not tested	(*12*)
Blue monkey (*Cercopithecus mitis*)	Kakamega Forest, Kenya	0.005	(*11*)	Not significant	(*40*)
Blue monkey (*C. mitis*)	Kakamega Forest, Kenya	0.02	(*12*)	Not tested	(*12*)
Blue monkey (*C. mitis*)	Kakamega Forest, Kenya	0.041	(*29*)	Not significant	(*86*, *87*)
Japanese macaque (*Macaca fuscata*)	Yakushima and Kinkazan Islands, Japan	0.005	(*11*)	Not significant	(*88*)
Rhesus macaque (*Macaca mulatta*)	Community name not listed by (*29*)	0.007	(*29*)	Not significant	No source listed by (*29*)
Northern muriqui (*Brachyteles hypoxanthus*)	Caratinga, Brazil	0.06	(*12*)	Not tested	(*12*)
Olive baboon (*Papio anubis*)	Gombe, Tanzania	0.02	(*11*)	Not significant	(*89*)
Ring-tailed lemur (*Lemur catta*)	Berenty, Madagascar	0.001	(*11*)	Not significant	(*90*)
Verreaux's sifaka (*Propithecus verreauxi*)	Beza Mahafaly, Madagascar	0.003	(*11*)	Not significant	(*40*)
Verreaux's sifaka (*P. verreauxi*)	Beza Mahafaly, Madagascar	0.02	(*12*)	Not tested	(*12*)
White-headed capuchin (*Cebus capucinus*)	Santa Rosa, Costa Rica	0.004	(*11*)	Not significant	(*40*)
White-headed capuchin (*C. capucinus*)	Santa Rosa, Costa Rica	0.04	(*12*)	Not tested	(*12*)
Yellow baboon (*Papio cynocephalus*)	Amboseli, Kenya	0.036	(*11*)	Not significant	(*40*)
Yellow baboon (*P. cynocephalus*)	Amboseli, Kenya	0.01	(*12*)	Not tested	(*12*)
		Captive nonhuman primates			
Chimpanzee (*P. troglodytes*)	Zoo-living composite	0.224	(*29*)	Significant	*
Western gorilla (*Gorilla gorilla*)	Zoo-living composite	0.214	(*1*)	Significant	*
Sumatran orangutan (*Pongo abelii*)	Zoo-living composite	0.231	(*1*)	Significant	*
Bornean orangutan (*Pongo pygmaeus*)	Zoo-living composite	0.192	(*1*)	Significant	*
Japanese macaque (*M. fuscata*)	Provisioned sanctuary, Arashiyama West, Texas	0.054	(*29*)	Significant	(*91*, *92*)
Japanese macaque (*M. fuscata*)	Zoo-living composite	0.247	(*29*)	Significant	*
Rhesus macaque (*M. mulatta*)	Zoo-living composite	0.178	(*29*)	Significant	*
		Humans			
Human (*Homo sapiens*)	Hadza hunter-gatherers, Tanzania	0.443	(*11*)	Significant	(*84*)
Human (*H. sapiens*)	!Kung hunter-gatherers, Botswana	0.426	(*1*)	Significant	(*93*)
Human (*H. sapiens*)	Ache hunter-gatherers, Paraguay	0.439	(*1*)	Significant	(*94*)
Human (*H. sapiens*)	Historical Sweden 1751–1755	0.477	(*1*)	Significant	(*95*)
Human (*H. sapiens*)	Plantation slaves of Trinidad	0.302	(*1*)	Significant	(*60*)

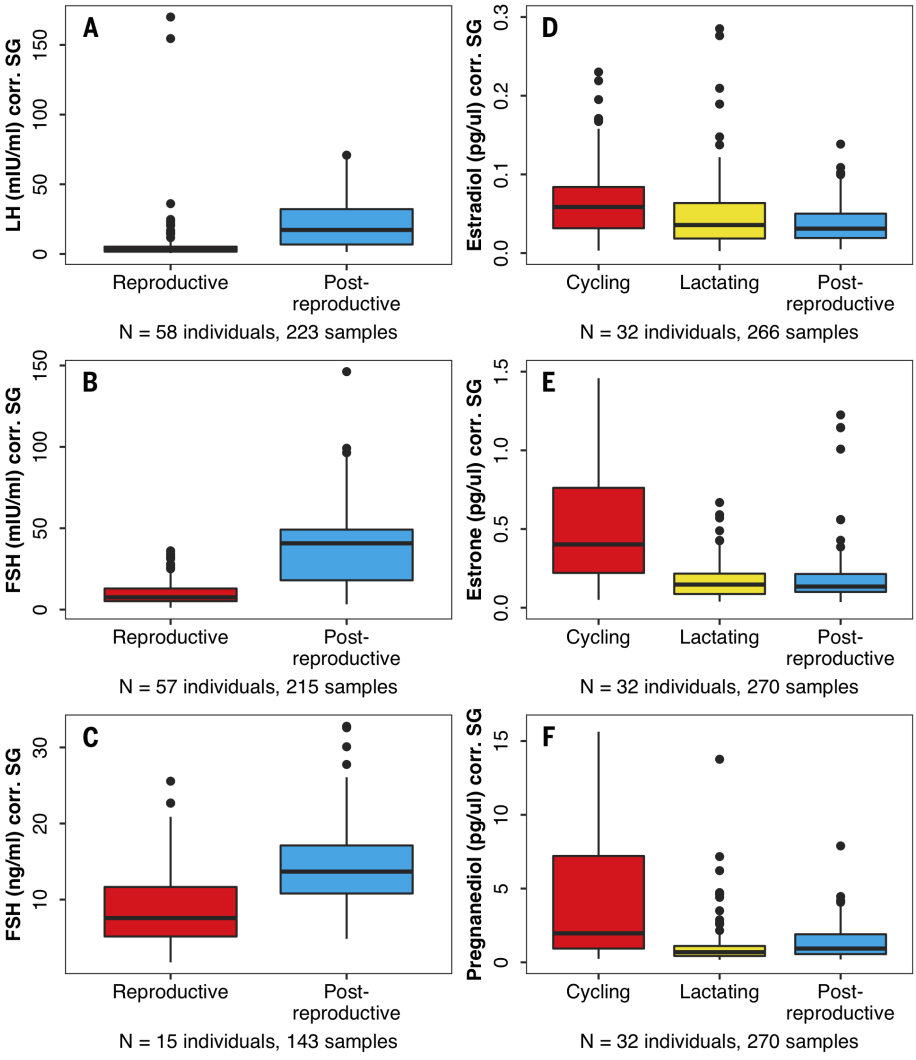

Fig. 3. Urinary hormone concentrations in females of different reproductive states. (**A**) LH in samples collected in 2016–2018, (**B**) FSH 2016–2018, (**C**) FSH 2006–2007, (**D**) estradiol 2016–2018, (**E**) estrone 2016–2018, and (**F**) pregnanediol 2016–2018. Boxplots are based on all urine samples rather than averages per female. Black dots indicate data points above the third quartile by >1.5× the interquartile range. Median values by reproductive category are provided in table S2. For legibility, a few extreme measures have been excluded from (D) to (F), but full data ranges are displayed in fig. S3. corr. SG, corrected specific gravity.

shape of changes in ovarian steroid hormones and gonadotropins with age. If Ngogo female chimpanzees follow a humanlike pattern of FSH changes accompanying menopause at around 50 years of age, there should be modest increases in their FSH levels beginning in their 30s, continuing into their early 40s, and then followed by a more acute increase starting around age 45 (*13*, *17*, *18*). LH concentrations are expected to remain relatively stable before rising in the mid to late 40s (*18*).

Consistent with the human pattern, FSH and LH increased with age (Fig. 4, A to C). To examine the shapes of these relationships, we evaluated alternative regression fits using Akaike's information criteria corrected for small sample sizes (AICc). For the larger 2016–2018 sample, changes in both FSH and LH exhibit steeply increasing nonlinear relationships with age beginning in the mid-40s, rather than linear relationships (Fig. 4, A and B, and table S3). In the 2006–2007 sample, the positive association between age and FSH is best described as a linear relationship (Fig. 4C and table S4), although this result probably stems from a smaller sample size and the inclusion of very few samples from females between the ages of 20 and 50 years. As in humans, gonadotropin production accelerated around the age of typical last reproduction at 50 years.

As Ngogo females age, they produce lower levels of ovarian steroids, especially after the age of 40. Figure 4, D to F, illustrates these age-related decreases in urinary estradiol, estrone, and pregnanediol among nonbreastfeeding Ngogo females. Post-reproductive Ngogo females exhibited uniformly low levels of ovarian hormones compared with reproductive females, a pattern also observed in human females (*19*).

Figure 5 compares standardized measures of gonadotropins and ovarian hormone concentrations in Ngogo females with those of women between the ages of 25 and 60. Because laboratory techniques and units of measure differ across these studies, we have standardized the measures of hormone concentration by their mean values within each study across the age range of 25 to 60, the age range of the human studies. Both species exhibit broadly similar patterns of increasing FSH and LH and declining estrogens and progestins as they undergo menopause.

We recognize that while our samples sizes are large by primatology standards, they are much smaller than those in human studies. Also, our age estimates—particularly those for older females—contain error that is not present in studies of human females (see Materials and methods section "Age estimation"). But even considering these limitations, the similarity across species in the age trends of urinary FSH, LH, estrogens, and progestins is considerable. In sum, the combination of demographic and hormonal data indicates that reproductive cessation in both humans and chimpanzees is caused by a common physiological factor, which is menopause, and that this occurs at a similar age of around 50 years in both species.

Discussion and conclusions

Although the occasional presence of old, nonreproductive females has been reported in other wild chimpanzee communities (*4*, *9*), demographic studies have not previously provided evidence for substantial post-reproductive survival in any wild population. The primary reason for this contrast is that the Ngogo chimpanzees exhibit higher survival rates than those in other populations studied in the wild, where only a few female chimpanzees have been observed to live beyond the age of 50. This demographic difference can be interpreted in two distinct ways, both of which have implications for understanding the evolution of this rare trait in humans.

The first possibility is that the long life spans of the Ngogo chimpanzees are a temporary demographic response to unusually favorable ecological circumstances. In captivity, or so-called "protective" environments, some mammal species, including chimpanzees, exhibit significant female post-fertile reproductive survivorship (Table 1). Leopards were extirpated from Kibale National Park by human hunters in the 1960s, and leopard predation may historically have been an important source of chimpanzee mortality, although this claim is contested (*20*–*22*). While hunters from the high-density human population surrounding the park have occasionally killed Ngogo chimpanzees using snares, metal spears, and trained

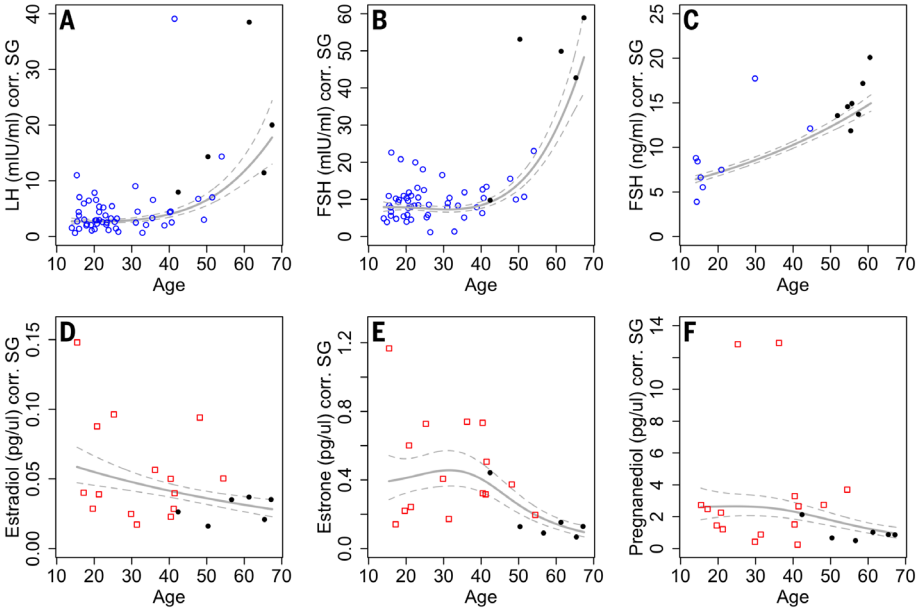

Fig. 4. Urinary concentrations of gonadotropins and ovarian hormones in females by age. (**A**) LH in 223 samples from 58 individuals collected 2016–2018, (**B**) FSH in 215 samples from 57 individuals collected 2016–2018, (**C**) FSH in 143 samples from 15 individuals collected 2006–2007, (**D**) estradiol in 160 samples from 21 individuals, (**E**) estrone in 162 samples in 21 individuals, and (**F**) pregnanediol in 162 samples from 21 individuals. Points represent average levels aggregated by individual female and reproductive status at the time of sample collection. Black points in (A) to (F) represent post-reproductive females, blue points in (A) to (C) represent reproductive females, and red squares in (D) to (F) represent nonbreastfeeding cycling females. The gray age curves in (A) to (F) represent mean expected values (±1 SE) from fit models 10 to 15, respectively (tables S4 to S7). See fig. S3 for sample-level plotting of gonadotropin concentrations and fig. S4 for age trends in ovarian hormone concentrations in all females, including those who were breastfeeding.

packs of dogs, no such cases were known to occur during the study period (*23*). It is thus possible that hunting by humans was a larger source of mortality in chimpanzees' overall long-term evolutionary history than currently occurs at Ngogo. Dietary quality is also high at Ngogo; the local fruit supply is more abundant and stable, and chimpanzees there consume considerably more meat than those in the nearby Kanyawara community (*24*–*26*). The Ngogo chimpanzees have been more successful in between-group competition than chimpanzees in other communities. They expanded their territory by 22% in 2010 after a decade of intense territorial boundary patrolling and killing in the area of the expansion (*27*). Finally, the Ngogo chimpanzee population grew over the study period, with a net reproductive rate (R_0) of 2.0, which suggests that their high survivorship cannot be representative of the long-term evolutionary history of the species.

If the substantial post-reproductive life spans observed in chimpanzees are only a temporary response to favorable and protective ecologies, this would offer insight into the early stages of its evolution as a species-typical trait in humans. Population genetic theory predicts, and empirical research confirms, that the power of selection to modify a trait depends on the amount of its standing genetic variation present in the population (*28*). Substantial post-reproductive life spans cannot evolve as a species-typical trait unless there already exist in the population individuals who outlive their own reproduction (i.e., post-reproductive viability) (*29*). In populations where there are individuals surviving beyond their reproductive years, natural selection can effectively translate the potential indirect fitness benefits of ceasing reproduction into a life stage for the species. The capacity for this demographic response in the last common ancestor humans share with chimpanzees would thus provide modest support for the plausibility of substantial post-reproductive life spans evolving earlier in hominin evolution [around 1.8 million years ago, at the emergence of *Homo erectus*, as discussed in (*30*)] rather than only very recently (around 50 thousand years ago, at the dispersal of *Homo sapiens* from Africa).

The second possibility is that substantial post-reproductive survivorship has been common in the evolutionary history of chimpanzees, but it is not exhibited by contemporary populations elsewhere because of recent environmental changes caused by humans. With one exception [Tai Forest (*31*)], previous demographic studies of chimpanzees have been conducted on populations living in habitats that were more heavily logged for forestry or agriculture within the past 100 years than Ngogo and are thus composed of less primary, old-growth forest (*32*–*35*). In addition, their close evolutionary relationship to humans makes chimpanzees extremely vulnerable to respiratory viruses that originate in humans and to which they have little developmentally acquired adaptive immunity or evolutionarily acquired genetic immunity [see review in (*36*)]. The devastating impact of human diseases was made clear by an outbreak of human metapneumovirus at Ngogo that killed 12.2% of the individuals in the community in early 2017, after the period under study here (*37*). The Ngogo chimpanzees had not previously suffered any known major disease outbreaks, and they have lower viral richness and load than the Kanyawara chimpanzee community, which lives at the edge of the park and has more contact with humans (*38*). Although more systematic research is needed, one study (*39*) concluded that anthropogenically caused habitat loss and disease epidemics are likely the main drivers of the substantial variability in survival rates observed across chimpanzee populations and for the fact that most have recently experienced devastating declines. The negative growth rates of non-Ngogo chimpanzee populations, like the positive growth rate of Ngogo, indicate that their patterns of survivorship and fertility also cannot represent the long-term average for the species. Comparative data suggest that favorable ecological conditions promoting population growth do not alone account for the emergence of substantial post-reproductive representation, as six other species of wild primates studied by Bronikowski and colleagues (*40*) have also grown at robust rates similar to those of the Ngogo chimpanzees (R_0 range: 1.51 to 2.37) yet do not show substantial post-reproductive life spans.

If substantial post-reproductive life spans were more common in the evolutionary history of chimpanzees, this would have additional implications for evaluating arguments for the evolution of prolonged post-reproductive life spans. A prominent adaptive evolutionary explanation for the evolution of substantial post-reproductive life spans in humans is the grandmother hypothesis (*41*, *42*), some versions of which state that the indirect fitness benefits that post-reproductive females gain by helping their daughters to reproduce or their grandoffspring to survive are greater than the direct fitness costs of ceasing reproduction. However, whether such indirect fitness benefits are sufficiently large to outweigh the direct fitness costs of ceasing direct reproduction in humans is controversial (*43*, *44*). Such indirect fitness benefits would likely be even smaller in chimpanzee-like social contexts. Chimpanzee males typically remain in the communities in which they were born for their entire lives. By contrast, females typically disperse from their natal community to reproduce

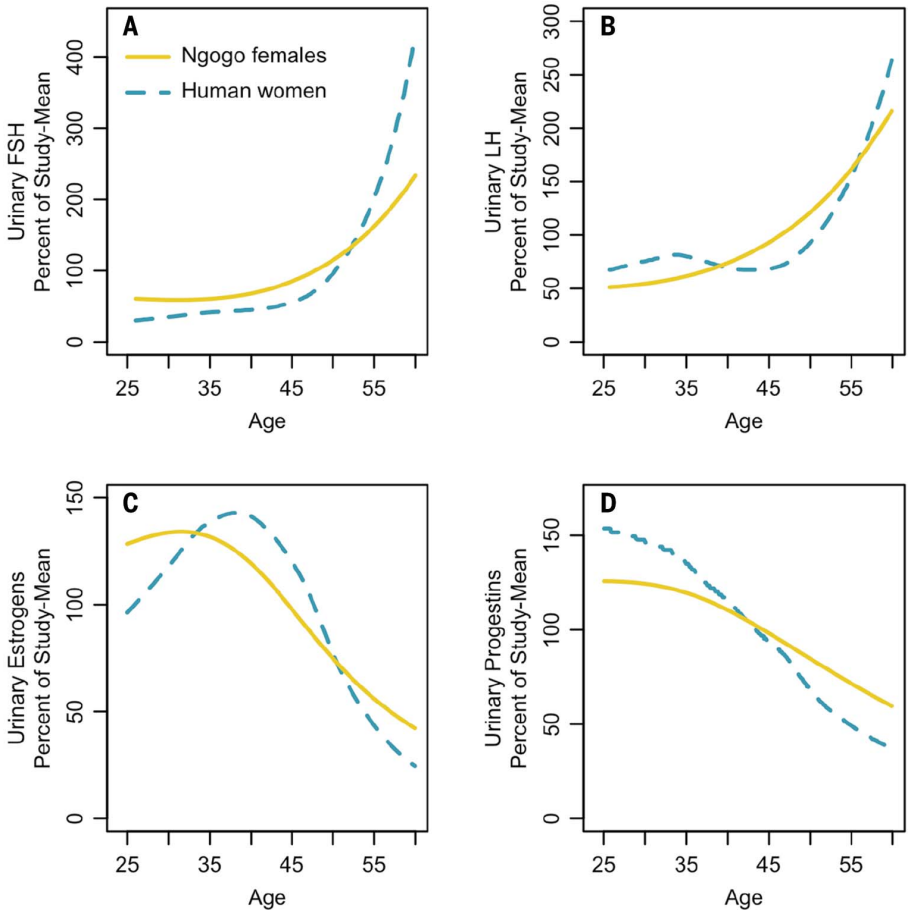

Fig. 5. Standardized age trends in female urinary hormones in Ngogo chimpanzees and human females. (**A**) FSH, (**B**) LH, (**C**) estrogens, and (**D**) progestins, as observed at Ngogo and in studies of human females by Ferrell *et al*. (*18*, *19*). The expected values for Ngogo females in all panels were generated from our study's data and the fit models 10, 11, 14, and 15 (tables S4 and S6). The analytes from Ngogo females shown in (C) and (D) are estrone and pregnanediol, respectively. Median age trends for human females in urinary FSH, LH, estrone glucuronide (a conjugated metabolite of estrone), and pregnanediol glucuronide (a conjugated metabolite of progesterone) are derived from (*18*, *19*).

in others, where they remain until they die (*45*). Unlike humans, chimpanzees cannot maintain long-distance helping relationships with individuals who reside in different communities (*46*, *47*). Thus, female dispersal limits opportunities for chimpanzee grandmothers to help either their daughters or their daughters' offspring, who will generally live elsewhere. Post-reproductive chimpanzee females could potentially help their sons reproduce, but current evidence suggests that unlike female bonobos, they do not do so. The difficulties of recognizing paternal kin under a highly promiscuous, polygynadrous mating system probably limits the ability of females to help their sons' offspring survive (*48*–*51*).

Another prominent adaptive evolutionary explanation for substantial post-reproductive life spans is the reproductive conflict hypothesis (*52*). This hypothesis suggests that in certain contexts, older females will compete with younger females for limited reproductive opportunities. In dispersal and mating systems where female relatedness to other breeders and their offspring increases with age, the net benefits of continued reproduction at advanced ages are low, because reproduction imposes costs on relatives. This pattern of age-graded local relatedness occurs under the system of male philopatry and female dispersal that characterizes chimpanzees and, more controversially, may be ancestral in humans and their hominin relatives (*52*–*56*). Some of the assumptions of the reproductive conflict model have been supported in empirical tests with killer whales (*57*), but similar investigations have led to mixed results in humans (*58*) and long-finned pilot whales (*59*).

The grandmother hypothesis and the reproductive conflict hypothesis are not mutually exclusive alternatives, and both may be required to explain why humans have evolved a robust post-fertile life stage. Even in the harshest socio-ecological conditions, human communities still have a higher representation of post-reproductive individuals compared with Ngogo chimpanzees. For example, under the extreme conditions of plantation era slavery in Trinidad, historical demographers estimate that a PrR of 0.302 was maintained (*1*, *60*), which is about 1.5 times that seen at Ngogo.

In sum, while the evolution of menopause and a substantial post-reproductive life span remains unclear, our results show that these traits can emerge in a chimpanzee population that has experienced a low level of human impact. The capacity for long post-reproductive life spans observed in contemporary humans may not have evolved de novo in our hominin ancestors but instead built on existing genetic variation in the last common ancestor we shared with chimpanzees. Our documentation of the survival of post-reproductive females at Ngogo required an extensive and ongoing research effort, and it will be crucial to invest in long-term studies across diverse ecological settings to better understand whether substantial post-reproductive life spans in chimpanzees emerge only rarely in particularly favorable ecological circumstances or are actually more common. There are currently insufficient data on survival and fertility in chimpanzees' sister species bonobos (*Pan paniscus*) to ascertain whether substantial post-reproductive life spans also occur in this species, and if so, under what conditions. Filling these gaps in knowledge about post-reproductive life spans in humans' two closest living relatives will be important for our understanding of the evolution of this rare and puzzling trait.

Materials and methods

Study site

All demographic and endocrinological data from females came from the Ngogo community of chimpanzees. Ngogo is located in Kibale National Park, southwestern Uganda. The 795 km^2 park is centered at ~0.5°S and 30.4°E. The vegetation of the park is mostly moist evergreen or semi-deciduous forest, transitional between lowland and montane forest (*33*). The Ngogo study area is in the center of the park, at altitudes between 1400 and 1470 m. Ngogo receives ~1500 mm of rainfall annually, concentrated in March to May and September to December (*61*, *62*). The vegetation is typically dry-ground forest that includes large tracts of old growth forest and early- to mid-stage colonizing forest regenerating from anthropogenic grassland (*63*). Anthropogenic grasslands still cover some of the study area, which also includes swamp forest, bush dominated by *Acanthus pubescens*, and a papyrus (*Cyperus papyrus*) swamp (*64*). The chimpanzees at Ngogo use the old-growth forest predominately and stay entirely within the park. Ngogo is surrounded on all sides by other communities of chimpanzees and does not

abut areas occupied by humans. Consequently, the chimpanzees do not eat crops but instead rely entirely on wild foods. Veterinary interventions have been limited to the removal of a snare from one adult male.

Demographic data

Our prior study of mortality (*65*) provides in-depth descriptions of our methods of demographic data collection, age assessment, and parentage assignment. Information on Ngogo chimpanzee births, interbirth intervals, age-specific fertility rates (ASFR), and post-reproductive survivorship are reported here for the first time, using data that span the years 1995–2016, including 306 individual chimpanzees and 3108 chimpanzee-risk-years of observation. Owing to the large size of the Ngogo community, the Ngogo demographic database is among the largest available for any wild chimpanzee community. This sample includes 185 females and 1611 risk years of observation.

Because the Ngogo chimpanzees have only been observed continuously since 1995, the ages of the oldest chimpanzees were necessarily estimated through morphological comparisons with known-aged individuals and genetically determined kinship relationships [as in (*65*, *66*) and see Materials and methods section "Age estimation"]. However, our demographic and physiological data on reproduction provide a strong internal validation of these age estimates. First, ASFR at Ngogo closely accord with data from other chimpanzee populations (Fig. 1). Second, the ages of post-reproductive females and ages at which FSH and LH levels increased exceeded the maximum ages of the majority of chimpanzees previously described from the wild, suggesting why long post-reproductive life spans were found at Ngogo but not elsewhere (Fig. 4). As a robustness check, we created a simulation model that generates alternative schedules of mortality and fertility, on the basis of a reasonable model of age estimation error. Including these influences of age estimation error, Ngogo post-reproductive representation (PrR) remains substantial and significant (Materials and methods section "The statistical significance of post-reproductive representation is robust to error in age estimates"). When we subsample our data and focus only on individuals who were precisely aged, we also find that Ngogo survivorship is high, consistent with our general results (Materials and methods section "Precisely aged individuals show low mortality among the Ngogo chimpanzees").

Interbirth intervals

Seventy-four females gave birth at Ngogo during the study, to a total of 175 offspring. We also identified 37 additional mother-offspring dyads through genetic analyses and observations of especially close interactions between females and young. Births that occurred before our observation period were not included in our analyses of ASFR or interbirth intervals, but they did allow us to infer whether some females had ever given birth. We identified closed, successful interbirth intervals at Ngogo—that is, those closed interbirth intervals in which first offspring survived to at least 4 years of age (n = 75). The mean duration of these intervals was 5.5 years (SEM = 0.14) and the standard deviation was 1.2 years.

Demographic measure of post-reproductive life span

We sought to identify older females who had not reproduced for an extended period and who were thus strong candidates for having experienced menopause (Fig. 1). We defined "older" as estimated to be at least 40 years and "extended period" as the population mean interbirth interval plus two standard deviations (7.9 years).

The PrR statistic is constructed from the observed life tables of survivorship and fertility and should be near zero for species in which a substantial female post-reproductive life span does not occur. However, owing to sampling variation, a nonzero PrR value is insufficient evidence to conclude that post-reproductive representation is generally greater than zero in a study population. Levitis and Lackey (*1*) present a demographic simulation procedure that generates PrR values from the null hypothesis that age-specific fertility rates (m_x) and survivorship (l_x) decline in parallel after the age of peak fertility. The observed age-specific fertility rates (m_x) are used to construct a smoothed model of monotonically declining fertility after the age of peak fertility. This fertility decline model is then used to construct the predicted survivorship $(\widehat{l_x})$ that would be anticipated if the null hypothesis were true, that is, if declines in survivorship were in parallel with and directly proportional to declines in fertility. Using the observed m_x and null-predicted survivorship values $(\widehat{l_x})$, 1000 simulated populations are then created, each with its own corresponding null-predicted $\widehat{\mathrm{PrR}}$ statistic. The distribution of these null $\widehat{\mathrm{PrR}}$ values is then compared with the observed PrR. The P value for this significance test is the number of null-simulated populations with higher $\widehat{\mathrm{PrR}}$ values than the observed PrR, divided by 1000. In our case, no null-simulated populations had a $\widehat{\mathrm{PrR}}$ value higher than 0.196; thus, our P value is <0.001.

Collection and measurement of urinary reproductive hormones

Most urine samples were collected opportunistically from individually identified female chimpanzees between 1 March 2016 and 11 May 2018. A second smaller dataset of urine samples from which we only analyzed FSH was collected between 28 May 2006 and 25 November 2007. Urine was pipetted from plastic sheets or leaves (*67*) and transferred to collection vials. Vials were stored in a cooled thermos until the collector returned to camp then deposited in a solar-powered freezer at −20°C. Samples designated for steroid measurements were transported on dry ice from Uganda to Germany, where they were stored at −80°C until they were analyzed. Similarly, samples designated for gonadotropin measurements were transported on ice from Uganda to the United States and stored at −80°C.

Urine samples collected in 2016–2018 were analyzed for gonadotropins FSH and LH in the Comparative Human and Primate Physiology Center at the University of New Mexico, Albuquerque, NM, USA. FSH and LH data from 2016–2018 were assayed using commercial enzyme-linked immunosorbent assays from MP Biomedicals with a sensitivity of 1.5 mIU/ml FSH and 1.0 mIU/ml LH. Interassay coefficients of variation (CVs) were 9.1% (low control) and 8.6% (high) for the FSH assay and 6.7% (low) and 13.4% (high) for the LH assay. Intrassay CVs, calculated as the mean CV of duplicate determinations, averaged 10.1% for both assays.

Urine samples from 2006–2007 were analyzed for FSH at the Smithsonian Conservation Biology Institute, Front Royal, VA, USA. FSH (LER-1976A) was iodinated following the protocol described in (*68*). Urine FSH was quantified by a iodine-125 double-antibody radioimmunoassay (RIA), with some modifications. The RIA used an anti-ovine FSH primary antibody (JADLER 178) and ovine LH label and standards (NIDDK-FSH-S16) in a phosphate-buffered saline (PBS)–based (0.01 M PO_4, 0.9% NaCl, 0.5% bovine serum albumin, 2 mM EDTA, 0.01% thimerosal, pH 7.4) buffer system. It was incubated in a total volume of 500 μl in 12 mm by 75 mm borosilicate tubes at room temperature. Standards (100 μl) and/or sample were added to PBS (200 μl), followed by addition of primary antibody (1:25,000, 100 μl) and incubation for 24 hours. Iodine-125 FSH tracer (~25,000 counts per minute, 100 μl) was added and the tubes incubated for an additional 24 hours. Antibody-bound complexes were precipitated by centrifugating at 3000g for 25 min after a 1 hour incubation with goat anti-mouse gamma globulin (1:300, 1 ml in PBS containing 5% polyethylene glycol, 8000 molecular weight, Sigma Chemical Co., St. Louis, MO). The antibody typically bound 30% of the iodinated tracer with ~5% nonspecific binding. Assay sensitivity was 0.25 ng/ml. The assay was validated for chimpanzee urine by demonstrating (i) parallelism between dilutions of pooled urine and the standard curve and (ii) significant recovery (>98%) of FSH standard added to non-estrous chimpanzee urine (y = 0.912x + 4.998, r = 0.993).

Urine samples collected in 2016–2018 were analyzed for steroid hormones in the Department

of Primatology at the Max Planck Institute for Evolutionary Anthropology, Leipzig, Germany. Each sample underwent extraction before measurement using a modification of Hauser *et al.* (*69*) in which we combined each sample with an internal standard, performed hydrolysis with β-glucuronidase derived from *Escherichia coli*, and performed solvolysis with ethyl acetate and sulfuric acid. Estradiol, estrone, and pregnanediol were measured by liquid chromatography–tandem mass spectrometry (LC-MS) using a modification of (*69*). We used a Waters ACQUITY UPLC separation module for liquid chromatography, using water (with 0.1% formic acid) and acetonitrile as eluents. We then used a Xevo TQ-S tandem quadrupole mass spectrometer (Waters, Milford, MA, USA) for mass spectrometry and examined the output with MassLynx (QuanLynx-Software).

We corrected for variation in urine concentration by measuring the specific gravity (SG) of each sample. We measured SG with a digital handheld refractometer (2016–2018 samples for gonadotropins: PAL-10S, ATAGO, Bellevue, WA, USA; 2006–2007 samples for FSH: Urisystem, Fisher Scientific, Waltham, MA, USA; 2016–2018 samples for steroids: TEC, Ober-Ramstadt, Germany). Hormone values were adjusted for SG following (*70*). We present estradiol, estrone, and pregnanediol concentrations in picograms per microliter of SG, and FSH and LH concentrations from 2016–2018 samples in milli–international units per milliliter of SG.

Of the eight reproductive females in the 2006–2007 FSH dataset, six were also included among the reproductive females in the 2016–2018 FSH dataset. Of the nine post-reproductive females in the 2006–2007 FSH dataset, four were also among the post-reproductive females in the 2016–2018 FSH dataset.

All analyses were based on individuals ≥14 years at the time of sample collection, which is close to the average age of first pregnancy among natal female chimpanzees (*71*). The oldest individual was estimated at 67 years. All analyses were based on samples from females that were known (via negative pregnancy tests) or inferred not to be pregnant because their next births occurred more than 253 days after sample collection, which corresponds to the mean gestation length plus two standard deviations in a sample of 118 captive chimpanzees (*72*). Finally, we did not include samples from females who died within 253 days of sample collection and did not have offspring younger than 3 years old, as they might have been pregnant.

Analysis of hormone levels by reproductive category

We assigned females to one of three reproductive categories: (i) post-reproductive, (ii) cycling (i.e., females who were experiencing regular sexual swellings), or (iii) lactating. To compare hormone levels among females in these categories, we used linear mixed models (LMMs) with Gaussian error structures and fitted with restricted maximum likelihood using the "lmer" function in package lme4 (*73*) in R version 4.0.3. For the analysis of gonadotropins (LH and FSH), cycling and lactating females were lumped into a single "reproductive" category whose levels were then compared with post-reproductive females (table S2, models 1 to 3). For the analysis of the steroid hormones estradiol, estrone, and pregnanediol, hormone levels were compared among all three reproductive categories, using 2016–2018 samples (table S2, models 4 to 6). To control for multiple sampling of individuals, we included subject ID as a random intercept in all models. Sexual swellings in chimpanzees coincide with the periovulatory period when sex-steroid and gonadotropin levels are strongly elevated. To compare baseline levels of the different reproductive categories, we omitted samples of females when they exhibited maximal sexual swellings, thereby making statistical comparisons considerably more conservative.

To produce conservative probability values (*74*), we generated Satterthwaite approximations for degrees of freedom using package *lmerTest* (*75*). We set alpha to 0.05 and used the "confint" function to calculate bootstrapped 95% confidence intervals of fixed effects from 1000 simulations. The regression assumption of residual normality was largely satisfied by Box-Cox transforming hormone values before analysis (*76*, *77*). We visually inspected residual plots and qq-plots (*78*) of models 1 to 6 (table S3) fit to all the hormone datasets to assess the normality of residuals and the presence of outliers. This assessment indicated that there were a few extreme observations that fell well outside the model-predicted ranges of hormone values. We then applied a conservative procedure described in Kutner *et al.* (*79*) to identify and exclude outliers from the datasets used in the final analyses. We first calculated the semi-studentized residuals of each observation in the raw data with respect to models 1 to 6. We then excluded any data in which the absolute value of the semi-studentized residual was greater than or equal to 4. This process led us to exclude one sample from the estradiol data, one sample from the estrone data, and one sample from the pregnanediol dataset. No outliers were excluded from the LH or FSH data. The sample sizes in the final hormone datasets are tallied in table S2. The structure of the statistical models (models 1 to 6) used in the analysis of hormone concentrations by reproductive category are listed in table S3.

Analysis of LH and FSH by age

We conducted generalized additive modeling to estimate linear and nonlinear continuous relationships between age and gonadotropin levels (table S4, models 7 to 12). To adjust for repeated samples of individuals, subject ID was treated as a random intercept in all models. Models were fit using the "gam" function of the R package *mgcv* (*80*). The *mgcv* package estimates relationships between predictor and outcome variables using penalized regression splines in which smoother relationships are preferred to more complex or "wigglier" relationships to prevent over-fitting.

In models 7 to 12, the hormone data were log-transformed before analysis, owing to the non-normal distribution and strictly positive ranges of these measures. In models 7 to 9, we specified that the relationship between age and hormone measures must obey a strictly linear relationship, whereas in models 10 to 12, we specified that a nonlinear relationship, constructed using a cubic spline, could characterize the age relationship. We then compared the estimated out-of-sample predictive accuracy of the linear and nonlinear models fit with each sample using AICc.

Comparison of models 7 to 9 to models 10 to 12 shows that treating age as a smoothed predictor variable leads to lower AICc values, representing improved out-of-sample predictive accuracy, for models 10 and 11. In the case of model 12, the penalized regression estimation process in *mgcv* shrunk the spline to a perfectly linear relationship between age and FSH, equivalent to the linear model 9, with an equivalent AICc value. AICc values are reported in table S4, and model coefficients are reported in table S5.

Analysis of estradiol, estrone, and pregnanediol concentrations by age

To investigate the continuous relationship between age and steroid hormone levels, we fit six generalized additive mixed models (models 13 to 18) in which the relationship between age and the hormone value was estimated as a smooth cubic spline using penalized regression estimation. Subject ID was entered into the model as a random intercept to model the possibility of subject-specific differences in mean hormone measures. The structure of these models is reported in table S6, and the coefficients estimated from models 13 to 18 are summarized in table S7. The model-predicted relationships between age and average steroid hormone measures are displayed in Figs. 4 and 5 and figs. S3 and S4.

Comparison of age trends in urinary hormone concentrations in human females and Ngogo females

We calculated average Ngogo female age trends for urinary FSH using model 11 (Fig. 5A), for LH using model 10 (Fig. 5B), for estrone using model 14 (Fig. 5C), and for pregnanediol using model 15 (Fig. 5D). The median age trends for human females for urinary FSH were carefully traced from figure 1B of (*18*), for LH figure 1A

of (*18*), for estrone-glucuronide figure 2A of (*19*), and for pregnanediol-glucuronide figure 2B of (*19*) using WebPlotDigitizer (*81*). Estrone-glucuronide is a metabolite of estrone that humans and chimpanzees excrete in their urine (*82*). In our analyses, following the protocol of (*69*), estrone metabolites were deconjugated to estrone before measurement by LC-MS, whereas in (*19*), immunoreactive estrone-glucuronide was measured directly. Likewise, the major urinary metabolite of progesterone, pregnanediol-glucuronide, was deconjugated to pregnanediol before measurement using LC-MS in our study, whereas immunoreactive pregnanediol was measured directly in (*19*).

There are several data limitations to Fig. 5. First, the error in chimpanzee age estimation would be expected to flatten out steep changes in age trends that might otherwise be detected with more-precise age estimates. Second, Fig. 5 displays only model-expected central trends for each population, ignoring considerable between-individual variability in base rates and change over time that is known to exist from studies of human females [see figure 2 of (*18*)]. Finally, our sample sizes are large by primatology standards but much smaller than those of the human studies.

Age estimation

Ages of the oldest females in the sample, including those found to be post-reproductive, were necessarily estimated because they were adults when first identified. Nevertheless, these estimates are predicated on critical data that together suggest that any error in age estimation does not affect our conclusions.

First, age estimations for females in the study were made when females were first identified, rather than at the time of the current study. For the oldest females in the sample, including those found to be post-reproductive, these determinations were made in 1995–1998, 11 to 23 years before sampling for this study. In other words, it was not the case that females in the study were visually estimated to be >50 years old, but rather they were identified when still young or middle-aged and subsequently observed over many years. Age estimations at the outset of the study benefitted from understandings of visual signs of aging (e.g., gray hair, prominent hips and shoulders, worn or missing teeth) generated by previous chimpanzee research [e.g., long-term research at Gombe (*83*)], and the distribution of these original age estimations for chimpanzees in the Ngogo community were consistent with population structures observed for wild chimpanzees elsewhere. Fortunately, females disperse into new communities within a narrow age range during adolescence, which means that those females who joined the Ngogo community over the course of the study could be aged with minimal error.

In most cases, contextual information allows us to identify the minimum age of females when first observed, on the basis of the presence and size of dependent offspring and/or genetic pedigree data that can identify adult offspring. For example, a female observed with two dependent offspring when first observed should be at least 20 years old. For 4 of the 11 post-reproductive females, they must have already been in their 30s to 40s when first identified in 1995 because they already had genetically identified adult-aged offspring at this time. These estimates will tend to be conservative because first-born offspring may have died or emigrated before the period of genetic sampling. As an illustrative example, consider the oldest post-reproductive female in our sample, MARL. We identified DO as a middle-aged male in 1995 and assigned him the age of 28 on the basis of his physical appearance, making his birth year 1967. Genetic parentage analyses subsequently confirmed our suspicion that adult female MARL was DO's mother. On average, immigrant female chimpanzees have their first offspring at age 16.2 years, which would make MARL's birth year 1951 if we assume DO was her first offspring. However, we instead assigned MARL a birth year of 1950 (and thus age of 45 years in 1995) instead of 1951 in recognition of the possibility that DO was not her first offspring, and because she looked a bit older than females whose age estimates were similarly genealogically informed in 1995. Our age estimate of 28 years for DO in 1995 could not have been considerably overestimated, because when he died in 2014 at an estimated age of 47 years, he had lost considerable muscle mass and all of his canines. Similarly, when MARL died in 2019 at the estimated age of 69, she was very thin, had sparse gray hair, very worn teeth, and had multiple great-grandoffspring.

As noted above, although our age estimates were derived long before this study was conceived, the results help reinforce the validity of the age estimates because ASFR at Ngogo align closely with patterns observed in other chimpanzee populations (Fig. 1), and ages of the menopausal transition conform to the expectations from prior studies of ASFR and follicular depletion rate (Fig. 4).

The statistical significance of post-reproductive representation is robust to error in age estimates

To assess the possible impacts of age estimation error on our measure of post-reproductive representation (PrR) at Ngogo, we created a simulation model that generates alternative schedules of mortality and fertility, embodying a reasonable model of age measurement error. Our model of age estimation error starts with the intuition of human demographers and primatologists that as the actual age of an individual increases, the error in age estimations also increases (*65*, *84*). We treated age measurement error as a process arising from uniform distributions, with ranges that increased proportional to our best point estimate (BPE) of an individual's age when they entered our study. Following (*65*), we used the formula below to specify the relationship between the range of the uniform distribution and our best point estimates

$$\text{Range of uniform distribution} = \text{BPE} * 0.2$$

Applying this model of age estimation error, we generated 1000 data permutations. Among permutations, the simulated ages were distributed across intervals with a minimum value of BPE − BPE∗0.1 and a maximum value of BPE + BPE∗0.1. The scaling factor of 0.2 was chosen because it produces estimates that accord with experience. For example, a chimpanzee with a real age of 10 years might have been visually estimated as being between 9 and 11 years old, while chimpanzees estimated to be ages 20 or 40 when first identified were treated in this model as having probabilistic ages uniformly distributed across the intervals 18–22 and 36–44 years, respectively.

By simulating 1000 permutations of the demographic data, we created 1000 schedules of mortality and fertility. Using these schedules, we then calculated the PrR statistic for each permutation, which resulted in the distribution of values shown in fig. S5.

Among all 1000 data permutations, the minimum PrR value recorded was 0.132. We then applied the analysis procedure described in (*1*) to assess whether even this minimal-PrR value of 0.132 was nevertheless greater than expected under the null hypothesis of parallel declines in survivorship (l_x) and fertility (m_x). We found that the PrR value of 0.132 is indeed higher than expected under the null hypothesis of there being no post-reproductive representation in this community ($P < 0.001$). In summary, while our age estimates may be off by a few years, even when making allowances for such error, Ngogo PrR values are significantly higher than expected if survivorship and fertility declined in parallel in this study population.

Precisely aged individuals show low mortality among the Ngogo chimpanzees

When we subsample our data and only focus on those individuals who were two years or younger when they first entered our mortality risk pool, we see that Ngogo survivorship to young adulthood is higher than in the nearby community of Kanyawara, also located in Kibale National Forest [see figure 2 in (*65*)]. This analysis is particularly informative because age estimation error is minimized in this sample, and comparative demographic analysis indicates that high survivorship in early life

is positively associated with high survivorship across the life course (*85*).

In summary, when making reasonable data adjustments for age estimation error (fig. S6), and when restricting analyses to only those individuals with the highest age accuracy, we find that survivorship among the Ngogo chimpanzees is high and post-reproductive representation is significant. Both age measurement error and sampling variation are thus not adequate alternative explanations for our results.

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ACKNOWLEDGMENTS

We are grateful to the Makerere University Biological Field Station, Uganda Wildlife Authority, and Uganda National Council for Science and Technology for sponsoring our research in Uganda. We thank the Ngogo students, postdocs, and other researchers who have contributed to the chimpanzee demographic database and the late J. Lwanga and S. Angedakin, who served as camp managers for the Ngogo Chimpanzee Project. We are indebted to R. Davenport for sample collection, and to project field assistants C. Aligarya, C. Birungi, D. Kalunga, B. Kamugyisha, D. Kamweri, A. Magoba, G. Mbabazi, L. Ngandizi, A. Tumusiime, and A. Twineomujuni. **Funding:** We are grateful to the funders of research at Ngogo, including the US National Science Foundation (BCS-9253590, IOS-0516644, BCS-0215622, BCS-9253590, BCS-1613393), the National Institute on Aging and the Office for Research on Women's Health (R01-AG049395), the Fulbright Foundation, the Explorer's Club, the National Geographic Society (9824-15), the Detroit Zoological Society, Underdog Films, Silverback Films, University of Michigan, Yale University, Arizona State University President's Strategic Initiative Fund, the Institute of Human Origins, the Max Planck Society, the Wenner-Gren Foundation, and the L.S.B. Leakey Foundation. **Author contributions:** Conceptualization: J.C.M., D.P.W., K.E.L., and B.M.W. Field data collection: J.C.M., D.P.W., K.E.L., S.G., and J.D.N. Hormone measurement: J.L.B., T.D., M.E.T., S.G., and J.D.N. Demographic data analyses: B.M.W. Categorical hormone data analyses: J.D.N. and B.M.W. Continuous hormone data analyses: B.M.W. Writing of original draft: B.M.W. and K.E.L. Major contributions to subsequent drafts: B.M.W., K.E.L., M.E.T., J.C.M., D.P.W., and T.D. Editing: All authors. **Competing interests:** The authors have no competing interests to declare. **Data and materials availability:** The data and code ("all_functions.R") necessary to reproduce all the analyses in this paper are provided in the supplementary materials.

SUPPLEMENTARY MATERIALS

science.org/doi/10.1126/science.add5473
Figs. S1 to S5
Tables S1 to S7
MDAR Reproducibility Checklist
Data S1 to S16

Submitted 17 June 2022; accepted 5 September 2023
10.1126/science.add5473

Demographic and hormonal evidence for menopause in wild chimpanzees

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Science **382** (6669), eadd5473. DOI: 10.1126/science.add5473

Editor's summary

Menopause occurs in all known human societies; however, it is not common to all mammals and has so far only been observed in humans and a few toothed whale species. Wood *et al*. looked at demographic and endocrine data in a long-studied population of chimpanzees in Uganda and found clear evidence for menopause in females living past the age of 50 (see the Perspective by Cant). Unlike the case for humans and toothed whales, however, postreproductive chimps in this population are not involved in the raising of related offspring, suggesting that a different process is driving its development. —Sacha Vignieri