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RESEARCH ARTICLE

Developmental Canalization in Squirrel Monkey (*Saimiri bolivienesis*) Body Mass

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ABSTRACT

Animal growth is often described as self-correcting or canalized, with most vertebrate evidence coming from laboratory rodents, domesticated livestock, and human auxological research. Non-human primate models for developmental canalization are necessary given the long duration and multi-phasic nature of primate growth that limit comparisons with these taxa. We explored patterns of body size growth in captive female and male squirrel monkeys (*Saimiri boliviensis*) through calculation of age- and sex-specific coefficients of variation (CV). These are further decomposed with quantitative genetic models to estimate the heritability of and maternal effects on mass at each age. We also deployed a recently proposed method by Mitteroecker and Stansfield (2021) to estimate the strength of canalization at each age (θ). We found that canalization changed across ontogeny in squirrel monkeys, but the pattern was largely similar between the sexes. Strong canalization resulting in a reduction of phenotypic variance only appeared entering the juvenile period. Prior to this, growth was more variable. Size differences among neonates became greater during infancy (i.e., "negative canalization"). Quantitative genetic models showed significant additive genetic variation for body mass at all ages (h^2, 0.27–0.74), but maternal identity effects were only prominent on neonate mass ($m = 0.28$). This reduction in maternal variance with age and increased canalization likely results from changes in the hormonal regulation of growth and attainment of nutritional independence late in the infant period.

1 | Introduction

Somatic development has often been characterized as target-seeking or self-correcting. Individuals relatively large or small at one age tend to be less extreme at later ages. There is abundant evidence for this phenomenon from human auxological research (Tanner 1986; Tanner 1963) where this self-correcting tendency is referred to as catch-up or catch-down growth following some nutritional, hormonal, or other pathological perturbation. Similar patterns are documented in the animal breeding and animal ecology literature, although most of this work on mammals has been performed with laboratory rodents and domesticated ungulates (Marcil-Ferland et al. 2013; Willmore et al. 2007; Wilson and Réale 2006; Flatt 2005; Riska et al. 1984).

We follow Flatt (2005) in defining developmental canalization as "reduced sensitivity of a phenotype to changes or perturbations in the underlying genetic and nongenetic factors that determine its expression." Theoretical models and empirical work on developmental canalization emphasize a complex interplay between genetic and environmental inputs that ultimately generate phenotypes that are robust to perturbations (Hallgrimsson et al. 2019; Wagner et al. 1997; Waddington 1957; Waddington 1942; Schmalhausen 1949). While the precise mechanisms underlying developmental canalization remain

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unclear, they likely mask abundant cryptic genetic variation that can be expressed under altered environmental conditions or genetic background (Nijhout 2025; Flatt 2005). Stabilizing selection for an intermediate optimum phenotype, such as body size, is often implied or assumed to be the ultimate cause of developmental canalization, though the complexity of developmental processes likely plays an important role as well (Siegal and Bergman 2002; Rice 1998).

Primates are unusual among mammals for their especially long and multi-phasic growth periods (Schmitt et al. 2018; Pereira and Leigh 2003; Zullinger et al. 1984; Watts and Gavan 1982). The longer growth period might act as a buffer against developmental perturbations by allowing greater opportunity for catch-up or catch-down growth and decoupling of structures growing at different times (Riska and Atchley 1985). On the other hand, a longer growth period may be a risk as it presents additional duration for perturbations to occur (Williams 1966). Prolonged, multi-phasic growth periods may also allow primates to mitigate some of the frequently documented fitness costs of catch-up growth (Hector and Nakagawa 2012), although the effects of early life stressors likely vary tremendously among growth periods (Berghänel et al. 2017). Approximately, ontogenetic changes in hormone concentrations are a possible mechanism for phase transitions and for modulating the growth rates that contribute to canalization (Bernstein et al. 2020; Sabbi et al. 2020; Conley et al. 2012; Karlberg et al. 2003).

Squirrel monkeys (*Saimiri*) are a well-characterized taxon for studying primate growth with a long history of captive biomedical study and solid knowledge of their natural history in the wild (Stone and Williams 2021; Blomquist and Williams 2013; Zimbler-DeLorenzo and Stone 2011). They are small, frugi-insectivorous Neotropical primates weighing about 1 kg as adults. Compared to other Neotropical primates, they are relatively large-brained and late maturing with very high maternal investment pre- and post-natally (Hartwig 1996) and marked seasonal changes in diet and social organization in the wild (Zimbler-DeLorenzo and Stone 2011; Jack 2011). Previous growth studies on captive animals showed adult sexual dimorphism is achieved primarily by a longer male growth period (bimaturism) with smaller sex differences in growth rates (Garber and Leigh 1997; Kaack et al. 1979; Long and Cooper 1968). Adult male size is further complicated by temporary buildup of fluid and fat in the thorax, shoulders, and arms ("fattening") along with increased testicular size and function during the breeding season (Vasconcelos Sampaio et al. 2022).

Experimental studies of developmental canalization have typically relied on comparison of variance between wild-type and mutant strains subjected to perturbing environmental factors (Willmore et al. 2007; Waddington 1942). Observational research often relies on changing variance across age or stage classes, perhaps with additional comparisons among taxa or populations (Willmore et al. 2006; Young 2006; Hallgrimsson et al. 2002). However, because of large-scale changes in the mean through ontogeny, this requires a standardized measure such as a coefficient of variation (CV). Mitteroecker and Stansfield (2021) developed novel univariate and multivariate methods to describe developmental canalization in longitudinal growth measurements. While their model envisions development as a complex Ornstein-Uhlenbeck, mean-reverting stochastic process, it provides simple predictions about the regression of trait (x_t) and trait change ($x_{t+1} - x_t$) between different time points (t) during development. They demonstrated this with human radiographic data, but it has yet to gain broader use.

We applied the univariate method of Mitteroecker and Stansfield (2021) to a large database of squirrel monkey body mass measurements collected over four decades. In addition to describing canalization with their method, we explored causes for developmental trajectory heterogeneity, including maternal and additive genetic effects. Captive data offer many advantages for this analysis. All individuals are reared in relatively standardized environments, very large numbers can be measured, and known parentage links measured individuals through a multigenerational pedigree that allows estimation of heritabilities and other quantitative genetic statistics.

We had three objectives in this study. First, to interface with previous observational research on developmental canalization, we used comparisons among ages to gauge how phenotypic variation in body mass changed through normal ontogeny in squirrel monkeys. Because of long, multi-phasic primate growth periods, we hypothesized age-heterogeneity in amounts of variation. Second, we compared traditional measures of variation (CVs) used in canalization research to the new measure (θ) for the strength of canalization. Given the novelty of the method and mixture of results with different craniofacial traits studied by Mitteroecker and Stansfield (2021), we made no predictions about how these would correspond to one another for squirrel monkey body mass. Finally, we used quantitative genetic statistics to describe the age course of different sources of variation. We hypothesized maternal effects would decline with offspring age as they increased in independence, while genetic effects would likely remain prominent throughout postnatal life.

2 | Methods

All animals used in this research were maintained in accordance with the regulations set by the U.S. Department of Agriculture and the National Institutes of Health Guide for the Care and Use of Laboratory Animals. Protocols for this study adhered to the Principles for the Ethical Treatment of Non-Human Primates established by the American Society of Primatologists and were approved by The University of Texas MD Anderson Cancer Center Institutional Animal Care and Use Committee.

We used body mass and associated demographic records of captive Bolivian squirrel monkeys (*S. boliviensis*) from the Squirrel Monkey Breeding and Research Resource (SMBRR) housed at the University of Texas MD Anderson Cancer Center Michael E. Keeling Center for Comparative Medicine and Research (KCCMR) in Bastrop, Texas. Before giving birth, monkeys are housed in social groups of 15-35 animals containing one adult male and 10-15 adult females with their offspring. Current housing consists of indoor pens measuring

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approximately 4.3 × 1.2 × 2.0 m, with an opening to allow large groups access to multiple pens. Social groups have access to one to three pens, depending on their size. Previous housing at University of South Alabama was similar (Williams et al. 2002; Nehete et al. 2021; Abee 2000).

A commercial New World Primate diet supplemented with chopped vegetables (celery, bell peppers, squash, beans) is provided to all animal, with grapes, peanuts, and mealworms fed sparingly as positive reinforcers when animals present for clinical observations. Water is available *ad libitum*. The light-dark schedule is maintained to track the local sunrise and sunset, so animals are exposed to long and short days annually.

Animals were weighed on a commercial small animal scale to the nearest gram. Neonate mass was collected within the first 72 h post-partum to assess the clinical condition of the dam and newborn (Blomquist and Williams 2013). Later juvenile and adult masses were recorded during routine or clinical veterinary exams or animal processing events (i.e., tagging or tattoos).

We limited our analysis to 1165 monkeys who were live, singleton, non-Cesarean birth from January 1980 to September 2024. Any measurements from pregnant females or fattened males were omitted. All animals belonged to the *S. boliviensis boliviensis* subspecies (Lynch Alfaro et al. 2015). An additional 381 monkeys were included as members of the pedigree for quantitative genetic analysis.

Five age classes were constructed based on dental and skeletal criteria and practical considerations of sex- and age-specific sample sizes with the database. The age classes were neonate (0–3 days), infant (≈3 months, 0.2–0.3 years), juvenile (≈9 months, 0.65–0.85 years), dentally adult (≈2.4, 2.3–2.6 years), and skeletally adult (≈5, 4.4–5.4 years) (Galliari 1988; Galliari and Colillas 1985; Manocha 1979). The second and third age classes relate to skeletal, dental, and brain size juvenility that are achieved by 18 weeks (0.35 years). These two classes capture size just before and a few months afterwards. Weaning also falls between these two classes, occurring around 5 months within SMBRR. Males may continue to gain mass after the final age class used here, but very few are kept in the colony for breeding, which severely limits sample size.

We initially removed any measurements less than 20 g and greater than 2000 g as unrealistic data entry errors. We then removed any remaining measurements that had modified *z*-scores for their sex and age class greater than 5 (Iglewicz and Hoaglin 1993). These were considered likely data entry errors or reflective of pathology and this is a common threshold for data filtering in human growth research (e.g., Heude et al. 2019). Finally, because some animals had more than one measurement within an age class, we selected their individual median for that age class. All analyses were performed in R v4.6 (R Core Team 2025). The final set of 1165 monkeys were measured in at least two of the five age classes.

Mass measurements for all but the neonatal age class were collected over a variety of ages within an age class. These could span several weeks to several months. We tested for this effect using separate linear regressions of mass on age in days with a sex interaction for each non-neonatal age class (the lm() function in R). These were significant for all but the final skeletally adult age class. Residuals for each monkey from these significant regressions were then added to the predicted value at the midpoint age of the age class to generate "age corrected" data.

To describe canalization, we report age- and sex-specific coefficients of variation ($CV = 100\frac{\sqrt{Var(x)}}{\bar{x}}$). We tested for differences in CVs between sexes with functions in the cvequality R package (Marwick and Krishnamoorthy 2019). We also carried out the univariate canalization analysis proposed by Mitteroecker and Stansfield (2021). This is a series of linear regressions of mass increase on previous mass (i.e., $mass_{t+1} - mass_t = \beta_0 + \beta_1 mass_t$). The slope ($\beta_1$) from each regression is interpreted as the strength of canalization $\theta = -1 \cdot \beta_1$ with positive values of θ indicating self-correcting, canalized development. Negative regression slopes (β_1) describe a case in which relatively small monkeys gain relatively more mass (catch-up growth) and relatively large ones gain relatively less (catch-down growth). Because this is what is predicted under canalization, it has a positive sign for the measure of canalization, θ. Negative θ suggest inverse canalization, though interpretation is complex. We checked for heavily influential points in the regressions with scatterplots and influence statistics but did not remove any. We used a custom R function for this portion of the analysis. It is available in the supplementary material along with anonymized data and additional R code.

Four different canalization analyses can be carried out depending on how sex is treated. Two separate sex analyses can be compared to results where both sexes' data are simply combined ("pooled") or when they are adjusted for mean differences in each age class ("adjusted"). Because heterogeneity in target growth trajectories will depress θ, sexual dimorphism makes pooled analysis particularly problematic. Sexual size dimorphism is very limited in *Saimiri* neonates but becomes dramatic at later ages (Table 1).

TABLE 1 | Descriptive statistics for Saimiri body mass (g) by sex and age classes.

	Females					Males				
Age class	***N***	**Mean**	**SD**	**Min**	**max**	***N***	**Mean**	**SD**	**Min**	**Max**
Neonate	385	106.37	12.73	66	146	458	112.28	14.17	73	182
Infant	156	254.56	46.89	126	342	195	271.08	50.55	156	416
Juvenile	385	454.42	41.28	306	592	437	498.10	49.98	244	637
Dental adult	284	642.94	54.43	480	824	316	752.99	78.17	456	991
Skeletal adult	403	722.76	63.10	492	927	109	1015.28	132.44	682	1442

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We addressed individual variation in growth trajectories in two ways. First, we considered maternal age effects through the regression of mass on maternal age. This was carried out with orthogonal polynomial quadratic regression with a sex interaction. Animal age within age classes was also included for the classes where needed (see above). The analysis was necessarily restricted to a subset of those monkeys in the canalization analysis because not all measured animals had known dams and some dams did not have known ages.

For animals with known dams, we also explored quantitative genetic models. These decompose phenotypic variation in body mass into additive genetic and maternal identity variance components (Moore et al. 2019; Wilson et al. 2010). These were run separately for each age class and included sex as a fixed effect for all age classes. Age within the class was also included for those that required adjustment. Models were run with functions from the brms package (Bürkner 2021). For comparison with previous studies, we also calculated the CVs for these variance components for each age class. Because sex was a fixed effect in these models, we used the sex midpoint for standardization. All statistics reported were modes and credible intervals from their full posterior distributions.

3 | Results

Sex differences and phenotypic CVs. Squirrel monkey body mass growth at KCCMR was similar to that reported previously. On average, both sexes were born about 100 g and reached adult mass of about 800 g for females and 1000 g for males (Table 1). Mean sex differences were subtle at birth and increased dra-

TABLE 2 | Sex-specific means and coefficients of variation (CV) in Saimiri body mass (g). Measurements were adjusted for age within age classes. Cohen's *D* indicates the effect size of sex differences in means with a 95% confidence interval. Sex differences in CVs are measured by an asymptotic test with corresponding *p*-value.

	mean			CV			
Age class	**Female**	**Male**	**Cohen's *D***	**Female**	**Male**	***D***	***p***
neonate	106.366	112.275	0.437 (0.299, 0.574)	11.971	12.622	1.129	0.288
infant	256.633	274.799	0.396 (0.183, 0.608)	17.523	16.938	0.188	0.664
juvenile	454.524	497.051	0.980 (0.835, 1.124)	8.665	9.387	2.558	0.110
dental adult	652.013	763.313	1.661 (1.474, 1.846)	8.177	10.124	13.123	< 0.001
skeletal adult	722.759	1015.275	3.533 (3.230, 3.836)	8.731	13.045	33.450	< 0.001

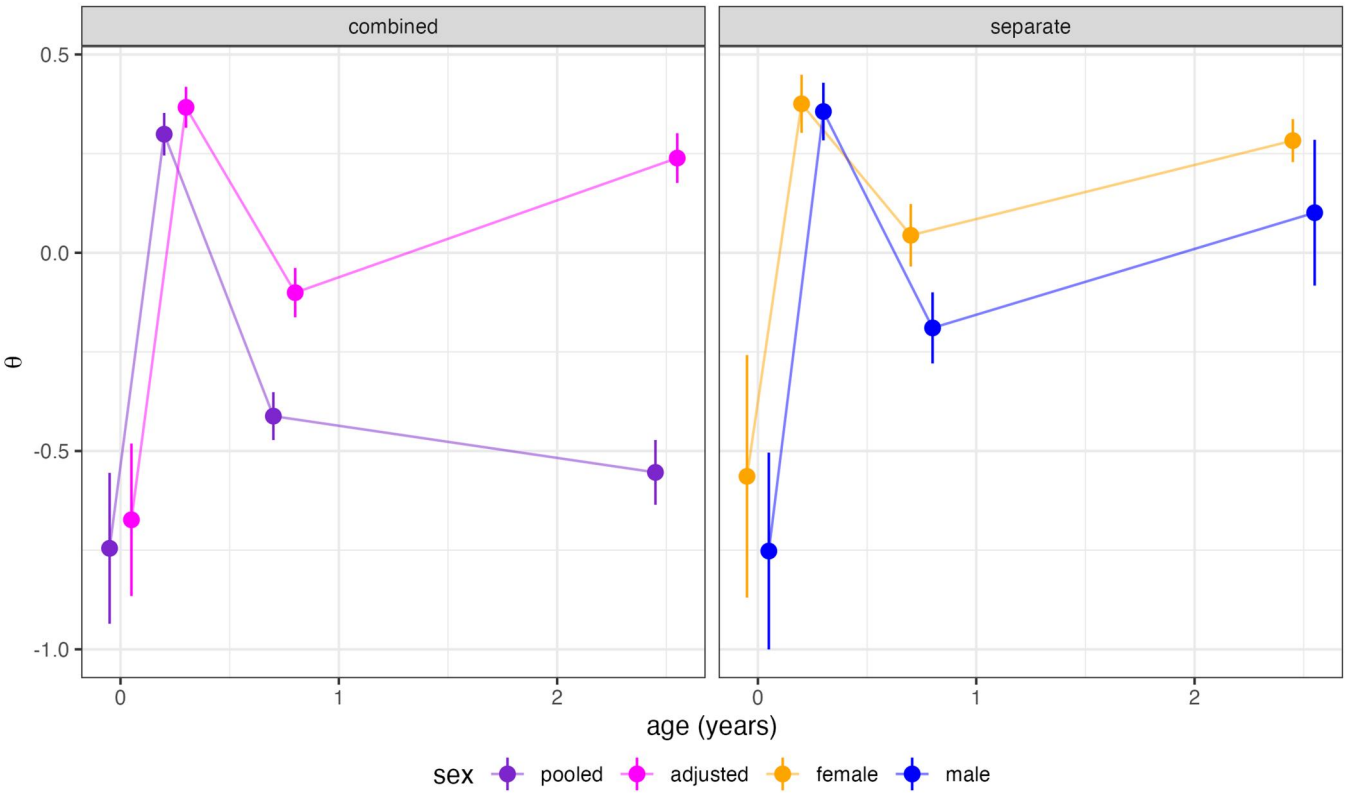

FIGURE 1 | Strength of canalization (θ) in Saimiri body mass from 4 different treatments of sex. Age classes are the same as the rows of Tables 1–3 (birth, infant, dental adult, skeletal adult). Positive values for θ indicate developmental canalization.

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matically in the juvenile stage and later ages (Cohen's D: 0.44, 0.40, 0.98, 1.66, 3.53). The variation in mass relative to the mean (CV) was relatively high at birth and infancy (Table 2). In females, it declined and remained low through adulthood. In males, the CV rebounded to significantly higher values than females in the dental and skeletal adult age classes.

The strength of canalization θ. The Mitteroecker and Stansfield (2021) canalization results were largely compatible with the patterns documented in CVs. For both sexes, there was strong "negative canalization" from birth to infancy followed by a switch to clear canalization from infancy to juvenility (Figure 1). This matches the high birth CV, increase in CV to infancy, and subsequent decline to juvenility. The final two θs documented a drop to roughly 0 or negative for juvenility to dental adulthood followed by a rebound to weak canalization for dental to skeletal adulthood. The weak positive θs for the final age class pair is unexpected because there was an increase in CV between them that was particularly strong in males. Male θs were lower than females at all ages, which may reflect greater heterogeneity among males in growth trajectories. The pooled analysis was seriously compromised by sexual dimorphism (Figure 2). At later ages, when dimorphism was pronounced, this pulled θs down to negative values because of heterogeneity in growth trajectories (i.e., mixture of female and male averages with individual variation around them). The sex mean-adjusted analysis did not suffer from this problem and was very similar to the sex-specific ones (Figure 1).

Individual and maternal sources of variation. We found little evidence for maternal age effects on offspring mass in any of the age classes. None of the regressions reached statistical significance, but trends for the neonate and infant classes suggested reasonable patterns of peak offspring mass from prime-aged mothers (8–10 years old, Figure 3).

In contrast, quantitative genetic mixed models showed clear changes in maternal identity effects with offspring age. They were substantial ($m = 0.28$) for neonatal mass but non-existent for the later age classes (Table 3, Figure 4). Heritabilities documented clear genetic heterogeneity in growth trajectories that became strong with infancy. The neonatal heritability was

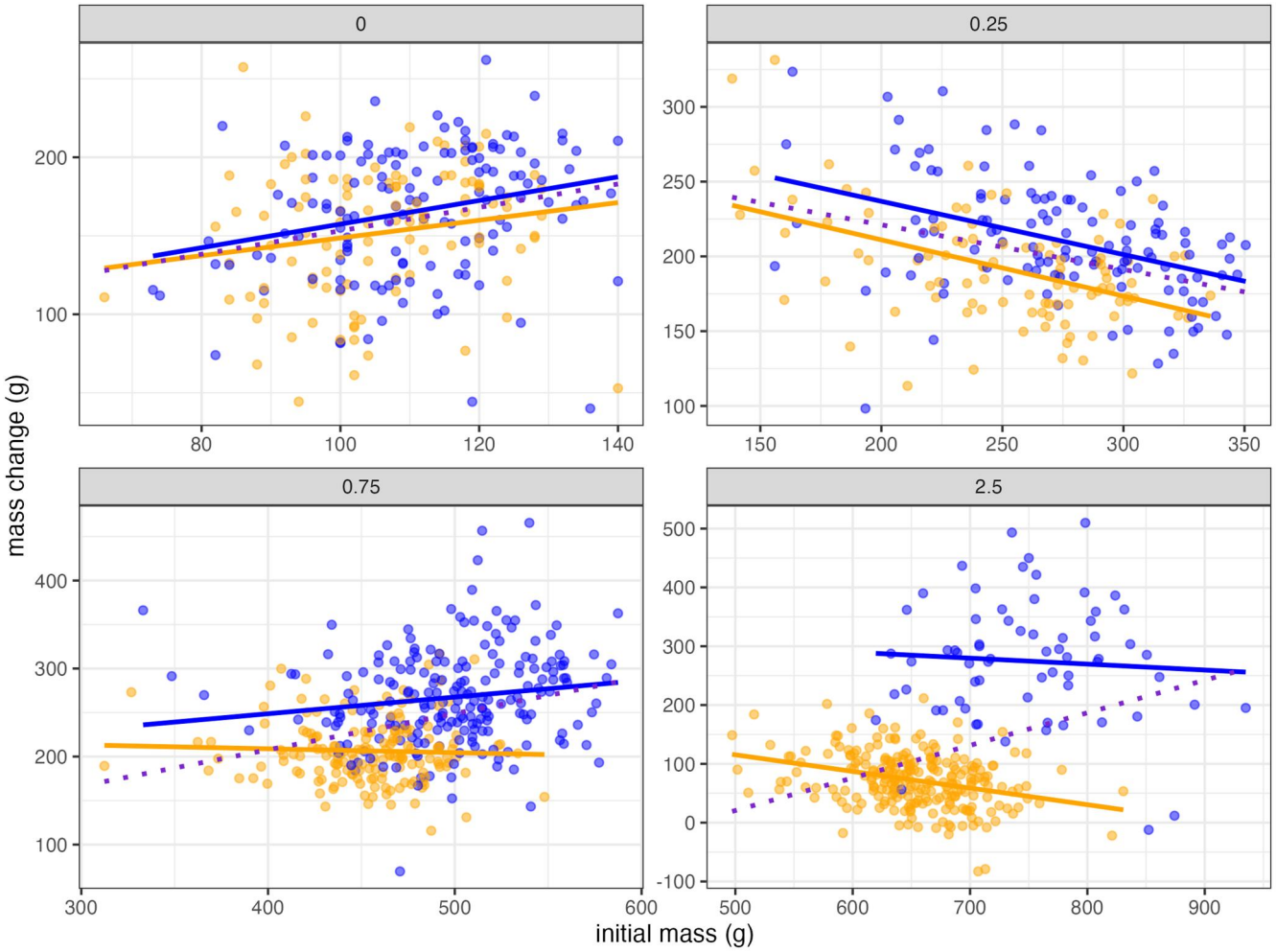

FIGURE 2 | Scatterplots of change in Saimiri body mass on initial mass. Each panel is labeled by the initial age in years. Male (blue) and female (orange) points are fit with corresponding regression lines. The slope of these regression lines is the inverse of a measure of the strength of canalization ($\theta = -1 \cdot \beta_1$) in Figure 1. Dashed purple regression lines are the estimate of canalization when naively pooling the sexes without adjustment for the sex-specific means.

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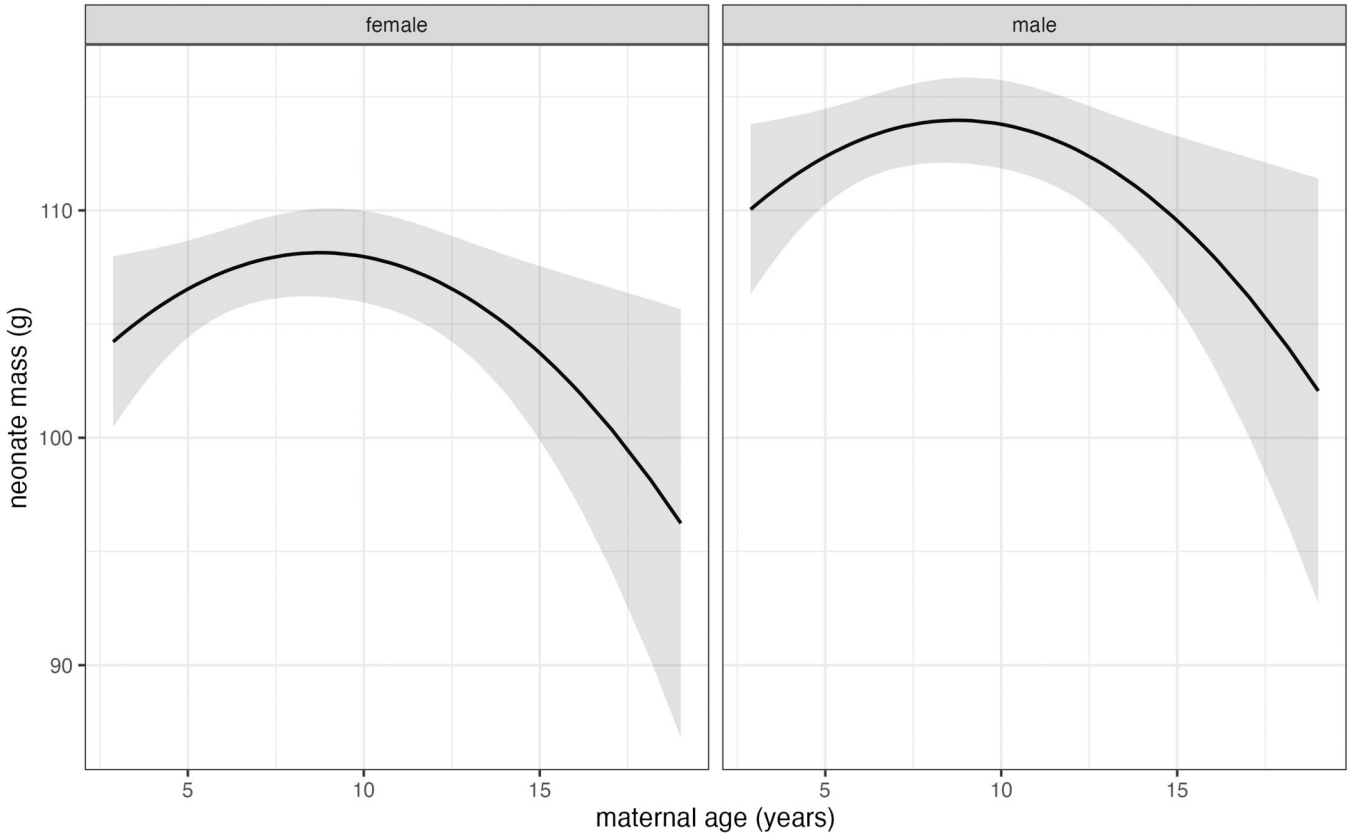

FIGURE 3 | Effect plot illustrating non-significant trend in neonate mass with maternal age for female and male Saimiri.

TABLE 3 | Variance components with ratios and their 95% credible intervals from quantitative genetic models for Saimiri body mass.

Age class	V_P	V_A	V_M	V_R	h^2		m	
Neonate	182.18	49.23	49.76	77.51	0.271	(0.129, 0.451)	0.284	(0.175, 0.376)
Infant	2304.00	1773.40	1.43	16.83	0.742	(0.450, 0.962)	0.001	(0.000, 0.213)
Juvenile	1814.23	1117.51	1.18	680.76	0.597	(0.475, 0.718)	0.001	(0.000, 0.105)
Dental adult	4551.94	2844.12	3.08	1644.43	0.597	(0.416, 0.792)	0.001	(0.000, 0.120)
Skeletal adult	6720.45	2495.05	7.54	3405.23	0.386	(0.250, 0.559)	0.001	(0.000, 0.205)

much lower ($h^2 = 0.27$) and roughly equal to the maternal identity effect. All other heritabilities were greater in infancy, highest in juvenility and dental, slightly lower in adulthood, and lower still in skeletal adulthood (h^2 range: 0.39–0.74). CVs for these variance components were somewhat similar. There was a spike in additive genetic variance from the neonate to the infant period. This was responsible for the large increase in observed phenotypic variance. The other components had less change overall. The maternal and residual variance were similar for the neonate and infant ages, but both contributed to the decline in phenotypic variance entering the juvenile period (Figure 5).

4 | Discussion

We found squirrel monkey body mass growth to have shifting patterns of canalization across ontogeny. Initial post-natal growth magnified differences present at birth, suggesting a continuation of fetal growth patterns. This was followed by a period of strong canalization that reduced size variation by juvenility. Later growth periods were more difficult to characterize, with female growth remaining somewhat canalized but males becoming more variable as they approached skeletal adulthood.

While evidence for developmental canalization has often been emphasized, there is notable heterogeneity among studies that likely reflects some combination of true biological heterogeneity and methodological differences. For example, related research on captive rhesus macaques (*Macaca mulatta*) found no evidence for catch-up growth by infants born small but within the normal range of variation. Coe and Shirtcliff (2004) argued that small, average, and large monkeys mostly remained in those categories across the first 3 years of life with stable developmental trajectories. However, these results were derived from a latent factor growth model for comparison with a prior human study (dos Silva 2002) and are difficult to compare with the methods used here. In contrast, earlier work showed obvious catch-up growth by rhesus infants subjected to temporary protein or calorie

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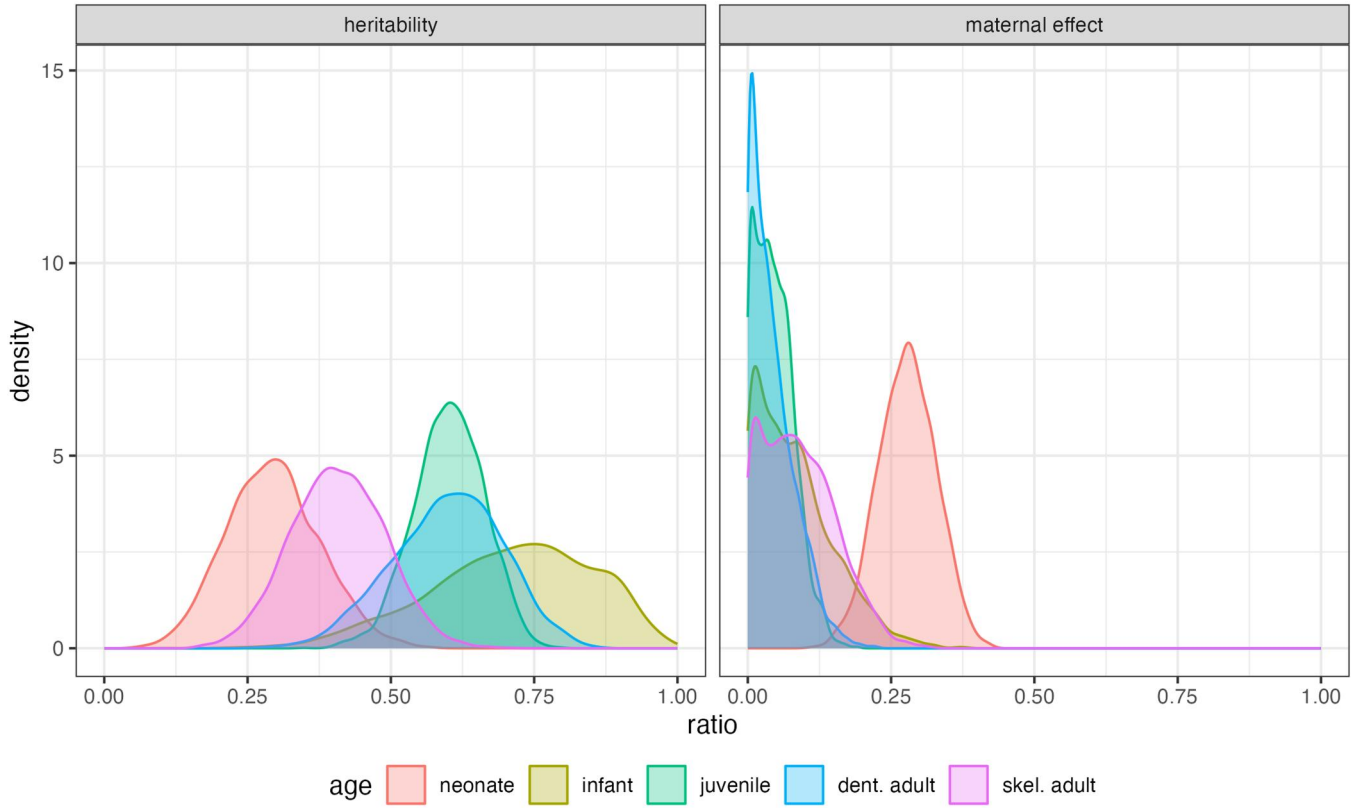

FIGURE 4 | Density plot for the heritabilities and maternal identity effects in quantitative genetic models for Saimiri body mass. Each posterior distribution corresponds to a different age class.

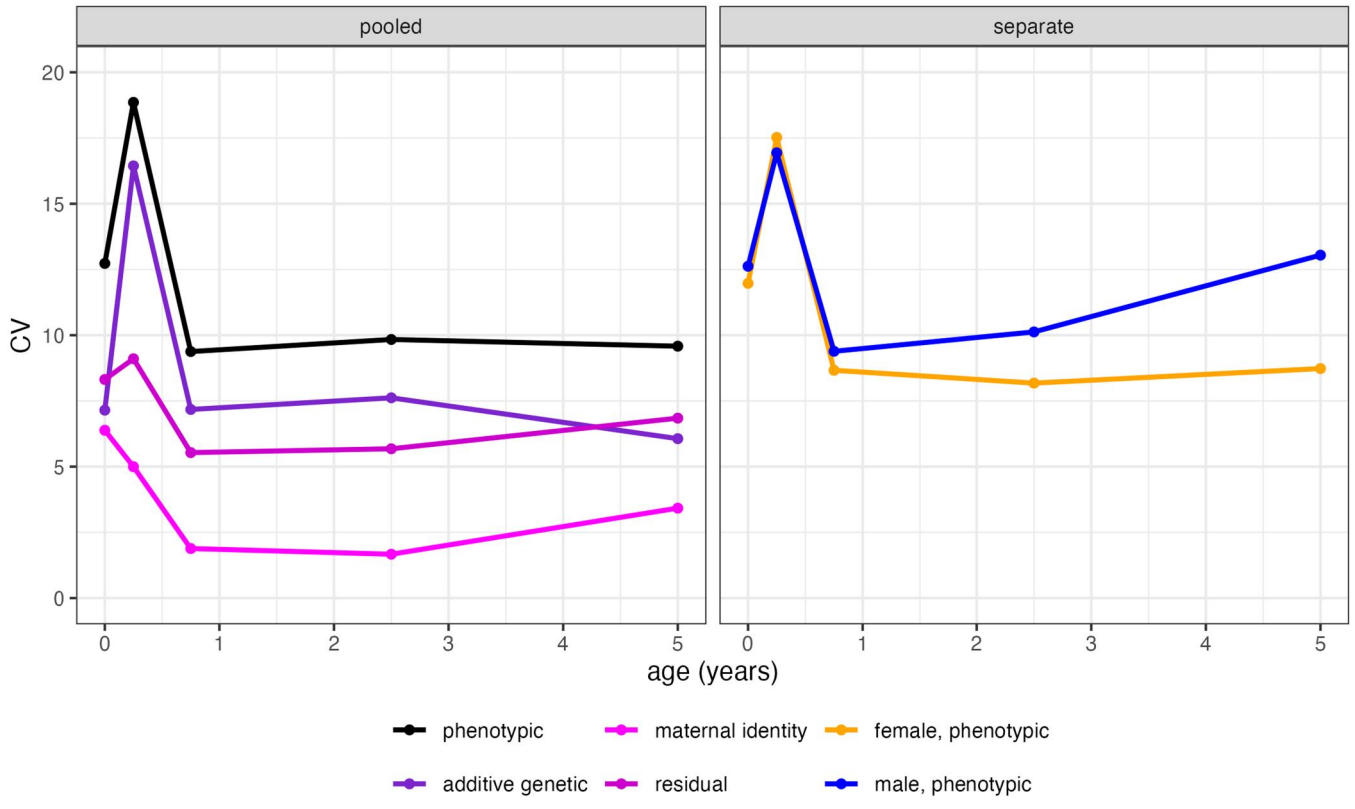

FIGURE 5 | Coefficients of variation (CV) across different age classes for Saimiri body mass as sex-pooled CVs from variance components in quantitative genetic models (left) or sex-specific phenotypic CVs (right).

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deprivation (Kerr et al. 1975). Similarly, published longitudinal growth records for captive chimpanzees highlighted catch-up growth in body mass and length typically through a spurt approaching adulthood (Hamada and Udono 2002).

Our results have some parallels with much earlier work on laboratory mice (*Mus*) which showed an initial post-natal increase in body size variation until weaning and later reduction due to targeted growth (Riska et al. 1984). This was primarily due to age-specific changes in genetic and maternal (foster) variance in their sample. Meta-analysis of domesticated cattle (*Bos*) and sheep (*Ovis*) growth studies provided general support for compensatory growth, but found this was typically due to declining age-specific maternal *genetic* and residual variance alone (Wilson and Réale 2006). Our results with squirrel monkeys emphasize ontogenetic change in additive genetic variance with less prominent changes in residual and maternal variance. However, comparing this work to ours is complicated because of different resolution of age measurements and estimation of maternal identity versus genetic effects. The closest parallel of our results to those with mice is surprising given the drastic difference in duration of growth and body size between species. Phenotypically, human growth charts for body mass also show similar high CVs at birth followed by a steep decline in the first year of life and gradual rebound (Cole et al. 1998), suggesting this is a fundamental feature of mammalian growth.

The patterns of canalization we described in squirrel monkey growth were largely similar between methods. Coefficients of variation have a long history of use in growth analysis, including those of canalization. The new regression-based method of Mitteroecker and Stansfield (2021) offers some additional quantification and theory to explain patterns of changing variation. They noted that the interpretation of θ is complicated when there is short- or long-term variation in target trajectories or timing of growth acceleration. Our ability to describe this heterogeneity was mixed. We were unable to account for much variation with maternal age. However, we did identify large maternal effects on neonate size and individual genetic differences contributed significantly to size variation at all ages. A practical limitation of their method we encountered was that many monkeys did not have measurements in adjacent time intervals. They could contribute to CVs for any ages measured, but dropped out of the regression analysis when measurements were not from adjacent age classes. This meant that direct comparison of the methods is difficult because the sample compositions changed with age, although CVs for the sample used in calculation of θ were very similar to our full dataset. The overall similarity between our CVs and θ results contrasts with their comparisons of measurements from human cranial radiographs. This could be because we used a more labile trait (mass) rather than skeletal dimensions or because we included early post-natal measurements that could have a very different pattern of canalization that became the main signal in the results. We note that Cole et al. (2016) developed a somewhat similar analysis to Mitteroecker and Stansfield (2021) using the correlation matrix of serial growth measurements treated as z-score deviations from WHO growth charts. They focused on identifying shifts between infant and childhood growth phases (Karlberg et al. 2003; Karlberg 1989). We did not attempt to use this technique because of the small sample of monkeys with measurements at all ages and no standard growth chart for *Saimiri* to generate reliable z-scores.

Some of the variation in neonate mass and early infant growth might be attributed to differences in gestational age among offspring. Squirrel monkey gestation is typically 152 days with relatively little variation for full-term deliveries ($\bar{x} = 152.5$, SD = 3.9, $N = 46$; Stolzenberg et al. 1979). Gestational ages were not available for our sample. This would have required a timed breeding or pregnancy monitoring program throughout the decades of data collection. Because one would expect gestation length to contribute primarily to variation in neonate size, the *increase* in variance from birth to infancy we found would likely become more dramatic if we were able to account for gestation length.

The hormonal regulation of growth may offer some explanation for these changing patterns of canalization (Aref et al. 2024; Murray and Clayton 2013). The pre-natal and immediate post-natal hormonal milieu makes for largely nutritional control of growth mediated by levels of insulin-like growth factors (IGF−1 and IGF-2), insulin, and thyroid hormones. Later in infancy there is a transition to more intrinsic hormonal feedbacks from the hypothalamus triggering pulsatile secretion of growth hormone (GH) from the anterior pituitary and subsequent IGF-1 from the liver (Benyi & Sävendahl 2017). This characterizes subsequent post-natal growth and is later enhanced by gonadal sex steroids during adolescence. Earlier gonadal hormone activity prenatally and in newborns is also well-known in humans, even if the mechanisms underlying its quiescence are not (Lanciotti et al. 2018).

Weaning likely coincides with many of these changes in infant hormonal regulation of growth in *Saimiri*. We were limited by a lack of individual information on weaning ages. Moreover, a large number of measurements would be required at several time points in the infant period to better describe at what ages canalization shifts truly occur. Recent models of mammalian growth response to early life experiences, such as intrauterine growth restriction, have emphasized contrasts in growth pre- and post-independence from mothers. Early gestation stressors often recalibrate offspring life history for a "faster" pace, but it only becomes evident after weaning. Late gestation stressors often result in modest-sized neonates that continue to grow slowly after weaning (Berghänel et al. 2017). The complexity and age-specificity of these responses emphasize the need for further longitudinal studies of maternal effects on primate growth (Schmitt et al. 2018).

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Conflicts of Interest

The authors declare no conflicts of interest.

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Data Availability Statement

The data that supports the findings of this study are available in the supplementary material of this article.

References

Abee, C. R. 2000. "Squirrel Monkey (*Saimiri* spp.) Research and Resources." *ILAR Journal* 41, no. 1: 2–9. https://doi.org/10.1093/ilar.41.1.2.

Aref, Y., S. C. Fat, and E. Ray. 2024. "Recent Insights into the Role of Hormones During Development and Their Functional Regulation." *Frontiers in Endocrinology* 15: 1340432. https://doi.org/10.3389/fendo.2024.1340432.

Berghänel, A., M. Heistermann, O. Schülke, and J. Ostner. 2017. "Prenatal Stress Accelerates Offspring Growth to Compensate for Reduced Maternal Investment Across Mammals." *Proceedings of the National Academy of Sciences* 114, no. 50: E10658–E10666. https://doi.org/10.1073/pnas.1707152114.

Bernstein, R. M., G. K. O'Connor, and E. A. Vance, et al. 2020. "Timing of the Infancy-Childhood Growth Transition in Rural Gambia." *Frontiers in Endocrinology* 11: 142. https://doi.org/10.3389/fendo.2020.00142.

Blomquist, G. E., and L. E. Williams. 2013. "Quantitative Genetics of Costly Neonatal Sexual Size Dimorphism in Squirrel Monkeys (*Saimiri boliviensis*)." *Journal of Evolutionary Biology* 26, no. 4: 756–765. https://doi.org/10.1111/jeb.12096.

Bürkner, P.-C. 2021. "Bayesian Item Response Modeling in R With Brms and Stan." *Journal of Statistical Software* 100, no. 5: 1–54. https://doi.org/10.18637/jss.v100.i05.

Coe, C. L., and E. A. Shirtcliff. 2004. "Growth Trajectory Evident at Birth Affects Age of First Delivery in Female Monkeys." *Pediatric Research* 55, no. 6: 914–920. https://doi.org/10.1203/01.pdr.0000125259.45025.4d.

Cole, T. J., J. V. Freeman, and M. A. Preece. 1998. "British 1990 Growth Reference Centiles for Weight, Height, Body Mass Index and Head Circumference Fitted by Maximum Penalized Likelihood." *Statistics in Medicine* 17, no. 4: 407–429. https://doi.org/10.1002/(SICI)1097-0258(19980228)17:4%3C407::AID-SIM742%3E3.0.CO;2-L.

Cole, T. J., A. Singhal, M. S. Fewtrell, and J. C. Wells. 2016. "Weight Centile Crossing in Infancy: Correlations Between Successive Months Show Evidence of Growth Feedback and An Infant-Child Growth Transition." *American Journal of Clinical Nutrition* 104, no. 4: 1101–1109. https://doi.org/10.3945/ajcn.116.139774.

Conley, A. J., R. M. Bernstein, and A. D. Nguyen. 2012. "Adrenarche in Nonhuman Primates: The Evidence for It and the Need to Redefine It." *Journal of Endocrinology* 214, no. 2: 121–131. https://doi.org/10.1530/JOE-11-0467.

Flatt, T. 2005. "The Evolutionary Genetics of Canalization." *Quarterly Review of Biology* 80, no. 3: 287–316. https://doi.org/10.1086/432265.

Galliari, C. A. 1988. "A Study of Postnatal Appendicular Skeletal Maturation in Captive-Born Squirrel Monkeys (*Saimiri boliviensis*)." *American Journal of Primatology* 16, no. 1: 51–61. https://doi.org/10.1002/ajp.1350160107.

Galliari, C. A., and O. J. Colillas. 1985. "Sequences and Timing of Dental Eruption in Bolivian Captive-Born Squirrel Monkeys (*Saimiri sciureus*)." *American Journal of Primatology* 8, no. 3: 195–204. https://doi.org/10.1002/ajp.1350080302.

Garber, P. A., and R. Leigh. 1997. "Ontogenetic Variation in Small-Bodied New World Primates: Implications for Patterns of Reproduction and Infant Care." *Folia Primatologica* 68: 1–22. https://doi.org/10.1159/000157226.

Hallgrimsson, B., R. M. Green, D. C. Katz, et al. 2019. "The Developmental-Genetics of Canalization." *Seminars in Cell & Developmental Biology* 88: 67–79. https://doi.org/10.1016/j.semcdb.2018.05.019.

Hallgrimsson, B., K. Willmore, and B. K. Hall. 2002. "Canalization, Developmental Stability, and Morphological Integration in Primate Limbs." Supplement, *American Journal of Physical Anthropology* 119, no. S35: 131–158. https://doi.org/10.1002/ajpa.10182.

Hamada, Y., and T. Udono. 2002. "Longitudinal Analysis of Length Growth in the Chimpanzee (*Pan troglodytes*)." *American Journal of Physical Anthropology* 118, no. 3: 268–284. https://doi.org/10.1002/ajpa.10078.

Hartwig, W. C. 1996. "Perinatal Life History Traits in New World Monkeys." *American Journal of Primatology* 40, no. 2: 99–130. https://doi.org/10.1002/(SICI)1098-2345(1996)40:2%3C99::AID-AJP1%3E3.0.CO;2-V.

Hector, K. L., and S. Nakagawa. 2012. "Quantitative Analysis of Compensatory and Catch-Up Growth in Diverse Taxa." *Journal of Animal Ecology* 81, no. 3: 583–593. https://doi.org/10.1111/j.1365-2656.2011.01942.x.

Heude, B., P. Scherdel, A. Werner, et al. 2019. "A Big-Data Approach to Producing Descriptive Anthropometric References: A Feasibility and Validation Study of Paediatric Growth Charts." *Lancet Digital Health* 1, no. 8: e413–e423. https://doi.org/10.1016/S2589-7500(19)30149-9.

Iglewicz, B., and D. C. Hoaglin. 1993. *How to Detect and Handle Outliers*. ASQ Quality Press.

Jack, K. M. 2011. "The Cebines: Toward an explanation of variable social structure." In *Primates in Perspective*, edited by C. J. Campbell, A. Fuentes, K. C. MacKinnon, S. K. Bearder, and R. M. Stumpf, 2nd ed., 108–122. Oxford University Press.

Kaack, B., L. Walker, and K. R. Brizzee. 1979. "The Growth and Development of the Squirrel Monkey (*Saimiri sciureus*)." *Growth* 43, no. 2: 116–135.

Karlberg, J. 1989. "A Biologically-Oriented Mathematical Model (ICP) for Human Growth." *Acta Paediatrica* 78, no. s350: 70–94. https://doi.org/10.1111/j.1651-2227.1989.tb11199.x.

Karlberg, J., C. Kwan, L. Gelander, and K. Albertsson-Wikland. 2003. "Pubertal Growth Assessment." *Hormone Research in Paediatrics* 60, no. Suppl. 1: 27–35. https://doi.org/10.1159/000071223.

Lanciotti, L., M. Cofini, A. Leonardi, L. Penta, and S. Esposito. 2018. "Up-to-Date Review About Minipuberty and Overview on Hypothalamic-Pituitary-Gonadal Axis Activation in Fetal and Neonatal Life." *Frontiers in Endocrinology* 9. https://doi.org/10.3389/fendo.2018.00410.

Long, J. O., and R. W. Cooper. 1968. "Physical Growth and Dental Eruption in Captive-bred Squirrel Monkeys, *Saimiri sciureus* (Leticia, Colombia)." In *The Squirrel Monkey*, edited by L. A. Rosenblum and R. W. Cooper, 193–205. Academic Press.

Lynch Alfaro, J. W., J. P. Boubli, F. P. Paim, et al. 2015. "Biogeography of Squirrel Monkeys (Genus Saimiri): South-Central Amazon Origin and Rapid Pan-Amazonian Diversification of a Lowland Primate." *Molecular Phylogenetics and Evolution* 82: 436–454. https://doi.org/10.1016/j.ympev.2014.09.004.

Manocha, S. L. 1979. "Physical Growth and Brain Development of Captive-Bred Male and Female Squirrel Monkeys, Saimiri Sciureus." *Experientia* 35, no. 1: 96–98. https://doi.org/10.1007/BF01917901.

Marcil-Ferland, D., M. Festa-Bianchet, A. M. Martin, and F. Pelletier. 2013. "Despite Catch-Up, Prolonged Growth Has Detrimental Fitness Consequences in a Long-Lived Vertebrate." *American Naturalist* 182, no. 6: 775–785. https://doi.org/10.1086/673534.

Marwick, B., and K. Krishnamoorthy. 2019. *cvequality: Tests for the Equality of Coefficients of Variation from Multiple Groups*. https://doi.org/10.32614/CRAN.package.cvequality.

Mitteroecker, P., and E. Stansfield. 2021. "A Model of Developmental Canalization, Applied to Human Cranial Form." *PLoS Computational Biology* 17, no. 2: e1008381. https://doi.org/10.1371/journal.pcbi.1008381.

10982345, 2026, 10, Downloaded from https://onlinelibrary.wiley.com/doi/10.1002/ajp.70217 by Gregory Blomquist - University Of Missouri Columbia , Wiley Online Library on [12/10/2026]. See the Terms and Conditions (https://onlinelibrary.wiley.com/terms-and-conditions) on Wiley Online Library for rules of use; OA articles are governed by the applicable Creative Commons License

Moore, M. P., H. H. Whiteman, and R. A. Martin. 2019. "A Mother's Legacy: The Strength of Maternal Effects in Animal Populations." *Ecology Letters* 22, no. 10: 1620–1628. https://doi.org/10.1111/ele.13351.

Murray, P. G., and P. E. Clayton. 2013. "Endocrine Control of Growth." *American Journal of Medical Genetics, Part C: Seminars in Medical Genetics* 163, no. 2: 76–85. https://doi.org/10.1002/ajmg.c.31357.

Nehete, P. N., B. P. Nehete, G. K. Wilkerson, S. J. Schapiro, and L. E. Williams. 2021. "Effects of Relocation on Immunological and Physiological Measures in Female Squirrel Monkeys (*Saimiri boliviensis boliviensis*)." *PLoS One* 16, no. 2: e0240705. https://doi.org/10.1371/journal.pone.0240705.

Nijhout, H. F. 2025. "Genetic Assimilation, Robustness and Plasticity are Key Processes in the Development and Evolution of Novel Traits." *Developmental Biology* 523: 132–138. https://doi.org/10.1016/j.ydbio.2025.04.011.

Pereira, M. E., and S. R. Leigh. 2003. "Modes of Primate Development." In *Primate Life Histories and Socioecology*, edited by P. M. Kappeler and M. E. Pereira, 149–176. University of Chicago Press.

R Core Team. 2025. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing. https://www.R-project.org/.

Rice, S. H. 1998. "The Evolution of Canalization and the Breaking of von Baer's Laws: Modeling the Evolution of Development With Epistasis." *Evolution* 52: 647–656. https://doi.org/10.1111/j.1558-5646.1998.tb03690.x.

Riska, B., and W. R. Atchley. 1985. "Genetics of Growth Predict Patterns of Brain-Size Evolution." *Science* 229, no. 4714: 668–671. https://doi.org/10.1126/science.229.4714.668.

Riska, B., W. R. Atchley, and J. J. Rutledge. 1984. "A Genetic Analysis of Targeted Growth in Mice." *Genetics* 107, no. 1: 79–101. https://doi.org/10.1093/genetics/107.1.79.

Sabbi, K. H., M. N. Muller, and Z. P. Machanda, et al. 2020. "Human-Like Adrenal Development in Wild Chimpanzees: A Longitudinal Study of Urinary Dehydroepiandrosterone-Sulfate and Cortisol." *American Journal of Primatology* 82, no. 11: e23064. https://doi.org/10.1002/ajp.23064.

Silva, I. S. 2002. "Prenatal Factors, Childhood Growth Trajectories and Age at Menarche." *International Journal of Epidemiology* 31, no. 2: 405–412. https://doi.org/10.1093/ije/31.2.405.

Schmalhausen, I. I. 1949. *Factors of Evolution: The Theory of Stabilizing Selection*. Blakiston Company.

Schmitt, C. A., S. K. Service, A. J. Jasinska, et al. 2018. "Obesity and Obesogenic Growth Are Both Highly Heritable and Modified by Diet in a Nonhuman Primate Model, the African Green Monkey (*Chlorocebus aethiops sabaeus*)." *International Journal of Obesity* 42, no. 4: 765–774. https://doi.org/10.1038/ijo.2017.301.

Siegal, M. L., and A. Bergman. 2002. "Waddington's Canalization Revisited: Developmental Stability and Evolution." *Proceedings of the National Academy of Sciences* 99, no. 16: 10528–10532. https://doi.org/10.1073/pnas.102303999.

Stolzenberg, S. J., D. C. L. Jones, J. N. Kaplan, R. A. Barth, G. D. Hodgen, and S. M. Madan. 1979. "Studies With Timed-Pregnant Squirrel Monkeys (Saimiri Sciureus)." *Journal of Medical Primatology* 8, no. 1: 29–38. https://doi.org/10.1159/000460173.

Stone, A. I., and L. Williams. 2021. "Behavioral biology of squirrel monkeys." In *Behavioral biology of laboratory animals*, 395–407. CRC Press.

Tanner, J. M. 1963. "The Regulation of Human Growth." *Child Development* 34, no. 4: 817–847. https://doi.org/10.1111/j.1467-8624.1963.tb05970.x.

Tanner, J. M. 1986. "Growth as a target-seeking function." In *Human Growth: A Comprehensive Treatise Volume 1 Developmental Biology Prenatal Growth*, edited by F. Falkner and J. M. Tanner, 167–179. Springer.

Vasconcelos Sampaio, W., D. Leite Leão, P. da Cunha Sousa, H. Lima de Queiroz, and S. Farhayldes Souza Domingues. 2022. "Male Fattening Is Related to Increased Seminal Quality of Squirrel Monkeys (Saimiri Collinsi): Implications for Sperm Competition." *American Journal of Primatology* 84, no. 2: e23353. https://doi.org/10.1002/ajp.23353.

Waddington, C. H. 1942. "Canalization of Development and the Inheritance of Acquired Characters." *Nature* 150, no. 3811: 563–565. https://doi.org/10.1038/150563a0.

Waddington, C. H. 1957. *The strategy of the genes: A discussion of some aspects of theoretical biology*. Allen & Unwin.

Wagner, G. P., G. Booth, and H. Bagheri-Chaichian. 1997. "A Population Genetic Theory of Canalization." *Evolution* 51, no. 2: 329–347. https://doi.org/10.1111/j.1558-5646.1997.tb02420.x.

Watts, E. S., and J. A. Gavan. 1982. "Postnatal Growth of Nonhuman Primates: The Problem of the Adolescent Spurt." *Human Biology* 54, no. 1: 53–70.

Williams, G. C. 1966. *Adaptation and natural selection: A critique of some current evolutionary thought*. Princeton University Press.

Williams, L. E., A. G. Brady, S. V. Gibson, and C. Abee. 2002. "The Squirrel Monkey Breeding and Research Resource: A Review of Saimiri Reproductive Biology and Behavior and Breeding Performance." *Primatologie* 5: 303–334.

Willmore, K. E., L. Leamy, and B. Hallgrímsson. 2006. "Effects of Developmental and Functional Interactions on Mouse Cranial Variability Through Late Ontogeny." *Evolution & Development* 8, no. 6: 550–567. https://doi.org/10.1111/j.1525-142X.2006.00127.x.

Willmore, K. E., N. M. Young, and J. T. Richtsmeier. 2007. "Phenotypic Variability: Its Components, Measurement and Underlying Developmental Processes." *Evolutionary Biology* 34, no. 3/4: 99–120. https://doi.org/10.1007/s11692-007-9008-1.

Wilson, A. J., and D. Réale. 2006. "Ontogeny of Additive and Maternal Genetic Effects: Lessons From Domestic Mammals." *American Naturalist* 167, no. 1: E23–E38. https://doi.org/10.1086/498138.

Wilson, A. J., D. Réale, M. N. Clements, et al. 2010. "An Ecologist's Guide to the Animal Model." *Journal of Animal Ecology* 79, no. 1: 13–26. https://doi.org/10.1111/j.1365-2656.2009.01639.x.

Young, N. M. 2006. "Function, Ontogeny and Canalization of Shape Variance in the Primate Scapula." *Journal of Anatomy* 209, no. 5: 623–636. https://doi.org/10.1111/j.1469-7580.2006.00639.x.

Zimbler-DeLorenzo, H. S., and A. I. Stone. 2011. "Integration of Field and Captive Studies for Understanding the Behavioral Ecology of the Squirrel Monkey (*Saimiri* Sp)." *American Journal of Primatology* 73, no. 7: 607–622. https://doi.org/10.1002/ajp.20946.

Zullinger, E. M., R. E. Ricklefs, K. H. Redford, and G. M. Mace. 1984. "Fitting Sigmoidal Equations to Mammalian Growth Curves." *Journal of Mammalogy* 65, no. 4: 607–636. https://doi.org/10.2307/1380844.

Supporting Information

Additional supporting information can be found online in the Supporting Information section.
Supporting File 1: ajp70217-sup-0001-research_highlights_rev.txt.
Supporting File 2: ajp70217-sup-0002-saimiri_canalization.Rmd.
Supporting File 3: ajp70217-sup-0003-saimiri_data.csv.
Supporting File 4: ajp70217-sup-0004-saimiri_pedigree.csv.

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