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RESEARCH ARTICLE

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Anthropometric heritability and child growth in a Caribbean village: A quantitative genetic analysis of longitudinal height, weight, and body mass index in Bwa Mawego, Dominica

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Abstract

Objectives: Body size and composition vary widely among individuals and populations, and long-term research in diverse contexts informs our understanding of genetic, cultural, and environmental impacts on this variation. We analyze longitudinal measures of height, weight, and body mass index (BMI) from a Caribbean village, estimating the extent to which these anthropometrics are shaped by genetic variance in a small-scale population of mixed ancestry.

Materials and Methods: Longitudinal data from a traditionally horticultural village in Dominica document height and weight in a non-Western population that is transitioning to increasingly Westernized lifestyles, and an 11-generation pedigree enables us to estimate the proportions of phenotypic variation in height, weight, and BMI attributed to genetic variation. We assess within-individual variation across growth curves as well as heritabilities of these traits for 260 individuals using Bayesian variance component estimation.

Results: Age, sex, and secular trends account for the majority of anthropometric variation in these longitudinal data. Independent of age, sex, and secular trends, our analyses show high repeatabilities for the remaining variation in height, weight, and BMI growth curves (>0.75), and moderate heritabilities (h^2_{height} = 0.68, h^2_{weight} = 0.64, h^2_{BMI} = 0.49) reveal clear genetic signals that account for large proportions of the variation in body size observed between families. Secular trends show increases of 6.5% in height and 16.0% in weight from 1997 to 2017.

Discussion: This horticultural Caribbean population has transitioned to include more Westernized foods and technologies over the decades captured in this analysis. BMI varies widely between individuals and is significantly shaped by genetic variation, warranting future exploration with other physiological correlates and associated genetic variants.

KEYWORDS
anthropometric, Caribbean, child growth, heritability, longitudinal

1 | INTRODUCTION

Variation in body size and child growth is shaped by combinations of biological, cultural, and environmental factors that are often ecologically dependent and population-specific. Body size and growth patterns vary widely among populations in developing countries and small-scale societies (Walker et al., 2006), many of whom are transitioning nutritionally and behaviorally to more Westernized, processed foods and decreased

physical activity (Popkin, Adair, & Ng, 2012). Metabolic health is of increasing concern in the Caribbean, yet we know little of the specific biological, cultural, and environmental influences on anthropometric variation in this region, particularly in rural areas (Boyne, 2009; Rueda-Clausen, Silva, & López-Jaramillo, 2008). Longitudinal measures of height, weight, and body mass index (BMI) reflect secular trends in human growth and development (Cole, 2003) and inform public health concerns of undernutrition and overnutrition (Monteiro, Conde, & Popkin, 2004). We assess the repeatabilities and heritabilities of longitudinal height, weight, and BMI in a remote Caribbean village during a period of nutritional transition to quantify variation within-individual growth trajectories and to estimate the proportion of variation between individuals attributed to genetic versus nongenetic variance.

Body size and composition vary among geographic regions (de Onis, Blössner, Borghi, Frongillo, & Morris, 2004), in part reflecting climate adaptations such that temperature and BMI generally show inverse relationships in indigenous populations (Wells, 2012). Small-scale societies that share similar tropical climates show considerable variation among their growth patterns, shaped partially by life-history trade-offs that balance growth rates with mortality risk and fertility to produce taller individuals through faster growth (Walker et al., 2006). The extent to which height, weight, and BMI are phenotypically plastic depends upon genetic variants, environmental inputs, and developmental/epigenetic backgrounds (Godfrey, Gluckman, & Hanson, 2010). The relative impacts of these factors vary within and between populations as well as across age over the course of the human lifespan (Visscher, Hill, & Wray, 2008).

Heritabilities quantify the proportions of variation in observed phenotypes that are explained by genetic variation in a population (Falconer, 1960; Lynch & Walsh, 1998). Heritability estimates do not reflect the extent to which a trait's phenotypic outcome is determined by an individual's genes, but instead partition the variance in an observed trait into genetic and nongenetic components (Lewontin, 1974; Vitzthum, 2003). Methods for estimating heritabilities use known genetic relationships to assess the extent to which the proportion of alleles shared among individuals associates with phenotypic variation (Vandemark, Kelly, & Eckhardt, 1985), and larger pedigrees with many generations provide varied kinship coefficients that produce more robust estimates of genetic variance than those based on only ancestor–descendant pairs, sibships, and so on (Kruuk, 2004; Wilson et al., 2010).

Quantities of phenotypic and genotypic variation are population-specific, and heritability estimates range from 0.26 to 0.90 for height, 0.22 to 0.85 for weight, and 0.17 to 0.90 for BMI (Dubois et al., 2012; Elks et al., 2012; Nan et al., 2012; Starkweather & Keith, 2018; Yang et al., 2015). Few heritabilities are published from Caribbean populations, but an analysis from Jamaica reports estimates of 0.74 for height, 0.63 for weight, and 0.53 for BMI (Luke et al., 2001). Larger heritabilities can result from larger quantities of genetic variation or from relatively low amounts of environmental variance. The proportional impact of genetic variance generally increases under stable and more favorable environmental conditions (Hoffmann & Merila, 1999); however, heritabilities may also decrease when environmental conditions change drastically between generations. Lower anthropometric heritabilities in some immigrant populations reflect the context-dependent nature of genetic variance components when environmental conditions differ dramatically between ancestors and their descendants (Gravlee, Bernard, & Leonard, 2003). Across the lifespan, Elks et al. (2012) found higher heritabilities for BMI in twin children than adult twins but found no detectable relationships between BMI heritability and age among other types of family studies. We estimate heritabilities for height, weight, and BMI in a Caribbean village population to capture the proportional influence of genetic variance on body size and child growth during a population-wide period of nutritional transition using longitudinal anthropometric data.

Longitudinal data require within-individual analyses to account for variation in repeated measures over time in addition to between-individual analyses of phenotypic variation. Repeatabilities reflect how consistent traits remain for an individual as they age by regressing an individual's measurements against themselves over time, and any aspects of an individual's identity (including genetic and nongenetic attributes) that impact the observed phenotypes are captured in repeatability ratios (Wilson et al., 2010). Repeatability estimates generally indicate the upper limits for heritability estimates in a population given that individuals share 100% of their genetic variation with themselves (Dohm, 2002; Falconer & Mackay, 1996). Our repeatability estimates of height, weight, and BMI measure phenotypic variation within-individual growth curves during a period of nutritional and behavioral transition.

We expect repeatabilities and heritabilities to be higher for height than for weight or BMI given that height is less variable as an additive, long-term measure that remains constant once adult height is reached until substantial bone loss occurs at elderly ages (Dey, Rothenberg, Sundh, Bosaeus, & Steen, 1999). Weight and BMI are constrained by height but can fluctuate in response to short-term conditions across the lifespan. BMI is a composite measure of height and weight used to define clinical underweight, overweight, and obese categories despite its variable relationship with adiposity and metabolic health across ethnicities/ancestries (Carroll et al., 2008; Hall & Cole, 2006; Prista, Maia, Damasceno, & Beunen, 2003). BMI remains a readily available metric of body size that may be more useful in diverse populations when tracked longitudinally over time to assess population-specific trends in changing body mass rather than relying on standardized cut-offs to categorize metabolic status (Hall & Cole, 2006). We capture secular trends in height, weight, and BMI from 1997 to 2017 in rural Dominica to assess how the global nutrition transition (Popkin et al., 2012) has influenced growth and body size in a small-scale horticultural population.

Body size, composition, and metabolic health in the Caribbean are uniquely impacted by sugar cultivation, other aspects of historic colonialism, alcohol production, tourism, and recent economic transition (Cherry, Serieux, Didier, Nuttal, & Schuster, 2014; Mintz, 1985). Metabolic health is an increasing concern in this region as cardiovascular disease and type II diabetes climb in prevalence (Rueda-Clausen et al., 2008). Sugar was widely cultivated throughout the Caribbean from the 18–20th centuries, mostly for export to European countries who

transported slave labor to the islands from west Africa in the 17–18th centuries (Mintz, 1985). Sugarcane is still grown throughout much of the Caribbean, and gene flow from Europe and Africa into indigenous Caribbean communities continues to shape genetic variation throughout the region. Middle-income/wealthier Caribbean nations such as St. Lucia report negative metabolic outcomes characteristic of the global nutrition transition (Popkin et al., 2012) that result from decreasing physical activity levels, increased alcohol consumption, urbanization, and changing diets due to imports and increased tourism (Cherry et al., 2014). Poorer, less developed Caribbean nations appear to be suffering similar health outcomes, but their data are sparse and secular trends poorly understood, particularly in rural areas (Boyne, 2009).

The Commonwealth of Dominica is one of the least developed Caribbean islands, and the village of Bwa Mawego is one of the most remote communities on the island. There are approximately 500–600 residents in Bwa Mawego, most of whom continue to practice traditional horticulture in tandem with increased access to cash goods and modern technology (e.g., cell phones, high-speed internet) (Decker & Flinn, 2011). Several varieties of taro, yams, and other root vegetables are the primary components of the traditional diet, which are boiled and eaten with plantains, other crops, and sometimes fish (Quinlan, 2004). Observational data from several decades of research at this field site indicate that processed foods, sweets, sugary beverages, and meat are increasingly available in local rum shops, transforming diets population-wide to include a combination of horticultural products and foods with more caloric sweeteners, oils, and animal products since the 2000s. Coinciding with dietary shifts characteristic of the global nutrition transition (Popkin et al., 2012), the transport of piped water and electricity to most homes in the village as of the early-mid 2000s has decreased physical activity demands (Decker & Flinn, 2011). We analyze longitudinal anthropometric data spanning 20 years (1997–2017) by capturing secular trends in growth curves during this transitional period and estimating the relative influence of genetic variation on observed variation in height, weight, and BMI.

2 | MATERIALS AND METHODS

Longitudinal anthropometric data were collected in Bwa Mawego, Dominica at varying time points between 1997 and 2017 in accordance with procedures approved by the University of Missouri's Institutional Review Board. All participants provided informed consent, and parental consent was also obtained for all individuals under the age of 18 at the time of data collection.

This study includes data for 260 individuals (126 males and 134 females) for whom there were repeated measures of height and weight over the study period that met our quality-control criteria, and ages of this sample range from birth to 27 years (Table 1). The number of repeated measures per individual ranges from 2 to 16 with a mean of 7.56, and the average time between a person's data points is 0.90 years, ranging from 4 months to 10 years (Table 1). The height of individuals old enough to stand upright was measured with a stadiometer on a flat surface; those too young to stand were laid on a flat surface and measured to the nearest millimeter by stretching a tape measure from heel to crown. Weight was measured using an electronic scale, and children too young to be weighed independently were weighed with a parent, whose weight was then subtracted from the combined total.

Growth data were visually inspected and cleaned using the Sitar package in R v.3.4.3 to account for errors in data collection over the 20-year study period and to remove outliers exceeding three standard deviations in an individual's growth curve (Cole, 2015; R Core Team, 2017). BMI was calculated using the standard equation (weight (kg)/height $(m)^2$). A pedigree that includes 11 generations was compiled for this village in the 2000s from interview data and historical records (Quinlan & Hagen, 2008), providing kinship coefficients needed for estimating trait heritabilities (Table S1; Figure S1).

We used Bayesian linear mixed models (LMMs) to analyze repeatabilities, heritabilities, and secular trends in longitudinal height, weight, and BMI among the sample of Bwa Mawego residents described above. Height, weight, and BMI were log-transformed to account for heteroscedasticity as variation increases in these variables with age. All LMMs included three fixed effects as control variables: age modeled as a cubic spline with knot points at 7 and 12 years, sex, and year of data collection (*z*-scored). This cubic polynomial spline allowed us to control for age across different stages of growth such that the relationships between anthropometrics and age later in adolescence were not impacted by trends very early in childhood, balancing complexity in different stages of growth with flexibility in a smooth curve that eases linear model fit (Buja, Hastie, & Tibshirani, 1989; Harrell, 2001). Knot points at 7 and 12 years divide the age range approximately into thirds, also bracketing the beginning and end of the juvenile period (Bogin & Smith, 1996). The inclusion of data collection year in these LMMs controlled for secular trends across the 1997–2017 timespan during which these longitudinal data were collected. This captured period effects independently from the effects of individual aging and indicates how height, weight, and BMI have changed among younger generations across this population during a period of nutritional and behavioral transition.

Repeatability LMMs included individual ID as a random effect, producing variance component estimates that measure the amount of variation in growth curves for height, weight, and BMI explained by an individual's ID, thereby capturing within-individual variation over time. Heritability models included two random effects: an ID variable to control for repeated measures and a second "identity" variable to connect each individual to the population's pedigree. This second random effect produced estimates of additive genetic variance by using Mendelian rules of allele sharing between individuals to explain the observed variation between their heights, weights, and BMIs independent of any variation within-individual growth curves.

This method of estimating heritabilities is referenced as the "animal model" and uses complex multigenerational relationships (parents, siblings, half-siblings, grandparents, cousins, etc.) to capture the extent to which proportions of shared alleles influence the variance in observed traits (Kruuk, 2004; Teplitsky et al., 2008). This provides

TABLE 1 Sample characteristics of longitudinal data

N	260
Males	126 (48.5%)
Females	134 (51.5%)
Age range (years)	0–27
Data collection period	1997–2017 (mean = 2002, *SD* = 3.5 years)
Number of repeated measures	2–16 (mean = 7.56, *SD* = 4.85)
Time between data points (years)	0.36–10.01 (mean = 0.90, *SD* = 1.01)

more robust heritability estimates than other common methods that rely on only two generations of parent-offspring relationships or single-generation twin studies. The pedigree for this village population in Dominica goes back 11 generations, ensuring that we capture as many kinship coefficients as possible, including relationships between more extended kin that are less likely to share common household environments (Table S1; Figure S1).

Although animal models allow for the inclusion of random household effects (Thomson et al., 2018), we do not include common household environment as a variance component in our models because of the flexibility and fluctuation in household composition in this matrifocal community. Households in Bwa Mawego fluctuate in composition as both children and adults change residence frequently related to short-term economic opportunities, temporary migrations, and changing family dynamics (Quinlan, 2004). Many people in this sample resided in more than one household over the timespan of their data points. In many cases, "households" are not discrete units, as many dwellings are organized to varying degrees into larger compounds with extended kin (Quinlan & Flinn, 2003). Additionally, the extensive depth of this population's pedigree reduces confounding effects of common environments since many smaller kinship coefficients contribute to estimates of genetic variance between relatives who do not share household environments.

Bayesian LMMs produce posterior probability distributions of fixed effect beta coefficients and random effect variance components by updating prior probability distributions with data (McElreath, 2015). This method of linear modeling is robust to sample size and accommodates complexity in regression-based analyses by controlling for repeated measures and accounting for other sources of heterogeneity that vary among individuals (Zhao, Staudenmayer, Coull, & Wand, 2006). Fixed effects such as age and sex are simultaneously incorporated in a multivariable fashion such that the coefficients of each variable are measured independently from the rest. We captured Bayesian posterior distributions of repeatability and heritability variance components using Markov chain Monte Carlo sampling with the MCMCglmm package in R (Hadfield, 2010). This method samples posteriors in a step-wise fashion rather than directly computing distributions in their entirety, which becomes less feasible as models increase in their complexity (McElreath, 2015).

All LMMs ran for 5,200,000 iterations with a burn-in of 200,000 and thin of 5,000 to produce 1,000 estimates of within-individual variance and additive genetic variance of height, weight, and BMI from the posteriors. We used parameter expanded priors for all models to facilitate chain mixing and obtain sufficient effective sample sizes (>900) by setting prior means to 0 (alpha.mu = 0) and prior covariance matrices to 1,000 (alpha.V = 1,000) (Hadfield, 2010). Repeatabilities and heritabilities were calculated as variance component ratios, also representing 1,000 retained samples from the posteriors. Repeatability estimates reflect the proportion of total variation in each outcome due to the random effects of individual IDs (Equation 1), controlling for the fixed effects of sex, age, and data collection year. Heritability estimates reflect the proportion of phenotypic variation in each outcome captured by the additive genetic variance components derived from each individual's relatedness to everyone in the pedigree, also controlling for repeated measures, sex, age, and data collection year (Equation 2).

$$r = \frac{V_I}{V_I + V_e} \quad (1)$$

$$h^2 = \frac{V_A}{V_A + V_I + V_e} \quad (2)$$

V_I is the vector of 1,000 retained ID variance components that capture within-individual variation, V_A is the vector of additive genetic variance components, and V_e is the vector of residuals in each model. Ratios of these vectors produced 1,000 estimates of either repeatability or heritability for each outcome from which we report posterior modes and credible intervals.

3 | RESULTS

The pedigree from Bwa Mawego, Dominica includes 1,455 individuals, spans 11 generations, and dates back to 1899 (Figure S1). We have longitudinal growth data for 260 of those individuals, and the 662 people marked by dots in Figure S1 show the members of the pedigree who are related to them such that they contribute to estimates of genetic variance. Pedigree statistics calculated with the Pedantics package in R (Morrissey, 2018) show that inbreeding is negligible in this population despite the small founding structure of the community (Table S1).

Individual growth curves from this population are plotted with World Health Organization (WHO) percentiles overlaid for comparison (Figures 1–3). WHO growth percentiles range from birth-19 years for height and BMI, and from birth-10 years for weight (de Onis et al., 2007). Therefore, height and BMI plots include only individuals with

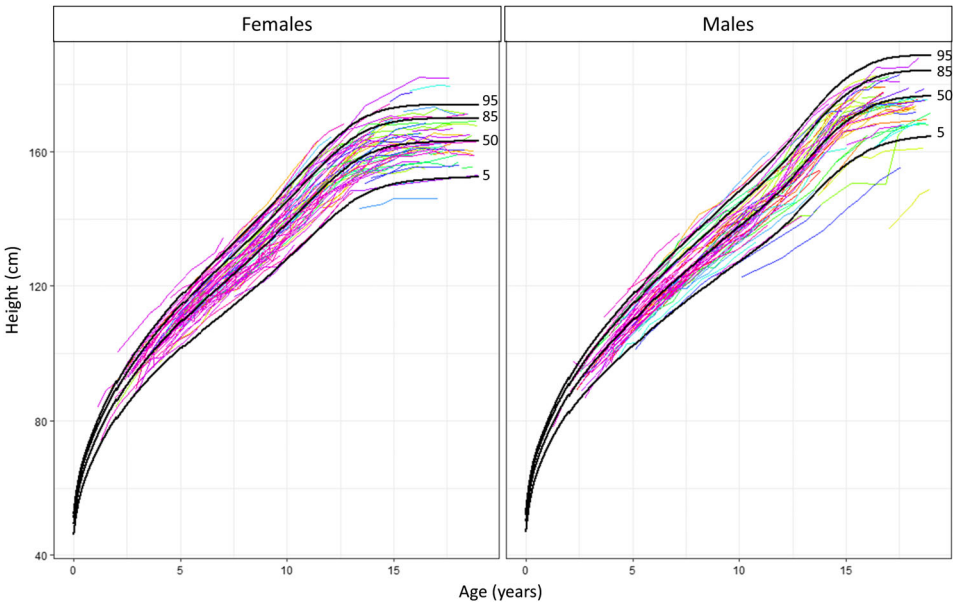

FIGURE 1 Height (cm) curves for 251 children ages 0–19 years plotted with WHO percentiles for comparison (de Onis et al., 2007). WHO, World Health Organization

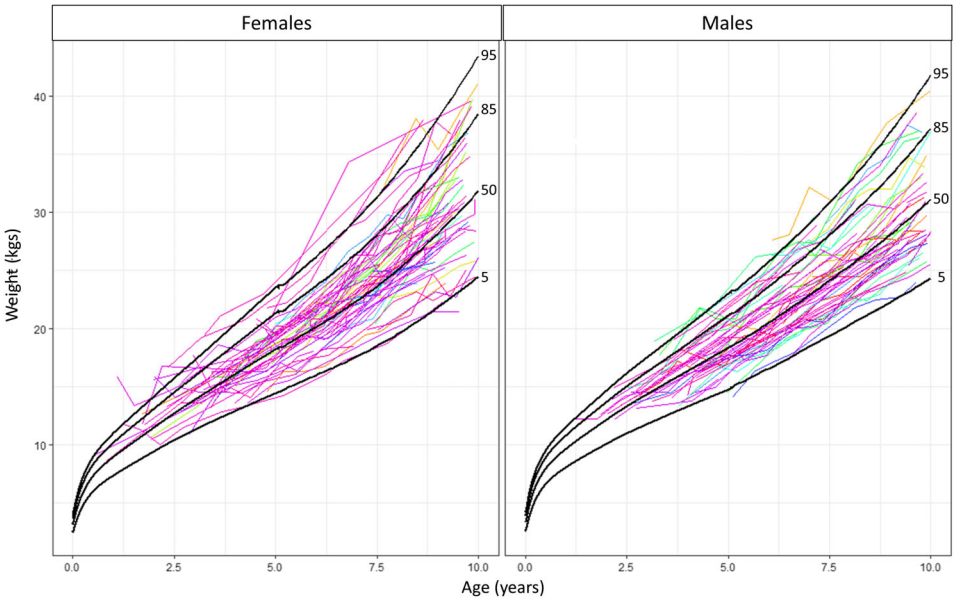

FIGURE 2 Weight (kg) curves for 158 children ages 0–10 years plotted with WHO percentiles for comparison (de Onis et al., 2007). WHO, World Health Organization

2 or more measurements recorded by age 19, and the weight plot includes only those with 2 or more measurements by age 10. We plot WHO curves for the 5th, 50th, 85th, and 95th percentiles due to their significance in reference to BMI (Barlow, 2007).

Growth curves show that males are slightly taller than females and females slightly heavier than males across all ages (Figures 1 and 2), and female BMI appears to increase substantially around age 12 (Figure 3). BMI is the most variable of the three traits, as we expect for such a composite trait with a less direct relationship to age than either height or weight. This variation is characterized by both fluctuations in individual BMI curves and the large population-wide spread of BMI measurements that increases substantially throughout adolescence (Figure 3). Children and teens who fall below the 5th BMI percentile are considered underweight by one broadly accepted clinical standard, those between 5 and 85% are healthy, those between 85–95% are considered overweight, and those above 95% are considered obese (Barlow, 2007). Table 2 displays descriptive statistics for child height, weight, and BMI in Bwa Mawego alongside height-for-age, weight-for-age, and BMI-for-age *z*-score statistics based on the 2007 WHO reference tables (de Onis et al., 2007). Stunting (height-for-age < $-2SD$), underweight (weight-for-age <$-2SD$), and wasting (BMI-for-age <$-2SD$) appear to be uncommon in this population given that no more than 2 children fall into any one of these categories in any age class (Table 2). Higher proportions of children fall above 1 standard deviation in BMI-for-age *z*-scores, but less than 10% are considered overweight (>+2*SD*) after age 3 by WHO standards (WHO, 2010).

Bayesian LMMs characterize the variation observed in height, weight, and BMI. Although sex and age were modeled as fixed effects in all LMMs, we do not report their coefficients since these control variables are better visualized (Figures 1–3), and age was modeled as

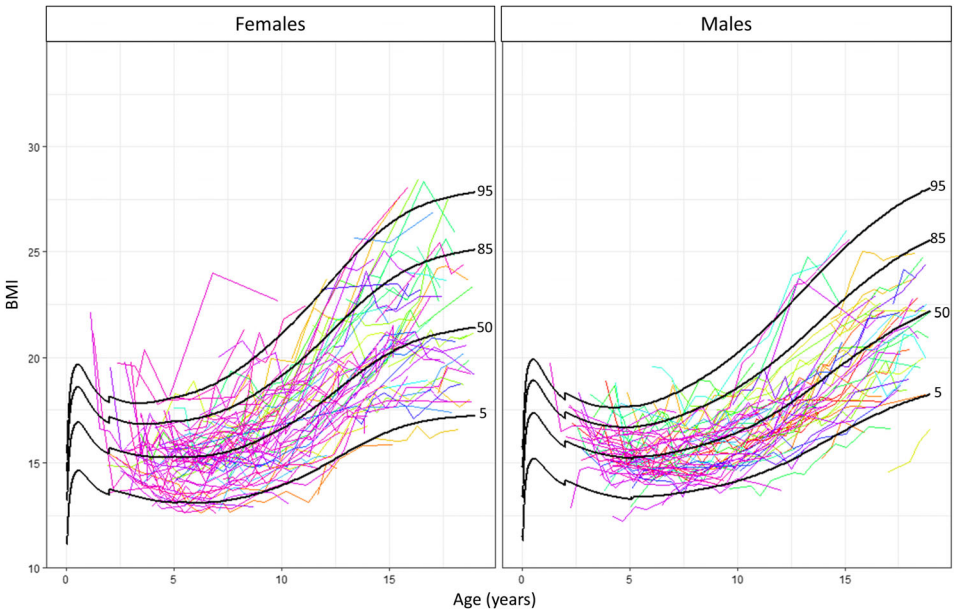

FIGURE 3 BMI curves for 251 children ages 0–19 years plotted with WHO percentiles for comparison (de Onis et al., 2007). BMI, body mass index; WHO, World Health Organization

a spline which complicates interpretations of its model coefficients. We report beta coefficient estimates for the period effect control (secular trends) in Table 3. Since height, weight, and BMI were log-transformed as outcome variables, these coefficients are interpreted on a multiplicative scale (Flanders, DerSimonian, & Freedman, 1992). The collection year variable used to measure this effect was *z*-scored (mean = 2002; *SD* = 3.5). Across the 20-year data collection period, exponentiating these beta coefficients and converting them to percentages indicates that height increased by 6.5%, weight increased by 16.0%, and BMI increased by 2.9% in this population (Table 3). Credible intervals indicate that height and weight show clearly increasing secular trends, whereas the 90% interval for BMI spans zero and does not show a significantly positive trend. Height, weight, and BMI in 5-year-olds plotted from 1997 to 2007 show secular trends in the raw anthropometric data for a single age of significance and also support these model coefficients (Figures 4–6). We show secular trends at this age because body fat is typically at its lowest percentage between 5 and 6 years, and children who are overweight at this age show increased risks of metabolic disorders later in life (Moore et al., 2003; Nader et al., 2006). Height and weight show increasing secular trends in 5-year-olds during the 1997–2007 decade that are slightly higher in males than females (Figures 4 and 5). BMI trends are less clear, and there is a larger spread of variation in this metric than for either height or weight at this specific age (Figure 6). BMI appears to increase slightly in 5-year-old females between 1997 and 1999 before plateauing, and males show a modest increase between 2005 and 2007.

LMM results show that height, weight, and BMI are all highly repeatable for the 260 individuals in this analysis (Table 3; Figure 7). We report modes and credible intervals to characterize posterior probability distributions. Unlike confidence intervals that reflect accuracy in reference to theoretical probability distributions, credible intervals denote ranges of variation in parameter estimates to describe the shape of posterior distributions that have been produced by updating prior probability distributions with observed data (McElreath, 2015). Modes reflect the most probable beta coefficient and variance component values, and 90% credible intervals encompass 90% of the values sampled from posterior distributions. High posterior modes with small credible intervals indicate that approximately 82% of the variation in height and 81% of the variation in weight are explained by variance within-individual growth curves in this population when also controlling for sex, age, and secular trends in these longitudinal data. BMI is less predictable than height or weight as individuals age, but variance within individuals still explains 77% of the population-wide variation not captured by age, sex, or secular trends (Table 3).

Heritability estimates reveal that, after accounting for the impacts of sex, age, and secular trends, genetic variation explains substantial proportions of variation observed between individuals' height, weight, and BMI, and also explains large proportions of the repeatabilities modeled in the set of LMMs with only one random effect (Table 3). Heritability estimates reflect only the proportions of variation between individuals that are explained by shared genes because these LMMs also included individual IDs as a random effect to account for variation within-individual growth curves. Thus, heritability estimates are independent of repeated measures within individuals, but repeatability estimates do encompass what is measured in heritabilities because all aspects of an individual's identity (including genetic and nongenetic attributes) that impact the observed phenotypes are captured in repeatability ratios. Additive genetic variance accounts for approximately 68% of the observed variation between individuals in height, 64% for weight, and 49% for BMI when also controlling for repeated measures within individuals. Although 90% credible intervals are much wider for heritabilities than repeatabilities, the lower limits for all heritability intervals are greater than 0.20, indicating that genetic variation significantly impacts phenotypic variation for all three traits (Figure 7).

TABLE 2 Descriptive anthropometrics with WHO-reference *z*-score statistics

Height										
Age (years)	***N***	**Mean (cm)**	***SD* (cm)**	**Range (cm)**	**Mean HAZ**	**HAZ *SD***	**% <−2*SD***	**% <−1*SD***	**% >+1*SD***	**% >+2*SD***
Females										
0–3	21	90.13	6.35	(74.0, 100.3)	0.70	1.70	0 (0%)	3 (14.3%)	2 (9.5%)	4 (19.0%)
3–5	26	103.34	5.73	(95.4, 116.6)	0.00	0.97	1 (3.8%)	2 (7.7%)	3 (11.5%)	1 (3.8%)
5–10	32	119.95	9.49	(107.7, 142.2)	0.07	0.78	0 (0%)	1 (3.1%)	4 (12.5%)	0 (0%)
10–15	30	154.98	8.45	(142.5, 175.0)	0.03	1.03	1 (3.3%)	3 (10.0%)	6 (20.0%)	1 (3.3%)
15–19	18	162.38	5.94	(155.0, 178.1)	−0.06	0.91	0 (0%)	2 (11.1%)	1 (5.6%)	1 (5.6%)
Males										
0–3	13	90.62	5.27	(78.10, 98.55)	0.08	1.15	1 (7.7%)	1 (7.7%)	1 (7.7%)	1 (7.7%)
3–5	33	103.56	6.78	(90.6, 122.8)	0.00	1.19	1 (3.0%)	4 (12.1%)	3 (9.1%)	3 (9.1%)
5–10	29	124.30	10.30	(101.1, 138.5)	0.00	0.90	1 (3.4%)	2 (6.9%)	3 (10.3%)	1 (3.4%)
10–15	22	151.10	11.79	(122.7, 167.5)	−0.34	1.01	1 (4.5%)	4 (18.2%)	2 (9.1%)	0 (0%)
15–19	22	169.34	9.52	(137.3, 183.7)	−0.63	1.23	1 (4.5%)	4 (18.2%)	1 (4.5%)	0 (0%)
Weight										
Age (years)	***N***	**Mean (kg)**	***SD* (kg)**	**Range (kg)**	**Mean WAZ**	**WAZ *SD***	**% <−2*SD***	**% <−1*SD***	**% >+1*SD***	**% >+2*SD***
Females										
0–3	20	13.15	2.57	(8.62, 17.69)	0.99	1.37	0 (0%)	1 (5.0%)	6 (30.0%)	4 (20.0%)
3–5	27	16.70	2.55	(13.15, 21.77)	0.13	1.00	1 (3.7%)	3 (11.1%)	4 (14.8%)	1 (3.7%)
5–10	32	22.45	4.66	(15.06, 33.57)	−0.04	0.80	0 (0%)	3 (9.4%)	3 (9.4%)	0 (0%)
Males										
0–3	14	13.66	0.96	(12.25, 14.97)	0.53	0.59	0 (0%)	0 (0%)	2 (14.3%)	0 (0%)
3–5	33	16.74	2.56	(12.70, 24.04)	0.07	1.00	0 (0%)	7 (21.2%)	4 (12.1%)	1 (3.0%)
5–10	29	24.74	4.93	(14.06, 34.02)	0.03	1.04	1 (3.4%)	3 (10.3%)	5 (17.2%)	1 (3.4%)
BMI										
Age (years)	***N***	**Mean**	***SD***	**Range**	**Mean BAZ**	**BAZ *SD***	**% <−2*SD***	**% <−1*SD***	**% >+1*SD***	**% >+2*SD***
Females										
0–3	16	17.04	2.83	(13.29, 22.13)	0.88	1.73	0 (0%)	2 (12.5%)	1 (6.3%)	6 (37.5%)
3–5	26	15.72	1.54	(12.95, 18.59)	0.24	1.06	0 (0%)	4 (15.4%)	6 (23.1%)	1 (3.8%)
5–10	32	15.48	1.69	(12.71, 20.05)	−0.16	1.00	1 (3.1%)	5 (15.6%)	2 (6.3%)	1 (3.1%)
10–15	30	19.10	2.65	(13.47, 25.64)	−0.01	1.00	1 (3.3%)	1 (3.3%)	6 (20.0%)	0 (0%)
15–19	18	21.90	4.27	(17.73, 33.85)	0.12	1.09	0 (0%)	2 (11.1%)	4 (22.2%)	1 (5.6%)
Males										
0–3	13	16.82	1.70	(12.83, 19.72)	0.65	1.34	1 (7.7%)	0 (0%)	5 (38.5%)	1 (7.7%)
3–5	33	15.57	1.46	(12.44, 18.89)	0.11	1.10	2 (6.1%)	1 (3.0%)	3 (9.1%)	3 (9.1%)
5–10	29	15.87	1.57	(12.97, 19.55)	0.01	1.09	1 (3.4%)	3 (10.3%)	2 (6.9%)	1 (3.4%)
10–15	22	18.01	2.66	(14.61, 25.42)	−0.18	0.99	1 (4.5%)	4 (18.2%)	0 (0%)	1 (4.5%)
15–19	22	20.59	2.08	(14.56, 23.93)	−0.23	0.90	1 (4.5%)	1 (4.5%)	0 (0%)	0 (0%)

Abbreviations: BAZ, BMI-for-age z-score; BMI, body mass index; HAZ, height-for-age z-score; WAZ, weight-for-age z-score; WHO, World Health Organization.

We report two R^2 statistics defined specifically for LMMs by Nakagawa and Scheilzeth (2013) (Table 3). R^2_m values measure the proportion of variation in growth phenotypes explained by only the fixed effects of each model. Sex, age, and secular trends explain approximately 92% of the observed variation in height, 89% of the variation in weight, and 58% of the variation in BMI in this population. R^2_c values measure the proportion of variation in phenotypes explained by both fixed and random effects of each model (Nakagawa & Scheilzeth, 2013). We report conditional R^2 estimates from the set of heritability models, and the combination of sex, age, secular trends, within-individual variance, and additive genetic variance explains approximately 99% of the observed variation in height,

	Height	Weight	BMI
V_I	0.019 (0.016, 0.021)	0.199 (0.170, 0.233)	0.107 (0.097, 0.131)
Repeatability	0.817 (0.790, 0.838)	0.813 (0.787, 0.841)	0.772 (0.743, 0.802)
V_A	0.014 (0.009, 0.022)	0.162 (0.105, 0.246)	0.071 (0.028, 0.112)
Heritability	0.683 (0.450, 0.836)	0.640 (0.453, 0.837)	0.487 (0.213, 0.704)
Beta (period)	0.011 (0.008, 0.014)	0.026 (0.016, 0.035)	0.005 (−0.003, 0.012)
R^2_m	0.924 (0.914, 0.934)	0.891 (0.876, 0.905)	0.580 (0.534, 0.614)
R^2_c	0.986 (0.986, 0.987)	0.981 (0.979, 0.982)	0.905 (0.893, 0.913)

Note: R^2_m measures the proportion of observed variation explained by only the fixed effects of each heritability model (sex, age, and period effect), and R^2_c is that explained by all of the fixed and random effects (sex, age, period effect, within-individual variance, and additive genetic variance).
Abbreviations: BMI, body mass index; LMM, linear mixed model.

TABLE 3 LMM posterior modes and 90% credible intervals for within-individual variance (V_I), additive genetic variance (V_A), repeatability and heritability ratios, and secular trends

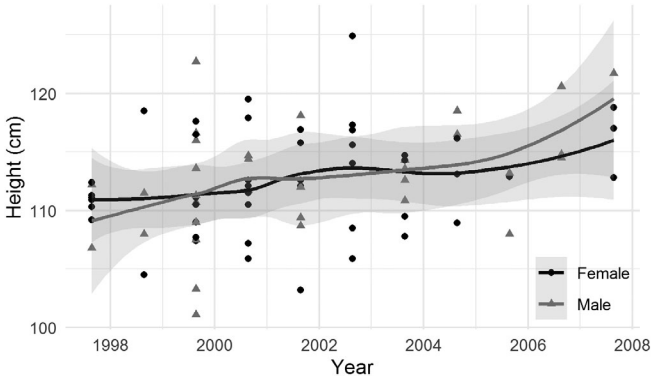

FIGURE 4 Height of children between ages 5 and 6 years from 1997 to 2007. One hundred and twenty-eight data points for 80 children (33 males and 47 females) are plotted with loess curves showing moving averages and 95% confidence intervals

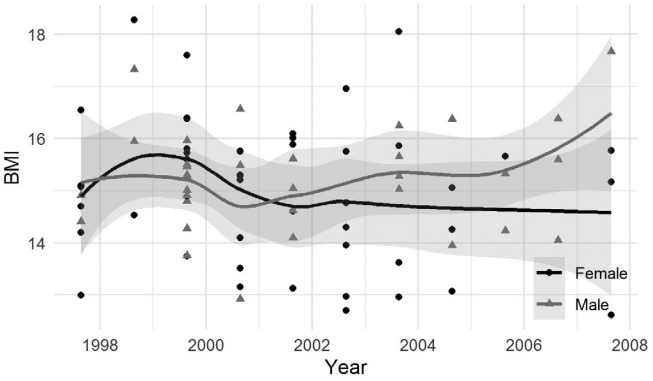

FIGURE 6 BMI of children between ages 5 and 6 years from 1997 to 2007. One hundred and twenty-eight data points for 80 children (33 males and 47 females) are plotted with loess curves showing moving averages and 95% confidence intervals. BMI, body mass index

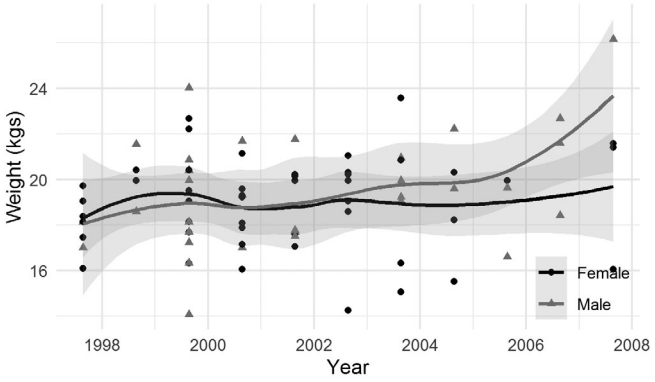

FIGURE 5 Weight of children between ages 5 and 6 years from 1997 to 2007. One hundred and twenty-eight data points for 80 children (33 males and 47 females) are plotted with loess curves showing moving averages and 95% confidence intervals

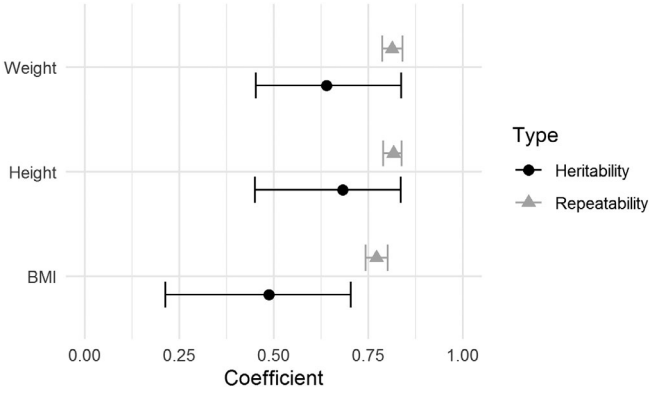

FIGURE 7 Repeatability and heritability estimates from Bayesian LMMs. Plotted modes and 90% credible intervals summarize 1,000 samples from posterior distributions of within-individual variance components and additive genetic variance components. LMM, linear mixed model

98% of the variation in weight, and 91% of the variation in BMI (Table 3). These statistics indicate that age, sex, and secular trends explain the majority of anthropometric variation, leaving relatively small amounts of variation to be explained by within-individual and additive genetic variances. However, repeatability and heritability variance components account for much more of the variation observed in BMI than for variation observed in height or weight (Table 3).

4 | DISCUSSION

We analyzed longitudinal measures of body size in a small-scale Caribbean population that has recently transitioned nutritionally and behaviorally to include more Westernized dietary products and technologies alongside traditional subsistence horticultural practices. Height, weight, and BMI measurements track growth for 260 individuals in a remote village in Dominica. Individual BMI growth curves show large increases for many females in adolescence and into adulthood while more males appear to be overweight earlier in childhood (Figure 3).

Sex-specific differences in growth and variation are population-specific and age-dependent, related to environmental stressors, morbidity, gender-biased resource distributions, and life-history trade-offs (Stinson, 1985). Growth phenotypes from Bwa Mawego follow general patterns observed in other small-scale tropical societies in which males exhibit less variation than females (Walker et al., 2006), a pattern also seen in BMI across Australia and several European countries (Schousboe et al., 2003). The combination of higher levels of adiposity in females with substantial genetic variation in different patterns of fat distribution may contribute to greater variation in female versus male BMI (Samaras et al., 1997). Additional data regarding more detailed body composition, morbidity, specific behavioral and dietary variables, and activity levels are needed to address potential underlying causes of patterns observed between male and female anthropometric variation in Bwa Mawego.

The individuals in this study range in age from birth to 27 years old throughout the 20-year data collection period (1997–2017). We modeled age, sex, and collection year simultaneously to capture secular trends independent of age or sex, and period effect beta coefficients show that height and weight have increased over these decades during which the population as a whole has gained access to imported and processed foods, piped water, electricity, and other resources such as internet and cell phones (Table 3). From 1997 to 2017, 15-year-olds in Bwa Mawego gained approximately 10.9 cm in height, 9.3 kg in weight, and 0.6 units in BMI. Similar data from the Seychelles that span a nutritional transition show gains of 10–13 cm in height and 9–15 kg in weight over a 50-year period in 15-year-old adolescents (Marques-Vidal, Madeleine, Romain, Gabriel, & Bovet, 2008). Secular trends among U.S. children and adolescents show similar increases in weight (+5–7 kg) and relatively smaller gains in height (+1.5–2.0 cm) from 1960–2002 (Ogden, Fryar, Carroll, & Flegal, 2004). Longitudinal data from Tsimane forager-horticulturalists show that adolescents gained 0.6 cm in height, 0.5–1.4 kg in weight, and 0.16–0.56 units in BMI per decade from 2002 to 2015 (Blackwell et al., 2017). Few individuals in Bwa Mawego fall into clinically defined overweight or obese categories at any time point in these longitudinal data (Table 2; Figure 3), and we do not find clear evidence of a population-wide increase in BMI in these younger generations (Table 3; Figure 6).

Age, sex, and secular trends account for the majority of variation in anthropometric phenotypes in Bwa Mawego, but far less in BMI than height or weight (marginal R^2 in Table 3). Repeatabilities and heritabilities measure the proportions of phenotypic variation explained by within-individual and additive genetic variances that are residual to the variation explained by sex, age, and secular trends. Repeatability estimates show that aspects of an individual's identity, including both genetic and nongenetic factors such as behavior, are highly predictive of these anthropometrics as individuals age. All repeatabilities are greater than 75% (Table 3), leaving low residual variances unexplained in these repeated measures.

Heritability estimates for height (0.68), weight (0.64), and BMI (0.49) in Bwa Mawego are lower than many published estimates from twin studies (Elks et al., 2012; Silventoinen, 2003; Silventoinen et al., 2017), but well within the range of estimates from other types of family-based designs that are likely less inflated from common developmental environments than those shared by twins (Elks et al., 2012). We acknowledge that common environments may inflate our estimates of heritability slightly; however, flexible and fluctuating residence patterns in Bwa Mawego diffuse much of the household/spatial clustering known to influence anthropometric variation and heritability estimates in other populations (Heckerman et al., 2016; Saunders & Gulliford, 2006). Much of the variance in anthropometrics left unexplained by age, sex, and secular trends is attributed to additive genetic variance in this Caribbean population (Table 3; Figure 7), and future molecular research is needed to characterize specific genetic influences on variable anthropometric and metabolic health outcomes. This is particularly warranted in reference to body mass index given that age, sex, and secular trends explain much less of the variation in BMI than in height or weight, bolstering the relative importance of genetic variation (Table 3).

Variation in BMI between populations is best explained by environmental, ecological, and behavioral factors, but most of the variation within populations appears to be explained by genetic variation (Bogardus, 2009). Despite estimating moderate to large heritabilities in a multitude of populations, geneticists have yet to account for most of this alleged genetic variation with specific variants, creating a problem of "missing" heritability. Diverse, small-scale populations that are underrepresented in the current genetic literature may be valuable resources for discerning how biological, cultural, and environmental factors intersect to shape anthropometric variation and health on a more inclusive, global scale (Popejoy & Fullerton, 2016). Substantial contributions of additive genetic variance to anthropometric variation in this Caribbean population of mixed ancestry (Moreno-Estrada et al., 2013) warrant further investigation, especially given the large amount of variation in body mass index between individuals and the lack of population-wide secular trends throughout this transitional period (Table 3; Figures 3 and 6).

We have assessed the impacts of age, sex, secular trends, within-individual variance, and additive genetic variance on phenotypic variation in height, weight, and BMI in a Caribbean community that has recently transitioned to include more Western foods and technologies into traditional horticultural diets and subsistence practices. Anthropometric heritabilities are moderate in this population and body mass index varies considerably between individuals, but metabolic health

correlates of anthropometric variation remain unknown at this time. Additional data regarding specific behavioral, dietary, environmental, and genetic factors will enhance our understandings of anthropometric variation and health in the future.

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DATA AVAILABILITY STATEMENT

The data for these analyses are not available due to privacy and ethical restrictions. Heritability estimates require linked identifiers between phenotypes and pedigree membership, precluding de-identification of personal and anthropometric data.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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