Report

Current Biology

Male-Mediated Maturation in Wild Geladas

Highlights

- Female geladas were 3× more likely to mature after a new dominant male arrives
- New dominant males can accelerate a female's maturation
- The presence of a female's father as the breeding male counters this effect
- New males caused an increase in estrogens for all immature females

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In Brief

This is the first report of male-mediated maturation in a wild primate. Lu et al. find that maturations in wild geladas are more likely after a new male arrives. While new males accelerate maturations, this is countered by overlap with her father. All females, even those too young to mature, showed elevations in estrogens upon new male arrival.

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Report

Male-Mediated Maturation in Wild Geladas

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SUMMARY

The timing of female maturation in wild mammals is often constrained by ecological variables that relate to food acquisition. However, maturational timing in female mammals can also respond to social variables. Specifically, the arrival of novel males can accelerate maturation while the presence of related males can inhibit it. Despite studies on more than two dozen mammalian taxa in captivity, evidence for male-mediated maturation has not been systematically demonstrated in any wild population. Here, we report the first evidence of male-mediated maturation in a wild primate, the gelada (*Theropithecus gelada*). After the arrival of a new breeding male in the group (a male takeover), young females were three times more likely to mature. We then examined these takeover-associated maturations in more detail: some were earlier than expected (a presumptive "Vandenbergh effect," or male-accelerated maturation), some were at the expected age for the average female gelada, and some were later than expected (a presumptive "inbreeding avoidance delay," or father-induced reproductive suppression). An examination of fecal estrogens, which rise just before visible signs of maturation in this species, revealed that male takeovers induced a surge in estrogens for immature females of all ages—even females that did not mature. These are the first data to demonstrate that specific males are associated with the onset of maturation in a wild primate and to provide a possible mechanism for this change. These results suggest that all male-mediated maturation (whether accelerated, on-time, or delayed) may be governed by similar neuroendocrine processes.

RESULTS AND DISCUSSION

More than 50 years ago, John Vandenbergh conducted a series of experiments demonstrating that female mice mature earlier if they are housed with an unrelated adult male.[1] This "Vandenbergh effect"—the acceleration of maturation by the presence of an unrelated male—has since been found in many other rodents,[2–12] one marsupial,[13] several domestic livestock,[14–18] galagos,[19] callitrichids,[20–22] and possibly in hamadryas baboons.[23] In a related (but as yet unnamed) phenomenon, female rodents are known to exhibit a delay in maturation when housed with their male kin.[8,24,25] This delay, generally attributed to inbreeding avoidance, is often immediately lifted upon the female's removal from male kin followed by exposure to a novel male.[8,24,25] However, studies of male-mediated maturation, which includes accelerated maturation due to the Vandenbergh effect and delayed maturation due to father presence, derive almost exclusively from captive studies (and overwhelmingly from rodents) where experimental setups may not accurately recapitulate the process of maturation in natural settings. The only evidence of male-mediated maturation in a wild mammal is from Cape ground squirrels (*Xerus inauris*), where females living in groups with two or more male relatives matured later than other females.[24] This dearth of studies is surprising given that female maturation to social cues has been used to explain a more controversial claim—that father-presence/absence in human families can alter the timing of maturation in young girls.[26] Critically, we have surprisingly limited comparative data from primates more generally and none from primates in natural settings.

Here, we used 14 years of data to test for male-mediated maturation in a wild primate, the gelada (*Theropithecus gelada*). Geladas are ideal candidates for this investigation because we already know that female reproduction is sensitive to the arrival of novel males in this species. For example, female geladas exhibit male-mediated pregnancy termination, or "the Bruce effect,"[27,28] following the arrival of a novel breeding male. Furthermore, novel males also prompt lactating females to resume signs of fertility (sexual swellings) in what appears to be an estrous condition.[29] Additionally, the Vandenbergh effect co-occurred with the Bruce effect in mice and possibly shares a common mechanism.[30] This prompted us to examine whether geladas exhibit a similar co-occurrence. We examined the effects of males on the timing of female maturation in geladas, including the acceleration of maturation due to novel males and the delay

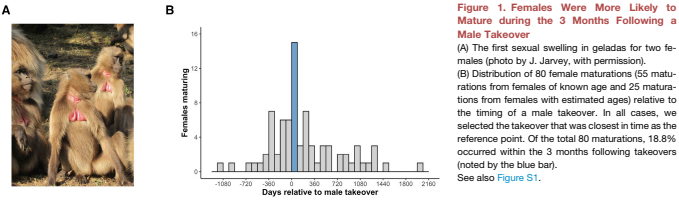

Figure 1. Females Were More Likely to Mature during the 3 Months Following a Male Takeover

(A) The first sexual swelling in geladas for two females (photo by J. Jarvey, with permission).

(B) Distribution of 80 female maturations (55 maturations from females of known age and 25 maturations from females with estimated ages) relative to the timing of a male takeover. In all cases, we selected the takeover that was closest in time as the reference point. Of the total 80 maturations, 18.8% occurred within the 3 months following takeovers (noted by the blue bar).

See also Figure S1.

of maturation due to the prolonged presence of fathers as primary breeding males.

To test for male-mediated maturation, we collected demographic and behavioral data from a population of geladas living in the Simien Mountains National Park, Ethiopia. Geladas are catarrhine primates that live in polygynous family units (''reproductive units'') comprising one dominant breeding male (''leader male''), 1–12 related adult females, and their dependent offspring.[31] Male reproductive success depends on maintaining reproductive control over the unit. Threats to leader males come from ''bachelor'' males residing in all-male groups, that may challenge and defeat leader males (''male takeover''), allowing successful bachelors to rise to the position of the leader male.[32] Male takeovers are semi-seasonal, and each male is replaced approximately every 2.74 years (89 takeovers across 228 unit-years of study)—usually well before his daughters reach maturity around 5 years of age. However, in this population, both male tenure (<1 month – 8.1 years, N = 67) and female age at maturation (3.5–6.5 years, N = 80) are highly variable, creating the potential for overlap between a father and his maturing daughter. We recorded female maturations based on the first signs of sex skin swelling on a female's chest.[33] Such swellings are conspicuous and are tightly correlated with changes in fecal estrogens (Figure 1A; see also Figure S1), indicating that they serve as an accurate morphological proxy for reproductive maturation.

First, we analyzed demographic data from 80 immature females across 28 male takeovers during a 14-year period (January 2006–July 2019) to determine if maturations increased during the 3 months after the arrival of a new male (''takeover window''). We selected this window because most changes in adult female reproductive physiology in response to male takeovers occurs within 3 months following the takeover.[29] Indeed, female maturations peaked following male takeovers (Figure 1B), occurring three times more often within the takeover window compared to other months (Cox proportional hazards: HR = 2.86, $z = 3.25$, $p = 0.001$). Females only matured in the takeover window if they were at least 3.5 years of age. Of all immature females (that were at least 3.5 years) that experienced a male takeover, 53.6% (15/28) matured in the next 3 months. Moreover, the effect of takeovers remained stable across the range of maturation ages (Schoenfeld residuals: $\chi^2 = 0.53$, $p = 0.47$).

By contrast, females were less likely to mature when their fathers were still the leader male in their social unit (Cox proportional hazards: HR = 0.66, $z = -4.49$, $p < 0.0001$; Figure 2A). The suppressive effect of fathers significantly weakened as females aged (Schoenfeld residuals: $\chi^2 = 4.66$, $p = 0.03$). Across all females, we found that male takeovers accelerated maturation by an average of 4.6 months (linear mixed model: $\beta = -0.383$, $t = -2.80$, $p = 0.007$; N = 80; Figure 2B; see also Table S1), and father-presence (at the minimum age at maturation, 3.5 years) delayed maturation by an average of 5.2 months ($\beta = 0.434$, $t = 3.61$, $p = 0.0005$). Taken together, while male takeovers generally accelerate the onset of female maturation, the presence of their father as the primary breeding male counters this effect.

Estrogens Increased for All Immature Females Immediately after Male Takeover

Next, we investigated one possible mechanism for male-mediated maturation in geladas—male-induced elevation in female estrogens. In prepubertal mammalian females, estrogens generally exert negative feedback on the hypothalamic-pituitary gonadal (HPG) axis, preventing the gonadotropin releasing hormone (GnRH) surges that are necessary to initiate maturation.[34] As females approach the pubertal transition, however, the influence of estrogens on the HPG axis switches from an inhibitory to a stimulatory one, a switch that initiates the development of the female reproductive tract, uterine growth,[35] functional menstrual cycles, sexual behavior, and—for some primates—the development of sexual swellings.[36] The surge in estrogens that accompanies reproductive maturation is largely the result of mature ovaries; however, evidence suggests that administering estradiol to prepubertal females can actually hasten this process. In separate experiments, administering exogenous estradiol to juvenile female mice promoted the growth of the reproductive tract[37] and induced the Vandenbergh effect.[38] Currently, it is unclear whether estradiol directly influences GnRH sensitivity or simply acts after the central ''switch'' has occurred.

To examine whether a similar estradiol-based mechanism might be occurring in geladas, we analyzed hormonal data from 51 juvenile females, including 42 females with a known date of maturation and 9 females that had not yet matured by the end of this study. Using radioimmunoassay of fecal estrogens,[27] we tested if the arrival of a new male (a male takeover) increased female fecal estrogen metabolites for immature females during the immediate 30 days following a male takeover (the physiological effects of males are expected to be immediate, preceding subsequent effects on sex skin swellings).

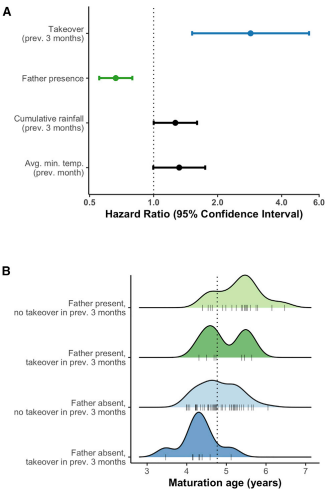

Figure 2. Females Are More Likely to Mature in the 3 Months Following Male Takeovers and Less Likely to Mature when Fathers Are Still Present in the Social Unit

(A) Forest plot of survival model-predicted hazard ratio and 95% confidence intervals. Ecological control variables showed slight trends: females tended to mature at times when there was more cumulative rainfall and higher minimum temperatures (i.e., when grass is highly available and when cold stress is minimal). (B) Distributions of age of female maturations (N = 80). Maturations are separated into four groups based on whether females' fathers were absent (blue) or present (green) at the minimum age at maturation (41.7 months, or 3.5 years) and whether females matured within the 3 months following (dark blue/green) or not following (light blue/green) a male takeover. The dashed line indicates the median age at maturation for all females.
See also Table S1.

Outside of male takeovers, estrogen metabolites for these juvenile females ranged from 0.06–10.31 ng/g; however, following male takeovers, these values more than doubled (pre-takeover mean = 1.47; post-takeover mean = 3.15 ng/g; Figure 3A). This observation parallels those in mice,[38,39] where novel males trigger an immediate rise in estrogens for immature females. These effects were not limited to females of maturational age. We found that estrogens increased in immature females of all age classes ($\beta = 0.54$, $t = 8.54$, $p < 0.0001$; Figure 3B; see also Table S2)—even females as young as 1 year old. Notably, no gelada females under 3.5 years of age matured in response to male takeovers, indicating that this brief rise in estrogens is not, by itself, sufficient to induce maturation in the youngest females.

Although the surge in estrogens that accompanies typical mammalian maturation derives from endogenous sources (e.g., ovaries, adrenals), most examples of Vandenbergh-stimulated maturation identified in mice derive from exogenous sources (e.g., male urinary estrogens).[30,40,41] We currently do not know whether the gelada spike in estrogens after male takeovers is due to *endogenous* or *exogenous* sources. However, we propose that they are endogenous for two reasons. First, catarrhine primates, like geladas, do not have a functioning vomeronasal organ,[42] the primary structure that is known to process chemosignals.[43] Second, geladas live in large societies of up to 1,200 individuals,[41] making it difficult for a system of chemical communication to target a specific individual. Catarrhine primates have largely replaced chemosensory processing used by most other mammals with cognitive processing of the surrounding information.[44] Therefore, it is more likely that the increase in estrogens derives from socio-cognitive input (e.g., understanding that a new breeding male has arrived), followed by downstream neuroendocrine changes in the HPG axis that parallel those confirmed in other mammals (e.g., the stimulation of GnRH surges).[45] Regardless of the source, it appears that females must be physiologically "ready" (i.e., have reached some critical age/size threshold) before males can stimulate reproductive maturation, since the youngest females did not mature despite the rise in estrogens. Moreover, even for immature females that have reached the critical threshold (where presumably estrogens have switched to a stimulatory role), they may nonetheless stall reproductive maturation a bit longer until optimal sociosexual cues are present.

Based on these results, we propose that all forms of male-mediated maturation (that is, *accelerated* maturation characterized by previous definitions of the Vandenbergh effect and *delayed* maturation characterized by prolonged exposure to male kin) may be governed by the same (or similar) neuroendocrine processes, regardless of whether maturation takes place early, on-time, or late.

Possible Adaptive Explanations for Male-Mediated Maturation

Despite being documented in about two dozen mammalian taxa, we currently have no evidence that male-mediated maturation is adaptive for females. Here, we speculate on three possible explanations that may be at work in geladas.

First, if a sexually mature male is available, females that accelerate maturation should reduce the time to their first birth.[46] Although it has not been tested with empirical data, this has been the putative adaptive explanation for the Vandenbergh effect in short-lived species, where current reproduction is favored over longevity (i.e., survival).[47] However, long-lived species, like primates, are largely characterized by investment strategies that maximize longevity.[48] Indeed, gelada females that matured earlier did have an earlier first birth ($\beta = 0.86$, $t = 7.75$, $p < 0.0001$; $N = 62$) and a ~4-month head start on reproduction. However, this translates to a meager 0.14 offspring advantage (based on an interbirth interval of 2.44 years) across a lifetime, all else being equal. Therefore, we are unconvinced by this explanation for geladas (or any other primate).

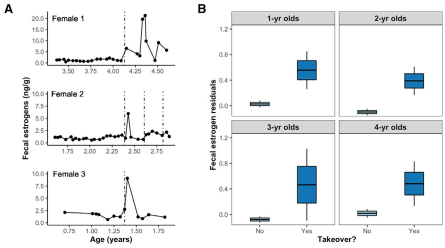

Figure 3. Male Takeovers Induced an Increase in Estrogen Levels in all Prepubescent Females

(A) Fecal estrogen metabolites for three representative adolescent females at different ages (Female 1 is 4 years old, Female 2 is 2.5 years old, and Female 3 is 1.5 years old) relative to the timing of male takeovers (indicated by a dashed line). More than one dashed line indicates multiple takeovers.

(B) Residual fecal estrogen metabolites for females at different ages (1–4 years of age) either outside of ("no") or within the 30-day window after a male takeover ("yes") Fecal estrogens presented as residuals controlling for female age, cumulative rainfall (previous 90 days), average minimum temperature (previous 30 days), whether the sample was collected within the 100 days before the female's first swelling, and methodological differences in sample extraction. Mean = solid line, standard error = box outline, 95% confidence interval = whiskers.

See also Table S2.

The second explanation focuses on optimal timing. Females that sync their maturation with the arrival of a new male may be able to mitigate two potential sources of intersexual conflict. One source of conflict is the potential cost of inbreeding if females mate with their fathers. Under this scenario, females suppress maturation when their fathers are present to prevent inbreeding,[49,50] but sensitivity to novel males then allows them to lift reproductive suppression the moment an unrelated male is available. Their fathers often remain in the group even after being taken over, so although there is potential for reproductive suppression to continue, females that are simultaneously sensitive to cues from a novel male can lift this suppression immediately.

Optimal timing may mitigate another source of intersexual conflict—the costs paid by pregnant and lactating gelada females after a male takeover. More than 80% of pregnant females immediately abort (i.e., the Bruce effect, spontaneous abortion in response to a novel male[27]) and nearly 50% of lactating females lose their infants to infanticide (i.e., males killing dependent infants[51]). Selection may, thus, favor females that are able to initiate a reproductive event in response to a new male[27] since this maximizes their time for gestation and weaning an infant before the next male takeover. We found modest support for this hypothesis: females that matured following a male takeover did indeed gain more time to conceive, gestate, and wean their first offspring before the next male takeover (linear mixed model: $\beta = 359.4$; $t = 2.56$, $p = 0.01$; $N = 63$). Such females were takeover-free for 1,087 days (2.98 years, SE = 160 days) following their maturation, while all other females ("control females") were takeover-free for only 727 days (1.99 years, SE = 116 days). Based on the average length of adolescent sterility and gestation,[33] these "takeover-free females" are expected to have about a 1-year-old infant at the time of the next takeover while control females are expected to have (vulnerable) newborns. Although 1 year of age is younger than the average age at weaning (~1.5 years) for geladas, the likelihood of infanticide in the first 3 months following a takeover dramatically decreases after an infant has reached 1 year of age (Figure 4). This suggests that maturational sensitivity to novel males may indeed provide these females with a timing advantage that gets them through the most vulnerable period.

However, none of these explanations is entirely satisfactory. Moreover, a recent study on yellow baboons (*Papio cynocephalus*) demonstrated that the fitness effects of a longer life far outweigh the fitness effects of an accelerated reproductive schedule.[52] This opens the door to a third hypothesis: that male-mediated maturation is not adaptive but simply the

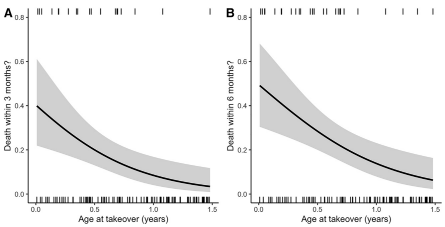

Figure 4. The Risk of Infanticide Following Male Takeover Decreases with Age

This result holds true when including deaths within 3 months (A; N = 17 deaths) or 6 months (B; N = 21) of a takeover event (N = 63 takeover events across 95 individuals under 1.5 years of age). Black lines indicate the predictions from a binomial generalized linear model.

byproduct of selection for females to retain sensitivity to new breeding males. Specifically, the Bruce effect (male-mediated pregnancy loss) can mitigate the costs of infanticide across the life course.[53] We have already demonstrated that the Bruce effect is adaptive for pregnant geladas.[27] Moreover, studies in mice have suggested that the Vandenbergh and Bruce effects are mediated by a similar neuroendocrine mechanism.[30,41] Male-mediated maturation could therefore be driven by strong selection for the Bruce effect. Testing this "piggy-back" hypothesis will depend on further characterization of the neuroendocrine mechanism for both Vandenbergh and Bruce effects as well as a proper comparative analysis that depends on data from additional mammalian taxa.

Conclusions

We found that (1) male-mediated maturation occurred in a wild primate, and (2) the effect is accompanied (and possibly mediated) by a surge in estrogens. These data indicate that maturation in a non-human primate is highly sensitive to the identity of potential male mates, with novel males accelerating and biological fathers delaying pubertal onset. Taken together, we propose that all forms of male-mediated maturation (whether early, on-time, or late) be considered together in future studies on mechanism and function (and that it may be necessary to reconsider a new definition for the Vandenbergh effect altogether). We also lay out several hypotheses for the evolution of this phenomenon that can be tested with further comparative data.

STAR★METHODS

Detailed methods are provided in the online version of this paper and include the following:

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- EXPERIMENTAL MODEL AND SUBJECT DETAILS
- METHOD DETAILS
  - Hormone collection, extraction, and analysis
- QUANTIFICATION AND STATISTICAL ANALYSIS

SUPPLEMENTAL INFORMATION

Supplemental Information can be found online at https://doi.org/10.1016/j.cub.2020.10.003.

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AUTHOR CONTRIBUTIONS

Conceptualization, A.L. and J.C.B.; Data Curation, A.L., N.S.-M., T.J.B., and J.C.B.; Formal Analysis, A.L., J.A.F., and J.C.B.; Funding Acquisition, A.L., N.S.-M., T.J.B., and J.C.B.; Investigation, A.L., J.A.F., and J.C.B.; Methodology, A.L., J.A.F., and J.C.B.; Project Administration, A.L., N.S.-M., T.J.B., and J.C.B.; Resources, A.L., N.S.-M., T.J.B., and J.C.B.; Supervision, A.L., N.S.-M., and J.C.B.; Validation, A.L. and J.C.B.; Visualization, J.A.F.; Writing – Original Draft, A.L., J.A.F., and J.C.B.; Writing – Review & Editing, A.L., J.A.F., N.S.-M., T.J.B., and J.C.B.

DECLARATION OF INTERESTS

The authors declare no competing interests.

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STAR★METHODS

KEY RESOURCES TABLE

REAGENT or RESOURCE	SOURCE	IDENTIFIER
Critical Commercial Assays		
17β-estradiol Double Antibody RIA kit	MP Biomedicals	0714020-CF
Experimental Models: Organisms/Strains		
Theropithecus gelada	Simien Mountains National Park, Ethiopia	N/A
Software and Algorithms		
R 3.6.0	Tinsley Johnson et al.[29]	https://www.r-project.org/
survival v2.44-1.1	Lu et al.[54]	https://cran.r-project.org/
lme4 v1.1-20	Snyder-Mackler et al.[55]	https://cran.r-project.org/
lmerTest v3.0-1	Jarvey et al.[56]	https://cran.r-project.org/

RESOURCE AVAILABILITY

Lead Contact

Further information and requests for resources and reagents should be directed to and will be fulfilled by the Lead Contact, Jacinta C. Beehner (email: jbeehner@umich.edu).

Materials Availability

This study did not generate any new products.

Data and Code Availability

Data and code are available at https://github.com/GeladaResearchProject/Lu_Vandenbergh_2020

EXPERIMENTAL MODEL AND SUBJECT DETAILS

The data in this study were collected from a population of wild geladas in the Simien Mountains National Park, Ethiopia. The Simien Mountains Gelada Research Project has monitored this population on a near-daily basis since January 2006, collecting demographic, behavioral, and hormone data. All gelada subjects are individually recognized and habituated to the presence of human observers.

METHOD DETAILS

Hormone collection, extraction, and analysis

Fecal samples (N = 1506) were collected within minutes after deposition from positively-identified individuals. Hormones were then extracted from feces using a method described previously.[54] Specifically, the entire fecal sample was mixed thoroughly with a wooden spatula, and an aliquot of the mixed sample (~0.1 g wet feces) was placed in 3 mL of a MeOH/acetone solution (4:1). The solution was immediately homogenized for 1 min using a battery-powered vortexer (BioVortexer, BioSpec Products, Inc., Bartlesville, OK). Approximately 6-8 h later, 2.5 mL of the fecal homogenate was filtered through a 0.2 μm polytetrafluoroethylene (PTFE) syringeless filter (Whatman, Florham Park, NJ), and the filter was subsequently washed with an additional 1 mL of MeOH/acetone (4:1). We then added 7 mL of distilled water to the filtered homogenate, capped and mixed the solution, and loaded it onto a reverse-phase C_{18} solid-phase extraction cartridge (Sep-Pak Plus, Waters Corporation, Milford, MA). Prior to loading, Sep-Pak cartridges were prepped according to the manufacturer's instructions (with 2 mL MeOH followed by 5 mL filtered water). After the sample was loaded, the cartridge was washed with 1 mL of a sodium azide solution (0.1%). A subset of samples were washed with a 20% MeOH solution (N = 371), and this difference was accounted for in all statistical models ("methodological differences"). After loading hormone metabolites on the cartridges, cartridges were allowed to dry at ambient temperature for one week, after which they were stored in Whirl-Pak bags containing ~2 g of silica beads at subzero temperatures (−10ϸC) until transported to the University of Michigan for analysis. In the laboratory, steroids were eluted from cartridges with 2.5 mL 100% MeOH and subsequently stored at −20ϸC until the time of assay. Dry fecal weights from all samples were obtained to the nearest 0.001 g.

All samples were assayed for 17b-estradiol (E2) using a radioimmunoassay (RIA) kit produced by MP Biomedicals. Prior to RIA, all samples were incubated at room temperature for one h. Then, an aliquot of each sample was evaporated to dryness under nitrogen.

Sample aliquots were determined such that hormone metabolite values were within the range of optimal precision of the assay. Kit protocols were followed except that all reagents were halved from the amount suggested by the manufacturer (a common technique employed by researchers measuring fecal steroids to maximize the use of each kit). Internal controls were run in every assay and consisted of a high (binding at 30%) and a low (binding at 70%) ''pool'' (a composite of many fecal samples). All standards were run in triplicate, all controls and samples were run in duplicate, and mean concentrations are expressed as ng per dry gram of fecal material (ng/g). The MP Biomedicals E2 antibody is known to have minor cross-reactivities with other estrogen metabolites (estrone: 20%; estriol: 1.5%; estradiol-17α: 0.7%). Inter-assay CVs for a low and high sample were 13.4% and 14.2% (N = 40) respectively, and intra-assay CVs for the equivalent were 8.74% and 14.73%, respectively (N = 10). Assays were conducted in laboratories at both the University of Michigan (N = 777 samples) and Stony Brook University (N = 729 samples), and this difference was accounted for in all statistical models (''methodological differences'').

QUANTIFICATION AND STATISTICAL ANALYSIS

First, to assess the influence of male takeovers on the timing of first sexual swelling (N = 80), we conducted survival analysis using a time-varying Cox proportional hazards model. For this analysis, females entered the dataset at 3.4 years (40.8 months) of age, just prior to the earliest age at maturation and were modeled on a monthly basis until their maturation. Females' birth dates were either known (N = 55) or estimated based on coat color (N = 23) or juvenile size (N = 2).[54] For each female-month, we included male takeover status and father presence as binary fixed effects. Based on previous studies,[29] we assigned ''takeover status'' as ''yes'' if a female had been taken over within the previous three months. We assigned ''father presence'' for each female-month. Because leader males sire between 83%–100% of all unit offspring,[55] a female's father was assigned as the leader male at the time of her conception. In some cases (N = 6 females), females experienced multiple male takeovers in quick succession, resulting in more than three consecutive months of ''yes'' for her takeover status. Also, given that the effect of father presence was non-proportional (as determined via Schoenfeld residuals), we used a time transformation on this predictor, assuming linear change in its hazard ratio over female age. To control for environmental conditions, we also included cumulative rainfall (previous 3 months) and average minimum temperature (previous month) as fixed effects, as these respectively are reliable proxies for grass availability and thermoregulatory stress.[29,56] To control for repeated-measures of individual females, we included a cluster option on female identity. Survival models were constructed using the R package 'survival'[57]. In a parallel analysis, we determined whether females that matured in response to males (i.e., those that mature within three months of a male takeover) matured earlier than others, using a linear mixed model (LMM). Here, we used two fixed effects: whether fathers were present at the earliest documented age at maturation (3.48 years) and whether the female matured within three months following a male takeover. For all mixed models, reproductive unit was included as a random effect.

Second, to determine the immediate physiological effect of male takeovers, we constructed a linear mixed model (LMM), using logged fecal estrogens concentrations (ng/g) as the outcome. Takeover status (previous 30 days), cumulative rainfall, (previous 90 days), average minimum temperature (previous 30 days), subject age, wash step (sodium azide versus methanol), and laboratory (UM or SBU) were included as fixed effects. Additionally, in order to control for whether the female was in the process of maturing, we included whether the sample was collected within 100 days prior to the female's maturation as a fixed effect. This allowed us to confirm whether all females experienced increased fecal estrogens following male takeovers, regardless of whether they were in the process of maturing. Both individual and unit ID were included as random effects. The residuals from the resulting model were normally distributed. All LMMs were constructed and assessed using the R packages 'lme4' and 'lmerTest'[58,59].

Next, we quantified three potential benefits of male-mediated maturations: First, we examined whether females with male-mediated maturation gave birth earlier on average than females without male-mediated maturation To do this, we determined the relationship between age at maturation and age at first birth using a linear model. We then extrapolated the net effect of male-mediated puberty by multiplying the earlier maturation advantage conferred by male-mediated puberty (4.6 months) by the delay in first birth for every month of delayed maturation (i.e., the slope from the linear model). Second, we investigated two potential consequences of *timing* maturations to the arrival of novel males. Specifically, we constructed an LMM using male-mediated status (Y/N) as a fixed effect to determine whether females with male-mediated maturation (N = 11) had longer intervals than control females (N = 52) from the day of maturation until the next male takeover, using unit as a random effect. We excluded male takeovers within 60 days of maturation, since no females had conceived within such a short interval following maturation, and therefore these females would not incur reproductive costs.

For all survival models and linear models, variance inflation factors (VIFs) were less than 2.0. All statistical analyses were performed in R v.3.6.0.[60]