Received: 2 July 2023 | Revised: 24 December 2023 | Accepted: 9 February 2024

DOI: 10.1002/ajhb.24056

ORIGINAL ARTICLE

American Journal of Human Biology WILEY

Variation in diurnal cortisol patterns among the Indigenous Shuar of Amazonian Ecuador

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Funding information
National Science Foundation; Ryoichi Sasakawa Young Leaders Fellowship Fund; University of Oregon; Wenner-Gren Foundation

Abstract

Objectives: The hypothalamic–pituitary–adrenal (HPA) axis and its primary end product, the glucocorticoid cortisol, are major components of the evolved human stress response. However, most studies have examined these systems among populations in high-income settings, which differ from the high pathogen and limited resource contexts in which the HPA axis functioned for most of human evolution.

Methods: We investigated variability in diurnal salivary cortisol patterns among 298 Indigenous Shuar from Amazonian Ecuador (147 males, 151 females; age 2–86 years), focusing on the effects of age, biological sex, and body mass index (BMI) in shaping differences in diurnal cortisol production. Saliva samples were collected three times daily (waking, 30 minutes post-waking, evening) for three consecutive days to measure key cortisol parameters: levels at waking, the cortisol awakening response, the diurnal slope, and total daily output.

Results: Age was positively associated with waking levels and total daily output, with Shuar juveniles and adolescents displaying significantly lower levels than adults ($p < .05$). Sex was not a significant predictor of cortisol levels ($p > .05$), as Shuar males and females displayed similar patterns of diurnal cortisol production across the life course. Moreover, age, sex, and BMI significantly interacted to moderate the rate of diurnal cortisol decline ($p = .027$).

Overall, Shuar demonstrated relatively lower cortisol concentrations than high-income populations.

Conclusions: This study expands the documented range of global variation in HPA axis activity and diurnal cortisol production and provides important insights into the plasticity of human stress physiology across diverse developmental and socioecological settings.

1 | INTRODUCTION

The human stress response has been shaped by natural selection to regulate cognitive, biological, and behavioral responses to adaptively relevant threats posed by ancestral environments (Bribiescas & Muehlenbein, 2010; Nepomnaschy & Flinn, 2009; Nesse & Young, 2000; Worthman, 2015). Notably, neuroendocrine activity associated with the hypothalamic–pituitary–adrenal (HPA) axis and its primary end product, the glucocorticoid steroid hormone cortisol, have been identified as key components of the evolved human stress response (Rao & Androulakis, 2019; Sapolsky, 2021; Selye, 1956). For example, upon encountering evolutionarily relevant cues of a real or perceived stressor, the HPA axis activates a state of physiological and behavioral preparedness by releasing cortisol, which in turn orchestrates energy mobilization toward immediate biological demands and away from investments in maintenance, growth, and reproduction (Finch & Rose, 1995; Kadmiel & Cidlowski, 2013; Sapolsky et al., 2000).

While short-term activation of the HPA axis evolved to respond to immediate threats, chronic and repeated stimulation of this system contributes to negative physical and mental health outcomes, including cardiovascular disease, type 2 diabetes, immune suppression, impaired growth, depression, and cognitive impairment (Björntorp & Rosmond, 2000; Cohen et al., 2006; Flinn & England, 1995; Gunnar & Vazquez, 2001; Juster et al., 2010; Lupien et al., 2018; McEwen, 1998; O'Connor et al., 2021). In particular, many chronic psychosocial stressors (e.g., occupational pressures, living among strangers in high population densities) are relatively recent phenomena that differ from brief, episodic threats in evolutionary-relevant contexts and are recognized as major triggers of HPA axis activity with downstream effects on health (Boyce & Ellis, 2005; Flinn et al., 2011; Nesse & Young, 2000; Sapolsky, 2021).

Despite important advances in stress research over recent decades (e.g., Epel et al., 2018; Kirschbaum et al., 1993; McEwen & Seeman, 1999), most studies examining variability in HPA axis activity and cortisol production have been conducted among children and adults from high-income countries (HICs), with few studies evaluating the basic biological mechanisms of these systems among heterogeneous populations in resource-limited settings, such as those in low- and middle-income countries (LMICs). In particular, HPA axis function is understudied among Indigenous peoples in subsistence-based societies who face various physiological burdens (e.g., high parasitic infections, caloric restriction, high fertility) along with psychosocial stressors associated with culture change such as increasing market integration (MI; degree of production for and consumption from a market-based economy; Gildner et al., 2020; Liebert et al., 2013; Lu, 2007; Urlacher, Liebert, et al., 2016). The narrow focus on high-income populations consequently provides a limited understanding of population variation in fundamental components of the human stress response across different ecological, psychological, and developmental contexts and obscures interpretations of these complex physiological processes and their associations with chronic stress and disease (Nyberg, 2012; Urlacher, Liebert, et al., 2018; Worthman & Panter-Brick, 2008). To expand the documented range of global variation in HPA axis activity, the present study investigates diurnal cortisol patterns among the Indigenous Shuar, a forager-horticulturalist population from Amazonian Ecuador living in a high pathogen and limited resource setting, with a specific focus on the effects of age, reported sex, and body composition in shaping individual differences in cortisol activity.

1.1 | Diurnal cortisol patterns, human variation, and health

1.1.1 | Diurnal cortisol patterns

Cortisol is among the most studied neuroendocrine biomarkers of physiological and psychological stress (Brewis et al., 2021; Condon, 2018). Cortisol measurements from saliva (compared to other biospecimens like hair) present opportunities to capture real-time, biologically active levels of unbound cortisol in momentary and naturalistic settings (DeCaro & Helfrecht, 2022; Djuric et al., 2008;

Hellhammer et al., 2009). Although studies of salivary cortisol have been conducted predominantly among high-income populations, this body of research shows that cortisol production typically exhibits a strong diurnal rhythm anchored by person-specific sleep-wave schedules, resulting in levels that are elevated upon waking, increase to peak concentrations approximately 30–45 min post-waking, and subsequently decline to nadir before sleep (Adam et al., 2017; Adam & Kumari, 2009; Kirschbaum & Hellhammer, 1989; Kudielka et al., 2012; Pruessner et al., 1997; Stalder et al., 2016). Tightly regulated by negative hormonal feedback processes, the presence of this robust diurnal curve tends to signify healthy HPA axis functioning; however, deviations from the more common diurnal cortisol pattern are recognized as potential mechanisms and markers of multisystemic biological dysregulation caused by chronic stress (Adam et al., 2017; Gunnar & Vazquez, 2001; Karlamangla et al., 2022; Miller et al., 2007).

To elucidate the links among cortisol activity, human variation, and health, researchers have measured several parameters from the diurnal cortisol rhythm, including *cortisol levels at waking*, the *cortisol awakening response (CAR)*, the *diurnal slope*, and *total daily cortisol output* (Adam et al., 2017; Adam & Kumari, 2009; Ross et al., 2014; Stalder et al., 2016). Studies conducted in HICs provide evidence that each cortisol index offers unique information on the diurnal rhythm and may be associated with specific physical and psychological conditions (O'Connor et al., 2021; Saxbe, 2008). For example, *cortisol levels at waking*, which are collected as soon as possible upon awakening in the morning (i.e., immediately after opening eyes), establish the baseline for the subsequent rhythm of cortisol activity throughout the remainder of the day (Adam & Kumari, 2009; Stalder et al., 2016). Lower cortisol levels at waking, in particular, compress the dynamic range of diurnal cortisol production and are significantly correlated with current and future health outcomes (e.g., fatigue, obesity risks) (Karlamangla et al., 2022; Kumari et al., 2009; Ruttle et al., 2013; Saxbe, 2008; Yu et al., 2020).

Another common marker of HPA axis activity is the *CAR*, defined as the increase in cortisol levels from waking to 30–45 min post-waking (Fries et al., 2009; Pruessner et al., 1997). The function of the CAR remains incompletely understood; however, current evidence suggests that this parameter represents a discrete index superimposed on the diurnal cycle that is under different regulatory control from the cortisol released throughout the rest of the day (Clow et al., 2010; Kudielka et al., 2012). Notably, both blunted and heightened CARs are associated with negative physical and psychological health conditions in HICs, including cancer risks and burnout (Chida & Steptoe, 2009; Sephton et al., 2000).

Additionally, the *diurnal cortisol slope* (i.e., degree of change in cortisol levels from early morning to late evening) is a central feature of the daily pattern of cortisol activity, with a growing body of literature suggesting that it is an important indicator of stress-related circadian dysregulation across multiple biological systems (Adam et al., 2017; Karlamangla et al., 2022). Specifically, a daytime cortisol rhythm demonstrating a flat decline (i.e., a more positive slope), which commonly emerges from low cortisol levels at waking and subsequently high evening levels, has been linked to psychosocial stress and poorer health outcomes in HICs (e.g., inflammation and major depressive disorder) (Adam et al., 2017; Adam & Kumari, 2009; Jarcho et al., 2013; Kumari et al., 2009; Sephton et al., 2000; Sephton & Spiegel, 2003).

Studies of diurnal cortisol also frequently include estimates of *total daily cortisol output*, or more specifically, *total area under the cortisol curve with respect to the ground (AUC_g)*. Researchers hypothesize that this index may denote cumulative tissue exposure to cortisol throughout the day as well as hypo- and hyperactivity of the HPA axis (Ross et al., 2014; Saxbe, 2008), with both low and high AUC_g levels demonstrating significant associations with adverse physical and mental health outcomes (Gunnar & Vazquez, 2001; Vreeburg et al., 2013).

1.1.2 | Key factors associated with variation in diurnal cortisol patterns

Indices of diurnal cortisol production are often associated with sociodemographic characteristics, such as age and reported sex, as well as anthropometric factors related to chronic disease risks (e.g., proxies of body composition like body mass index [BMI]) (Adam et al., 2017; Champaneri et al., 2013; Darnall & Suarez, 2009; Miller et al., 2016; Ruttle et al., 2013; Yu et al., 2020). These person-based variables are considered important moderators of individual differences in reactivity to acute and chronic stress and may consequently impact diurnal cortisol rhythms and disparities in health (Jessop & Turner-Cobb, 2008; Kudielka et al., 2012; Novais et al., 2017; Saxbe, 2008). For example, some studies among high-income populations suggest that the magnitude of circadian cortisol levels, including waking cortisol, the CAR, and AUC_g, increase gradually with age, yet the change in cortisol levels throughout the day (i.e., diurnal slope) may become blunted over time due to adrenocortical dysfunction associated with senescence and delayed termination of the stress response from chronic wear and tear (Adam et al., 2006; Dmitrieva et al., 2013; Gaffey &

Martinez, 2019; Karlamangla et al., 2013; Miller et al., 2016; Nater et al., 2013).

Recent evidence further suggests that diurnal cortisol patterns are shaped by developmental, physiological, and psychosocial factors related to sex and gender (Darnall & Suarez, 2009; DuBois & Shattuck-Heidorn, 2021; Juster et al., 2019). Studies based on binary categories of biological sex demonstrate that differences in cortisol levels between males and females may become evident during key stages of ontogeny (Almeida et al., 2009; Gaffey et al., 2016), with females displaying higher morning cortisol levels and steeper diurnal cortisol slopes than age-matched males during early stages of development (e.g., infancy and adolescence) (Rolfsjord et al., 2017; Shirtcliff et al., 2012), whereas males have flattened cortisol profiles (Adam et al., 2006; Dmitrieva et al., 2013) as well as higher morning and total daily cortisol levels compared to females at older ages (Karlamangla et al., 2013; Miller et al., 2016). Overall, these results indicate that age and biological sex may interact to moderate diurnal cortisol activity among males and females across the human life course, although the causal biological and social pathways underlying these associations remain incompletely understood (Darnall & Suarez, 2009; DuBois & Shattuck-Heidorn, 2021).

Additional studies have investigated potential links between diurnal cortisol variability and anthropometric measurements of body composition to illuminate the relationship between HPA axis functioning and the etiology of stress-related chronic diseases, including obesity and type 2 diabetes (Adam et al., 2017; Bose et al., 2009; Rosmond, 2005; Rutters et al., 2012; Ruttle et al., 2013). For example, research conducted among obese and non-obese children and adults demonstrates that relatively lower morning cortisol concentrations and flatter diurnal cortisol slopes are significantly correlated with increased measurements of generalized and abdominal adiposity (e.g., BMI and waist circumference, respectively) (Champaneri et al., 2013; Kumari et al., 2010; Morita et al., 2016; Ruttle et al., 2013; Therrien et al., 2007; Wallerius et al., 2003; Yu et al., 2020). While the literature to date remains inconclusive (Rodriguez et al., 2015), these findings provide evidence that indices of diurnal cortisol activity may represent important markers of HPA axis dysregulation, obesity, and other chronic diseases (Adam et al., 2017; Björntorp et al., 1999).

1.2 | Limitations of previous research

While previous research sheds light on the complexity of HPA axis activity and diurnal cortisol production in relation to sociodemographic and anthropometric variables, most studies examining these relationships have been conducted among children and adults from HICs. For example, recent work by Miller et al. (2016) presents sex-specific normative reference ranges for diurnal cortisol levels across the lifespan, thereby affording an opportunity for researchers and clinicians to interpret deviations from the "normal" circadian pattern. However, these reference values are exclusively based on studies among North American and European children and adults, which provides a narrow understanding of the ways in which stress-responsive physiological mechanisms are regulated across a range of diverse ecologies and may consequently result in biased interpretations of "abnormal" or "dysregulated" diurnal cortisol profiles when used as a reference for populations outside of HICs (DeCaro & Helfrecht, 2022; Wiley, 2021).

Several noteworthy studies have examined cortisol activity among resource-limited populations in LMICs, including in Dominica (Decker, 2000; Flinn & England, 1995; Ponzi et al., 2015), Nepal (Worthman & Panter-Brick, 2008), Mongolia (Hruschka et al., 2005), Guatemala (Nepomnaschy et al., 2004), Paraguay (Amir et al., 2015), the Philippines (Gettler et al., 2011), Kenya (Pike & Williams, 2006), Honduras (García et al., 2017), and the Republic of Congo (Sarma et al., 2020); however, this research is often constrained to one biological sex, narrow age ranges, and specific sampling times and frequencies. Providing more comprehensive analysis, studies by Nyberg (2012) and Urlacher, Liebert, et al. (2018) analyzed diurnal cortisol rhythms, namely cortisol levels at waking and diurnal cortisol slopes, across the lifespan among the Indigenous Tsimane' of Bolivia and Garisakang of Papua New Guinea, respectively, revealing that these forager-horticulturalist groups have the lowest recorded cortisol levels to date across all population-based studies. Similar to findings documented in HICs, Nyberg (2012) and Urlacher, Liebert, et al. (2018) demonstrated that variation in diurnal cortisol production among the Tsimane' and Garisakang is associated with sociodemographic factors, specifically age and sex, with males and females in each population displaying unique ontogenetic patterns of HPA axis activity. Given that Indigenous populations in resource-limited locations endure distinct physical and psychosocial stressors (e.g., high pathogen exposure, marginal nutrition, limited access to health services, increased integration into market-based economies) (Gracey & King, 2009; Snodgrass, 2013; Valeggia & Snodgrass, 2015), more research among these groups is greatly needed to fully capture global diversity in HPA axis activity and to elucidate its impact on chronic stress and disparities in health.

1.3 | Objectives and hypotheses of the present study

In the present study, we examine diurnal salivary cortisol rhythms among the Shuar, an Indigenous, forager-horticulturalist population from the neo-tropical region of Amazonian Ecuador living in a high pathogen and limited resource setting and currently experiencing a range of cultural and economic changes associated with MI (Gildner et al., 2020; Liebert et al., 2013; Urlacher, Liebert, et al., 2016). By investigating sociodemographic and anthropometric factors and their associations with multiple diurnal cortisol parameters, including cortisol levels at waking, the CAR, the diurnal slope, and AUC_g, this study addresses the following objectives and hypotheses:

- **Objective 1**: To examine associations between age and diurnal cortisol rhythms among the Shuar. **Hypothesis 1.1**: Based on available evidence (e.g., Adam et al., 2006; Dmitrieva et al., 2013; Karlamangla et al., 2013; Miller et al., 2016; Nater et al., 2013), we hypothesize that age will be positively associated with flatter diurnal cortisol slopes and higher levels of waking cortisol, the CAR, and AUC_g.
- **Objective 2**: To evaluate sex differences in diurnal cortisol patterns among the Shuar. **Hypothesis 2.1**: In accordance with previous findings (e.g., Almeida et al., 2009; Karlamangla et al., 2013; Nyberg, 2012; Shirtcliff et al., 2012), we hypothesize that there will be a significant interaction between sex and age on diurnal cortisol levels. Specifically, we posit that younger females will display elevated levels of waking cortisol, the CAR, and AUC_g and steeper diurnal cortisol slopes than age-matched males, whereas males will have higher levels of waking cortisol, the CAR, and AUC_g and flattened cortisol slopes compared to females at older ages.
- **Objective 3**: To investigate the relationship between BMI (a proxy measure of body composition and generalized adiposity) and diurnal cortisol levels among the Shuar. **Hypothesis 3.1**: Aligning with recent studies demonstrating links between BMI and diurnal cortisol patterns (e.g., Adam et al., 2017; Champaneri et al., 2013; Kumari et al., 2010; Ruttle et al., 2013; Yu et al., 2020), we hypothesize that higher BMI levels will be associated with lower morning, CAR, and AUC_g concentrations as well as flatter diurnal slopes. **Hypothesis 3.2**: Based on documented age-associated changes (Ferraro et al., 2003) and biological sex differences (Stevens et al., 2010; Wells, 2007) in BMI, we further predict that the effects of BMI on diurnal cortisol levels will be significantly moderated by age and sex.

2 | MATERIALS AND METHODS

2.1 | Study population

The present study was conducted under the Shuar Health and Life History Project (SHLHP; http://www.shuarproject.org), a longitudinal research endeavor involving faculty and students from several universities as well as local Ecuadorian organizations, including the *Federación Interprovincial de Centros Shuar* (FISCH), the Ecuadorian *Ministerio de Salud* (Health Ministry), and community members. Shuar are a Jivaroan-speaking, Indigenous population concentrated in the lowland Amazonas region of southeastern Ecuador. Historically, Shuar lived in small, scattered households between the eastern Andean foothills and Cordillera de Cutucú, wherein traditional subsistence practices of horticulture, foraging, hunting, and fishing served as the foundations of their economy (Harner, 1984; Karsten, 1935; Rubenstein, 2001; Stirling, 1938). Today, the Ecuadorian Shuar are a rapidly growing population with an estimated 40 000–110 000 individuals living in 668 communities across ~900 000 hectares of land in the Morona-Santiago, Pastaza, and Zamora-Chinchipe provinces of Ecuador (CODENPE, 2012; Rubenstein, 2001). Across their territory, Shuar are currently undergoing a wide range of cultural and economic changes due to the establishment of modern infrastructure and the emergence of and increased dependence on market-based systems of exchange (Gildner et al., 2020; Liebert et al., 2013; Lu, 2007; Urlacher, Liebert, et al., 2016). While exposure to MI has led to alterations in subsistence practices, household resources, and health (e.g., chronic disease risks, infectious disease patterns, and childhood growth), many Shuar continue to consume a diet based on staple carbohydrates, such as plantains and sweet manioc, and endure high rates of infectious/parasitic diseases and childhood growth faltering (Blackwell et al., 2009; Cepon-Robins et al., 2014; Gildner et al., 2020; Houck et al., 2013; Urlacher et al., 2021; Urlacher, Blackwell, et al., 2016; Urlacher, Ellison, et al., 2018).

2.2 | Participants and sampling

The present study used a cross-sectional design and included 298 Shuar participants (147 males, 151 females; age 2.7–86.3 years; mean age of 18 years), with data collection occurring over four field seasons (July–September 2011–2014) from six communities in the Morona-Santiago province of Ecuador. Volunteers from 50 households participated in the study, with an average of six individuals per household. These households reflected a

spectrum of engagement with MI, with some households exhibiting reliable access to regional market centers and formal medical care, whereas others had more limited access to these services. Pregnant women were excluded from analyses due to the documented links between pregnancy and elevated cortisol concentrations (Kirschbaum et al., 1992).

Individual ages were self- or parent-reported and cross-checked by birthdates on government identification cards, school matriculation records, and more extensive genealogical information gathered via face-to-face interviews (Blackwell et al., 2009; Liebert et al., 2013). Estimates of age were represented as a continuous variable as well as a categorical variable, in which participants were classified into the following age groups: children (2.7–6.9 years; $n = 59$), juveniles (7.0–9.9 years; $n = 60$), adolescents (10.0–15.9 years; $n = 76$), young adults (16.0–34.9 years; $n = 54$), and older adults (35+ years; $n = 49$). The cut-off criteria for these age categories were based on biologically meaningful developmental patterns (Bogin, 1999; Urlacher, Blackwell, et al., 2016) and created relatively equal sample sizes across groups.

This study was approved by village leaders upon participant consensus, the FISCH, and the Office for the Protection of Human Subjects at the University of Oregon. Informed verbal consent was acquired from all adult participants, and parental verbal consent and child assent were obtained for all participants under 15 years of age (the local age of consent).

2.3 | Anthropometrics: Body mass index

We assessed height (m) and weight (kg) using standard anthropometric procedures (Lohman et al., 1988). Stature was recorded to the nearest 1.0 mm using a field stadiometer (Seca Corporation, Hanover, MD), and body weight was measured to the nearest 0.1 kg using a Tanita BF-558 electric scale (Tanita Corporation, Tokyo, Japan). Body mass index (BMI; kg/m^2) was calculated from measured height and weight values. Age- and sex-specific BMI z-scores were calculated from Shuar growth models (Urlacher, Blackwell, et al., 2016) and were used as proxy measures of body composition and generalized adiposity.

From the total sample, 13.8% of participants had missing anthropometry data or anthropometry data collected out of optimal time ranges (>1 month between cortisol and anthropometry collections); these individuals were therefore excluded from the BMI analyses. The pattern of missing anthropometry data was not systematically related to age and sex, with proportional amounts of missing data occurring across all groups. For the remaining sample (86.2%), anthropometric measurements were collected concurrently with cortisol sampling, on average within ±5.7 days.

2.4 | Salivary cortisol collection and assay procedures

On-site, the lead author (MAL) and SHLHP team members (SSU, FCM, TEG, TJC-R, CJH, RGB, LSS) directly collected saliva samples from each participant three times per day for three consecutive days – at time of waking, 30 minutes post-waking, and in the evening—for a maximum of nine cortisol samples per individual. We designed an in-person research protocol to verify all waking and sampling times and to enhance participant compliance and saliva collection accuracy, which are important concerns when measuring and interpreting diurnal cortisol patterns (Adam et al., 2017; Golden et al., 2014; Laufer et al., 2022; Stalder et al., 2016; Stalder et al., 2022). On average, 8.3 samples were collected per participant.

The exact time of sample collection as well as the sleep and wake time of each participant were noted for all days. For each sample, the time between waking and collection (i.e., *hours since waking*) was calculated as absolute hours in decimals (e.g., 12:30 = 12.5 h). To obtain the first morning samples immediately upon waking, we followed a specific protocol, which included: scheduling collection days with households in advance, noting their usual morning wake times, and describing our collection procedures in detail; arriving at their homes in the morning and waiting outside until waking; and then collecting samples from participants in their homes as they successively arose in the morning. Despite our best efforts, logistical challenges precluded the timely collection of some measurements. The mean wake time across all participants was 5:45 AM with first morning samples collected, on average, 11.3 min after waking. In the *Salivary Cortisol Indices* section (below), we discuss strategies for dealing with missing values and samples collected outside of the targeted time ranges.

To collect saliva, participants pooled saliva in their mouths and then gently "drooled" through a short plastic drinking straw into a 2.0 mL vial, which was pre-labeled with the individual ID number, day, and time of collection. Samples were then stored in a portable field freezer (at −30°C) and transported frozen on dry-ice to the United States for analysis in the Global Health Biomarker Laboratory at the University of Oregon.

All cortisol samples were analyzed in duplicate using commercially available, high-sensitivity enzyme immunoassay (ELISA) kits from Salimetrics (Kit #1-3002; State College, PA), which have been well-validated and extensively used for quantitative measurements of salivary

cortisol. Samples assayed in duplicate that did not meet the coefficient of variation (CV) and/or absolute value difference criterion were re-analyzed (Kim et al., 2015). The average intra-assay and inter-assay coefficients were 4.3% and 9.0%, respectively. Twelve percent of cortisol concentrations were below the lower limit of detection, with all occurring in evening samples; for statistical analyses, these values were substituted with the median lower limit of detection calculated across all plates (0.019 μg/dL).

2.5 | Salivary cortisol indices

To capture variation in HPA axis activity, key cortisol indices were extracted from specific portions of the diurnal rhythm including: (1) cortisol levels at waking and the decline in cortisol levels across the day (i.e., the diurnal cortisol slope); (2) the cortisol awakening response (CAR); and 3) total daily cortisol output, or total area under the cortisol curve with respect to the ground (AUC_g). Figure 1 in the Supplementary Materials illustrates these parameters in detail.

2.5.1 | Cortisol levels at waking and the diurnal cortisol slope

The diurnal slope was estimated by a best fit line resulting from a regression of the cortisol values collected across the day onto *hours since waking*, with cortisol levels at waking reflecting the intercept in these models (Adam, 2006; Ong et al., 2011). These calculations excluded the CAR based on evidence that the morning awakening response may be regulated by different biological mechanisms compared to the remaining diurnal rhythm (Clow et al., 2010; Stalder et al., 2016). Accordingly, the diurnal slope was anchored on the first sample of the day, and all positive CAR values (i.e., increase in cortisol levels from waking to 30 minutes post-waking) were not included in subsequent analyses. Of the remaining cortisol values, 7.6% were missing. Additional analyses revealed that missing data did not vary by age; however, there were systematic differences between males (67.4%) and females (32.6%). These discrepancies were accounted for with robust data techniques and were acknowledged when interpreting the results.

2.5.2 | Cortisol awakening response (CAR)

In accordance with recommended consensus guidelines (Stalder et al., 2016; Stalder et al., 2022), the CAR reflected the change in the post-waking increase in cortisol from waking to 30 min after waking. Using an area-under-the curve method, CAR values were calculated from the area of a right-angle triangle formed from the change in time (horizontal) and cortisol concentrations (vertical) from waking to 30 min post-waking (Ross et al., 2014). The CAR was not directly calculated for a specific day if either the waking or 30 min post-waking measurements were missing or if the cortisol concentrations decreased from waking to 30 minutes post-waking. The following criteria were further considered to ensure CAR sampling sufficiency: (1) all first morning samples were collected less than 15 min post-waking; (2) second morning samples were collected less than 50 min post-waking; and (3) the time from first to second morning collection occurred within 25–40 min (Cohen et al., 2006; Stalder et al., 2016; Stalder et al., 2022). For additional verification, we objectively monitored and recorded all waking and sampling time points associated with the CAR. While these conditions reduced the sample size for CAR estimates, such that 51.5% were regarded as missing in subsequent analyses, this conservative strategy ensured that issues of delayed sampling did not bias the results (Smyth et al., 2016). The pattern of missing data among CAR estimates did not differ by age or sex.

2.5.3 | Area under the curve with respect to ground (AUC_g)

Area Under the Curve with Respect to Ground (AUC_g) provided an approximation of total daily cortisol output, or more specifically, the total area under the cortisol values across the waking day with respect to the ground (Adam & Kumari, 2009). Following standard procedures, this index was quantified using a trapezoidal function, with all CAR samples excluded from calculations (Pruessner et al., 2003). Thus, AUC_g values reflected the area of the trapezoid formed from waking and evening cortisol levels (two adjacent vertical lines) and the time between their collection (one horizontal line) (Ross et al., 2014). This parameter was not directly calculated for a specific day if either the waking or evening samples were missing (16.1%). This pattern of missing data did not differ by age; however, there were significant differences in missing data between males (65.3%) and females (34.7%). As previously noted, robust data techniques were implemented to manage these discrepancies and were recognized when interpreting results.

2.6 | Exclusion criteria

Additional information on current illness, medication use, alcohol consumption, and smoking were collected

each day. Participants displaying overt infectious morbidity at the time of collection were not included in the present study. No participants reported use of steroid-based medications, which are known to affect cortisol levels, and all adult participants reported no alcohol or cigarette use within 2 h for each sample collected. Efforts were made to collect all samples prior to morning and evening mealtimes; however, in cases where participants had consumed food within the last 30 min, they were asked to rinse their mouths with water prior to saliva collection. Samples containing known contaminants (e.g., blood, food, dirt) were excluded.

2.7 | Analytic strategy

Prior to analysis, cortisol outliers, defined as +3 SD above the mean, were examined for CAR, AUC_g, and each sample time (at waking, 30 min post-waking, and evening) by age category and sex. A small proportion of cortisol concentrations (<2% for each index) were defined as outliers across all groups, and preliminary tests revealed non-significant differences in the results with outliers excluded. Thus, all sample values were included in subsequent analyses to investigate naturally occurring variation in diurnal cortisol rhythms. Moreover, due to a positive skew in the distribution of cortisol values, all indices were natural log-transformed and retested for normality prior to analysis. Retested transformed variables were normal, with skewness values between ±2; transformed measures of cortisol were therefore used in all analyses.

2.7.1 | Descriptive statistics

Descriptive statistics were calculated for sociodemographic and anthropometric factors (*age*, *BMI*, and *BMI z-scores*) and diurnal cortisol measurements (*waking*, *evening*, *diurnal slope*, *CAR*, and *AUC_g* levels) by sex and age category. One-Way Analysis of Variance (ANOVA) tests were used to evaluate differences in BMI and BMI z-scores by age category and between males and females from the same age category, with *p*-values for post-hoc multiple comparisons adjusted using the Games-Howell method. All statistical analyses were performed in R (R Development Core Team, Version 4.2.2, 2022).

2.7.2 | Multilevel growth curve models

In accordance with previous literature (e.g., Adam, 2006; Hruschka et al., 2005; Nyberg, 2012; Urlacher, Liebert, et al., 2018), we used a series of multilevel growth curve models to describe the diurnal cortisol patterns of the Shuar. Specifically, three-level models were used to estimate cortisol indices (at waking/diurnal slope, CAR, AUC_g), whereby each level represented a different source of variation: within-individuals (Level-1), between-individuals (Level-2), and between-households (Level-3). Preliminary tests identified significant correlations between cortisol values across days for each index: at waking/diurnal slope ($r = .28–.59$; $p < .05$), CAR ($r = .23–.33$, $p < .05$), and AUC_g ($r = .39–.57$, $p < .05$). Given that cortisol measures were significantly correlated across days and testing between-day variance was not a central objective of the present study, day-to-day variance was not estimated in these models.

For the waking/diurnal slope analyses, we first estimated a partially unconditional growth model, which included *hours since waking* as a fixed predictor at Level-1. This model was used to calculate intraclass correlation coefficients (ICCs) and regarded as the baseline model in subsequent analyses. This procedure has been implemented in previous studies examining diurnal cortisol patterns with multilevel growth models because it affords an opportunity to control for the diurnal rhythm (Shirtcliff & Essex, 2008). Accordingly, Level-1 in the waking/diurnal slope model involved the extraction of repeated measures of cortisol at waking and across the day, wherein the natural log-transformed cortisol values were included as the outcome variable and *hours since waking* was the predictor variable. Cortisol levels at waking therefore represented the intercept and the change in cortisol secretion across the day reflected the linear growth coefficient (i.e., slope), which was estimated by fitting a best fit line through the morning and evening cortisol measures (Adam, 2006; Ong et al., 2011).

For the CAR and AUC_g analyses, fully unconditional models, which did not include predictor variables at any level, were used as baseline models to estimate ICC values. In the CAR and AUC_g models, Level-1 included the repeated measures of each cortisol index across 3 days of measurement, in which the CAR and AUC_g were independent outcomes. Time was not added as a predictor at Level-1 in the CAR and AUC_g models because it was included in their calculations.

For all models, Level-2 captured between-individual differences in each cortisol index as predicted by person-based variables. Specifically, *age*, *sex*, and *BMI z-scores* were added as fixed predictors at Level-2 to test the study hypotheses. In these models, *age* and *BMI z-scores* were continuous variables whereas *sex* was a binary variable (0 = male, 1 = female). The *age* and *BMI z-score* variables were centered on the mean values across all participants (18 years and 0.15 z-scores, respectively). The interaction terms (*age*sex, age*BMI z-scores, sex*BMI*

*z-scores, age*sex*BMI z-scores*) were also included at Level-2 to examine their effects on each cortisol parameter. Between-household differences were estimated at Level-3 in each model, with no predictor variables included at this level. Person-level predictors from Level-2 were not allowed to vary at Level-3 because this study focused on the effects of these variables on cortisol variation between-individuals rather than between-households. The equation for the full model is outlined in the Supplementary Materials.

All models were analyzed in R (R Development Core Team, Version 4.2.2, 2022) with the function *lmer* provided by the package *lme4* (Bates et al., 2015). The *lmertest* package was further used to calculate *p*-values for fixed effects based on the Satterthwaite's method for approximating degrees of freedom (Kuznetsova et al., 2017), and normal bootstrap confidence intervals were estimated for fixed effects using the *lmeresampler* package with 2000 residual resamples set at a 95% confidence level (Loy & Korobova, 2021). The *ranova* function of the *lmertest* package provided *p*-values for random effects via likelihood ratio tests (Kuznetsova et al., 2017). All models were assessed with Full Information Maximum Likelihood (FIML) estimation and partial to bound optimization by quadratic approximation (BOBYQA). Diagnostic tests indicated that statistical assumptions for linear models (e.g., normality and homogeneity of variance) were not violated. Results were considered statistically significant at $p < .05$ and were interpreted based on several statistical criteria, including the effect sizes and bootstrap confidence intervals of fixed effects (i.e., intervals that did not include zero indicated that coefficients were significantly different from zero) (Ferron et al., 2008; Lorah, 2018). When appropriate, the *p*-values of all post-hoc, pairwise comparisons were adjusted using the Holm-Bonferroni method.

3 | RESULTS

3.1 | Descriptive statistics

In Table 1, descriptive statistics are provided for sociodemographic and anthropometric predictors (*age*, *BMI* and *BMI z-scores*) and diurnal cortisol measurements (*waking*, *evening*, *diurnal slope*, *CAR*, and AUC_g levels) by sex and age category. To enhance interpretation, the cortisol values in Table 1 reflect averages of raw, non-transformed estimates. One-Way ANOVA tests evaluated differences in BMI and BMI z-scores by age category and between males and females from the same age category. Results demonstrated that BMI levels increased with age and were significantly different

TABLE 1 Sample sizes, means, and standard errors (SE) for sociodemographic characteristics, anthropometric variables, and diurnal cortisol measurements by biological sex and age category.[a]

	Children (2.7–6.9 years)				Juveniles (7.0–9.9 years)				Adolescents (10.0–15.9 years)				Young adults (16.0–34.9 years)				Older adults (+35 years)			
	Males		Females		Males		Females		Males		Females		Males		Females		Males		Females	
	n	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)	*n*	Mean (SE)
Age (Years)	32	5.08 (0.17)	27	5.39 (0.17)	34	8.42 (0.13)	26	8.38 (0.17)	39	12.52 (0.24)	37	12.65 (0.29)	16	25.33 (1.54)	38	25.86 (0.95)	26	43.56 (1.22)	23	46.31 (2.27)
BMI (kg/m^2)	31	16.22 (0.15)	25	15.98 (0.14)	33	16.57 (0.26)	25	16.46 (0.28)	29	18.37 (0.32)	33	20.12 (0.55)	10	23.24 (0.70)	30	24.58 (0.49)	21	25.84 (0.68)	20	25.14 (0.67)
BMI z-scores	31	−0.052 (0.111)	25	0.177 (0.104)	33	−0.045 (0.173)	25	0.136 (0.214)	29	0.197 (0.161)	33	0.469 (0.202)	10	−0.373 (0.345)	30	0.121 (0.218)	21	0.540 (0.381)	20	0.121 (0.296)
Cortisol at Waking (μg/dL)	32	0.233 (0.013)	27	0.245 (0.012)	34	0.213 (0.014)	26	0.208 (0.013)	39	0.226 (0.011)	37	0.222 (0.012)	16	0.262 (0.030)	38	0.262 (0.024)	26	0.288 (0.026)	23	0.254 (0.019)
Evening Cortisol (μg/dL)	32	0.033 (0.003)	27	0.034 (0.003)	33	0.065 (0.029)	26	0.035 (0.005)	39	0.045 (0.005)	37	0.042 (0.005)	14	0.058 (0.007)	38	0.042 (0.007)	25	0.047 (0.008)	23	0.034 (0.004)
Diurnal Slope (%/h)[b]	32	−15.64 (0.60)	27	−16.05 (1.08)	33	−14.45 (1.32)	26	−14.86 (1.80)	39	−14.21 (1.30)	37	−14.62 (1.77)	14	−14.34 (1.37)	38	−14.75 (1.85)	25	−15.54 (1.38)	23	−15.94 (1.86)
CAR[c] (μg/dL/h)	22	0.020 (0.005)	22	0.020 (0.003)	29	0.033 (0.014)	23	0.018 (0.002)	33	0.020 (0.002)	34	0.023 (0.003)	15	0.021 (0.004)	32	0.030 (0.005)	19	0.026 (0.005)	20	0.026 (0.008)
AUC_g[d] (μg/dL/h)	32	1.52 (0.09)	27	1.67 (0.10)	33	1.66 (0.23)	26	1.43 (0.10)	38	1.62 (0.09)	37	1.56 (0.10)	14	1.97 (0.23)	38	1.85 (0.19)	24	2.04 (0.20)	23	1.76 (0.12)

[a]Cortisol values are presented here in original, non-transformed units.
[b]Diurnal cortisol slope values were estimated from multilevel models. %/h = percent per hour.
[c]CAR, cortisol awakening response. μg/dL/h = μg per dL per hour.
[d]AUC_g area under the curve with respect to ground. μg/dL/h = μg per dL per hour.

among all age categories ($F = 178.52$, $\eta^2 = 0.74$, $p < .001$), except between children and juveniles ($p = .330$) and between young and older adults ($p = .284$). BMI z-scores were not significantly different by age. Additionally, BMI levels and BMI z-scores were not significantly different among males and females from the same age category, except for adolescent females ($M = 20.12$, SE = 0.55) displaying significantly higher BMI levels than age-matched males ($M = 18.37$, SE = 0.32), ($F = 7.07$, $\eta^2 = 0.11$, and $p = .010$).

3.2 | Multilevel growth models of diurnal cortisol rhythms

3.2.1 | Cortisol levels at waking and the diurnal cortisol slope

A three-level model was used to examine cortisol levels at waking and the diurnal cortisol slope, with variance apportioned to the within-individual (Level-1), between-individual (Level-2), and between-household components (Level-3). As previously noted, a partially unconditional model with *hours since waking* as a fixed predictor at Level-1 was used to calculate ICC values and regarded as the baseline model for analyses of cortisol levels at waking and the diurnal slope. Of the total variance in this model, within-individual differences in cortisol levels comprised 68.35%, between-individual differences 20.50%, and between-household differences 11.15%. *Hours since waking* was added as a random effect to the model at Level-2 and Level-3, and likelihood ratio tests indicated that the models with random effects at Level-2 [χ^2 (2) = 98.98, $p < .001$] and Level-3 [χ^2 (4) = 112.42, $p < .001$] were a better fit than the model with *hours since waking* as a fixed effect; thus, this predictor remained a random effect at these levels in subsequent analyses. In this model, *hours since waking* accounted for 84.19% of the variance at Level-1.

The results of the waking/diurnal slope model with *hours since waking* as a random effect at Level-2 and Level-3 (Model 1) are presented in Table 2. As shown, average cortisol levels at waking were significantly different than zero ($\gamma_{000} = -1.547$, SE = 0.033, 95% bCI [−1.570, −1.520], $p < .001$). Because all cortisol values were logarithmically transformed prior to analysis, the exponential function of that transformation was used to return this intercept to its original scale of measurement, resulting in a mean cortisol level of 0.21 μg/dL at waking. Furthermore, *hours since waking* was a significant predictor of cortisol levels ($\gamma_{100} = -0.163$, SE = 0.003, 95% bCI [−0.166, −0.159], $p < .001$). Logarithmic outcome variables maintain special properties that permit the effect sizes of coefficients predicting that outcome to be interpreted as percent change in the outcome per unit change in the independent variable after applying the following transformation: $B_{\%change} = [(\exp(B_{raw}) - 1)*100]$ (see Adam, 2006; Ong et al., 2011). Accordingly, the mean rate of change indicated that cortisol levels declined 15.04% per hour.

In a series of models (Models 2, 3, and 4), sociodemographic and anthropometric predictors, including *age*, *sex*, *BMI z-scores*, and their hypothesized interactions (*age*sex, age*BMI z-scores, sex*BMI z-scores, age*sex*BMI z-scores*), were entered as fixed effects at Level-2 to examine their influence on cortisol levels at waking and the rate of cortisol change across the day (see Table 2). In Model 2, *age* was independently added, demonstrating that *age* was a significant predictor of cortisol levels at waking ($\gamma_{010} = 0.0059$, SE = 0.0013, 95% bCI [0.0035, 0.0084], $p < .001$) but not the diurnal slope ($\gamma_{110} = -0.0002$, SE = 0.0002, 95% bCI [−0.0005, 0.0001], $p = .141$). These findings indicate that age is positively associated with cortisol levels at waking, which increased by 0.59% per year of age; however, age did not significantly affect the change in cortisol levels across the day.

To elaborate on the effects of age on cortisol levels at waking, post-hoc pairwise comparisons between all categorical age groups (children, juveniles, adolescents, young adults, older adults) were tested using the *glht* (generalized linear hypothesis test) command in the *multcomp* package in R (Hothorn et al., 2008); all *p*-values included adjustments for multiple comparisons using the Holm-Bonferroni method. These analyses revealed that both juveniles and adolescents had significantly lower cortisol levels at waking compared to young adults ($\gamma_{010} = 0.219$, SE = 0.062, $p = .004$ and $\gamma_{010} = 0.163$, SE = 0.060, $p = .045$) and older adults ($\gamma_{010} = 0.251$, SE = 0.063, $p = .001$ and $\gamma_{010} = 0.195$, SE = 0.059, $p = .008$), even after adjusting for multiple comparisons. Specifically, cortisol levels at waking were 24.48% lower for juveniles and 17.70% lower for adolescents compared to young adults, while cortisol levels at waking were 28.53% lower for juveniles and 21.53% lower for adolescents compared to older adults. All remaining differences between age categories in cortisol levels at waking were non-significant.

In Model 3, *sex* and the *age*sex* interaction were added as fixed effects (see Table 2). These results indicated that *sex* was a non-significant predictor of cortisol levels at waking ($\gamma_{020} = -0.0316$, SE = 0.0381, 95% bCI [−0.1040, 0.0400], $p = .409$) and the diurnal slope ($\gamma_{120} = -0.0031$, SE = 0.0047, 95% bCI [−0.0122, 0.0059], $p = .505$). While the coefficient for *sex* was not significant, the results indicate that males, on average, display relatively higher waking levels and flatter (i.e., more

TABLE 2 Three-level models with sociodemographic and anthropometric variables (main effects and interactions) predicting cortisol levels at waking and diurnal cortisol slopes among the Shuar.

	Model 1			Model 2			Model 3			Model 4			
Fixed effect	**Coefficient (SE) [95% bCI[a]]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-value**	***p*-Value**	**Interpretation**
Cortisol Intercept: Mean levels at waking	−1.547 (0.033) [−1.570, −1.520]	−47.48	< .001	−1.549 (0.033) [−1.570, −1.520]	−47.52	< .001	−1.531 (0.038) [−1.570, −1.490]	−40.29	< .001	−1.532 (0.041) [−1.580, −1.480]	−37.57	< .001	0.21 μg/dL at +0 hours[b]
Age (Years)				0.0059 (0.0013) [0.0035, 0.0084]	4.46	<.001	0.0073 (0.0019) [0.0036, 0.0109]	3.81	< .001	0.0082 (0.0021) [0.0041, 0.0123]	3.81	< .001	+0.59% per year of age
Sex (Female = 1)							−0.0316 (0.0381) [−0.1040, 0.0400]	−0.83	.409	−0.0397 (0.0431) [−0.1190, 0.0417]	−0.92	.358	N.S.
Age*Sex							−0.0026 (0.0026) [−0.0075, 0.0023]	−0.98	.327	−0.0041 (0.0029) [−0.0096, 0.0014]	−1.41	.160	N.S.
BMI z-scores										−0.0126 (0.0339) [−0.0759, 0.0505]	−0.37	.711	N.S.
Age*BMI z-scores										−0.0019 (0.0018) [−0.0759, 0.0505]	−1.05	.293	N.S.
Sex*BMI z-scores										0.0072 (0.0447) [−0.0768, 0.0907]	0.16	.871	N.S.
Age*Sex*BMI z-scores										−0.0012 (0.0023) [−0.0056, 0.0032]	−0.53	.600	N.S.
Hours Since Waking: Mean diurnal slope	−0.163 (0.003) [−0.166, −0.159]	−50.88	< .001	−0.163 (0.003) [−0.166, −0.159]	−51.04	< .001	−0.161 (0.004) [−0.167, −0.155]	−40.00	< .001	−0.160 (0.004) [−0.166, −0.154]	−37.58	< .001	−15.04% decline per hour[c]
Age (Years)				−0.0002 (0.0002) [−0.0005, 0.0001]	−1.48	.141	−0.0003 (0.0002) [−0.0007, 0.0002]	−1.16	.248	−0.0001 (0.0002) [−0.0006, 0.0003]	−0.40	.693	N.S.
Sex (Female = 1)							−0.0031 (0.0047) [−0.0122, 0.0059]	−0.67	.505	−0.0042 (0.0049) [−0.0135, 0.0049]	−0.85	.395	N.S.
Age*Sex							0.0000 (0.0003) [−0.0005, 0.0007]	0.27	.788	0.0001 (0.0003) [−0.0006, 0.0007]	0.16	.875	N.S.
BMI z-scores										0.0007 (0.0039) [−0.0065, 0.0081]	0.18	.856	N.S.
Age*BMI z-scores										−0.0004 (0.0002) [−0.0008, 0.0000]	−1.81	.071	N.S.
Sex*BMI z-scores										0.0032 (0.0051) [−0.0064, 0.0124]	0.63	.528	N.S.
Age*Sex*BMI z-scores										0.0006 (0.0003) [0.0001, 0.0011]	2.22	.027	+0.06% per unit change

(Continues)

TABLE 2 (Continued)

Random effect	Variance (SD)	χ^2	*p*-Value	Variance (SD)	χ^2	*p*-Value	Variance (SD)	χ^2	*p*-Value	Variance (SD)	χ^2	*p*-Value	Interpretation
Level-1: Within-individual variation	0.1829 (0.4277)	–	–	0.1829 (0.4277)	–	–	0.1828 (0.4276)	–	–	0.1790 (0.4231)	–	–	–
Level-2: Between-individual variation													
Intercept	0.0608 (0.2465)	53.57	< .001	0.0528 (0.2230)	55.89	< .001	0.0521 (0.2282)	55.76	< .001	0.0590 (0.2429)	33.18	< .001	–
Hours since waking	0.0007 (0.0259)			0.0007 (0.0257)			0.0007 (0.0257)			0.0006 (0.0236)			
Level-3: Between-household variation													
Intercept	0.0338 (0.1838)	13.44	.001	0.0352 (0.1875)	13.33	.001	0.0353 (0.1878)	13.33	.001	0.0334 (0.1827)	12.21	.002	–
Hours since waking	0.0002 (0.0155)			0.0002 (0.0154)			0.0002 (0.0154)			0.0002 (0.0157)			

[a]95% bCI = 95% bootstrap confidence intervals.
[b]Since all cortisol values were logarithmically transformed, the exponential function of that transformation returned this intercept to its original scale of measurement.
[c]Logarithmic outcome variables maintain special properties that permit the effect sizes of coefficients predicting that outcome to be interpreted as percent change in the outcome per unit change in the independent variable, after applying the following transformation: $B_{\%change} = [(\exp(B_{raw}) - 1)*100]$ (see Adam, 2006; Ong et al., 2011).

positive) slopes compared to females. The *age*sex* interaction was also a non-significant predictor of cortisol levels at waking ($\gamma_{040} = -0.0026$, SE = 0.0026, 95% bCI [−0.0075, 0.0023], $p = .327$) and the diurnal slope ($\gamma_{140} = 0.0000$, SE = 0.0003, 95% bCI [−0.0005, 0.0007], $p = .788$), suggesting that the previously described pattern of sex differences is maintained across the life course. The diurnal cortisol rhythms of males and females from each age category are displayed in Figures 1 and 2, respectively.

As shown in Table 2, *BMI z-scores* and the associated interactions (*age*BMI z-scores, sex*BMI z-scores, age*sex*BMI z-scores*) were entered into Model 4. The main effect of *BMI z-scores* was not a significant predictor of cortisol levels at waking ($\gamma_{030} = -0.0126$, SE = 0.0339, 95% bCI [−0.0759, 0.0505], $p = .711$) and the diurnal slope ($\gamma_{130} = 0.0007$, SE = 0.0039, 95% bCI [−0.0065, 0.0081], $p = .856$). Additionally, the *age*BMI z-scores* and *sex*BMI z-scores* interactions were non-significant predictors of cortisol levels at waking ($\gamma_{050} = -0.0019$, SE = 0.0018, 95% bCI [−0.0759, 0.0505], $p = .293$; $\gamma_{060} = 0.0072$, SE = 0.0447, 95% bCI [−0.0768, 0.0907], $p = .871$) and the diurnal slope ($\gamma_{150} = -0.0004$, SE = 0.0002, 95% bCI [−0.0008, 0.0000], $p = .071$; $\gamma_{160} = 0.0032$, SE = 0.0051, 95% bCI [−0.0064, 0.0124], $p = .528$). The three-way interaction between *age*, *sex*, and *BMI z-scores* was a non-significant predictor of cortisol levels at waking ($\gamma_{070} = -0.0012$, SE = 0.0023, 95% bCI [−0.0056, 0.0032], $p = .600$); however, it was a significant predictor of the diurnal slope ($\gamma_{170} = 0.0006$, SE = 0.0003, 95% bCI [0.0001, 0.0011], $p = .027$), which suggests that *age*, *sex*, and *BMI z-scores* interact to moderate the rate of cortisol decline across the day.

To interpret the effects of the *age*sex*BMI z-scores* interaction on the diurnal slope, simple intercepts and slopes were plotted using conditional values of *age* (±1 SD from mean age of 18 years), *sex* (0 = male, 1 = female), and *BMI z-scores* (±1 SD from mean BMI *z*-score of 0.15) in a regression of cortisol values on *hours since waking* (see Figure 3). Accordingly, participants below and above the mean age of 18.0 years were classified as "younger" and "older," respectively, and participants with BMI z-scores below and above the mean BMI z-score of 0.15 were defined as "lower BMI" and "higher BMI," respectively. In general, these results revealed that younger males (−15.24% per hour) and younger (−15.14% per hour) and older females (−15.88% per hour) with lower BMI levels had relatively steep cortisol declines across the day. In contrast, younger males (−14.11% per hour) and younger (−15.05% per hour) and older females (−14.53% per hour) with higher BMI levels displayed relatively flat cortisol slopes. The anomalies to these general patterns are older males (see Figure 3, Panel C), whereby those with

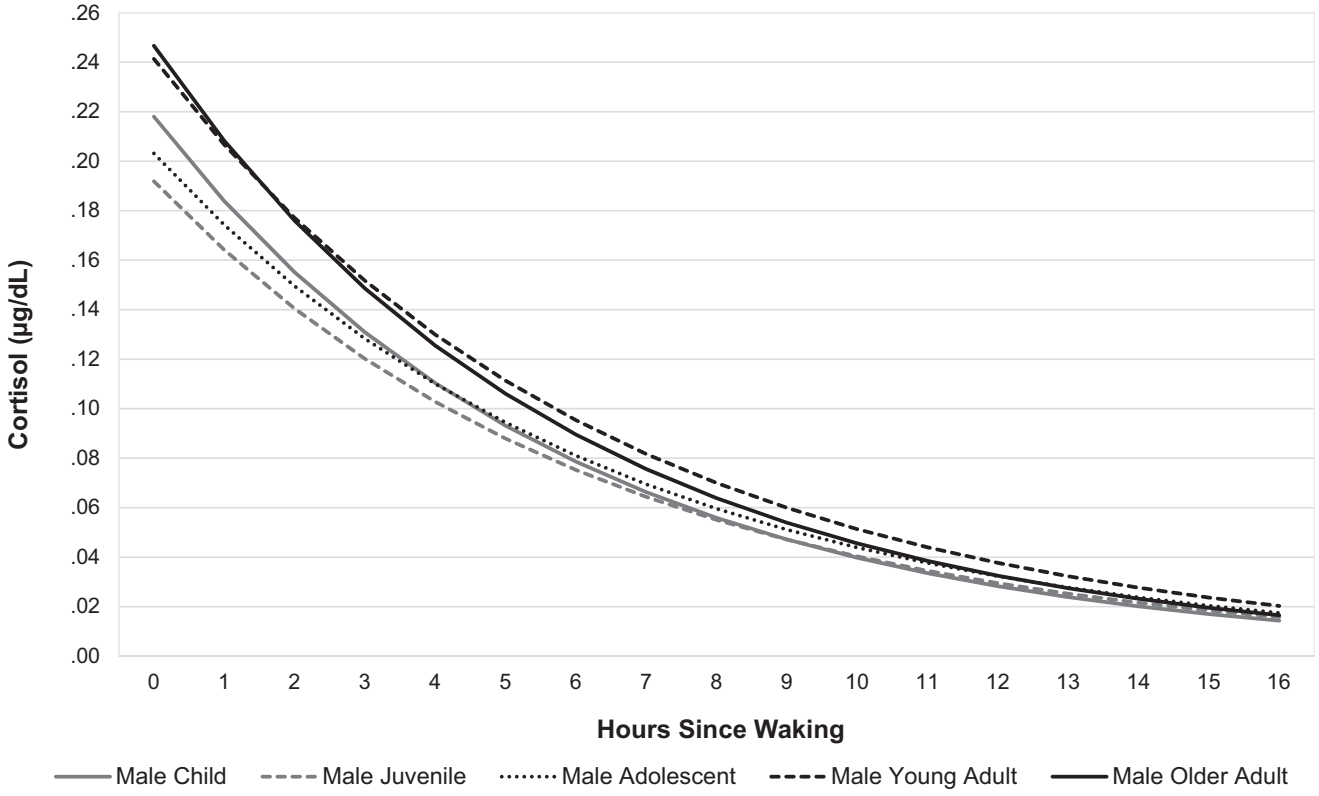

FIGURE 1 Diurnal cortisol rhythm for males by age category. Cortisol values are presented in original, non-transformed units (µg/dL). Solid gray line represents male children; dashed gray line represents male juveniles; dotted black line represents male adolescents; dashed black line represents male young adults; and solid black line represents male older adults.

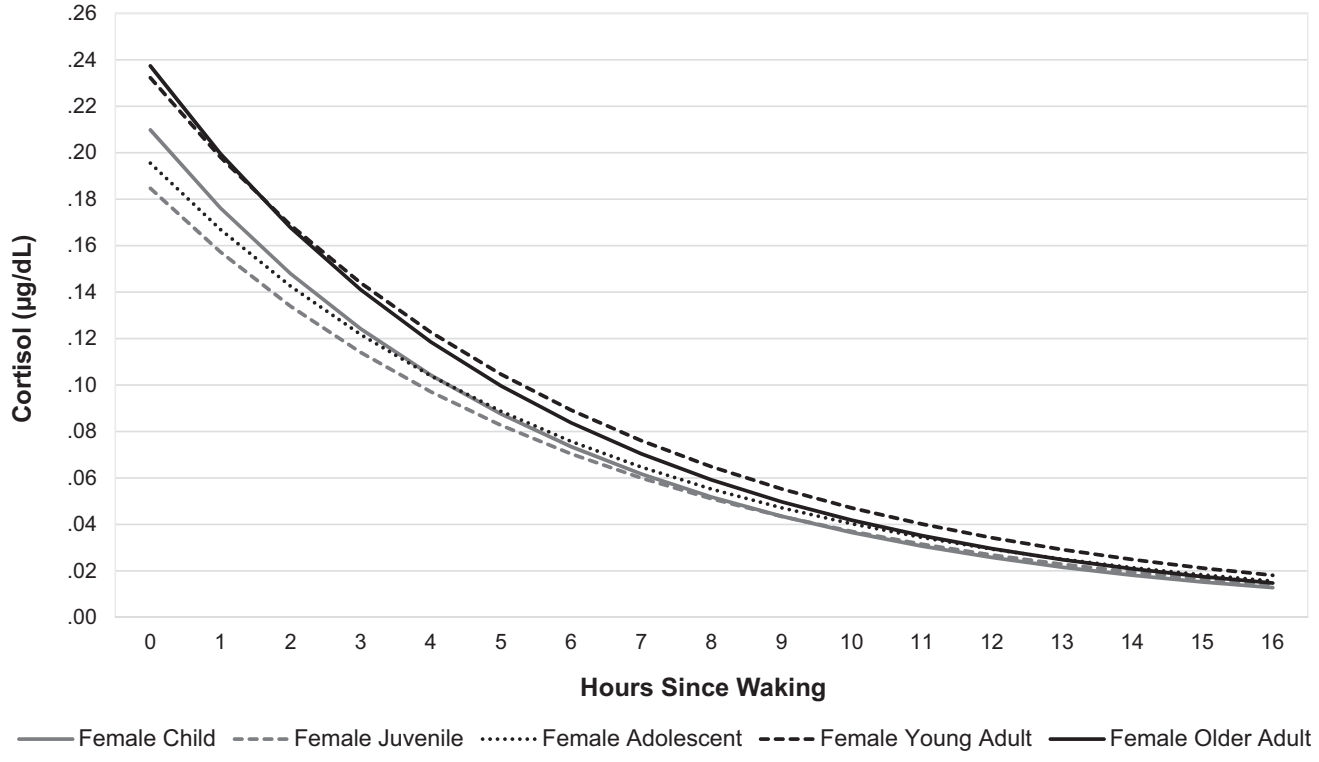

FIGURE 2 Diurnal cortisol rhythm for females by age category. Cortisol values are presented in original, non-transformed units (µg/dL). Solid gray line represents female children; dashed gray line represents female juveniles; dotted black line represents female adolescents; dashed black line represents female young adults; and solid black line represents female older adults.

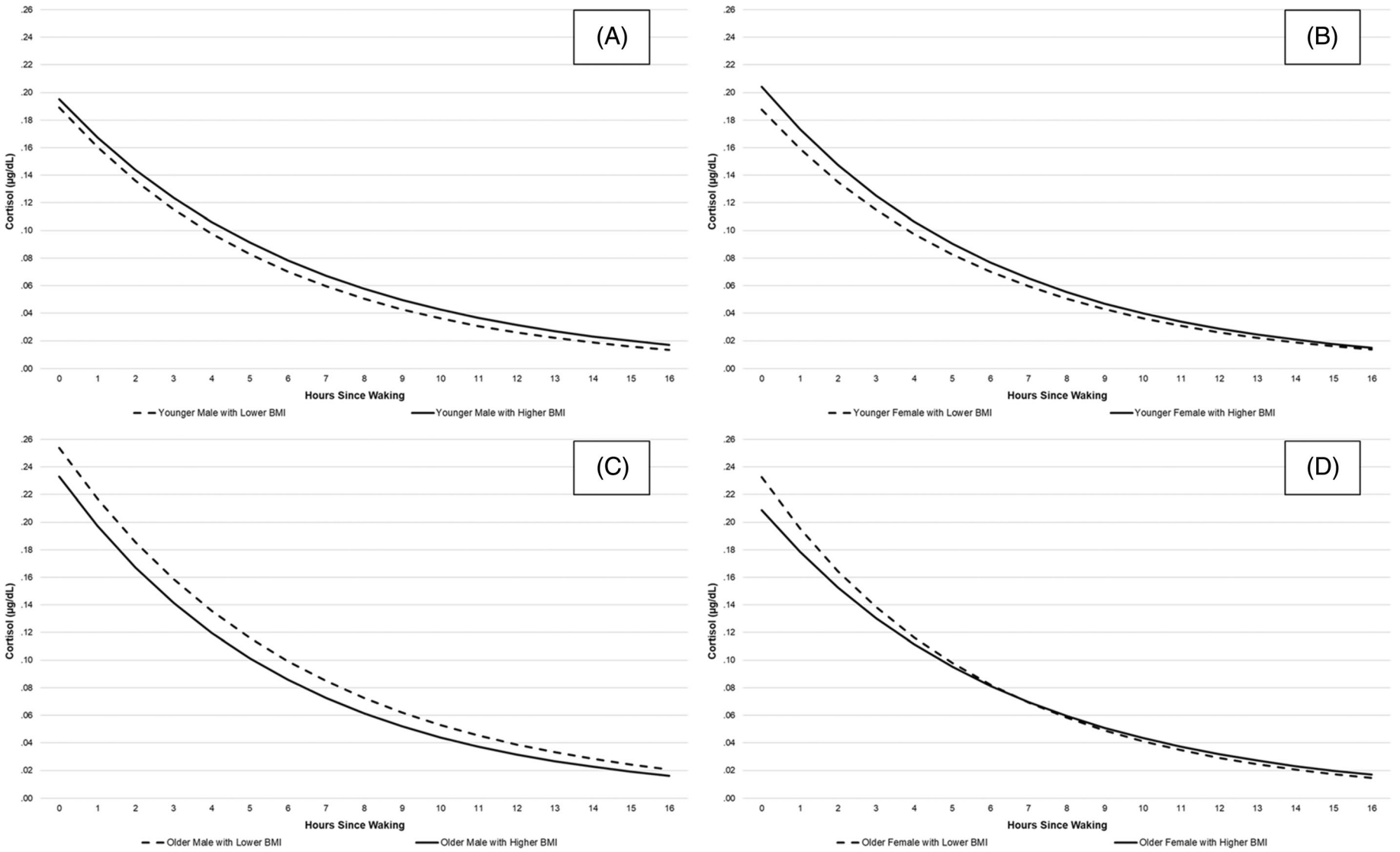

FIGURE 3 (A–D) Simple intercepts and slopes based on conditional values of *age* (±1 SD from mean age of 18 years), *sex* (0 = male, 1 = female), and *BMI z-scores* (±1 SD from mean BMI z-score of 0.15) in a regression of cortisol values on *hours since waking*. Cortisol values are presented in original, non-transformed units (µg/dL). Participants below and above the mean age of 18.0 years were classified as "younger" and "older," respectively, and participants with BMI z-scores below and above the mean BMI z-score of 0.15 were defined as "lower BMI" and "higher BMI", respectively. (A) Diurnal cortisol slopes for younger males with lower (−15.24% per hour; dashed black line) and higher (−14.11% per hour; solid black line) BMI; (B) Diurnal cortisol slopes for younger females with lower (−15.14% per hour; dashed black line) and higher (−15.05% per hour; solid black line) BMI; (C) Diurnal cortisol slopes for older males with lower (−14.48% per hour; dashed black line) and higher (−15.35% per hour; solid black line) BMI; and (D) Diurnal cortisol slopes for older females with lower (−15.88% per hour; dashed black line) and higher (−14.53% per hour; solid black line) BMI.

lower BMI levels exhibited a relatively flat cortisol decline across the day (−14.48% per hour), whereas those with higher BMI levels had a relatively steep diurnal cortisol slope (−15.35% per hour). Thus, the discrepancies among older males with lower and higher BMI levels appear to drive the significant *age*sex*BMI z-scores* interaction on the diurnal cortisol slope.

3.2.2 | Cortisol awakening response (CAR)

A three-level model was used to examine differences in the CAR, in which variance was allocated into within-individual (Level-1), between-individual (Level-2), and between-household components. A fully unconditional model was used to calculate ICC values and regarded as the baseline model for all CAR analyses. Accordingly, within-individual differences in CAR values comprised 73.98%, between-individual differences 11.35%, and between-household differences 14.67% of the total variance in this model. The results of the baseline model, presented as Model 1 in Table 3, demonstrated that average CAR levels were significantly different than zero ($\gamma_{000} = -4.202$, SE = 0.074, 95% bCI [−4.280, −4.120], $p < .001$), with average levels equaling 0.015 μg/dL.

Sociodemographic and anthropometric predictors were added as fixed effects at Level-2 in a series of models (Models 2, 3, and 4) to examine variability in CAR estimates (see Table 3). In Model 2, *age* ($\gamma_{010} = 0.0030$, SE = 0.0036, 95% bCI [−0.0038, 0.0097], $p = .407$) was not a significant predictor of CAR levels. Similarly, *sex* ($\gamma_{020} = -0.0107$, SE = 0.0998, 95% bCI [−0.1910, 0.1680], $p = .914$) and the interaction between *age* and *sex* ($\gamma_{040} = -0.0070$, SE = 0.0074, 95% bCI [−0.0204, 0.0065], $p = .345$) were non-significant predictors of the CAR (see Model 3 in Table 3). Finally, *BMI z-scores* ($\gamma_{030} = -0.0643$, SE = 0.0784, 95% bCI [−0.2120, 0.0834], $p = 0.413$) as well as the *age*BMI z-scores* ($\gamma_{050} = -0.0024$, SE = 0.0044, 95% bCI [−0.0106, 0.0059], $p = .596$), *sex*BMI z-scores* ($\gamma_{060} = 0.0427$, SE = 0.1048, 95% bCI = [−0.1530, 0.2410], $p = .684$), and *age*sex*BMI z-scores* ($\gamma_{070} = 0.0090$, SE = 0.0064, 95% bCI [−0.0028, 0.0208], $p = .159$) interactions were non-significant (see Model 4 in Table 3). In general, these results suggest that the magnitude and direction of the CAR were not significantly related to *age*, *sex*, and *BMI z-scores*.

3.2.3 | Area under the curve with respect to ground (AUC_g)

A three-level model was used to explore variability in AUC_g, with variance distributed into within-individual (Level-1), between-individual (Level-2), and between-household levels (Level-3). First, a fully unconditional model was utilized to calculate ICC values, indicating that within-individual differences in AUC_g comprised 53.79% of the total variance in this model, between-individual differences 27.77%, and between-household differences 18.44%. In Table 4, the results of the baseline model (Model 1) revealed that AUC_g levels were significantly different than zero ($\gamma_{000} = 0.391$, SE = 0.035, 95% bCI [0.367, 0.415], $p < .001$), whereby average levels across all participants totaled 1.48 μg/dL.

Sociodemographic and anthropometric predictors were then added at Level-2 to examine individual differences in AUC_g values (Models 2, 3, and 4 in Table 4). In Model 2, *age* ($\gamma_{010} = 0.0055$, SE = 0.0014, 95% bCI [0.0029, 0.0081], $p < .001$) was a significant predictor of AUC_g estimates, in which values increased by 0.55% per year of age. Similar to previous analyses, post-hoc pairwise comparisons between all categorical age groups (children, juveniles, adolescents, young adults, older adults) were used to further interpret the effects of age on AUC_g levels; all *p*-values included adjustments for multiple comparisons using the Holm-Bonferroni method. These results revealed that juveniles had significantly lower AUC_g values compared to young and older adults ($\gamma_{010} = 0.192$, SE = 0.064, $p = .021$ and $\gamma_{010} = 0.250$, SE = 0.065, $p = .001$). Moreover, adolescents had significantly lower AUC_g levels than older adults ($\gamma_{010} = 0.196$, SE = 0.062, $p = .014$). Specifically, AUC_g values were 21.17% and 28.40% lower for juveniles compared to young and older adults, respectively, while AUC_g values were 21.65% lower for adolescents compared to older adults. Comparisons between the remaining age categories were non-significant.

As shown in Model 3 (Table 4), *sex* was not a significant predictor of AUC_g ($\gamma_{020} = -0.0371$, SE = 0.0390, 95% bCI [−0.1110, 0.0375], $p = .342$). Although non-significant, these results demonstrate that females, on average, have lower AUC_g levels than males. The *age*sex* interaction was also not significant ($\gamma_{040} = -0.0040$, SE = 0.0028, 95% bCI [−0.0092, 0.0111], $p = .147$). Figure 4 presents AUC_g values for males and females from each age category. Lastly, *BMI z-scores* ($\gamma_{030} = 0.0098$, SE = 0.0351, 95% bCI [−0.0560, 0.0769], $p = .780$) as well as the *age*BMI z-scores* ($\gamma_{050} = -0.0025$, SE = 0.0018, 95% bCI [−0.0059, 0.0009], $p = .175$), *sex*BMI z-scores* ($\gamma_{060} = -0.0081$, SE = 0.0459, 95% bCI [−0.0944, 0.0804], $p = .859$), and *age*sex*BMI z-scores* ($\gamma_{070} = 0.0019$, SE = 0.0025, 95% bCI [−0.0026, 0.0064], $p = 0.437$) interactions were non-significant (see Model 4 in Table 4).

TABLE 3 Three-level models with sociodemographic and anthropometric variables (main effects and interactions) predicting Cortisol Awakening Response (CAR) levels among the Shuar.

	Model 1			Model 2			Model 3			Model 4			
Fixed effect	**Coefficient (SE) [95% bCI[a]]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Interpretation**
CAR intercept: Mean CAR levels	−4.202 (0.074) [−4.280, −4.120]	−56.67	< .001	−4.199 (0.074) [−4.280, −4.120]	−56.69	< .001	−4.189 (0.092) [−4.310, −4.060]	−45.60	< .001	−4.201 (0.098) [−4.350, −4.050]	−42.92	< .001	0.015 μg/dL increase from waking to 30 minutes post-waking[b]
Age (Years)				0.0030 (0.0036) [−0.0038, 0.0097]	0.83	.407	0.0071 (0.0056) [−0.0035, 0.0174]	1.26	.209	0.0094 (0.0061) [−0.0018, 0.0203]	1.55	.123	N.S.
Sex (Female = 1)							−0.0107 (0.0998) [−0.1910, 0.1680]	−0.11	.914	0.0320 (0.1084) [−0.1710, 0.2370]	0.30	.768	N.S.
Age*Sex							−0.0070 (0.0074) [−0.0204, 0.0065]	−0.95	.345	−0.0045 (0.0080) [−0.0187, 0.0104]	−0.56	.575	N.S.
BMI z-scores										−0.0643 (0.0784) [−0.2120, 0.0834]	−0.82	.413	N.S.
Age*BMI z-scores										−0.0024 (0.0044) [−0.0106, 0.0059]	−0.53	.596	N.S.
Sex*BMI z-scores										0.0427 (0.1048) [−0.1530, 0.2410]	0.41	.684	N.S.
Age*Sex*BMI z-scores										0.0090 (0.0064) [−0.0028, 0.0208]	1.41	.159	N.S.
Random effect	**Variance (SD)**	χ^2	***p*-Value**	**Variance (SD)**	χ^2	***p*-Value**	**Variance (SD)**	χ^2	***p*-Value**	**Variance (SD)**	χ^2	***p*-Value**	**Interpretation**
Level-1: Within-individual variation	0.7632 (0.8736)	–	–	0.7636 (0.8739)	–	–	0.7635 (0.8738)	–	–	0.7768 (0.8813)	–	–	–
Level-2: Between-individual variation													
Intercept	0.1170 (0.3421)	3.23	.072	0.1150 (0.3391)	3.09	.079	0.1127 (0.3357)	2.98	.084	0.0794 (0.2818)	1.39	.238	–
Level-3: Between-household variation													
Intercept	0.1513 (0.3889)	21.08	< .001	0.1507 (0.3883)	20.90	< .001	0.1510 (0.3886)	21.12	< .001	0.1444 (0.3800)	16.75	< .001	–

[a]95% bCI = 95% bootstrap confidence intervals.

[b]Since all cortisol values were logarithmically transformed, the exponential function of that transformation returned this intercept to its original scale of measurement.

TABLE 4 Three-level models with sociodemographic and anthropometric variables (main effects and interactions) predicting Area Under the Curve with Respect to Ground (AUC_g) levels among the Shuar.

	Model 1			Model 2			Model 3			Model 4			
Fixed effect	**Coefficient (SE) [95% bCI[a]]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Coefficient (SE) [95% bCI]**	***t*-Value**	***p*-Value**	**Interpretation**
AUC_g intercept: Mean AUCg levels	0.391 (0.035) [0.367, 0.415]	11.20	< .001	0.393 (0.035) [0.368, 0.417]	11.23	< .001	0.416 (0.041) [0.369, 0.461]	10.20	< .001	0.405 (0.044) [0.350, 0.459]	9.30	< .001	1.48 μg/dL of total cortisol output[b]
Age (Years)				0.0055 (0.0014) [0.0029, 0.0081]	4.03	< .001	0.0078 (0.0020) [0.0040, 0.0118]	3.84	< .001	0.0083 (0.0023) [0.0042, 0.0124]	3.70	< .001	+0.55% per year of age[c]
Sex (Female = 1)							−0.0371 (0.0390) [−0.1110, 0.0375]	−0.95	.342	−0.0338 (0.0440) [−0.1190, 0.0527]	−0.77	.443	N.S.
Age*Sex							−0.0040 (0.0028) [−0.0092, 0.0111]	−1.45	.147	−0.0045 (0.0030) [−0.0100, 0.0011]	−1.48	.141	N.S.
BMI z-scores										0.0098 (0.0351) [−0.0560, 0.0769]	0.28	.780	N.S.
Age*BMI z-scores										−0.0025 (0.0018) [−0.0059, 0.0009]	−1.36	.175	N.S.
Sex*BMI z-scores										−0.0081 (0.0459) [−0.0944, 0.0804]	−0.18	.859	N.S.
Age*Sex*BMI z-scores										0.0019 (0.0025) [−0.0026, 0.0064]	0.78	.437	N.S.
Random effect	**Variance (SD)**	χ^2	***p*-Value**	**Variance (SD)**	χ^2	***p*-Value**	**Variance (SD)**	χ^2	***p*-value**	**Variance (SD)**	χ^2	***p*-Value**	**Interpretation**
Level-1: Within-individual variation	0.1190 (0.3450)	–	–	0.1193 (0.3454)	–	–	0.1191 (0.3451)	–	–	0.1193 (0.3455)	–	–	–
Level-2: Between-individual variation													
Intercept	0.0614 (0.2479)	61.49	< .001	0.0540 (0.2323)	50.53	< .001	0.0530 (0.2302)	49.79	< .001	0.0590 (0.2429)	50.44	< .001	–
Level-3: Between-household variation													
Intercept	0.0408 (0.2020)	35.97	< .001	0.0423 (0.2058)	40.56	< .001	0.0425 (0.2062)	41.27	< .001	0.0413 (0.2032)	30.42	< .001	–

[a]95% bCI = 95% bootstrap confidence intervals.

[b]Since all cortisol values were logarithmically transformed, the exponential function of that transformation returned this intercept to its original scale of measurement.

[c]Logarithmic outcome variables maintain special properties that permit the effect sizes of coefficients predicting that outcome to be interpreted as percent change in the outcome per unit change in the independent variable, after applying the following transformation: $B_{\%change} = [(\exp(B_{raw}) - 1)*100]$ (see Adam, 2006; Ong et al., 2011).

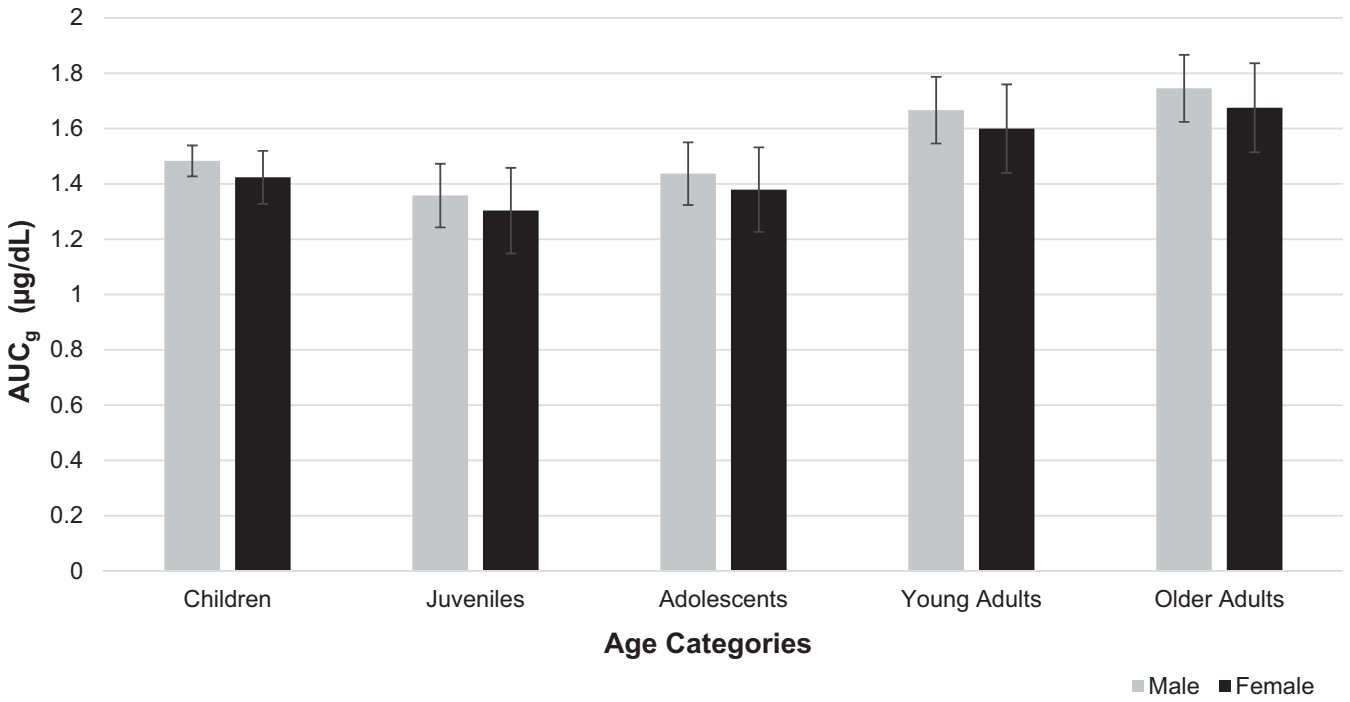

FIGURE 4 Estimates for Area Under the Curve with Respect to Ground (AUC_g) for males and females by age category. AUC_g values are presented in original, non-transformed units (µg/dL). Gray bars represent males, and black bars represent females. Error bars reflect standard errors estimated by biological sex and age category.

4 | DISCUSSION

To offer insights into global variation in HPA axis activity, we examined diurnal cortisol rhythms in relation to sociodemographic and anthropometric factors, including age, biological sex, and BMI, among the Indigenous Shuar, a forager-horticulturalist group from Amazonian Ecuador living in a high pathogen and limited resource environment and currently experiencing a range of cultural and economic changes associated with MI. Following the general circadian pattern documented in high-income populations, Shuar participants displayed identifiable diurnal cortisol rhythms, in which levels were highest in the morning, decreased precipitously across the day, and continued to decline to their lowest values in the evening. Across all participants, the average cortisol concentrations were 0.21 µg/dL at waking, 15.04% per hour rate of decline across the day, 0.015 µg/dL CAR, and 1.48 µg/dL AUC_g.

4.1 | Age is positively associated with cortisol levels at waking and total cortisol output among the Shuar, with juveniles and adolescents displaying significantly lower levels

The first objective of the present study was to examine associations between age and diurnal cortisol rhythms among the Indigenous Shuar. Consistent with our hypotheses, age was positively correlated with higher levels of waking cortisol and AUC_g, indicating that cortisol production at waking and total daily cortisol output increase with age among the Shuar. While previous research examining the effects of age on diurnal cortisol activity among high-income populations has presented inconsistent results (Gaffey & Martinez, 2019), our findings align with a growing body of literature that suggests waking and total daily cortisol levels increase throughout the human lifespan (Adam et al., 2006; Dmitrieva et al., 2013; Ice, 2005; Karlamangla et al., 2013; Miller et al., 2016; Nater et al., 2013). For example, using a large dataset of salivary cortisol samples from over 18 000 North American and European participants across a broad age range (1–99 years), Miller et al. (2016) demonstrated that diurnal cortisol concentrations gradually increase with age, particularly during the transition to puberty and adolescence, and remain relatively elevated throughout adulthood.

Age-related increases in diurnal cortisol production, including at waking and throughout the day, are hypothesized to result from biological maturation and senescence of the HPA axis (Gaffey et al., 2016). Glucocorticoid receptors, in particular, are a primary mechanism implicated in age-related changes in HPA axis function; with increasing age, they become less sensitive to stressors due to prolonged and repeated activation throughout the lifespan (de Kloet et al., 1998;

Kudielka et al., 2009). This process consequently reduces the efficiency of the negative feedback response of the HPA axis and its termination of cortisol production, leading to higher diurnal cortisol output over time (Gaffey et al., 2016; Sapolsky et al., 2000). Age-related increases in diurnal cortisol activity may also be shaped by the cumulative experiences of physical and psychosocial stress across the lifespan, such that chronic wear and tear of the HPA axis may further impact its feedback mechanisms and result in higher cortisol concentrations with older age (Gaffey et al., 2016; McEwen, 1998; Sapolsky, 2021). The similarities in age-related increases in waking cortisol and total daily cortisol output among the Shuar and high-income societies may reflect common biological changes associated with maturation and aging of the HPA axis in humans, although longitudinal research among diverse populations is further required to better understand the links between age and HPA axis functioning across the human life course.

In general, our results suggest that older age is associated with higher cortisol levels at waking and throughout the day among the Shuar; however, additional analyses highlight more nuanced relationships between age and diurnal cortisol indices in this population. In contrast to prior research among high-income populations suggesting that diurnal cortisol levels tend to increase with pubertal maturation and adolescent development (Gunnar et al., 2009; Kiess et al., 1995; Miller et al., 2016; Netherton et al., 2004), our findings indicate that cortisol levels at waking and total cortisol output across the day are dampened during the juvenile and adolescent stages among the Shuar. Although the metabolic costs of adrenarche, puberty, and adolescent growth are incompletely understood, the dramatic changes in morphology, endocrinology, and neurological development during these life history transitions are hypothesized to require substantial energetic resources (Caldwell et al., 2023; Cheng et al., 2016; Ellison et al., 2012). Accordingly, the relatively lower cortisol concentrations among Shuar juveniles and adolescents may reflect population-specific strategies for energy conservation in resource-limited settings to buffer the costly metabolic requirements of rapid skeletal growth, reproductive maturation, and somatic investments in muscle and fat deposition while also maintaining energy flow to homeostatic priorities and other physiological functions like immune activity. The relatively lower cortisol concentrations among Shuar juveniles and adolescents could also be impacted by other biological and behavioral changes that occur during these developmental stages, including steroid hormone production by the adrenal gland and hypothalamic–pituitary-gonadal (HPG) axis (e.g., dehydroepiandrosterone [DHEA], testosterone, estrogen) (Ellison et al., 2012; Goodyer et al., 2001; Matchock et al., 2007; Netherton et al., 2004; Romeo, 2010) in addition to shifts in sleep schedules (Dahl & Lewin, 2002; Wolfson & Carskadon, 1998; Zeiders et al., 2011). Future research that incorporates additional biological and behavioral measures of juvenile and adolescent development (e.g., assessments of growth, energy status, reproductive hormones, sleep and physical activity patterns) would help to address outstanding questions.

Finally, we did not support our hypothesis that age would be positively associated with flatter diurnal cortisol slopes and higher CAR levels; thus, age does not affect the early morning increase in cortisol production and its decline throughout the day among the Shuar. These results align with previous studies in HICs demonstrating null or inconclusive relationships between age and these cortisol parameters (Clow et al., 2004; Kudielka & Kirschbaum, 2003). However, these findings contrast with evidence from high-income populations (Adam et al., 2006; Dmitrieva et al., 2013; Ice et al., 2004; Nater et al., 2013), as well as the Indigenous Tsimane' (Nyberg, 2012) and Garisakang (Urlacher, Liebert, et al., 2018), which demonstrate relationships between increasing age and flatter diurnal cortisol slopes. The non-significant associations between age, the CAR, and the diurnal cortisol slope may be attributed to methodological challenges associated with capturing these cortisol indices (e.g., collecting the first sample upon waking) in addition to the small sample size of older Shuar participants (only 12 males and females over the age of 49), which limited our ability to examine the effects of aging and biological senescence on diurnal cortisol patterns. Additional studies among subsistence-based populations that examine diurnal cortisol activity among a larger sample of individuals from older age cohorts (e.g., over the age of 60) are needed to further elucidate these relationships.

4.2 | Diurnal cortisol patterns are similar among Shuar males and females across the life course

Our second objective was to evaluate sex differences in diurnal cortisol activity among the Shuar. Previous population-based studies of sex differences in diurnal cortisol rhythms demonstrate unique ontogenetic patterns among males and females, with younger males (<20 years) often exhibiting lower diurnal cortisol levels than age-matched females (Miller et al., 2016; Rolfsjord et al., 2017; Shirtcliff et al., 2012), whereas, older males (≥50 years) display elevated cortisol levels and flatter diurnal slopes compared to females of the same age

(Adam et al., 2006; Almeida et al., 2009; Dmitrieva et al., 2013; Gaffey & Martinez, 2019; Karlamangla et al., 2013; Miller et al., 2016). Research among subsistence-based societies, for example the Indigenous Tsimane' and Garisakang, further demonstrate distinct patterns of diurnal cortisol activity across the lifespan among males and females (Nyberg, 2012; Urlacher, Liebert, et al., 2018). In the present study, Shuar males generally had flatter diurnal cortisol slopes and higher levels of waking cortisol, the CAR, and AUC_g compared to females, which aligns with some studies in HICs, yet the differences among Shuar males and females were not statistically significant. Similarly, the interaction between sex and age was non-significant, suggesting that, in contrast to our hypothesis, Shuar males and females displayed relatively similar patterns of diurnal cortisol production across the life course.

In our interpretation of these results, we recognize that examining differences in diurnal cortisol activity based on binary, sex-based categories is limited and does not fully capture the complexity of sex, gender, and gender identity within sociocultural contexts and the ways in which these embodied experiences may influence stress-responsive physiological systems (DuBois & Shattuck-Heidorn, 2021; Juster et al., 2019). Notably, there are numerous developmental, physiological, and psychosocial factors that may shape variation in HPA axis functioning and diurnal cortisol production within and between males and females across the human lifespan. For instance, sex differences in diurnal cortisol patterns may result in part from dimorphism in neurological and biological characteristics, including brain structure and function, body composition and adiposity, metabolic activity, immune function, and gonadal steroid hormone production, which are suggested to explain the higher diurnal cortisol levels observed in older males compared to females (Darnall & Suarez, 2009; Gaffey & Martinez, 2019; Muehlenbein & Bribiescas, 2005; Worthman, 2015). Variation in diurnal cortisol rhythms among males and females may be further explained by differential experiences of sociocultural roles and identities, daily life stressors, and habitual behaviors that fluctuate throughout human development and may influence stress reactivity and diurnal cortisol production in dynamic ways (Almeida et al., 2009; DuBois & Shattuck-Heidorn, 2021; Juster et al., 2019). Ethnographic and empirical studies by the SHLHP have documented differences in biological outcomes and behavioral activities among Shuar males and females, including variation in anthropometric measurements, energy expenditure, immune function, pathogen exposure, metabolic health, dietary intake, family and community roles, and daily household and foraging practices (Christopher et al., 2019; Gildner et al., 2016; Liebert et al., 2013; Madimenos et al., 2011; Urlacher et al., 2019, 2021; Urlacher, Ellison, et al., 2018; Urlacher, Liebert, et al., 2016). Systematically accounting for some of these variables, in addition to their intersections with sociocultural experiences and expressions of sex and gender, may elucidate the non-significant sex differences presented here and may offer more nuanced understandings of the pathways shaping diurnal cortisol activity in this population.

4.3 | Age, sex, and BMI significantly interact to moderate the rate of diurnal cortisol decline among the Shuar

The final objective of the present study was to investigate the relationship between BMI and diurnal cortisol levels among the Shuar. Previous studies in HICs have examined associations between diurnal cortisol indices and anthropometric measurements of body composition to identify plausible biological pathways that may shape human variation in HPA axis functioning, often with the goal of better understanding the etiology of stress-related chronic diseases such as obesity and type 2 diabetes (Adam et al., 2017; Bose et al., 2009; Champaneri et al., 2013; Rodriguez et al., 2015; Rosmond, 2005; Rutters et al., 2012; Ruttle et al., 2013; Yu et al., 2020). However, research to date shows inconsistent and inconclusive results, with some studies indicating positive correlations between diurnal cortisol parameters and proxy indicators of body composition, including BMI (marker of generalized adiposity) and waist circumference (marker of abdominal adiposity), whereas others demonstrate negative or null conclusions (Rodriguez et al., 2015). These discrepant findings may be partially attributed to limitations in cortisol measurement methodology (e.g., sampling and assay protocols) and failure to account for interactions among participant characteristics (e.g., age and sex) that may modulate the relationships between diurnal cortisol activity and measures of body composition in important ways.

Considering the inconsistent findings of previous studies, we proposed hypotheses that directly tested the main effect of BMI on diurnal cortisol levels, while also analyzing its interactions with age and sex. Our analyses revealed that the main effect of BMI was a non-significant predictor of all diurnal cortisol parameters, aligning with previous research demonstrating null associations between body composition measurements and diurnal cortisol indices (Clow et al., 2004; Rodriguez et al., 2015). Upon accounting for interactions with age and sex, our findings further indicated that these variables did not significantly moderate the effects of BMI on

cortisol levels at waking, the CAR, and AUC_g. However, the three-way interaction between age, sex, and BMI was a significant predictor of the diurnal cortisol slope, suggesting that these key factors interact to regulate the rate of cortisol decline across the day. Our probing of the significant three-way interaction showed that younger males and younger and older females with lower BMI levels had relatively steep cortisol declines across the day. In contrast, younger males and younger and older females with higher BMI levels displayed relatively flat cortisol slopes. The anomalies to these general patterns were older males, in which older males with lower BMI levels exhibited relatively flat cortisol declines across the day, whereas older males with higher BMI levels had relatively steep diurnal cortisol slopes. Thus, the discrepancies among older males with lower and higher BMI levels may be the relationship driving the significant three-way interaction effect on the diurnal slope.

In our assessment of these findings, we acknowledge the limitations of BMI as a proxy measurement of body composition that crudely indicates generalized adiposity (American Medical Association, 2023; Rodriguez et al., 2015). Here, we further note that most Shuar children and adolescents (88%) and adults (58%) in the present study have BMI levels classified within the "normal" range based on CDC and WHO standards (Kuczmarski, 2002; World Health Organization, 2000); however, the remaining children and adolescents (12%) and adults (42%) were classified as overweight or obese. These rates may reflect a "double burden of malnutrition" among the Shuar, whereby undernutrition (e.g., stunting, wasting, and food insecurity) coexists with overnutrition and increased chronic disease risks (e.g., obesity, cardiovascular disease, and type 2 diabetes) (Wells et al., 2020; World Health Organization, 2016). Indeed, recent studies by the SHLHP provide evidence of a "double burden of malnutrition" among the Shuar, in which rates of childhood growth faltering and infectious/parasitic diseases remain relatively high (Blackwell et al., 2009; Cepon-Robins et al., 2014; Gildner et al., 2020; Urlacher, Blackwell, et al., 2016), while rapid sociodemographic, lifestyle, and dietary transitions associated with MI have contributed to modifications in body size and nutritional status among children and adults (Liebert et al., 2013; Urlacher, Liebert, et al., 2016). The prevalence of generalized adiposity among Shuar participants in the present study further reflects these epidemiological changes and may therefore influence the links between elevated BMI levels and flatter (i.e., more positive) diurnal cortisol slopes, which is a pattern increasingly documented among children and adults in HICs (Adam et al., 2017; Champaneri et al., 2013; Kumari et al., 2010; Ruttle et al., 2013; Yu et al., 2020). For example, Champaneri et al. (2013) revealed that higher BMI levels were correlated with blunted diurnal cortisol slopes among a large sample of older US adults, even after controlling for various confounding variables (e.g., age, gender, race/ethnicity, and socioeconomic status). Analogous relationships have been documented among children and adolescents, proposing that the biological pathways linking higher BMI levels and flatter diurnal cortisol slopes could be established during early life (Ruttle et al., 2013; Yu et al., 2020). Our findings suggest that similar underlying physiological relationships between elevated BMI levels and blunted diurnal cortisol production may exist among the Shuar.

The causal mechanisms linking indicators of generalized adiposity like BMI and diurnal cortisol activity are complex and bidirectional, thereby making it challenging to discern the biological connections between elevated BMI levels and flatter diurnal cortisol slopes (Adam et al., 2017; Champaneri et al., 2013; Kumari et al., 2010; Rodriguez et al., 2015; Ruttle et al., 2013; Yu et al., 2020). For one, cortisol is a major component of the hormonal architecture of human energetics that can promote weight gain via regulatory effects on energy metabolism, appetite, lipid deposition, and the maturation of adipocytes (Björntorp & Rosmond, 2000; Epel et al., 2001). Chronic activation of the HPA axis may further initiate endocrine disruptions, including insulin resistance and dyslipidemia, contributing to higher BMI levels and excessive fat accumulation (Björntorp et al., 1999). In the other direction, elevated generalized adiposity may constitute a chronic stressor that can alter the circadian rhythmicity of diurnal cortisol metabolism and may in turn lead to blunted cortisol production over time (Champaneri et al., 2013; Kolbe et al., 2015; Rask et al., 2001). These findings provide evidence that flatter diurnal cortisol slopes may serve as both a precipitating mechanism and marker of HPA axis dysregulation, elevated generalized adiposity, and increased obesity risks (Adam et al., 2017; Björntorp et al., 1999). Future analysis by the SHLHP will incorporate more robust measures of body composition and adiposity (e.g., waist circumference, waist-to-hip ratio, and body fat percentage), in addition to measures of dietary composition and physical activity, to elucidate these complex biological pathways and downstream health outcomes.

As noted earlier, the significant three-way interaction between age, sex, and BMI on the diurnal cortisol slope further indicated that older Shuar males diverge from the previously described general pattern, such that older males with lower BMI levels exhibited relatively flat cortisol declines across the day, whereas older males with higher BMI levels had relatively steep diurnal cortisol slopes. Although speculative, these findings suggest that

higher BMI levels among older Shuar males may reflect an adaptive energy buffer (e.g., greater amounts of muscle mass) that allows for more robust HPA axis functioning and diurnal cortisol production in resource-limited settings, as reflected in steeper diurnal cortisol slopes. These results may also illustrate distinct features of Shuar male life history, including biological and behavioral variables related to metabolism, muscle and fat deposition, immune function, daily physical activity, gonadal steroid hormone production, and senescence (Bribiescas, 2010), that may lead to differential diurnal cortisol patterns among older males and females with higher BMI levels. Research conducted by the SHLHP has assessed factors associated with Shuar male life history (Gildner, 2018), and forthcoming studies will systematically account for these variables to illuminate the unique patterns of diurnal cortisol activity among older Shuar males.

4.4 | Indigenous Shuar demonstrate relatively lower cortisol levels compared to high-income populations

Although not directly tested in this study, it is important to emphasize that the average cortisol concentrations among the Shuar are markedly lower relative to estimates from population-based studies in HICs (see Table 5). For example, Shuar in all age categories have lower average waking and evening cortisol concentrations compared to those among children and adults from the United States; these lower cortisol levels, in turn, result in generally flatter (i.e., more positive) diurnal cortisol slopes among the Shuar. Notably, cortisol levels at waking and the rate of cortisol decline across the day among the Shuar are comparable to documented values from other small-scale, forager-horticulturalist groups, including the Tsimane' of Bolivia and Garisakang of Papua New Guinea, who have the lowest cortisol values on record (Table 5; Nyberg, 2012; Urlacher, Liebert, et al., 2018). Thus, the similarities in diurnal cortisol patterns among these populations indicate that the Indigenous Shuar of Ecuador also demonstrate characteristically low and flat cortisol levels compared to those found in HICs. Here, we should note that the cortisol levels among the Shuar and other subsistence-based groups reflect generally lower and flatter diurnal cortisol rhythms that differ from the dampened cortisol slopes linked to chronic stress and hypocortisolism in HICs, which commonly emerge from low cortisol levels at waking and subsequently high evening levels (Adam et al., 2017; Adam & Kumari, 2009; Jarcho et al., 2013; Kumari et al., 2009; Sephton

TABLE 5 Population comparisons of waking cortisol, evening cortisol, and the diurnal cortisol slope.[a]

Study population	Age (years)	*n*	Waking cortisol (μg/dL)[b]	Evening cortisol (μg/dL)[b]	Cortisol slope (μg/dL/h)[c]	References
Shuar	2.7–7.0	59	0.239	0.034	−0.013	Liebert et al. (this study)
Shuar	7.0–9.9	60	0.211	0.050	−0.010	Liebert et al. (this study)
Shuar	10.0–15.9	76	0.224	0.044	−0.011	Liebert et al. (this study)
Shuar	16.0–34.9	54	0.262	0.050	−0.013	Liebert et al. (this study)
Shuar	35–86.3	49	0.271	0.041	−0.014	Liebert et al. (this study)
Tsimane'	1.6–15.9	222	0.191	0.070	−0.008	Nyberg (2012)
Tsimane'	16.0–82.0	81	0.232	0.080	−0.010	Nyberg (2012)
Garisakang	4.3–15.9	83	0.180	0.101	−0.005	Urlacher, Liebert, et al. (2018)
Garisakang	16.0–70.1	86	0.176	0.112	−0.004	Urlacher, Liebert, et al. (2018)
US (Atlanta)	5.0–6.0	28	0.360	0.075	−0.018	DeCaro and Worthman (2008)
US (Chicago)	13.0–19.0	52	0.565	0.131	−0.027	Adam (2006)
US MIDUS	33.0–84.0	1694	0.553	0.126	−0.027	Stawski et al. (2013)
US CARDIA	33.0–45.0	781	0.736	0.281	−0.028	Cohen et al. (2006)

[a]Population comparisons of cortisol measures should be interpreted cautiously due to differences in lab analyses (e.g., enzyme-linked immunosorbent assay [ELISA] vs. dissociation-enhanced lanthanide fluorescence immunoassay [DELFIA]), study design (e.g., number of collection days and samples per day), cortisol parameter calculations (e.g., estimations of CAR and diurnal slope), and measurement units. The list of studies provided here is not exhaustive and was selected based on available data of diurnal cortisol estimates from males and females across the life course in line with data presented by Nyberg (2012) and Urlacher, Liebert, et al. (2018).

[b]Waking and evening cortisol values are presented in original, non-transformed units (μg/dL). Values initially presented as nmol/L were converted to μg/dL.

[c]For comparisons across studies and populations, the diurnal cortisol slope values presented here (μg/dL/h) were estimated as a simple difference between waking and evening cortisol concentrations divided by 16 h.

et al., 2000; Sephton & Spiegel, 2003). Additionally, we should mention that comparisons of diurnal cortisol levels across population-based studies should be interpreted cautiously due to variability in lab analysis, study design, and cortisol parameter calculations (Miller et al., 2016). For example, the diurnal cortisol slope values in Table 5 were estimated as simple differences between documented waking and evening cortisol concentrations divided by 16 hours to allow for comparisons across studies and populations. These methodological disparities may preclude direct contrasts of diurnal cortisol levels; however, there is growing evidence of notable variation in diurnal cortisol rhythms between populations.

Interpreting differential patterns of HPA axis activity across diverse populations, particularly the relatively lower and flatter diurnal cortisol levels among subsistence-based societies, is facilitated by evolutionary and biocultural theoretical frameworks. From an evolutionary perspective, the HPA axis is a major effector arm of the evolved human stress response that plays an essential role in the mobilization of cognitive, physiological, and behavioral reactions to changing physical and social environments (Rao & Androulakis, 2019; Sapolsky, 2021). As a highly plastic neurobiological system, the HPA axis and its production of cortisol enables human adaptability to an array of ecological and psychosocial stressors and structures human life history patterns based on local conditions (Flinn et al., 2011; Nepomnaschy & Flinn, 2009). Cortisol, in particular, is a powerful human glucocorticoid that orchestrates context-specific resource tradeoffs between competing physiological domains to buffer top-level metabolic priorities (e.g., brain and vegetal activities) from fluctuations in energy intake and level demands (Ellison, 2017). Accordingly, we would hypothesize that variation in diurnal cortisol patterns between populations would emerge based on differential exposure to salient environmental, sociocultural, and developmental factors throughout the life course (DeCaro & Helfrecht, 2022).

The relatively lower and flatter diurnal cortisol levels among the Shuar and other subsistence-based populations may therefore reflect context-specific calibration of the HPA axis to resource-limited settings, particularly to physical stressors that are typically characteristic of these ecologies (Nyberg, 2012; Urlacher, Liebert, et al., 2018). Due to historical and contemporary practices, Indigenous peoples living in remote regions are often medically underserved, socioeconomically marginalized, and geographically isolated in areas with insufficient infrastructure and a lack of basic sanitation and clean water, resulting in heavy infectious/parasitic disease loads, nutritional inadequacy, and high morbidity and mortality risks (Madimenos et al., 2022; Valeggia & Snodgrass, 2015). As previously discussed, the Indigenous Shuar experience a "double burden of malnutrition", in which they continue to endure high rates of childhood growth faltering and infectious diseases (e.g., soil-transmitted helminths and gastrointestinal parasites) despite recent changes in nutritional status, body composition, diet, and lifestyle (Cepon-Robins et al., 2014; Gildner et al., 2016, 2020; Jokisch & McSweeney, 2011; Liebert et al., 2013; Pan et al., 2010; Urlacher, Liebert, et al., 2016). Notably, Shuar households in rural regions face various socioeconomic and geographic barriers to medical services and have limited access to clean water and basic sanitation devices, which exacerbate infectious disease risks in these settings (Gildner et al., 2020; Jokisch & McSweeney, 2011). For example, a recent study by Gildner et al. (2020) revealed that over 60% of the 620 Shuar participants were infected with at least one species of soil-transmitted helminth (*Ascaris lumbricoides* or *Trichuris trichiura*), with parasite infection patterns varying based on salient factors from the local environment including household floor types and water sources. Moreover, Shuar residing in communities demonstrate high levels of daily physical activity (Christopher et al., 2019; Madimenos et al., 2011; Urlacher et al., 2019) and consume a diet largely based on low-nutrient carbohydrates such as plantains, sweet manioc, and papaya with some supplementation with small game, fish, and market-based products (Liebert et al., 2013; Urlacher et al., 2021).

Collectively, these findings suggest that Indigenous Shuar in rural communities frequently encounter physical stressors associated with resource-limited settings that may synergistically contribute to chronic physiological stress and may in turn lead to dampened diurnal cortisol patterns. Likewise, lower concentrations of metabolic and reproductive hormones, such as leptin and testosterone, have been documented among subsistence-based populations in response to energy limitations (Bribiescas, 2010; Ellison et al., 2002; Sharrock et al., 2008). Thus, the lower and flatter cortisol levels among the Shuar may reflect a similar sensitivity of HPA axis activity and diurnal cortisol production to nutritional constraints (Nyberg, 2012; Urlacher, Liebert, et al., 2018). In particular, chronic physiological stress may influence how cortisol mobilizes finite metabolic energy (i.e., carbohydrates and fatty acids) and allocates it to life history functions, including brain activity, immune function, energy storage, growth, and reproduction (Ellison, 2017), which may in turn shape the relatively lower and flatter diurnal cortisol rhythms among the Shuar. Dampened diurnal cortisol patterns among the Shuar may be further affected by additional energetic costs including high levels of habitual physical activity associated with food and economic

production (Christopher et al., 2019; Madimenos et al., 2011; Urlacher et al., 2019).

Additionally, the relatively lower and flatter diurnal cortisol patterns among the Shuar may be shaped by the activities and energetic costs of other somatic functions, including the immune system. As previously noted, many Shuar experience conditions of recurrent pathogen exposure, which may contribute to chronically elevated levels of immune activity. While the interactions between the human immune system and HPA axis are bidirectional and inherently complex (Turnbull & Rivier, 1999), there is evidence that immune function associated with inflammation may influence diurnal cortisol production over time. Specifically, inflammatory cytokines and their downstream signaling pathways have been found to impair the number and sensitivity of glucocorticoid receptors, which in turn influences the bioavailability of cortisol and may lead to the downregulation of diurnal cortisol activity (Marques et al., 2009; Pace et al., 2007; Silverman & Sternberg, 2012). Pro-inflammatory cytokines may also impact the expression of clock genes that regulate circadian biological cycles, resulting in long-term effects on the diurnal cortisol rhythm (Cavadini et al., 2007; Cermakian et al., 2013; Man et al., 2016). Notably, inflammatory disease states and biomarkers of inflammation, such as interleukin-6 and tumor necrosis factor-α, have demonstrated robust associations with flattened diurnal cortisol slopes (Adam et al., 2017; DeSantis et al., 2012; Knight et al., 2021). Together, these results suggest that inflammation is one pathway that may contribute to dampened diurnal cortisol patterns among the Shuar and other subsistence-based populations in resource-limited settings (Nyberg, 2012; Urlacher, Liebert, & Konečná, 2018). Importantly, immune-related energetic costs have been documented among the Shuar with pronounced effects of acute inflammatory activity on childhood growth faltering and basal energy expenditure (Blackwell et al., 2009, 2010; Urlacher, Ellison, et al., 2018; Urlacher et al., 2019, 2021). Although Shuar adults do not display evidence of chronic low-grade inflammation (McDade et al., 2012), tradeoffs between acute immune function and diurnal cortisol patterns may exist. Research among societies outside of HICs is needed to unravel the role of diurnal cortisol production in the endocrine architecture of human energetics and to elucidate its interactions with competing physiological tasks. Future studies by the SHLHP will investigate the links between diurnal cortisol levels, energetic parameters, immune system activity, childhood growth, infectious disease exposure, dietary composition, and other essential life history domains to offer insights into these complex patterns.

While the lower and flatter diurnal cortisol levels among the Shuar and other Indigenous, subsistence-based populations likely reflect context-specific responses to chronic physiological stress, we should emphasize that these diurnal cortisol rhythms are also shaped by psychosocial stressors from the anthropogenic environment. In fact, the physical insults commonly associated with resource-limited settings, including heavy infectious disease burdens, malnutrition, barriers to basic medical services, and poor health, are themselves psychosocially stressful and are often products of broader macropolitical-economic forces linked to structural inequalities (DeCaro & Helfrecht, 2022; Leatherman & Goodman, 2020). For example, the Shuar and other Indigenous populations worldwide have historically and continue to face psychosocial stressors related to geographic, socioeconomic, and political marginalization that stems from issues related to colonization, land displacement, and poverty (Madimenos et al., 2022; Rubenstein, 2001; Snodgrass, 2013; Valeggia & Snodgrass, 2015). Moreover, most Indigenous populations throughout the world, including the Shuar, are increasingly exposed to and engaging with capitalism and globalized markets, resulting in various demographic, sociocultural, economic, and lifestyle changes that have profound effects on chronic psychosocial stress, health, and wellbeing (Dressler, 1999; Lea et al., 2020; Liebert et al., 2013; Mattison et al., 2022; McDade & Nyberg, 2010; Snodgrass et al., 2007). Using a biocultural lens, we can therefore interpret the dampened diurnal cortisol levels among the Shuar as an example of "local biologies," whereby the effects of historical disadvantage and contemporary globalization, in combination with physical stressors from the proximate environment, are biologically embodied (i.e., "get under the skin") over time and across space to produce variation in population-specific patterns of diurnal cortisol activity (DeCaro & Helfrecht, 2022; Leatherman & Goodman, 2020; Lock, 2017; Worthman & Kohrt, 2005). By providing a deeper understanding of the complex ways in which socioecological adversity may impact chronic stress and HPA axis functioning among the Shuar, our work ultimately strives to mitigate global health inequities and improve health outcomes among underserved populations in resource-limited settings.

As a final note of evolutionary and biocultural importance, the results presented here add to growing discourse in human biology regarding the validity of normative reference ranges to describe and understand patterns of human phenotypic variation within and between populations (Wiley, 2021). Rooted in biomedical and public health frameworks, reference ranges are typically estimated from statistical measures of central tendency and dispersion (e.g., averages and variance) and are used to verify if biological measurements fall

within a defined set of upper and lower limits, primarily with the goal of determining increased vulnerability to disease and poor health; in most cases, measurements within the established range are deemed "normal" or "healthy," whereas measurements outside the range are classified as "abnormal" or "unhealthy." While valuable tools for clinical practice and epidemiological research, normative reference ranges are predominantly based on biomedical data from homogeneous, European ancestry populations, which captures a narrow scope of human biological variation across different ecological, social, and developmental contexts. Thus, reference ranges are not universally applicable and may contribute to biased interpretations of "abnormal" or "unhealthy" patterns of human biology when used as benchmarks for populations outside of high-income settings (Madimenos et al., 2022; Wiley, 2021). Compared to existing references from HICs, the relatively lower and flatter diurnal cortisol levels among the Shuar and other subsistence-based groups may therefore be construed as deviations from the "normal" or "healthy" diurnal cortisol rhythm. This perspective may consequently lead to devaluing and pathologizing patterns of HPA axis activity as "abnormal" or "unhealthy" rather than recognizing these differences as population-specific responses to local conditions. To move beyond these limitations, future research that enhances the participation and representation of heterogeneous populations in LMICs, while considering the cultural and ethical implications of engaging with understudied, socially vulnerable groups, is imperative to expand the documented range of variation in diurnal cortisol activity across diverse human ecologies.

4.5 | Study limitations

This study encountered several important limitations. First, due to the logistical challenges of collecting diurnal cortisol data in a remote field setting, only three saliva samples were collected across the day for three days to maximize participant adherence and minimize participant burden. While pragmatic, this sampling protocol inhibited the ability to capture some measurements of diurnal cortisol sufficiently. For example, minimal measurements of the CAR and AUC_g were used because of the limited number of samples collected across the day, especially during the early morning after waking. Assessments of the CAR and AUC_g typically include 4–5 samples per day to estimate the peak awakening response and total daily cortisol output (Clow et al., 2004; Stalder et al., 2016). Accordingly, our interpretation of diurnal cortisol rhythms among the Shuar would have been enhanced with a greater number of cortisol samples across the day. Despite these limitations, this study includes an unprecedented set of cortisol data from a remote Indigenous population, thereby expanding current understanding of population variability in HPA axis activity. Further, recent analyses indicate that multiple days of cortisol sampling may provide more information on its diurnal pattern than the addition of more cortisol measurements within a single day, and three to six samples may be the optimal number to collect per day to estimate diurnal cortisol parameters robustly (Adam et al., 2017; Adam & Kumari, 2009). By using three saliva samples per day and multiple days of collection, the present study corresponds with current guidelines for salivary cortisol research in naturalistic settings.

Second, this study relied on BMI as a measure of body composition and did not include more informative measures of adiposity (e.g., waist circumference, waist-to-hip ratio, and body fat percentage). Moreover, we did not account for additional covariates that may moderate variability in diurnal cortisol levels, such as gonadal hormone levels, menstrual phase, dietary composition, parasitic infection status, and physical activity. Although not reported here, these data have been collected in tandem with the current study, and future analyses will examine their effects on inter-individual variability in diurnal cortisol rhythms.

Finally, the present study was limited by a cross-sectional design, which provides a relatively narrow window into the complexity of diurnal cortisol rhythms, particularly during important developmental periods such as the aging process and the adolescent transition. Longitudinal, field-based studies that track changes in diurnal cortisol rhythms within individuals over time would offer an opportunity to elucidate how cortisol activity varies throughout human development.

5 | CONCLUSIONS

The HPA axis and diurnal cortisol production are essential components of the evolved human stress response; however, most studies to date have investigated these neuroendocrine systems among populations from industrialized HICs. This limited sample from the vast array of human experience across sociocultural, ecological, and developmental settings provides an incomplete picture of HPA axis function and diurnal cortisol activity and restricts current understanding of the role of these physiological processes as they relate to chronic stress and associated disease risks. The present study investigated variability in diurnal cortisol patterns among the Shuar, a forager-horticulturalist population from Amazonian Ecuador living in a high pathogen and limited resource environment, with a specific focus on the effects of

sociodemographic and anthropometric factors in shaping individual differences in diurnal cortisol parameters, including cortisol levels at waking, the CAR, the diurnal slope, and AUC_g. This research expands the documented range of global variation in diurnal cortisol production and provides important insights into the plasticity and adaptive regulation of human stress physiology across the lifespan. Ultimately, studies that examine HPA axis activity across diverse socioecological contexts help to illuminate the biocultural pathways that link global inequities with negative health outcomes, including those among Indigenous peoples in resource-limited settings who are increasingly experiencing chronic psychosocial stress and disparities in health due to rapid sociocultural, political-economic, and lifestyle changes.

AUTHOR CONTRIBUTIONS

Melissa A. Liebert: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; writing – original draft preparation. **Samuel S. Urlacher**: Funding acquisition; investigation; writing – review and editing. **Felicia C. Madimenos**: Investigation; writing – review and editing. **Theresa E. Gildner**: Investigation; writing – review and editing. **Tara J. Cepon-Robins**: Funding acquisition; investigation; writing – review and editing. **Christopher J. Harrington:** Investigation; writing – review and editing. **Richard G. Bribiescas**: Investigation; supervision; writing – review and editing. **Lawrence S. Sugiyama**: Investigation; project administration; supervision; writing – review and editing. **J. Josh Snodgrass**: Funding acquisition; project administration; resources; supervision; writing – review and editing.

ACKNOWLEDGMENTS

We wish to express sincere appreciation and gratitude to the Shuar families who participated in this research. We also express a heartfelt thanks to our Shuar and Ecuadorian friends who have supported our research: Berta and Guimo Fernandez, the Jempekat family, Marcia Salinas Puente, and Oswaldo Mankash. Additional thanks are extended to Mark Van Ryzin for statistical guidance and for engaging in supportive conversations about this work.

FUNDING INFORMATION

This project was supported by Wenner-Gren Foundation (8749, 8476); NSF Doctoral Dissertation Improvement Grant (BCS-1341165); NSF Graduate Research Fellowship (2011109300); Ryoichi Sasakawa Young Leaders Fellowship Fund; Bray Fellowship, University of Oregon; Institute of Cognitive and Decision Sciences, University of Oregon; Department of Anthropology, University of Oregon.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

The Indigenous Shuar that are the focus of this research have a history of marginalization and exploitation by early researchers, the Ecuadorian State, and many others. Ethical and legal restrictions therefore prevent us from posting our data to a third-party server. However, the complete de-identified dataset and code supporting this article will be made available to qualified researchers, clinicians, and others upon request. Researchers must agree to privacy and data use expectations that conform to IRB requirements and are central to the welfare of our Indigenous participants and their agreements to participate in research. This process is streamlined, and requests for data can be easily made via an online form on the Shuar Health and Life History Project (SHLHP) website (https://www.shuarproject.org). The SHLHP has a long record of collaborative research and making data available to qualified scientists and health practitioners. All necessary ethical and legal approvals are with the University of Oregon IRB (Protocol Number: #09012010.010, researchcompliance@uoregon.edu). Additional details regarding necessary data sharing restrictions are also provided in the online data request form on the SHLHP website.

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SUPPORTING INFORMATION

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

How to cite this article: Liebert, M. A., Urlacher, S. S., Madimenos, F. C., Gildner, T. E., Cepon-Robins, T. J., Harrington, C. J., Bribiescas, R. G., Sugiyama, L. S., & Snodgrass, J. J. (2024). Variation in diurnal cortisol patterns among the Indigenous Shuar of Amazonian Ecuador. *American Journal of Human Biology*, e24056. https://doi.org/10.1002/ajhb.24056