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Health Parameters Across the Lifespan Among the Ache of Paraguay

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HEALTH PARAMETERS ACROSS THE LIFESPAN AMONG
THE ACHE OF PARAGUAY

by

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B.S. Nutritional Sciences, M.A. Anthropology

DISSERTATION
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ABSTRACT

This work provides an integrative approach for assessing population health among a group of indigenous South American forager-farmers, the northern Aché of eastern Paraguay. The Aché were full-time hunting and gathering nomads up until the time of first peaceful contact in the early 1970s when they experienced a devastating virgin-soil population epidemic that killed approximately 40% of the population, where all age and sex groups were affected more or less equally with the exception of a higher survival rate among reproductive-aged women. The Aché are now settled on several reservations and have fully recovered their population numbers.

The Aché have experienced a secular increase in body size in making the transition from forest to reservation living. The more abundant nutritional resources following contact as well as the extremely stressful conditions of the contact period provide a natural means for examining how environmental conditions interact with developmental sex differences. Whereas both males and females are exquisitely sensitive
to environmental conditions, each sex responds differently in ways that are organized around intrinsic physiological differences. For example, with respect to body size and particularly height, males tend to be both taller and exhibit greater variation in adult body size than females. This pattern arises from principles of energy allocation towards reproduction such that males invest greater energetic resources towards physical growth, such as building bone and muscle mass, while females invest greater energetic resources towards other facets necessary for obligate reproduction, such as building lipid stores to meet offspring energetic requirements. It follows that resource variation will interact with each sex in ways that reflect these differences; for example, stature is expected to fluctuate more closely with resource availability in males than females, whereas lipid deposition, ovulation and conception rates are expected to vary as a function of resource availability among females and, with the possible exception of pre-pubescent lipid deposition, are non-issues among males.

Previous studies of developmental sex differences in response to environmental conditions among human populations have proven inconclusive due to numerous mitigating factors; the results presented in this study represent a well-controlled natural experiment by which to probe the effects of changing dietary status and psychosocial stress on several developmental parameters. The parameters assessed in this study, and particularly the anthropomorphic variables, are slanted towards observing developmental effects in males versus females. However, this should not be taken as evidence that males are in general more sensitive to environmental conditions than females; but simply that the data collected here were more informative of male than female physiological responses.
A main finding presented here is that Aché males were more responsive in terms of physical growth to dietary conditions such that adult males made relatively greater gains in height and weight in comparison to adult females across the transition from forest to reservation living. Independent sample T-tests between the cohort of adults that completed growth before the contact period, and the cohort of fully grown adults that were born after the contact period, revealed more significant gains in height among males \( (p = 0.000) \) versus females \( (p = 0.029) \). The same pattern was observed in terms of weight where males made more significant gains \( (p = 0.001) \) versus females \( (p = 0.005) \). Concomitantly, linear regression equations using age as the independent variable indicated steeper slopes for male \((-0.21)\) versus female \((-0.10)\) height across adulthood (cross-sectional), and male \((-0.22)\) versus female \((-0.18)\) weight across adulthood, where height and weight were dependent variables. The relative differences between the sexes amounted to an increase of 5.1% in male height versus a 2.4% increase in female height; and a 15.5% increase in male weight versus a 13.7% increase in female weight.

A second finding of this study concerned the effect of the contact experience on peri-adolescent growth. Aché males who experienced the onset of the adolescent growth spurt, about 11 years of age, during one of the more severe years of the epidemic showed evidence of permanent developmental perturbations in terms of being significantly shorter, lighter, and somewhat smaller-headed than either adjacent male cohort. Regression analyses for the entire adult sample using a dummy coded variable for the contact cohort and controlling for age revealed a significant effect of contact on male height \( (p = 0.002) \) and weight \( (p = 0.016) \).
Similar effects were not found for females of this same cohort, who, along with the preceding female cohort, or those who initiated take-off growth before contact but experienced at least two years of adolescent growth during contact, exhibited increases in height and weight, although the sample sizes within these cohorts were too limited to draw firm conclusions. Similarly, the male cohort who preceded the contact cohort, or those who initiated take-off growth before the epidemic but experienced at least two years of adolescent growth during contact, were also taller and heavier although, again, the sample size was limited.

One possible interpretation for the overall pattern found among these peri-adolescent cohorts of both sexes is that the contact period may have exerted a mortality bias towards the elimination of smaller individuals, such that larger bodied individuals survived to be measured in the current study. This effect could account for the relatively larger body masses observed in these cohorts with the addition that there appeared to be a specific and permanent developmental effect on only those males who initiated take-off growth under conditions of severe physical and psychosocial stress. Note that under this scenario, mortality would have removed even smaller males from the middle male cohort during the contact period. The perturbational effect on the development of this cohort gains some support from other developmental parameters described below.

Evidence from visual evoked potentials (VEP) and bioelectric impedance analysis (BIA) were generally consistent with the pattern observed in the male contact cohort although the statistical results were not as strong as they were for the anthropomorphic measures. Visual evoked potentials (VEP) assess the integrity of the neurovisual pathway by recording the latency between a visual stimulus and occipital processing where the
major marker is referred to as the P100 latency. This latency can be used in conjunction with head length to calculate an estimate of nerve speed, referred to as Central Nerve Conduction Velocity (CNCV). Phase angle (PA) is derived from BIA parameters and is believed to reflect cellular health and membrane function. P100, CNCV and PA evinced characteristic ontogenetic and senescent patterns among the population as a whole, and all were consistent with a developmental perturbation occurring among the male adolescent cohort that experienced take-off growth during one of the severe contact years, although, again, these trends did not reach the statistical strength that the anthropomorphic variables showed.

Senescent trends in VEP and PA measures complicate identification of secular trends although senescence itself can be compared between the sexes and here males were found to senesce more significantly across the lifespan than females for both VEP and PA, although some of this pattern may be attributable to males having higher values in these measures during early adulthood than females.

Linear regression analyses using a dummy coded variable for the male contact cohort and controlling for age were not significant for VEP measures or PA, although all of the coefficients were in the expected direction. Conducting independent sample T-tests for these measures would require that age be controlled; however, the male contact cohort was significantly different than the younger adjacent cohort for CNCV ($p = 0.041$) and PA ($p = 0.048$), but not the older adjacent cohort despite similar means for each of the adjacent cohorts, which can, in part, be attributed to the low sample size of the older adjacent cohort. However, evaluating significance at $p$-values of 0.05 or less increases the risk of Type II error, or rejecting a truth, and does not appear to be ideal for detecting a
real effect when comparing groups with small sample sizes. In general, the proposed
effect of contact on male take-off growth was most profound for body size parameters,
less for BIA parameters, and less still for VEP parameters, indicating that visual
maturation—as measured by the VEP parameters used in this study—was largely
complete and/or immune to the psychosocial stress that obtained during the contact
period.

To probe for potential correlations among the anthropomorphic, visual evoked
potential, and bioelectrical impedance variables, correlation analyses were performed on
all variables and subjects by calculating individual residual values from non-parametric
fit lines (LOWESS) within each sex such that age was controlled. The most pronounced
and unanticipated findings concerned BIA variables and body size. Phase angle (PA)
residuals were inversely correlated with body size residuals among children and this
pattern was stronger for weight than height, stronger in females versus males, and
stronger at younger versus older childhood ages. Reactance, which reflects the
capacitance of cells or their ability to hold an electrical charge and is used in the
calculation of PA, was significantly and inversely correlated with weight residuals for the
entire Aché population. These findings were novel and unexpected and suggest that being
smaller for age was associated with better health and/or capacitance among the Aché.

The general agreement among the anthropomorphic and physiological measures
with respect to the effect of contact on the male cohort that initiated take-off growth is of
interest because there is no obvious mechanism linking these various measures. While
highly informative, BIA measurements and particularly PA are not well understood
biologically; potential explanations for this state of affairs are briefly explored in
conjunction with the properties of electricity in biological systems that might account for
the pattern observed in this research.

The overarching goal of this project was to glean information from traditional
populations experiencing unique ecological conditions such that it may better inform the
understanding of the relation between ecology and health. By extension, factors relevant
to the unique trajectory of human emergence may be illuminated. The evidence presented
here adds to the body of knowledge concerning human physiological adaptation in
response to variable ecological conditions, including the occurrence of an acute, stress
episode in the form of a virgin soil contact.
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I. Introduction

The northern Aché of eastern Paraguay were full-time nomadic foragers before making first peaceful contact in the early 1970s when they proceeded to suffer a devastating virgin-soil epidemic. Roughly 40% of the population perished over a period of 4 to 5 years, primarily from respiratory illness and related sequelae (Hill and Hurtado 1996). Following the contact episode, the Aché have lived on several reservation settlements within the Mbaracayu game preserve, where they have recovered and slightly increased their population numbers. At the time of data collection in 2005, the Aché were settled across several semi-permanent reservations and practiced a mix of hunting, horticulture, and wage labor for subsistence.

For purposes of analysis and following Hill and Hurtado (1996), the Aché experience was separated into three (3) periods: forest living conditions (pre-contact) that existed up until the start of the contact episode in 1971; which was followed by the contact period (1971-1978); which was followed by the period of reservation or settlement living conditions (post-contact) that have obtained since 1978. The Aché experience thus provides an ideal natural experiment by which to test for the effects of changing ecological conditions in making the transition from forest to settled living, and for assessing the effects of an acute physical and psychosocial stressor in the form of the contact episode, and how that may be reflected in the parameters measured here: anthropomorphics, visual evoked potentials (VEP), and bioelectric impedance analysis (BIA), described in turn below.

Predictions of sex differences in response to environmental conditions arise from intrinsic physiological differences that exist, on average, between the sexes. Females
were originally interpreted to be more buffered, or less susceptible, to environmental perturbations than males based on life-history principals concerning energetic trade-offs between continued growth versus reproduction. However, this interpretation was primarily founded on physical growth parameters where males typically invest more energetic resources into somatic growth than females to achieve larger body sizes. It is now known that both males and females are exquisitely sensitive to environmental conditions (e.g., Ellison, 2001) where, broadly, male physiology is more attuned to adjustments in physical growth, and female physiology is more attuned to adjustments in fertility. Although data on Aché fertility were not collected as part of the current study, the dearth of surviving offspring, and presumably live births, during the contact period is evidence that Aché females adjusted their fertility—likely through suppression of ovulation—in response to the stressful conditions of the contact episode.

Results presented in this dissertation indicate that, at least in terms of physical growth parameters, Aché males were more sensitive to environmental conditions than Aché females. The secular trend towards increased body size in transitioning from forest living (pre-contact) to reservation living (post-contact) was more pronounced in males relative to females and the stress of the contact episode appears to have permanently affected the physical development of only those Aché males who initiated their adolescent growth spurt—about 11 years of age—during one of the worst years of the epidemic or contact period.

In addition to compromised adult body size, neurological integrity, as measured through visual evoked potentials (VEP), and bioelectrical impedance analysis (BIA), a measure of cellular health, were generally consistent with the anthropomorphic
measurements in terms of the putative developmental perturbation among the male
contact cohort. However, the evidence for the effect in these measures was not as strong
as it was for anthropomorphics; and the BIA parameters showed stronger evidence than
VEP.

Both VEP and BIA have higher inherent variability and both have characteristic
senescent trends that require age to be controlled for statistically, all of which weakens
the power of significance testing and for the most, but not entire, part, significant findings
were not found among the VEP and BIA measurements where significance was evaluated
at $p < 0.05$. However, small sample sizes obtained for many of the comparisons and using
$p = 0.05$ to judge the significance of these types of comparisons increases the risk of
Type II error, or rejecting a truth, such that higher p-values would be consistent with an
effect being present. Overall, the effect on the male contact cohort was strongest for
anthropomorphic variables, less strong for BIA variables, and weakest for the VEP
measures. The latter finding may be evidence that visual ontogeny—as reflected by the
VEP parameters used in this study—was largely completed and/or immune to the effects
of contact within the male contact cohort.

Why these seemingly disparate measures of health corresponded solely to
developmental perturbations within the male contact cohort is open to speculation but
may be linked to growth pressures faced by males at the onset of the adolescent growth
period that are more acute than those faced by females. This sex difference in growth
patterns, just discussed, results in overall larger body size among males and logically
suggests that the take-off growth event for males is more susceptible to disruption, at
least in ways that can be detected by the measurements used in this study, and that
growth deficits were not able to be overcome through subsequent catch-up growth.

The number of female participants within the contact cohort was limited to the extent that firm conclusions could not be drawn with respect to the impact that the contact experience may have exerted. However, there was some indication that rather than decreased body size, females who initiated take-off growth during the contact period were, on average, larger than what would be expected in relation to other female cohorts. These women appeared to be relatively unaffected by the contact experience in terms of VEP and BIA. The increased body size of females whose growth periods overlapped with the contact period, in addition to the older and larger male cohort who had already reached the age of take-off growth before contact, could be interpreted as evidence that selection removed smaller individuals across the contact experience; a conclusion that suggests even smaller males were removed from the male contact cohort.

Correlations among the anthropomorphic, VEP, and BIA variables were tested using individual residual values calculated from non-parametric fit lines (LOWESS) within each sex in order to control for age. An inverse correlation between phase angle (PA) residuals and body size residuals was found among children where the correlations were stronger for weight versus height, stronger in females versus males, and stronger at younger versus older childhood age ranges. The strongest correlation concerned reactance (Xc), a measure that reflects the capacitance of cells, which was strongly and inversely correlated with weight residuals for the entire population such that being small for one’s age was significantly correlated with higher Xc values. This finding was novel
and suggested that an inverse relationship between growth output and Xc obtained among the Aché population in general.

The general agreement among the anthropomorphic and physiological measures with respect to the effect of contact on the male cohort who initiated take-off growth was of interest because there is no obvious mechanism linking these various measures. While highly informative, BIA measurements and particularly PA are not well understood biologically; potential explanations for this state of affairs are briefly explored in conjunction with the properties of electricity in biological systems in ways that might be able to account for the consistent pattern observed within the male contact cohort.

The overarching goal of this project was to glean information from traditional populations experiencing unique ecological conditions such that it may better inform the understanding of the relation between ecology and health. By extension, factors relevant to the unique trajectory of human emergence may be illuminated. The evidence presented here adds to the body of knowledge concerning human physiological adaptation in response to variable ecological conditions, including the impact of an acute, physical and psychosocial stressor in the form of a virgin soil contact that visited the Aché in the early 1970s.

**a. Organization**

The next, or 2nd chapter, provides background information on the study population, a brief review of virgin-soil contact episodes, and ethnographic details of the Aché contact experience. An overview of developmental sex differences in response to environmental conditions is provided along with a review of secular trends in stature. The adolescent
growth period, the relationship between stress and growth, and the phenomena of catch-up growth are also discussed. The physiological measures of visual evoked potentials (VEP) and phase angle (PA) are introduced and a summary section connects these measures to the overall hypothesis regarding indices of physical growth and development.

The 3rd chapter presents anthropomorphic data including height, weight, body-mass index (BMI), and head circumference. Several analytical approaches are pursued to delineate patterns among the various cohorts and between the sexes. The 4th chapter presents evidence from visual evoked potentials (VEP) along with a derivative measure that incorporates head length and is referred to as central nerve conduction velocity (CNCV). These measures are interpreted with attention to senescence patterns and to the proposed developmental perturbation among Aché males who reached the age of take-off growth during one of the more severe contact years.

The 5th chapter presents evidence from bioelectric impedance analysis (BIA) including a derivative measure, phase angle (PA), that, while extremely informative of health, is not well understood biologically. Evidence for the developmental perturbation among the male contact cohort is evaluated and appears to be in agreement with the previous measures.

The 6th chapter expands the analysis to explore potential correlations among all of the variables. Bivariate correlations controlling for age were performed for the entire sample and the sample was broken down along age and sex categories to test for patterns within these subsamples of the Aché population. Several patterns emerged which were further evaluated.
The last, or 7th chapter, summarizes the results and future directions are adumbrated along with a brief discussion of the somewhat enigmatic role of bioelectricity and its potential role for tying together the various measures that point towards a developmental perturbation among the male contact cohort. The concluding section reviews the current findings and places them within the larger context of developmental sex differences and life history theory.
II. Background

This chapter provides general background information on the study population and reviews developmental parameters that apply to examining Aché growth patterns in response to variable environmental conditions. This chapter provides complete background information for the following chapter on anthropomorphic findings; however, additional background information and methodological details are provided at the beginning of the 4th and 5th chapters that are germane to the visual evoked potentials (VEP) and bioelectrical impedance analysis (BIA) tests described therein.

a. Study Population

The Northern Aché of eastern Paraguay represent one of the few remaining hunter-gatherer communities left in the world. The Aché have been exhaustively studied with a wealth of ethnographic and life-history information provided by Hill and Hurtado in their seminal work, *Aché Life History* (1996). Interested readers are referred to this invaluable text for extensive details regarding virtually every facet of Aché life from ethnography to the fitting of life history models with empirical data. As such, only a cursory review of the Aché is provided here with emphases placed on the contact episode and nature of life history landmarks related to adolescent growth.

The Aché have been found to be genetically distinct from the general Amerindian population and exhibit several distinct morphological characteristics including white skin, light eye and hair color, beards, baldness, and strong Asian characteristics (Callegari-Jacques et. al., 2008). The Aché existed as nomadic hunter-gatherers and did not practice horticulture before first peaceful contact was made with outsiders in the early 1970s;
although several tribes of other Achés did make earlier contacts in the 1950s and 1960s (Hill and Hurtado, 1996; Clastres, 1998). There is no evidence of friendly relations between the Aché and any other ethnic population in Paraguay before this period.

The Aché currently (2005) live within five major mission/reservation settlements with a total population of about 1,000 people. Reservations are located at the periphery or slightly within the boundaries of the Mbaracayu game preserve, to which they have exclusive hunting use and access rights—in principle if not always in practice. Information for this project was collected in the Aché communities of Arroyo Bandera and Kuetuvy.

The Aché practice a mixed economy with some communities heavily dependent on cultigens, farm animals, and wage labor, while others are still partially dependent on hunting and gathering in the nearby Mbaracayu Natural Reserve. Game animals comprise up to 80% of the Aché diet in the forest (Kaplan et al., 2000) but the reservation diet is based on a staple of sweet manioc planted in slash-and-burn fields. The older generation continues to spend considerable stretches of time in the forest, typically about two weeks, while the younger generation shows less inclination towards forest living and developing foraging skills, and consequently spend less time in the forest.

The Aché continue to suffer considerable mortality and morbidity with a high manifestation of tuberculosis symptoms (Hurtado et al., 2003). However, overall health conditions appear to have improved with the advent of settlement living as evidenced by increased growth rates and larger adult body sizes recorded among the more recent generation of Aché. Factors contributing to this pattern are not entirely clear but more ready access to nutritional staples such as rice and cooking oil have improved nutritional
indices, at least in the caloric sense. Increased access to medical supplies and doctoral ministrations are likely contributing factors as well.

b. Virgin Soil Contact Episodes

Virgin soil epidemics have produced incalculable suffering throughout history (McNeill 1977) with disease transfer from European colonists to Amerindian populations in the New World especially acute (Crosby, 1976; Dobyns, 1993). “Virgin soil” refers to groups of people who have not been exposed to specific disease-causing organisms and therefore could not have developed an acquired immunity against them. It has been variously argued that a long history of interaction with domesticated animals combined with living in dense and squalid cities may have provided Europeans with disease-specific immunity to various zoonotic pathogens, which they carried with them upon their arrival in the New World and were spread to native populations (e.g., Diamond, 1997).

Regardless of the specific underlying causes, disease epidemics such as influenza, smallpox, measles and bubonic plague proceeded to decimate Amerindian populations upon contact with European travelers, drastically altering the course of history (Cook, 1998; Diamond, 1997). Virgin soil epidemics have continuously occurred throughout the modern era; most recently in regions of South America where previously uncontacted human populations experience initial contact episodes with outsiders through the actions of timber extraction, ore mining, missionary work, or brokered settlement—as was the case for the Aché.

Aché Contact Episode

Between 1930—when the first reliable census figures are available—and the early 1970s,
the Aché experienced a rapid population expansion that was probably made possible due to the war of the Triple Alliance that had eliminated approximately two-thirds of the entire Paraguayan population (Bertoni and Gorham, 1973).

Before contact was established with the Northern Aché, earlier contacts took place with members from two other local Aché tribes—the Ypety and Yvytyruzu. These Aché were living under the protection of a local rancher, Jesus Manuel Pereira. The Ypety and Yvytyruzu had been harassed and attacked by Paraguayan colonists for two decades preceding contact and promptly suffered 50% mortality from contact-related respiratory infections between the years 1963 and 1968. This early reservation settlement was then moved north into the San Joaquin hills by Pereira, who had been granted a government post and salary to administer the reservation and to attempt pacification of the hostile Northern Aché. Periera was motivated to recruit additional Aché members to live at the reservation as his salary was dependent on the number of Aché living under his auspices. It took two years of searching before contact was finally made with the Northern Aché in 1970. The assimilation process proceeded quickly as the tribes were very closely related, although many older males of high status refused to come into the reservation and lived the remainder of their lives in the forest.

New arrivals quickly fell ill upon initial exposure to the reservation Aché. By 1972-73 respiratory illnesses were exacting a heavy toll at both forested and reservation locales. Forest bands of Aché that were aware of the danger would seek to avoid contact with reservation Aché. In one case, virtually every member of a forest band died while searching for other forest Aché:

The Aché report that “king vultures blackened the skies above their camps”. Because all the adults were ill at the time, none of the dead were buried, and the
trail was littered with corpses as stragglers attempted to follow the band each
time it moved. Many weak and dying Aché were eaten alive by vultures in a
death that the Aché describe as most horrifying. Because of this possibility, sick
Aché often asked to be buried alive before the band moved on. The tearful
description by one close friend of how he buried his mother alive during this
epidemic, so the vultures would not pick her eyes out when she was left behind,
is probably the saddest and most moving story we have ever heard. (pp. 53-54)

Conspecific violence emerged as the second leading cause of mortality during the
contact period, particularly violence committed on orphaned children. The occasional
Aché custom of burying orphaned children with their deceased parents was intensified
during the contact period due to the high mortality rate among parents and other close
family members, who might otherwise agitate for an orphan’s survival. In normal times,
orphans could be spared live burial if others objected vociferously enough for their
survival. Many Aché adults have memories of being taken to the edge of a grave at least
once in their lives only to be rescued at the last moment by a close relative. During the
chaos of the contact episode, this practice reached dizzying heights.

The following anecdote serves to underscore the extreme psychosocial stress that
must have been experienced by the Aché throughout the contact epidemic. In this
interview, an Aché man is recalling several children he had killed during the contact
epidemic in 1973:

“The man recounted a horrifying story of coming upon a dead woman and
her healthy 13 year-old daughter after a band had dispersed in the forest
because of an epidemic. After digging the hole and placing the woman’s
body in it, he turned to the girl. She ran shrieking in the forest, crying and
begging that he not kill her. He caught the girl, dragged her to the grave,
and strangled her to death. His own thirteen-year-old daughter was present
at the time and witnessed the entire event according to his account…The
man told the story with tears welling up in his eyes and explained that it
was the Aché custom to kill children after their parents died. We were

1 Information in this section drawn from Aché Life History by Hill & Hurtado (1996) unless
otherwise noted.
distressed by the interview and couldn’t help berating the man for what seemed like inhumane behavior (we had heard many tales of child homicide and had even been present in the Aché camps when some small children were suffocated, but we had never heard such gruesome details of the sacrifice of an older child who was described as beautiful, healthy, and happy). The killer asked for our forgiveness and acknowledged that he never should have carried out the task and simply “wasn’t thinking.” He finally explained that “the old powerful men told us we had to kill all the orphans, and we did as they said without thinking.” (pp. 436-437)

The contact period continued in severe form through 1975 with heavy loss of life. Before contact the Northern Aché numbered approximately 550 members. By 1976 42% of these people were dead with another 11% missing, mostly orphaned children and adolescents who left of their own accord, or were sold by Aché adults to Paraguayans for labor or sexual exploitation. Others may have been kidnapped by the Paraguayans for the same purposes.

Contact-related illness, primarily in the form of respiratory illness and related sequelae, accounted for 31% of all deaths, 85% of adult deaths (14+ years), and 40% of child deaths (0-14). Mortality was especially high for the young (<10) and old (>40) of both sexes with only a handful of women over the age of 50 surviving. In many cases, other diseases such as measles were contracted and the associative state of weakness brought about by this affliction resulted in pneumonia being the ultimate cause of death. Sick and weakened parents were unable to feed or protect their vulnerable children. According to informants, many children died from starvation, abandonment or homicide rather than actually being sick themselves.

The final two years of the contact period were marked by high morbidity but very few deaths occurred because medical care and nutritional supplements were made available. Apart from the elimination of disease-related mortality, the latter years of the
contact period were also likely less stressful than the earlier years because of psychological and behavioral habituation to the initially novel and hectic conditions. Low mortality rates among reproductive-aged females across the entire contact period allowed the Aché population to recover their numbers rapidly upon the cessation of the contact period. Although the causes of this mortality pattern are not clear, a sex difference with respect to energy metabolism may have provided females with greater protection in the face of food shortages (Hoyenga and Hoyenga, 1982). Further discussion of sex differences in response to environmental conditions is provided below.

**c. Developmental Sex Differences**

The human sexes exhibit a wide range of developmental differences that manifest across the lifespan (see Geary, 1998; 1999 for overview). Secondary sexual characteristics diverge conspicuously at the onset of puberty and extend across the adolescent growth period before maintaining into adulthood. A major sexual dimorphism to emerge at puberty involves the allocation of energy towards lipid, muscle and bone tissue, with females showing relatively greater increases in lipid deposition and males showing relatively greater increases in muscle and bone tissue (Tanner, 1989).

From a theoretical viewpoint, it is expected that in species with differential investment towards offspring and pronounced sexual dimorphism in body size, the larger sex will be less investing in offspring since they will have invested more energetic resources into their own growth. These investments in growth are expected to payoff dividends later in life in terms of increased production, improved resource defense, or increasing reproductive fitness through increased access to mates. In contrast, the smaller sex is expected to be more directly investing towards offspring. This pattern can also be
understood from a life-history perspective, where organisms face a trade-off between allocating energy towards continued body growth or commencing reproductive function as they approach reproductive maturity. Energy cannot be allocated to both at the same time, thus generating the trade-off; see Stearns (1992) and Hill (1993) for overviews of animals and humans respectively.

In most animal species, particularly those with internal gestation, it is the female sex which per force invests more energetic resources towards offspring and ends up being comparatively smaller in adult body size than the male sex. However, these sex differences can be reversed in species where male investment in offspring exceeds that of the female, as discussed shortly with reference to the great skua bird species.

Among anthropoid primates, larger body size in males has often been interpreted as a sexually selected adaptation for intrasexual competition for mates; however, polygynous anthropoid primates exhibit high variation in weight dimorphism that is not associated with levels of competition (Plavcan and van Schaik, 1997) and is therefore inconsistent with the actions of sexual selection. Among extant human populations, degree of sexual dimorphism has also not been linked to mating system (Wolfe and Gray, 1982). Further, humans exhibit much lower degrees of size dimorphism than would be expected according to Rensch’s Rule, which is the tendency for sexual dimorphism to scale with increases in body size (Dixson, 2009), suggesting that other factors are involved which determine the relatively low degree of sexual dimorphism observed among human groups.

A major difference in humans compared to other anthropoid primates is the sexual division of labor and extremely high levels of intergenerational resource transfers, which
enable the funding of unproductive periods of childhood through greater resource provisioning by adults (Kaplan et al., 2000; Lancaster and Kaplan, 2010). Human male activities such as hunting, performing hard manual labor, and protecting women and children are far more important among humans versus other anthropoid primates and appear to contribute to decreased sexual dimorphism irrespective of mate competition (Lancaster and Kaplan, 2010; Kaplan et al., 2000).

On average, human females are physically smaller than human males and exhibit lower variation in both adult body size and lifetime reproductive success, or reproductive fitness. Human males exhibit a greater range of variability in terms of both adult body size and lifetime reproductive fitness. Consider that a woman faces very few obstacles in finding sires and producing one or several children. Males on the other hand may face extreme obstacles in finding mating partners although they have the physiological capacity for siring enormous numbers of offspring given suitable circumstances.

One expectation from this model is that female investment towards reproduction—conceiving and maintaining pregnancies, post-natal lactation, etc.—will take precedence over investments in physical growth, and this energetic bias will become particularly acute in the face of adverse environmental conditions. Because of their smaller bodies, downward shifts in female adult body size should not be as noticeable as downward shifts in male adult body size under conditions of environmental duress. Conversely, when environmental conditions improve it is males who are expected to allocate the extra energetic resources towards physical growth whereas females will allocate a greater proportion of the extra resources towards other energetically costly reproductive functions such as lipid deposition.
The above pattern with respect to physical development has been interpreted by some researchers for females being the more “buffered” or less vulnerable sex and there is some empirical support for this among humans although a number of factors need to be taken into account including the timing of the environmental insult during development, the duration and type of stressor, and the effects on various body tissues (Stinson, 1985).

It is useful here to distinguish between adult body size as measured by stature, or height, which reflects skeletal growth and maturation, and adult body size as it relates to weight and particularly fat deposition. The deposition of fat stores for reproductive-aged females is particularly salient in humans, who exist at the high end of the continuum among primates in terms of the quantity of fat that is stored for reproductive purposes (Dufuor and Sauther, 2002; Pond, 1998). In fact, the sexual dimorphism in fat deposition is more pronounced in humans than it is for either stature or weight.

Evidence suggests that the human mother is extraordinarily capable of shielding her embryo and developing fetus from nutritional deficiencies throughout pregnancy (Stini, 1979). This observation fits with the somewhat counterintuitive finding that women in underdeveloped countries have higher rates of obesity than women in developed countries despite the seemingly lower availability of nutritional resources (Brown and Konner, 1987; Brown, 1991). Along the same lines, men from a rural Colombian village who experienced chronic nutritional stress during adolescence showed a larger decrease in lean muscle mass compared to women (Stini, 1969), likely an adaptation to improve energetic efficiency across the lifespan (Stini, 1979).

The above statements indicate that differential sex-specific responses to nutritional conditions or stressors exist; however, classifying one sex as being more
“vulnerable” or less “buffered” than the other may simply be a matter of which criterion is being investigated. For example, assessing the effect of nutritional stress on adult height, which can be easily measured and to which males invest more energetic resources, will more often indicate that males are more “vulnerable” because male growth is more obviously hampered by these conditions than females, although this pattern is not always found (Stinson, 1985). However, the effect of nutritional stress on the lifetime reproductive success of females or males – assuming that it could be effectively measured – might indicate that females are more “vulnerable” in terms of lowered fertility than males, who, for example, have become more energetically efficient across the lifespan by limiting growth. In any event, it has been conclusively shown that women are acutely sensitive—particularly with respect to fertility—to environmental conditions (Ellison, 2001), and attempts to classify a more “vulnerable” sex are unnecessary. Rather, the goal is more profitably directed towards identifying sex differences in response to various environmental conditions vis-à-vis stressors as a means for gaining insight into the physiological nature of the human sexes.

Examining changes in body size as a means of testing for sex differences in response to environmental conditions was first suggested by Hiernaux (1968) and several authors have argued that differences in sexual dimorphism should be less in stressed environments and increase with improved environments as males become relatively larger than females (e.g., Stini, 1972; Tobias, 1972). The classic study on environmental sex differences was reported by Gruelich (1951) who found that male Guamanian children exposed to nutritional deficiencies and other perturbations during World War II had greater growth deficits in height, weight, weight for height, and skeletal maturation
than female Guamanian children. However, other studies have not found this trend and in some cases have actually found an opposite trend with females showing greater developmental deficits in various measures of growth and development (Stinson, 1985). It is important to keep in mind that a number of factors interact with the extent of sexual dimorphism. In addition to those factors already mentioned, parental investment patterns, mating systems, subsistence practices and nutritional status will interact with degree of sexual dimorphism (Gray and Wolfe, 1980; Wolfe and Gray, 1982).

Stinson (1985) has provided the most extensive review of studies concerning sex differences in environmental sensitivity with respect to growth and found much stronger empirical support for growth retardation in males during prenatal versus postnatal growth in comparison to females. Fetal exposure to environmental stress in the form of limited nutrition has been found to permanently alter the physiology and metabolism of the developing embryo such that strong correlations are found in the adult among conditions such as coronary heart disease, hypertension, diabetes, and stroke (Barker, 1994; 1997). A functional or adaptive model linking early environments with adult physiology has been proposed, which predicts and finds evidence that sex differences emerge in the form of birth weight in females and somatic investments related to reproductive function in males (Kuzawa, 2007).

Human growth is most sensitive to nutritional insult from the fetal period through about 2-3 years of age (Martorell et al, 1995) and—as just discussed—these developmental or growth effects can be transmitted to future offspring through the matriline. Kuzawa (2005) has reviewed evidence that changes in development can take several generations to “wash out” once environmental or nutritional conditions stabilize,
an effect he terms “intergenerational phenotypic inertia”. However, because the Aché nutritional-psychological stress episode was concentrated over a relatively short time span (~4 years) during which almost no births occurred and of those who may have been born many would have died (Hill and Hurtado, 1996), this pattern is not likely to be obvious among the Aché. Lipson (2001) further notes that chronic as opposed to acute energetic stress tends to slow growth, delay maturation and decrease ovariarian function in women. Any current evidence of these patterns among extant Aché women would be minimal given the acute nature of the stressor, the minimal births during the contact period, and the secular increase in body size among women that has occurred following the contact episode; such that women exposed to contact during growth periods were able to subsequently compensate for any growth deficits that may have occurred as a result of contact.

The lack of consistent findings among postnatal growth indicators as reported by Stinson (1985) is likely due to the wide variety of methods employed across studies as well as the complex social milieu in which humans exist, which complicates post-partum growth patterns. One possibility of reconciling the conflicting results is to consider that males and females may react differently to the timing, duration, and severity of stressors as well as to the extent and/or amount of alleviation from the stressor. For example, Tanner (1962) has argued that female growth is more canalized than male growth such that an improvement from poor nutritional conditions will result in females returning more quickly to their normal growth curve than males. However, with increasing or continual improvement in nutritional conditions, males will ultimately exhibit greater
overall growth. Therefore, it is important to consider the time at which growth is measured and the context of environmental conditions (Brauer, 1982).

A more consistent pattern has been reported with respect to the detrimental effects of psychosocial stress on male growth patterns being more pronounced in comparison to females (reviewed in Rudolf and Hochberg, 1990). A bias in the human sex-ratio at birth towards males has also been interpreted to suggest that males are the more vulnerable sex, although again, this can also simply be interpreted as a sex difference in life history strategy where males engage in riskier activities that bring about higher mortality. Although substantial variation exists between and within populations for reasons not entirely understood (see Seiff, 1990, for review), on average, about 105 males are born for every 100 female births across human societies (Cavalli-Sforza and Bodmer, 1971; Shettles, 1961). An evolutionary model of sex allocation suggests that parents will allocate resources to male and female offspring in order to maximize their own fitness (Charnov, 1982), which suggests that males are a more risky, but offer a potentially higher return, investment.

While numerous factors have been linked to fluctuations in the human sex ratio (Blaffer Hrdy, 1987; James, 1987; Teitelbaum, 1972), the overall pattern of more males born relative to females would seem to be driven by higher post-natal male mortality (Emlen, 1968). The Aché had a particularly high birth sex ratio of 116:100 during the forest period (Hill and Kaplan, 1988), which was consistent with higher male mortality across adulthood (Hill and Hurtado, 1996; Kreuger and Nesse, 2006). The sex ratio has decreased significantly during the reservation period along with mortality rates for both sexes (Hill and Hurtado, 1996).
The root causes of higher male mortality have been much speculated upon. While behavioral factors related to higher testosterone, such as risk-taking and homicide, are clear contributors to higher male mortality, other physiological evidence suggests males might be more vulnerable at an intrinsic level. Males appear to be more prone to immunological problems, possibly related to higher testosterone (Hazzard, 1990; Zuk and McKean, 1996) and have been found to have higher parasite loads in comparison to females, although this effect may be mediated through larger body size (Moore and Wilson, 2002). Males are also more prone to a range of chronic diseases and psychological stress (Kraemer, 2000). Sex differences in mortality are present from the moment of conception as evidenced by a higher male prenatal mortality level (McMillen, 1979) and extend across the lifespan where females routinely outlive males across all cultures, conditions and causes of death (Austad, 2006). Sex differences were recently reported for early life exposure to infectious disease, where there was no effect on female survivability but males had higher mortality early in life and lowered mortality at later ages (above age 45; Stormer, 2011).

Proximate mechanisms that might account for sex differences in environmental sensitivity are unknown (Stinson, 2000). However, evidence from animal species suggests that greater male size is more important for producing increased male vulnerability rather than other aspects of the male phenotype, such as decreased immunocompetence owing to higher levels of male sex hormones. Strong support for this conclusion was provided by Kalmbach et al. (2005) who found that in the great skuas (Stercorarius skua – a bird species found in Shetland), where size dimorphism is
reversed, it was the larger females that experienced higher mortality in the face of environmental insults.

In aggregate, however, males and females are expected to be equally vulnerable or sensitive to environmental conditions but respond differently and in accordance with factors that most directly affect their relative reproductive success. For example, constraints on physical growth are more likely to emerge among males in response to environmental duress whereas females will adjust fertility downwards. The parameters assessed in the current study, and particularly anthropomorphics, are biased towards detecting male responses to environmental conditions although this should not be taken as evidence that males are more sensitive or vulnerable than females in general, but rather that each sex responds to the effects of stress differently. The effects of stress on growth are examined in the penultimate section of this chapter.

**Secular Trends in Stature and Dimorphism in Body Size**

Long term, or secular, changes in a population’s body size have received significant attention from anthropometric-minded investigators. For example, Pleistocene humans appear to have been bigger brained and physically larger than modern Europeans (Ruff et al., 1997). More recently, the late 19th and 20th centuries have witnessed a marked trend towards increased body size among western populations likely associated with improved access to nutritional and medical resources (Cole, 2000; Garn, 1987). This trend appears to have stagnated among Americans in recent years (Kolmos and Lauderdale, 2007) while continuing apace in other populations, most notably the Dutch (Fredriks et al., 2000).
Until the early 1980s, it was assumed that populations who made the transition from hunting and gathering to agriculture were primarily rewarded with improvements in health. However, a wealth of evidence now indicates that the agricultural transition mainly resulted in negative health outcomes reflected by skeletal and dental pathologies (Cohen, 1989; Cohen and Armelagos, 1984; Larsen, 1995). Agriculture has been termed “The worst mistake in the history of the human race” (Diamond, 1987) with the primary culprit being the difference in dietary breadth. Foraging populations typically choose from a much broader range of foods in comparison to agriculturalists that rely heavily on one or a few starch crops, which are often of marginal nutritional quality (Larsen, 1995). In recent times, dietary protocols modeled after our Paleolithic ancestors and consisting of fruits, vegetables, nuts and lean meats, have been found effective for alleviating a number of age-related degenerative diseases (e.g., Lindeberg et al., 2003).

Thus, dietary deficiencies in combination with other behavioral changes, such as the mechanical demands placed upon skeletal structures in a sedentary existence, have generally produced decreases in stature and skeletal robustness among settled populations, although a class of ruling elite tended to avoid these outcomes (Cohen, 1989; Cohen and Armelagos, 1984). The advent of agriculture has also been linked to a decrease in sexual dimorphism in body size, such that the skeletal remains of Paleolithic humans exhibit similar levels of dimorphism as those of modern hunter-gatherers (Ruff, 1987).

The Aché represent a somewhat counter-intuitive example of a population that made the transition from foraging to agriculture and experienced an increase in stature rather than a decrease (Hill and Hurtado, 1996). The Aché also experienced a
concomitant increase in sexual dimorphism in body size (reported here). Amid the widely reported cases of secular changes in stature, changing nutritional conditions stand out as the most important environmental factor affecting the direction of change. Additionally, the status of the population at the beginning of the change period is also important; Tobias (1985) refers to this as the difference between the “haves” and the “have-nots”, or more descriptively, a four category approach of “have-most”, “have-ample”, “have-little”, and “have-least”. Accordingly, a population at the have-ample or have-most status will not experience an increase in body size due to improved nutritional conditions because they will already be maximizing their genetic potential for physical growth and adult body size. Conversely, populations existing at the lower end of resource availability will have greater potential to maximize their genetic potential for height with improved nutritional access.

The pattern found among the Aché may be explained by the fact that foraging populations have been increasingly pushed into marginal areas for subsistence due to the encroachment of civilization. The pre-contact Aché were arguably existing towards the have-little end of the spectrum and thus, the transition from foraging to agriculture resulted in an overall increase in the consistency and availability of food resources. This change is now reflected by increased physical growth and body size for both sexes, although, as presented below, this pattern is more pronounced for Aché males than it is for Aché females.

**d. Adolescent Growth Spurt**

The adolescent growth spurt refers to a rapid increase in the rate of growth that accompanies the onset of puberty in humans. Adolescence begins at puberty and lasts
between 5 and 8 years, ending when physical growth and sexual maturation are complete, typically by about 20 years of age for humans (Bogin, 1999). An adolescent, or juvenile, growth spurt is not a universal feature of anthropoid primate ontogeny although this pattern has been identified in many primate species, particularly those species that achieve larger adult body sizes. The growth spurt also tends to be more pronounced in male rather female anthropoids and particularly those species with greater sexual dimorphism in body size (Leigh, 1996).

In humans, female take-off growth occurs earlier and is completed over a shorter time period in comparison to human males, although both sexes reach similar peaks in terms of maximum growth-rate velocity, or the amount of mass gained per year. In comparison to other primates, the human adolescent growth spurt occurs very late—especially for males—and the duration of the female growth spurt period is exceptionally short (Leigh, 1996). In terms of degree, the pattern of a long and slow childhood growth period followed by a rapid and intense period of juvenile growth appears to be a unique feature of the human life course (Bogin, 1999). This growth schedule may represent an evolved strategy to minimize metabolic risks during development by limiting energetic requirements during childhood growth (Janson and van Schaik, 1993; Gurven and Walker, 2006).

The adolescent growth spurt among the Aché has been well studied (Walker et al. 2003, 2006a). In comparison to a large US sample, Aché males start take-off growth about a year later, have a lower peak-growth velocity, and finish growing about a year later. Aché females show a similar relationship in comparison to a large sample from the United States as shown below (adapted from Walker et al. 2006a).
Figure 1a. Growth velocity for Aché and US females. 1b Growth velocity for Aché and US males. Adapted with permission from Walker et al. (2006a).

As shown in Figures 1a and 1b, take-off growth begins around 10 years of age for Aché females and 11 years of age for Aché males. The adolescent growth spurt lasts for about 5 years for females, who achieve adult body height by about 16 years of age. The male adolescent growth spurt begins about 11 years of age and lasts slightly longer, about
6.5 years, such that males are fully grown by 20 years of age (Walker et al. 2006a).

Generally, in comparison to the U.S. sample, the Aché exhibit slower growth across childhood, begin take-off growth slightly later and have an extended but slower adolescent growth period. Nearly identical results are found using height instead of weight (Walker et al., 2006a).

Aché boys and girls are, on average, at about the fifth percentile of US body mass and height. These differences are likely due to differing environmental vis-à-vis nutritional conditions. For example, several Aché children were adopted into families in the US and grew to be significantly taller and heavier than their age mates who grew up on the Paraguayan reservations (Hill and Hurtado, 1996). Testosterone levels among young adult Aché males are also significantly lower than western samples (Ellison et al., 2002), where testosterone suppression is most likely a result of energy shortage (Bribiescas, 1996).

Some researchers have noted a decrease in rate of childhood growth that precedes the onset of the adolescent growth spurt for several years (Stutzle et al., 1980). This dip in growth velocity is visible in Figure 1 for the U.S. male sample but is much less obvious for Aché males or females in general. The cause of these growth dips are not known and not all children display them (Bogin, 1999).

The pubertal growth spurt emerges temporally for different body regions; for example, Greil (1997) found that the peak velocity growth rate for feet occurred at 9 years of age, and at 12 years of age for arm length and standing height in a large sample of German girls. Importantly, the female pelvic inlet follows its own slow growth pattern and does not reach adult size until the end of the adolescent growth spurt. This is
consonant with the asynchrony between physical growth and the onset of sexual maturation in the female, marked by menarche, whereby females achieve much of their growth before becoming fecund. From an adaptive perspective, conducting a pregnancy before sufficient pelvic girth was achieved to emit the offspring would be severely selected against. In the case of males, the initiation of sexual maturity or gonadarche, sometimes called spermarche, begins much earlier during the adolescent growth spurt (Bogin, 1999). From an adaptive perspective, this allows males to become potential sires because they are subject to much less initial reproductive costs in comparison to females, and they also avoid the attention of older males in competition for mates (Alexander et al., 1979).

The mechanistic effects of stress on the adolescent growth spurt are discussed in the next section but with respect to the timing of the growth spurt, it has been found in females who enter into rigorous physical training before puberty, have delayed menarche. For example, Malina et al. (1973) found this pattern among female track athletes while Frisch et al. (1980) recorded it among highly competitive ballerinas. Prolonged and severe malnutrition, illness or physiological stress has the ability to obliterate the growth spurt (Bogin, 1999) although when this occurs there is a tendency for the adolescent growth spurt to be much slower and more extended (Frisancho, 1977). For example, in Stini’s investigation of rural Colombian villagers living under conditions of chronic nutritional stress (1969), he found that the male adolescent growth spurt is non-existent and growth is a slow linear process that occurs up until about the age of 26 (1971). The Aché appear to be somewhere between this extreme and the growth rates reported in US
samples, as shown above, where the Aché have a longer, slower growth trajectory that terminates in the early 20s more or less for both sexes.

Currently, Aché females who have grown up on the reservation are reaching menarche about a year earlier than those who grew up during the forest period (Hill and Hurtado, 1996). This pattern is akin to the secular trend found in western societies towards earlier menarche (Roche, 1979) and could be a result of increased access to food combined with less energetic output (Garn, 1987). However, the ultimate explanation for these wide-spread trends across the developed world remains opaque; recent attention has been given to the proliferation of estrogen-mimicking/endocrine disrupting hormones found in many industrial products (de Muinck Keizer-Scharma and Mul, 2001; Parent et al., 2003).

e. Mechanics of Stress and Growth

The mechanism between stress and growth is probably regulated by glucocorticoids, a family of regulatory molecules—hormones and neuropeptides—that coordinate a wide-range of physiological and metabolic processes across most animal species (Becker and Breedlove, 1992). In humans, a generalized stress response stimulates increased release of glucocorticoids from the adrenal glands. The various glucocorticoids (cortisol, aldosterone, dehydroepiandrosterone sulfate or DHEAS, etc.) have wide-ranging temporal and physiological effects such as increased vigilance, suppression/stimulation of various components of the immune system, and increased energy production (Sapolsky et al., 2000). As a response to stressors over the short term, the effects of glucocorticoids are highly adaptive; however, when stressors become chronic, the negative effects of glucocorticoid action begin to accumulate including loss of appetite, suppression of
digestive processes, poorer energy metabolism, cessation of protein production and cell replication, inhibitory affects on bone tissue growth, and suppression of growth hormone among many others (Canalis, 2007; Miller and Chrousos, 2001). The effects just mentioned are those that primarily concern us here with respect to disruptions in the typical growth rate of body tissue in response to a prolonged stressor. The role of DHEAS has been investigated with respect to the onset of adrenarche, or the prepubertal onset of adrenal production (Campbell, 2006) and could potentially provide a mechanism to account for growth suppression of Aché males that initiated their adolescent growth spurt during the contact period, although this has not been demonstrated to date.

Before discussing disruptions of childhood and adolescent growth it is worth mentioning that from a physiological viewpoint, only semantic differences exist between physical and psychological stressors. Stress brought on by psychosocial challenges can be as detrimental as physical stressors, such as extreme heat or cold for example, and in some regards can be far worse if the stress cannot be physically acted upon to correct it. Consider that the stress response generates and liberates energetic molecules for the body’s cells to take up from the bloodstream—an adaptation that provides immediate and increased energy supply—for use in fleeing from a predator for example. However, for a ruminative species like humans, who may be stressed about losing a job in the middle of the night, there is no opportunity to utilize this energy and the adaptive, short-term effects of glucocorticoids give way to the prolonged, detrimental effects (Sapolsky, 1994). The saying that “something is eating you” is literally true in the sense that a prolonged stress response stimulates the body to perform gluconeogenesis, or the formation of carbohydrates out of muscle protein and adipose tissue, which are then converted to
glucose by the liver (Kaplan, 2000). Over the long-term, psychological stressors have a tremendous ability to wreak havoc on primate physiology (Sapolsky, 1994).

**Catch-up Growth**

Catch-up growth refers to a phase of rapid linear growth that allows a growth stunted child to accelerate towards and potentially resume their expected growth curve (Prader et al., 1963). Infants and young children exposed growth retardation, from such causes as malnutrition, endocrine and metabolic disorders, or celiac disease, may be able to achieve complete or near complete catch-up growth once the cause of the deficit is removed (Boersma and Wit, 1997; Prader et al., 1963). However, for older children who experience a growth perturbation near or during puberty, clinical evidence suggests that completing catch-up growth is much more difficult than for younger children (Prader, 1978). In both cases, growth outcomes are highly dependent on the nature, duration, and timing of the growth perturbation as well as the follow-up treatment or ensuing environmental conditions, as the case may be. The mechanisms underlying catch-up growth remain elusive although evidence points towards the regulation of hormonal growth factors and their effects on epiphyseal growth plates located in the proximal and distal parts of the long bones (Boersma and Wit, 1997).

**f. Visual Evoked Potentials (VEP) and Bioelectric Impedance Analysis (BIA)**

**Visual Evoked Potentials (VEP)**

The measures of VEP and BIA are briefly introduced here in order to bring them closer to the physical growth patterns just outlined. More extensive background and
methodological details are provided for each measure in the background sections of the respective data chapters.

Visual evoked potentials (VEP) are primarily used to assess the integrity of the visual system by recording the shape and duration of a major cortical response to a visual stimulus. A positive, peak-wave form occurs about 100 milliseconds after the stimulus and the duration of this time period, termed the P100, becomes progressively shorter throughout growth coincident with the maturation of the visual system. Across adulthood and at older ages, P100 values may increase again in conjunction with senescence, although the extent of this pattern, as well as developmental pattern, will depend on the parameters of the stimulus, usually a reversing black and white checkerboard pattern (Emmerson-Hanover et al., 1994). A relatively bright pattern of large check sizes will give the appearance of rapid development and little senescence across the lifespan, while relatively less bright and smaller check sizes will indicate longer and slower development with more rapid senescence occurring across adulthood into old age (Chiappa, 1990; Tobimatsu et al., 1993). VEP data cannot be formally compared across laboratories because of poorly understood variation that arises from recording equipment and measuring techniques (Odom et al., 2004).

A number of neuroanatomical features influence P100 values including head size, which appears to account for the slightly longer P100 latency values observed in males relative to females due to their larger head sizes and greater length of the visual pathway. A mild correlation between head size and P100 has been reported (Gregori et al., 2006) and a correlation between head size and height might be expected, although to this author’s knowledge, a direct correlation between other anthropomorphic measurements,
such as height, and P100 have not been reported. VEP is sensitive to a range of adverse conditions such as chronic obstructive pulmonary disease (Gupta et al., 2010), Parkinson’s disease (Nightingale et al., 1986) and dementia (Pollock et al., 1989).

Whether or how acute stressors during development might affect P100 latency into adulthood has not been investigated to this author’s knowledge, although delayed P100 latencies were found for victims 6 months after being poisoned by the neurotoxin sarin during the Tokyo subway attack in 1995 (Murata et al., 1997).

In terms of individual differences, several psychometric research groups have interpreted the division of head length by P100 time to represent optic nerve speed, or Central Nerve Conduction Velocity (CNCV) and have reported correlations between this measure and various psychometric tasks (Reed and Jensen 1992; Reed et al. 2004a). Although a range of studies have investigated various evoked potentials in other parts of the brain and the body and their relation to various cognitive tasks (Reed and Jensen, 1993), the P100 is mainly an indicator of early visual processing that does not invoke higher cortical functioning. No other VEP studies analyzing the P100 latency and individual differences have been investigated to this author’s knowledge, and no studies have examined any potential connections between VEP and the bioelectrical impedance analysis (BIA) parameters discussed next.

**Bioelectric Impedance Analysis (BIA)**

Bioelectric impedance analysis (BIA) measures the flow of electricity as it passes through the body. Two bioelectrical parameters are recorded: resistance (R), which represents the opposition of the body to the electric current and is inversely proportional
to the water and electrolyte content of tissue; and reactance (Xc), which reflects the capacitance properties of tissues and particularly the integrity, function, and composition of cell membranes (Baumgartner et al., 1988). BIA parameters can be used to estimate fat-free mass and body composition through the generation of regression equations, although in practice these equations are population specific and are not generally applicable across populations (Kyle et al., 2004; Lupoli et al., 2004). Phase angle (PA) is a derivative measure calculated from resistance and reactance and holds great interest for researchers due to its accuracy as an indicator and predictive marker of health, although the reasons underlying this relationship are not well understood biologically (Barbosa-Silva and Barros, 2005a).

PA, R, and Xc have been found to vary with age, sex, body mass index (BMI), percentage body fat, and disease state (Barbosa-Silva et al., 2005; Dittmar, 2003; Kotler et al., 1996). R and Xc are influenced by the length of the conductor and can be standardized by height although this has no effect on the calculation of PA since the height term drops out (PA = arc-tangent Xc/R x 180º/Π). R decreases during development and shows only a slight decline if any across adulthood, remaining lower in males compared to females (Dittmar, 2003). PA and Xc increase across developmental ages and show marked declines with increasing age; females exhibit higher Xc values than males but males have higher PA on average than females at all ages (Barbosa-Silva et al., 2005; Dittmar, 2003). PA is more predictive of BMI in children versus adults and increases with BMI up to values of 40 (Bosy-Westphal et al., 2006), a finding that reflects greater cell mass and also accounts for higher PA in males compared to females due to their larger average body size. Although not all studies have found the above
relationships (Baumgartner et al., 1988; Seberg and Seberg, 2002), studies using much larger sample sizes are consistent with these patterns (e.g., Barbosa-Silva et al., 2005; Dittmar, 2003).

Individual differences in BIA parameters, and particularly PA, have mainly focused on various health conditions where PA has been found to strongly correlate with disease state (e.g., Rutkove, 2002) and as a very accurate prognostic indicator of health state (e.g., Toso et al., 2000). Further information regarding this relation is provided in the background section of the BIA data, or 5th chapter. To this author’s knowledge, no studies have investigated VEP and BIA parameters in conjunction with one another.

g. Summary

In summary, the Aché experience provides a well controlled natural experiment by which to examine sex differences in response to environmental conditions for several reasons. The Aché represent a genetically homogenous population where individuals of both sexes were exposed at the same time and for the same duration to an extreme stressor—the contact period—which was not subject to cultural modification or interpretation. The Aché then experienced a secular trend towards increased body size in both sexes in making the transition from forest to reservation living. The Aché therefore offer a two-pronged approach for examining sex differences in response to environmental conditions in that they responded to a severe environmental stressor and then subsequently experienced a secular trend towards increased body size. While the results of this study cannot be extrapolated to other populations, they do provide a significant case study informing the nature of sex differences in response to environmental conditions and they
provide unique neuro- and physiological data collected among a well-studied traditional population.
III. Anthropomorphic Evidence

a. Methods

Most people residing in the communities of Arroyo Bandera and Kuetuivy were recruited to participate in the study for a total of 161 individuals (86 males, age range 5-75, mean = 28.71, std. = 18.62; 75 females, age range 3-73, mean = 26.76, std. = 17.48). Participants were provided with consent forms for the measurements described here and for all other tests and measurements described in this work. All procedures were approved by the Human Research Review Committee at the University of New Mexico, School of Medicine prior to the collection of data.

Participants had basic anthropometrics collected in addition to other tests and measurements described in subsequent chapters. A genealogical approach with interview-generated age ranks was used to age all individuals in the population born before fieldwork commenced in the late 1970s (see Hill and Hurtado 1996 for details), while ages for individuals born during the fieldwork period are exact to the day.

Height was measured to the nearest millimeter using a pre-measured and marked section of an interior wall. Subjects were instructed to stand with the heels of their feet flush against the base of the wall and to stand up tall. A flat ruler was then placed on top of the head perpendicular to standing height and the measurement was recorded from the wall to the nearest millimeter. Weight was recorded on a spring scale to the nearest tenth of a kilogram. BMI was calculated as the weight (kg) divided by height squared (cm$^2$). Head circumference was measured with a tape measure to the nearest millimeter. All analyses presented below were based on information collected from the study population in 2005.
Analytical Methods and Strategy

To assess developmental sex differences in response to environmental conditions, the secular trend towards increased body size was analyzed along with changes among specific cohorts in relation to the contact episode. The latter effort paid particular attention to the male cohort that experienced take-off growth during one of the five most severe contact years (1971—1975). Recall that the epidemic continued in severe form through the end of 1975 with the last two years being marked by high morbidity but virtually no mortality. A variety of approaches were used to explore and test these patterns, which required the formation of various cohorts described below.

Cohort Formation

Age cohorts were created based on three criteria: the age at which growth was completed, whether the contact episode occurred during childhood or adolescent growth, and the age at which take-off growth commenced with respect to the contact period. As such, five (5) cohorts were created: (1) those who were fully grown or nearly so before the onset of the contact period; (2) those who initiated take-off growth prior to contact but experienced at least 2 years of their adolescent growth spurt during the more severe contact years; (3) those who experienced the onset of take-off growth during one of the severe contact years; (4) those who experienced at least two years of childhood growth during the contact period; and (5) those who were born in a less severe contact year or later and were fully grown at the time of data collection (2005).

There were other possible ways to divide the sample into age cohorts. However, these definitions were inclusive of the entire adult sample for which information was collected and were based on a number of factors, including: the differential ability for
children to more ably achieve catch-up growth versus adolescents (Prader, 1978); whether the adolescent growth spurt was initiated during the contact period; how many years of the growth spurt occurred during the contact period; and whether childhood growth occurred during the contact period or not.

The subgroups were further divided by sex with the first group including all adults who reached full adult stature before the onset of the contact period in 1971; about 18 years of age and older for females and 20 years of age and older for males. The second group was described above while the third cohort includes those who reached the age of take-off growth during one of the severe contact years—for males, those born 1960-1964 and for females, those born 1961-1965. The one year difference adjusts for earlier female maturation and hence, onset of take-off growth. These individuals would have reached 11 (male) and 10 (female) years of age respectively during one of the severe contact years.

The fourth cohort includes those adults who, as children, experienced at least 2 years of childhood growth during the severe contact years. As stated, the fifth group includes all adults born after the last severe epidemic year (1975) and who were fully grown at the time of data collection. Height and weight plots indicated that Aché males reach full adult height around 22 years of age (Figure 2a below). Therefore, the more recent male cohort included males who were born between the years 1976 and 1983.

The more recent female cohort included those born between the years 1976 and 1987. In the case of Aché females, height appeared to be stable by 18-years of age although weight appeared to increase slightly throughout the early 20s. Owing to the more variable nature of weight versus height measurements, the 18-year age limit seemed acceptable for completed female growth. Another issue was that a continual secular
increase in body size, to the extent that it was occurring, would have the effect of making completed adult body size appear to be occurring earlier in a cross-sectional sample such as this one, and this applied to both males and females. However, the growth velocity curves in Figures 1a and 1b, which were derived from repeated Aché weight measurements\textsuperscript{2}, suggested that weight growth was completed by 18 and 22 years of age for females and males, respectively. These ages of completed growth were consistent with the cross-sectional plots of height, weight and head circumference presented below.

A handful of Aché who were born during one of the less severe contact years are included in the last, or fifth, cohort that represents adults who were fully grown at the time of data collection (2005) and were presumably unaffected by the contact episode. It may be possible that being born during this stage of the contact period could have permanent affects on growth but this occurrence seemed remote because, as stated previously, childhood growth occurs over a much longer period in comparison to the more rapid adolescent growth spurt. Childhood growth therefore tends to be more resilient to the effects of short-term environmental perturbations in terms of being able to perform catch-up growth and reaching expected adult body size (Prader, 1978).

It would also be difficult to untangle any permanent effects of a disruption in childhood growth as a result of contact, from the rising statures associated with the secular increase in body size that have obtained since settlement. If these potential growth effects were permanent, both phenomena would generate the same pattern in moving from smaller to larger body sizes across time. In contrast, an acute growth perturbation that occurred at the onset of the adolescent growth period, which is much more rapid and

\textsuperscript{2} See Walker et al., 2006a for further details.
occurs over a shorter duration in comparison to childhood growth, would be more likely to generate a permanent and observable deficit in adult body size (Prader, 1978).

**Tests**

The data were initially plotted cross-sectionally by age across the entire lifespan and fit with moving average lines, or LOWESS (locally weighted scatterplot smoothing). LOWESS generates a “smoother” line that adjusts to individual data points across the lifespan, rather than acting as a function as in other types of regression equations. The standard, or default, technique for constructing smoother lines is to use 50% of the data points nearest the x-value of the “x, y” point and then apply some technique for lessening the weight or influence of outliers, either on the x- or y-axis or both. In SPSS, LOWESS parameters are kernel and multiplier and the default settings are: kernel = normal, and bandwidth multiplier = 1.0. A ‘normal’ kernel specifies that points closer to each x-value have greater weight in calculating the smoother line and this has the effect of discounting the influence of outliers or data points that are further from the moving average. A multiplier set to 1.0 will use 50% of the data points on either side of the x-axis to calculate the moving average as described above. Standard smoother lines were fit to across-the-lifespan plots, which consisted of the entire sample including children.

Means and standard deviations for each measure were provided in table form for each adult cohort by sex with mean sex differences and the sex ratio calculated within each cohort. Two-way independent ANOVA (analysis of variance) was performed on the

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3 Some LOWESS parameters specify a fraction (f) of data values that are used in calculating the fit line and the number of smoothing steps (n) to discount the effect of outliers. The parameters (f) and (n) are functionally equivalent to the bandwidth multiplier and kernel smoothing options available in SPSS and produce the same results. For details on the former method see Cleveland (1979; 1985) and for the latter method see Velleman and Hoaglin (1981).
adult sample for each anthropomorphic measure (dependent variable) to test for the main effects of sex and cohort (fixed factors), in addition to sex by cohort interaction effects. Cohort means for each sex and cohort were plotted in line graphs with 95% confidence intervals for interpretation. Independent-sample T-tests were conducted to compare cohort means within each sex and measure to examine changes across the adult lifespan.

To further probe the apparent effect of contact on the adolescent growth, regression analyses using a dummy coded variable for the contact cohort was created and regressed on each anthropomorphic measure within each sex. However, the secular trend towards increased body size (i.e., height, weight, and head circumference) occurring across the transition from the forest period to the reservation period needed to be controlled. To this end, age is also entered as an independent variable into the regression equations. Because only fully grown adults are included in these analyses, age was entered as a linear variable along with the dichotomous dummy variable indicating whether take-off growth occurred during a contact year (contact = 0) or did not (contact = 1).

A final set of figures was provided showing just smoother lines plotted across just the adult sample. The smoother parameters were adjusted to allow for more detail to emerge in the fit line in with respect to changes in body size dimensions. The bandwidth multiplier was reduced to 0.5, which used 25% of the data values on either side of the x-axis to calculate the moving average, and the kernel was set to ‘uniform’, which gave equal weight to all measurements regardless of their distance from the moving average. These adjustments made the LOWESS smoother lines more sensitive to the inclusion of each individual. The adjustment of the parameters was arrived at through trial and error.
in order to provide adequate sensitivity to fluctuations in body size measurements without being unnecessarily chaotic.

Statistical analyses and graphic productions were performed with SPSS (Version 15.0, 2006) and Microsoft Excel (Version XP, 2002). The regression analyses applied parametric regression (linear and quadratic) to the scatterplot graphs, while all other graphs were fit with non-parametric LOWESS smoother lines, as described above, in order to display subtle changes across the lifespan that are not typically captured in parametric models. ANOVA analyses were performed in SPSS using the univariate option under the general linear model.

b. Results

Figures 2a through 2d depict across-lifespan measures of height, weight, body-mass index and head circumference, plotted cross-sectionally by age.
Figures 2a – 2d. Cross-sectional scatter plots of height, weight, body mass index (BMI) and head circumference for males and females by age. Data were fit with LOWESS smoother, kernel = normal, bandwidth multiplier = 1.0.

All measures show predictable growth trends across the first two decades of life. Male height exceeds female height around middle adolescence, or approximately 15 years of age. The decline in heights across the adult age range represented the secular trend and will be analyzed more explicitly below with regression analyses. Note that the fit lines for each sex converged in the early 40’s age range, which was the approximate mean age of the 3rd cohort. For weight, females were slightly heavier than males during adolescence and up to about the age of 20 before the fit lines cross over. Males were heavier across the adult sample except for a drop in male weights centered around 42 years of age where the fit lines crossed over briefly.

Body mass index (BMI) diverged for the sexes at the onset of puberty consistent with females increasing their production of less dense lipid tissues, and males increasing production of more dense tissues in the form of lean muscle mass and bone growth. Female BMI remained higher than male BMI throughout virtually the entire adult sample.
with a greater separation observed during early adulthood up to about age 50 after which the fit lines for each sex converged. Female BMI was also markedly high during early adulthood, until about the age of 40, after which it decreased at later ages (cross-sectional data). This pattern likely reflected an increase in body mass in the form of fat related to reproductive function as older females were peri-, or post-menopausal at the time of data collection and we might otherwise expect the increased availability of calories during the reservation period to increase female body weight vis-à-vis BMI across the board. The elevated BMI for early adulthood females was consistent with a similar pattern observed in body weight and indicated that this weight gain was primarily in the form of less dense lipid tissue.

Head circumference was relatively static across adulthood except for the convergence in fit lines for each sex around age 42 and no discernable secular pattern was apparent. In preparation for statistical tests of these trends, descriptive data for each sex and age cohort are provided in Table 1 along with calculation of sex differences and ratios.
Table 1. Mean anthropomorphic data for each cohort and sex, including standard deviation (S.D), mean difference between males and females (M – F), and sex ratio (M / F). The sample size for each cohort (N) is given in parenthesis next to height means.

A rigorous interpretation of Table 1 was not provided here because the cohort means were explicitely tested and graphed in subsequent sections. Note here however, that several of the cohort sample sizes were of limited size and particularly the 2nd and 3rd female cohorts, which contained only 3 individuals each. In general, the handful of women in these two cohorts, who experienced some duration of their adolescent growth spurt during contact whether their take-off growth commenced during contact or not, were
distinguished by being considerably larger and even slightly taller than the more recent, or 5\textsuperscript{th}, cohort. The second cohort of males who started take-off growth before contact but experienced at least 2 years of adolescent growth during contact appeared to be appreciably larger as well, although the sample size (5) was also limited.

In terms of height, weight and to some extent head circumference, the ratio of sexual dimorphism decreased in the middle, or 3\textsuperscript{rd}, cohort who experienced take-off growth during contact. Some of this pattern was driven by the increase in female body size in combination with the decrease in male body size for this cohort. Males in the third cohort were also considerably smaller than any of the other cohorts except for the first cohort of adult males who finished growing before the onset of the contact period.

\textbf{ANOVA}

Two-way independent ANOVA (analysis of variance) were performed on each physical measure (dependent variable) with sex and cohort entered as independent fixed factors along with sex by cohort interaction effects. Results of these analyses are given in Table 2.
Table 2. Two-way independent sample ANOVA for height, weight, body mass index, and head circumference with sex and cohort entered as fixed factors.

Table 2 indicated that the main effect of sex was significant for all measures except weight, while cohort was a significant main effect for all measures except head circumference. The only significant interaction between sex and cohort was found with height. Further interpretation of these results is provided after the next set of figures, which plotted cohort means by sex for each measure with 95% confidence intervals.

<table>
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<tr>
<th>Source</th>
<th>Dependent Variable</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
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<td>Sex</td>
<td>Height (cm)</td>
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<td>1</td>
<td>969.180</td>
<td>61.162</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>Weight (kg)</td>
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<td>1</td>
<td>48.972</td>
<td>1.262</td>
<td>.265</td>
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<td></td>
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<td>53.083</td>
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<td></td>
<td>Head Circ (mm)</td>
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<td>906.111</td>
<td>4.638</td>
<td>.034</td>
</tr>
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<td>Cohort</td>
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<td>143.000</td>
<td>9.024</td>
<td>.000</td>
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<td></td>
<td>Weight (kg)</td>
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<td>325.400</td>
<td>8.384</td>
<td>.000</td>
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<tr>
<td></td>
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<td>16.050</td>
<td>3.143</td>
<td>.019</td>
</tr>
<tr>
<td></td>
<td>Head Circ (mm)</td>
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<td>96.506</td>
<td>.494</td>
<td>.740</td>
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<td>.012</td>
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<td>43.730</td>
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<td>.350</td>
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<td>Body Mass Index (kg/cm²)</td>
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<td>4</td>
<td>4.192</td>
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<td>1492.740</td>
<td>4</td>
<td>373.185</td>
<td>1.910</td>
<td>.116</td>
</tr>
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</table>

Table 2. Two-way independent sample ANOVA for height, weight, body mass index, and head circumference with sex and cohort entered as fixed factors.
Figures 3a – 3d. Line plot of cohort means by sex for height, weight, body mass index, and head circumference. Means bracketed by 95% confidence intervals.

The significant main effect of sex was represented by the higher male average for height and head circumference, and higher female average for BMI. For weight, the sexes were very similar across the cohorts except for the 3rd cohort where males weighed less than females and the 5th cohort where males had greater weight relative to females. The significant main effect of cohort represents fluctuation of the average combined mean for both sexes within a cohort around the overall mean for each measure. In other words, the mean for both sexes for each of the various cohorts were significantly different from the other sex-combined means within that particular measure. Some of this variation was attributable to the secular increase in body size where the later, or more recent cohorts, were larger than the earlier cohorts. However, up and down fluctuation occurred across the middle cohorts as well. The sex by cohort interaction was only significant for height, where the mean of the middle male cohort was substantially lower than adjacent cohorts.
Also, by the 4th and 5th cohorts the secular trend in height favored males over females to a much greater extent than was observed in the 1st and 2nd cohorts.

Any interpretations surrounding the 2nd and 3rd female cohorts, and to some extent the 2nd male cohort, must be tempered by the small sample sizes. A symptom of these small samples can be found in the relatively large error bars that surrounded each of these respective means. The next section statistically examines mean differences between all of the cohorts within each sex. Attention was given to the proposed effect on the middle male cohort by comparing it with earlier and later cohorts. The secular trend in body size was also addressed, although this matter was dealt with more directly in the regression section.

**T-Tests**

Independent sample T-tests were conducted to test for mean differences among each of the five cohorts within each sex for height, weight, BMI, and head circumference. To review, the cohorts were defined as: (1) adults who were fully grown or nearly so before the contact period began; (2) adults who experienced 2 years of adolescent growth during the contact period; (3) adults who experienced take-off growth during a contact year; (4) adults who experienced 2 years of childhood growth during the contact period; and (5) adults who were born during a less severe contact year or later and were fully grown at the time of data collection.

Although the direction of the secular trend was apparent and would justify the use of one-tailed significance tests, two-tailed p-values were reported below to reflect the ambiguity in the direction of change associated with the contact cohort. Power, student’s
and two-tailed, p-values were summarized for each cohort comparison by sex in Table 3.

<table>
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<th>Female Cohort</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Height</td>
<td>Weight</td>
<td>BMI</td>
<td>Head Circ</td>
</tr>
<tr>
<td>1</td>
<td>0.96, -2.926, 0.010**</td>
<td>0.29, -2.254, 0.040*</td>
<td>0.38, -1.618, 0.122</td>
<td>0.60, -2.301, 0.029*</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>0.07, 0.563, 0.603</td>
<td>0.42, 1.844, 0.102</td>
<td>0.46, 1.372, 0.189</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>0.14, 1.134, 0.320</td>
<td>0.10, 0.692, 0.508</td>
<td>0.32, 1.179, 0.256</td>
</tr>
<tr>
<td>4</td>
<td>-</td>
<td>0.09, -0.578, 0.579</td>
<td>0.08, -0.390, 0.702</td>
<td>0.06, -0.379, 0.708</td>
</tr>
<tr>
<td></td>
<td>Height</td>
<td>Weight</td>
<td>BMI</td>
<td>Head Circ</td>
</tr>
<tr>
<td>1</td>
<td>0.97, -3.592, 0.003**</td>
<td>0.84, -2.228, 0.042*</td>
<td>0.68, -2.677, 0.015*</td>
<td>0.84, -3.043, 0.005**</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>0.13, 1.119, 0.326</td>
<td>0.05, 0.062, 0.952</td>
<td>0.13, 0.573, 0.575</td>
</tr>
<tr>
<td>3</td>
<td>-</td>
<td>0.35, -3.700, 0.021*</td>
<td>0.27, -1.274, 0.238</td>
<td>0.09, -0.403, 0.693</td>
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<tr>
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<td>-</td>
<td>0.04, -0.210, 0.839</td>
<td>0.03, -0.002, 0.999</td>
<td>0.05, 0.330, 0.745</td>
</tr>
<tr>
<td></td>
<td>Height</td>
<td>Weight</td>
<td>BMI</td>
<td>Head Circ</td>
</tr>
<tr>
<td>1</td>
<td>0.88, 1.923, 0.072</td>
<td>0.27, 1.157, 0.281</td>
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<td>0.07, 0.546, 0.591</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.21, 1.129, 0.272</td>
</tr>
</tbody>
</table>
Table 3. Independent sample T-tests for female and male cohorts. Power, student’s t and two-tailed, p-values reported for each measure. (*) = Significant at the p < 0.05 level, (**) = Significant at the p < 0.01 level.

For females, the only notable pattern for BMI was that all cohorts with the exception of the 3rd had statistically higher BMI values than the first cohort. For males, only the 2nd cohort had higher BMI than the first. In terms of head circumference, the only significant trend for either sex was that the more recent Aché generation of males (5th cohort) had marginally significant larger head sizes than the first cohort, while the head circumference of the third female cohort was significantly larger than the previous, or 2nd cohort, although this finding may have been driven by the low standard of deviation in the 3rd female cohort rather than a substantial difference in means. Due to the
limited sample sizes and the fact that these two cohorts were similar in terms of weight and particularly height, further interpretation was not warranted.

The strongest pattern from Table 2 was found in the 5th cohort of males being markedly larger than than the 1st and 3rd cohorts. The 4th cohort was also markedly larger in both height and weight than the 1st and 3rd cohorts. The trend between the 1st and 4th/5th cohorts represented the secular trend towards increased body size in males and a similar trend was also observed in females, although not as pronounced as it was in males. The secular trend in body size was examined further in regression analyses that follow. The 3rd male cohort, who experienced take-off growth during the contact period, was virtually identical in terms of height and weight to the men who completed growth before the contact period, or the 1st cohort. The 3rd male cohort was also significantly smaller in terms of both height and weight than males from either adjacent cohort. Of potential interest was that the 2nd male cohort was in league with the 4th and 5th male cohorts in terms of height and weight. A potential culling effect during contact that could have eliminated less robust males from this cohort will be considered, although it can be noted here that this potential effect would not explain the pattern in the 3rd male cohort unless there were even smaller males that did not survive from this cohort. A complementary but not exclusive interpretation was that the stress of the contact episode uniquely disrupted the initiation of the male growth spurt for the 3rd cohort of Aché males.

For females, both the 3rd and 5th cohorts were substantially larger than the 1st cohort, although the 3rd cohort was actually slightly larger than the 5th in terms of height and head circumference, with the 5th cohort just slightly heavier than the 3rd. Of course, given that there were only 3 individuals in the 3rd cohort, any interpretations must be
drawn with great caution. For both sexes, there was strong support for a secular increase in body size in terms of height and weight for the transition from the 1st to the 5th cohorts, with a more dramatic increase in body size for males relative to females. Fluctuations in the middle cohorts will be examined more closely below.

**Regression Analyses**

Regression analyses can also be applied to test the secular change in body size. A variety of regression equations can be generated to fit the data; however, for the present interest in comparing the secular trend between the sexes, linear regression was applied to all variables for both sexes. In cases where another type of equation (quadratic, cubic, etc.) produced noticeably higher R-squared and lower significance values, the type of equation was indicated along with R-squared and p-values.

Simply entering age as a independent variable for each of the dependent anthropomorphic measurements we find that, for males, age was a significant predictor of height (p = 0.000), weight (p = 0.001) but not head circumference (p = 0.187). For females, age was a less so but still significant predictor of height (p = 0.034) and weight (p = 0.019), but not head circumference (p = 0.872). Linear regression equations for each measurement, R-squared and significance values can be found in Figures 4a through 4d0.
Females

\[ \text{Height (cm)} = 151.96 - 0.10 \times \text{Age} \]
\[ \text{R-Square} = 0.11, p = 0.034 \]

Males

\[ \text{Height (cm)} = 166.24 - 0.21 \times \text{Age} \]
\[ \text{R-Square} = 0.33, p = 0.000 \]

\[ \text{Weight (kg)} = 63.29 - 0.18 \times \text{Age} \]
\[ \text{R-Square} = 0.13, p = 0.019 \]
\[ \text{R-Square} = 0.11 \]

\[ \text{BMI (kg/cm}^2\text{)} = 27.56 - 0.05 \times \text{Age} \]
\[ \text{R-Square} = 0.07, p = 0.080 \]
The strongest pattern in the above graphs in terms of slope incline, R-Square coefficient, and significance values was for male height and male weight, indicative of the secular trend towards increased body size. Male head circumference gave the same pattern albeit with more variation, while male BMI remained relatively static across adult ages. For females, height and weight results mirrored the pattern found in males except with less steep slope values, lower R-Squared values and consequently lower significance values. The female trend for BMI was similar to that for height and weight although the regression failed to reach significance and the slope was less steep. Overall, the secular trend in increased body size was present in both sexes but more pronounced in males in comparison to females.

Regression analyses can also be used to evaluate the effect of contact on the adolescent growth spurt vis-à-vis final adult body size. The contact group, or cohort
three, was coded as a dummy variable (0) with members of the other cohorts coded as the
two one (1), thereby creating a dichotomous contact variable (0 or 1), which was
entered along with age as independent predictor variables and regressed on the various
dependent variables or physical measurements. As in the above regressions, all fully
grown adults were included in the regressions, such that the non-contact sample (code =
1) included all adults excepting only those who experienced take-off growth during the
contact period (code = 0).

For male height, the overall model was significant (p = 0.000, $R^2 = 0.448$) with
age being a significant predictor (p = 0.000, unstandardized beta coefficient, or B = -
0.216) and the contact variable was also significant (p = 0.002, B = 5.181), indicating that
contact males were significantly shorter. The interpretation of beta, or B, was that for
each additional year of age, beta represented the change in the dependent variable. In this
case, each additional year of age was associated with a decrease in height of 0.216
centimeters, or - 0.216. The contact beta value indicated that not being in the contact
group (code 1) was associated with an increase in height of 5.181 centimeters. In other
words, contact males (code 0) were about 5.181 centimeters shorter than non-contact
males where the effect of age on height was accounted for, or controlled statistically.

To continue, for male weight, the overall regression model was again significant
(p = 0.000, $R^2 = 0.288$) with age a significant predictor of weight across the lifespan (p =
0.001, B = - 0.220) along with contact (p = 0.016, B = 6.093). But for BMI and head
circumference, neither age nor contact were significant predictors although the direction
of the beta coefficients indicated that male BMI and head circumference for the contact
cohort was lower and smaller, respectively (BMI: age, p = 0.213, B = -0.023, contact, p =
0.224, B = 0.890; Head Circumference: age, p = 0.168, B = -0.196, contact, p = 0.061, B = 1.071). The overall models for BMI (p = 0.226, R² = 0.061) and head circumference (p = 0.071, R² = 0.106) were not significant.

For females, age was a significant predictor of height (p = 0.026, B = -0.100) but not contact (p = 0.159, B = -3.570), although the beta coefficient indicated that contact females were noticeably taller. The overall model was significant (p = 0.040, R² = 0.152). Female weight was very similar to height in that the overall model was marginally significant (p = 0.054, R² = 0.139) with age a significant predictor (p = 0.018, B = -0.185) but not contact (p = 0.542, B = -2.663). Although, again, the beta weight suggested that contact females were heavier. For BMI, the overall model was not significant (p = 0.221, R² = 0.075), age approached significance (p = 0.085, B = -0.052) but not contact (p = 0.976, B = -0.051). For head circumference, age had virtually no effect (p = 0.966, B = 0.006) and contact did not reach significance (p = 0.085, B = -1.472). The overall model was also not significant (p = 0.219, R² = 0.075) although the contact beta coefficient indicated that head circumference for contact females was slightly larger. The fact that there were only three females in the contact cohort may have contributed to the failure to find significance in the female regressions.

**Moving Averages**

A final approach for visualizing the changes in final adult body size across the lifespan was to repeat the LOWESS procedure for computing moving averages but to not discount, or give less weight to, values that were further from the moving average. We can also restrict the percentage or number of adjacent values that were used to compute the average. In this way, the resulting line was more responsive to each individual
measurement regardless of how far that individual was from the postulated average. For each of the following graphs, the bandwidth multiplier was reduced to 0.50, which incorporated 25% of the adjacent x-axis values and the kernel function was set to uniform, which gave equal weight to y-value measurements regardless of how far they were from the moving average.
Figures 5a – 5d. LOWESS smoother lines for height, weight, BMI and head circumference for Aché adults. Smoother parameters: kernel = uniform, bandwidth multiplier = 0.50. Arrow was set at the average age for the third cohort of males (average = 42.4) and females (average = 42.6), or 42.5 years.

The most marked pattern of Figures 5a - 5d was the dip in the male fit line marked with an arrow at the average age of the contact, or 3rd cohort, which was 42.45 years for both sexes. This dip or depression in the fit line at this age was apparent for all male measures with a smaller effect for BMI. Across this cohort—about 39 to 45 years of age—the fit lines for each sex converged with respect to all measures except BMI and in the case of weight and head circumference actually reversed, which produced the decrease in sexual dimorphism for the 3rd cohort as calculated in Table 1. In terms of height and weight, males who just preceded the contact cohort—that is males who had already reached the age of take-off growth at the time the contact period began—were appreciably larger than both the males who were fully grown before the contact period and the males who experienced take-off growth during contact. The males from this cohort approximated the more recent cohorts of post-contact males in terms of height and weight.

Female patterns were less pronounced but showed a stronger secular increase in weight versus height. The increase in weight was likely a result of increased access to food sources during the reservation period in comparison to the forest period. The secular increase in female height was less pronounced than it was for weight, but both height and weight were marked by considerable increases across the contact cohorts. The increase in body size for the contact cohorts was actually higher than female body size following contact although the difference was not statistically significant (Table 2). Note that the
spike in weight shown in Figure 5b at age 33 was driven by a single individual. Because the Aché produced very few births during the severe contact years, this type of graphical analysis was subject to wide variation where few individuals existed across an age span. The only females in the sample for this age range were 35, 33, 29, and 25 years of age, respectively. Similarly, the dip in female head circumference was driven by a 30 millimeter difference between the women aged 33 and 29 years in this sample.

The picture that emerged from head circumference measurements across the contact period for each sex was similar to that for height and weight, which was consistent with high correlations among the three measurements (height, weight, head circumference) within each sex. Of some interest, however, was that the secular trend in body size evidenced by height and weight was not reflected in head size. Except for a dip in head circumference across the contact cohort, male head circumference was essentially the same for all of the other cohorts. Female head circumference was also static across the lifespan excepting again for the slight increase across the contact cohort. The only additional information that emerged from these more sensitive graphs in terms of BMI was the spike in female BMI at age 33, which was again driven by a single female at this age.

c. Discussion

The evidence presented in this chapter supports males as being the more responsive sex to environmental conditions as they relate to physical growth parameters among the Aché. The secular trend towards an increase in body size was registered more significantly in males than females, and the detrimental effect of the contact experience on the initiation of the adolescent growth spurt was only found in males. To review,
males who experienced take-off growth during the conditions of nutritional deprivation and severe psychosocial stress that obtained during the contact period appear to have attained permanently smaller body sizes in comparison to both females of the same cohort and in comparison to either the antecedent or post-contact male cohorts. Following the contact episode, improvements in nutritional and environmental conditions were converted by Aché males into achieving larger adult body sizes relative to females during the same period. The percentage increase in male height between the 1st and 5th cohorts is 5.1%, more than double that for females who increased their height by 2.4% between the 1st and 5th cohorts. The same pattern holds for weight where males increased 15.5% versus 13.7% for females between the 1st and 5th cohorts.

Females who experienced the age of take-off growth during the contact period did not register a decrease in body size in comparison to adjacent cohorts but actually appeared to have increased their body size to some extent. These twin occurrences greatly reduced the sex-ratios for height, weight and head circumference across the middle cohort as indicated in Table 1 and Figures 3 and 5, respectively.

There are pitfalls for over-interpreting the changes in body size across the contact cohorts because of the small sample sizes, and particularly within the female cohorts. Consider that both the 2nd and the 3rd female cohorts, that is those with 2 years of the adolescent growth spurt during contact and those who experienced the onset of take-off growth during contact, contained only three individuals each. The small sample sizes for these cohorts were not the product of a sampling bias as evidenced by a more inclusive census of Aché weight measurements (see footnote 2 for further details) that indicated only one female in either of these two cohorts was missing from the current sample. Born
in 1962, this female reached take-off growth in 1972 and her adult weight was recorded 6
times between 1984 and 2001 for an average of 63 kilograms. This weight puts her right
at the mean for this cohort, which is consistent with the pattern that females from this
period were substantially larger than those who came before them and even somewhat
larger than those who came after.

Before discussing potential adaptive explanations for the male and female growth
patterns across contact, one caution should be kept in mind and that is the effect that
differential mortality may have exerted on those who survived the contact period. Recall
the extremely high mortality rate for the entire Aché population across the contact period;
larger body size, to the extent that it represents a more robust phenotype, could have been
selected for and would produce a pattern of larger individuals having survived the contact
period. Although detailed information for the Aché were not available from this time
period, it is conceivable that survivors of the contact period could have been selected for
because of their larger body size or traits that are linked to larger body size. There is
evidence that stature is positively associated with various health outcomes (Leon et al.,
1995; Peck and Vagero, 1989; Silventoinen et al., 1999) although the causative factors
underlying this relationship are not clear and may correlate through a third factor such as
socioeconomic status. It is noteworthy that female height has been correlated with
reproductive success in a non-western population (Sear et al., 2004) and it seems
plausible that surviving stressful environmental conditions might correlate with larger
body size if for nothing else than being able to obtain more food, which is known to
correlate with childhood growth (e.g., Kielmann et al., 1978).
It is of interest that the five males who initiated take-off growth prior to the onset of the contact period, but experienced at least 2 years of their adolescent growth spurt during the contact period, or the 2nd cohort, were considerably larger than either of their adjacent cohorts. This pattern supports the idea that there may have been selection for larger males during the contact episode. However, for males born between the years 1960 and 1965, who were significantly smaller than either of their adjacent cohorts, we are left with the possibility that differential mortality may have removed even smaller males from the population within this cohort. A pattern that suggests that a growth disruption occurring at the onset of the male adolescent growth spurt presented a specific developmental obstacle that was not able to be overcome through subsequent catch-up growth.

This interpretation fits with what is known about the catch-up growth being more difficult to achieve when a growth perturbation occurs near or during puberty (Prader et al., 1963; Prader, 1978). However, these findings cannot account for female growth appearing to be not only relatively unaffected by the stress of the contact period, but that female growth seems to have taken the direction of achieving larger adult body sizes in response to the contact episode. If the data presented here represent a legitimate pattern (recall the very small sample sizes across the middle cohorts) the ability of females to increase body size in the face of environmental duress is compelling. The general picture is that in the face of environmental stress, female mammals will tend to reduce energy allocation towards body growth in order to meet the energetic requirements of producing offspring (Pickford, 1986).
Although speculative, one interpretation of the female pattern of increased body size is that when resources, and particularly food, are scarce, females assume a greater burden in providing sustenance and probably protection to offspring directly, rather than relying on the more circuitous and otherwise prominent human trend of securing some amount of resources through male consorts. Clearly, more data are needed to answer this prospect.

The evidence for a secular trend with respect to increased body size was very strong in both sexes. There exists the possibility that some amount of this trend was driven by higher mortality on larger body size phenotypes at advanced ages. For example, Suh et al. (2008) found that higher serum levels of insulin-like growth factor I (IGF1) were associated with smaller stature in female offspring of Ashkenazi Jewish centenarians relative to controls. However, this pattern would not seem to have much of an effect on Aché women, who have much shorter lifespans than centenarians and, in any event, this finding does not apply to the strong secular trend found among Aché males.

There are several limitations to the current study. The small sample sizes across the contact cohorts have already been mentioned and restrict firm conclusions from being drawn with respect to patterns across these cohorts. Bear in mind however that this distribution was not the product of an under-sampling bias; there simply weren’t that many Aché present in the middle cohorts with which to better test the potential effects of the contact experience on final adult body size. The patterns found here, and most notably the reduction in male body size vis-à-vis a permanent developmental perturbation for the male Aché who experienced take-off growth during contact, draw additional support in the forthcoming chapters.
Another limiting factor was that the cohort assignments could have been affected by inter-individual variability in the age of onset of puberty. However, the Aché are genetically very closely related (Callegari-Jacques et al. 2008) and several studies indicate that 70%-80% of the variability in the timing of puberty is controlled by heritable factors (see Parent et al., 2003 for overview); van den Berg et al. (2006) have estimated that the probability that the heritability of menarche lies between 0.22 and 0.40 is 95%.

Pubertal timing is also affected by a myriad of factors, many of which are not pertinent here such as socioeconomic status and migration, but specific nutrients are probably involved in addition to the wide-scale introduction of estrogen-mimicking substances found in industrial products and foods (Parent et al., 2003). These “xenoestrogens” cause earlier menarche and appear to also shorten the duration of puberty (de Muinck Keizer-Schrama and Mul, 2001). It is of note that since the start of the reservation period, Aché females were reaching menarche about a year earlier than during the forest period (Hill and Hurtado, 1996); however, Aché females were likely to have had very similar nutritional inputs.

Although we do not have specific nutritional or environmental information prior to or during contact, it is certain that the contact period exerted a tremendous amount of psychosocial stress and nutritional deprivation based on the extensive ethnographies collected by Hill and Hurtado (1996). The overall picture presented here suggests that peri-pubescent Aché males and females may have different physiological and growth responses to environmental conditions. Specifically, males who reached the age of take-

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4 Note that the Aché exhibit a bias towards male offspring, which could affect relative nutritional inputs between the sexes.
off growth during the contact period appeared to have experienced significant and
permanent reductions in final adult body size, while their female counterparts did not and
may have even increased body size in the face of such duress. Following the transition
from forest to reservation living, Aché males converted the more abundant nutritional and
other resources into larger body sizes to a greater extent than females, although both
sexes exhibited an overall increase in body size.
IV. Visual Evoked Potentials

a. Background

Visual evoked potentials (VEP) are a standard clinical tool for assessing the integrity of the visual system. A visual stimulus is presented, which elicits a response from the visual system and this in turn is measured at the surface of the scalp with electrodes that detect electrical changes occurring over the occipital cortex. Note that this technique uses electroencephalography (EEG) to measure short-term electrical changes occurring in the brain in response to a specific stimulus; in other words, the stimulus “evokes” the “potential” firing of whatever neurons are under investigation. Other types of evoked potentials include auditory, sensory and motor.

Pattern-reversal VEP, or PR-VEP, is the most common technique for collecting VEP and uses a reversing black-white, checkerboard pattern as the stimulus. Each stimulus reversal elicits a corresponding response in the occipital cortex where electrodes attached to the scalp at the back of the head—over the occipital cortex—record the electrical activity of visual processing during the procedure. The most stable and major physiological response of the occipital cortex is observable as a characteristic positive peak waveform occurring about 100 milliseconds after the onset of the stimulus. This positive (P) peak is referred to as the P100 waveform. An earlier negative response occurs about 75 milliseconds after the onset of the stimulus (N75) but this measure tends to be more variable than the P100, which has higher within-subject stability and lower variability than the N75 (Chiappa, 1990).

Pattern-reversal VEP only requires subjects to focus their gaze on the surface of the display screen and is appropriate for all subjects except the very young or disabled, in
which case a flashing light is preferred (Flash VEP). The latency of the P100 waveform follows a U-shaped curve over the lifespan with early rapid decreases in P100 latency (faster processing) associated with myelination of the optic nerve, followed by gradual maturation through early adulthood coincident with continual maturation of the visual cortex (Brecelj, 2003). P100 increases slightly across adulthood (slower processing) and is probably reflective of changes in optic nerve conduction velocity (Fotiou et al., 2003) although unspecific decline in retinal structures and luminance response may also be involved (Fiorentini et al., 1996). Decay in visual cortex structure is probably not involved because, in contrast to some other brain regions, the visual cortex shows minimal if any degradation with increasing age (Raz et al., 2004).

Optic nerve conduction velocity depends on the fiber diameter of the axons, the number and form of ion channels in the axon membrane, and the quality (thickness and stability) of the myelin sheath generated by the oligodendrocytes (Kandel et al., 1991). However, estimates of this velocity using PR-VEP must consider test parameters known to affect P100 latency – particularly the size of individual checks in the checkerboard pattern as well as luminance. Check size has been shown to reliably affect P100 latency with larger checks (fewer white or black squares within the same viewing area) being associated with shorter P100 latencies relative to smaller checks (Moskowitz and Sokol, 1983). Luminance affects P100 latency such that brighter screen displays are associated with shorter latencies vis-à-vis faster processing (Tobimatsu et al., 1993).

The general pattern of visual ontogeny is that the ability to discriminate larger checks presented on bright screens is achieved earlier in development than the ability to discriminate small check sizes presented on dimly lit screens. Similarly, during visual
senescence, the loss of ability to discriminate small check sizes presented on dimly lit screens occurs before the loss of ability to discriminate larger checks presented on bright screens.

Because P100 latency is an indicator of visual system ontogeny and aging, P100 values will appear more adult-like at younger ages if the checkerboard squares are large and the screen is bright. Conversely, the use of smaller squares and a dimmer screen will produce a picture of visual maturation characterized by an extended period of continual development up through early adulthood. Logically, senescence will also appear more severe where small and dim checks are used versus large and bright checks (Mitchell et al., 1987; Porciatti et al., 1992).

**Central Nerve Conduction Velocity (CNCV)**

A related technique for estimating optic nerve conduction velocity is to divide head length by P100 latency, which theoretically corrects for head length. This approach has been used by psychometric researchers who have reported significant correlations between optic nerve conduction velocity – or Central Nerve Conduction Velocity (CNCV) – and various psychometric tasks (Reed and Jensen 1992; Reed et al. 2004a). There are a number of anatomical and conceptual issues involved in how to correctly interpret CNCV (head length/ P100) – either as a reliable indicator of nerve conduction velocity or its purported functional relation to so-called fluid intelligence – but these issues are beyond our current scope. CNCV is reported here because it lends greater analytical detail to the relationship between nerve speed and head length vis-à-vis the earlier findings with respect to head size reported among the various male cohorts.
CNCV can also be interpreted as a method for correcting, or standardizing, P100 values by the length of the visual pathway, which is approximated by head length. The evidence suggests a slight sex difference in P100 latency and CNCV (Reed et al. 2004b), which may arise from anatomical differences such as larger male head size (Gregori et al., 2006) or even hormonal influences (Celesia et al., 1987). Just as evoked potentials that involve peripheral limb measurements, known as somatic evoked potentials, are typically standardized by height or limb length because these measurements are known to be influenced by body measurements (e.g., Chu, 1986; 1989); the P100 latency can also be standardized by head length, which is a stronger determinant of P100 latency than sex (Gregori et al., 2006). However, among the adult Aché, no correlation was found between P100 and head length/head circumference once the effect of age was controlled. There was also no correlation between P100 and height, although height and head length were significantly correlated (p = 0.000) when age was controlled for the entire adult sample. These types of correlations were explored in more detail in Chapter 7.

b. Methods

Most people residing in the communities of Arroyo Bandera and Kuetuvy were recruited to participate in the study for a total of 161 individuals (86 males, age range 5-75, mean = 28.71, std. = 18.62; 75 females, age range 3-73, mean = 26.76, std. = 17.48); data presented here focus primarily on the adult portion of this sample. Participants had basic anthropometrics collected including head length measured with calipers to the nearest millimeter from the nasion to the inion. Subjects were then fitted with an electrode cap (Electro-Cap International, Eaton, OH) made of an elastic spandex-type material with
pure tin recessed electrodes attached to the fabric and arrayed in the international 10-20 system.

Recessed electrodes place a conductive layer between the scalp and the metal conductor and have the advantage of reducing movement artifacts (Cooper et al., 1980). Electrodes were filled with conductive gel using a blunted needle syringe at locations F3, F4, C3, C4, P3, P4, O1, O2, T4, T5, FZ with an average reference and a ground electrode positioned slightly below FZ. Impedances were always held below 10 kilo-ohms and typically below 5 kilo-ohms. Following collection of resting electroencephalography (EEG), pattern reversal and flash VEP were collected.

Electrophysiological signals were amplified with Neuron-spectrum-3 and analyzed with software Version 1.4.5.28 (Neurosoft Corporation, Ivanovo, Russia). Bandwidth was set from 0.5 to 35 Hz with a quantization frequency of 200 Hz. EEG signals for the VEP test were collected using the registration montage described above. The software program allows for off line re-referencing such that a VEP montage was created with active electrodes at O1 (left occipital) and O2 (right occipital) and a reference electrode at Fz (frontal sagittal) to better isolate occipital cortex recordings (Chiappa, 1990). The pattern-reversal checkerboard was presented on a 15-inch flat-screen monitor (LiquidVideo E15LCD1). Viewing distance was one meter and luminance remained constant at 30 cd/m2. Field size was $18^\circ \times 13.5^\circ$ with individual checks subtending an arc of 60°. Participants were directed to focus on a red dot at the center of the checkerboard pattern, which reversed at a rate of 1 Hz (two reversals per second) for 100 reversals. P100 values were taken as the average of O1 and O2 for although these values rarely differed by more than a few milliseconds. The analysis epoch was 400 ms.
Data were inspected offline and artifacts from eye blinks, which were clearly visible as muscle artifacts in the F3 and F4 electrodes, were excluded from the analyses as were any other obvious signs of major electrical interference. A mean of about 80 pattern reversals were used for averaging for each subject. It was discovered through experience that removing over half of the trials had minimal effect on the VEP waveform or P100 latency such that the P100 latency values here can be considered accurate.

Pattern-reversal VEP waveforms were identified for the characteristic P100 peak and marked to the nearest millisecond as shown in Figure 6a. Nine subjects were removed from the analyses due to uncertainty in identifying P100 waveforms. For example, one subject had obviously occluded vision and no discernable P100 peak as shown in Fig 4.1b and was therefore not included. Two other males had obvious indications of mental and/or physical disability and did not generated identifiable P100 waveforms.

Figure 6a. Representative PR-VEP waveforms recorded from left and right occipital (O1 and O2) and referenced to Fz. The earlier peak (N75) occurs about 75ms before the P100. X-axis is measured in milliseconds and vertical axis in microvolts. Figure 6b. Subject with occluded vision and no identifiable PR-VEP peak.
i. Analytical Methods and Strategy

The analytical approach pursued here is similar to that found in the previous chapter and identical with respect to the criteria used for the formation of the age cohorts. That explanation will not be repeated here except to note that the sample sizes for the 1st and 3rd male cohorts are one fewer each owing to the fact that one individual from each cohort did not have identifiable peak P100 latencies and were therefore excluded from the current analyses.

Sex differences were examined by evaluating relative aging trends in the P100 and CNCV measures across the adult lifespan. Scatterplots of P100, head length and CNCV across the entire lifespan were initially fit with non-parametric curve fitting (i.e., locally weighted scatterplot smoothing or LOWESS) to display subtle changes with age that may not be captured in a parametric model. A final set of figures plotted just the adult sample with the LOWESS parameters adjusted to allow for more individual variation to be exhibited in the fit line.

Overall changes in the cohorts and between the sexes were analyzed with two-way independent ANOVA (analysis of variance), which was performed on the adult sample for each measure (dependent variable) to test for the main effects of sex and cohort (fixed factors), in addition to sex by cohort interaction effects. Cohort means for each sex and cohort were plotted in line graphs with 95% confidence intervals for interpretation.

An important difference between the current set of measures and the anthropomorphic data presented in the previous chapter is that P100 and CNCV show age-specific changes across adulthood associated with senescence. Aging trends therefore
complicate the detection of a secular trend in these measures as well as within-sex cohort comparisons (T-tests) because the effect of age needs to be controlled. Therefore, complete T-test, cohort comparisons are not presented here as in the previous chapter.

Regression analyses were used to evaluate overall senescent trends in P100 and CNCV, and to test for a potential secular trend in head length. Regression analyses were also used to test the contact cohort through the creation of a dummy-coded contact variable, which was regressed on each neurophysiological measure along with age within each sex.

The reported slight sex differences in P100 latency and CNCV were also found among the Aché although our interest lies primarily in the relative change between the sexes across the adult lifespan. Head length was not appreciably different from the head circumference data presented in the previous chapter; head circumference and head length have a Pearson correlation of about 0.8 for both adults and children of both sexes. However, the head length data are shown below for greater transparency in the derivation of the CNCV measure, which was calculated from the P100 and head length values (CNCV = head length/P100).

c. Results

Figures 7a through 7c depict across-lifespan measures of P100 latency, head length, and Central Nerve Conduction Velocity (CNCV), plotted cross-sectionally by age.
Figures 7a – 7c. Cross-sectional scatter plots of P100, head length, and central nerve conduction velocity for males and females by age. Data were fit with LOWESS smoother, kernel = normal, bandwith multiplier = 1.0. Note that the y-axis was inverted for the P100 Figure to give a more intuitive feel for ontogenetic changes. Central Nerve Conduction Velocity (CNCV) was computed as head length divided by P100 latency.

Figures 7a – 7c provided the overall ontogeny patterns for P100, head length and CNCV as well as the senescent trends for P100 and CNCV. Females had slightly shorter P100
latencies and appeared to be more resilient to the effects of senescence across the adult life span. Males had appreciably longer head lengths and the difference remained relatively constant across the entire adult sample. CNCV was considerably higher, or faster, for younger adult males and the sexes were similar from around the age of 40 and older. The inflections in the fit lines across the middle male cohort will be examined in more detail below.

Descriptive data for each sex and age cohort are provided in Table 4 along with calculated values for sex differences and sex ratios.

Table 4. Mean neurophysiological data for each cohort and sex, including standard deviation (S.D), mean difference between males and females (M – F), and sex ratio (M / F). The sample size for each cohort (N) is given in parenthesis next to P100 means.

<table>
<thead>
<tr>
<th>P100 (ms)</th>
<th>Fully Grown Before Contact</th>
<th>At Least 2 Years of Growth Spurt During Contact</th>
<th>Take-Off Growth During Contact</th>
<th>At least 2 Years of Childhood Growth During Contact</th>
<th>Born After Contact, Fully Grown</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>123.9</td>
<td>116.8</td>
<td>118.3</td>
<td>115.9</td>
<td>112.2</td>
</tr>
<tr>
<td>S.D. (N)</td>
<td>9.03 (14)</td>
<td>3.77 (5)</td>
<td>4.55 (6)</td>
<td>5.11 (10)</td>
<td>4.17 (13)</td>
</tr>
<tr>
<td>Female</td>
<td>119.1</td>
<td>116.7</td>
<td>113.0</td>
<td>113.9</td>
<td>113.2</td>
</tr>
<tr>
<td>S.D. (N)</td>
<td>9.69 (14)</td>
<td>3.06 (3)</td>
<td>8.54 (3)</td>
<td>3.85 (7)</td>
<td>6.11 (15)</td>
</tr>
<tr>
<td>Difference (M - F)</td>
<td>4.86</td>
<td>0.13</td>
<td>5.33</td>
<td>2.04</td>
<td>-0.97</td>
</tr>
<tr>
<td>Sex Ratio (M / F)</td>
<td>1.04</td>
<td>1.00</td>
<td>1.05</td>
<td>1.02</td>
<td>0.99</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Head Length (mm)</th>
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<th></th>
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<th></th>
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<tr>
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<td>181.8</td>
<td>180.2</td>
<td>185.4</td>
<td>183.3</td>
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<tr>
<td>S.D.</td>
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<td>9.04</td>
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<tr>
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<td>177.3</td>
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<td>177.1</td>
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<tr>
<td>S.D.</td>
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<td>6.11</td>
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<td>4.04</td>
<td>5.28</td>
</tr>
<tr>
<td>Difference (M - F)</td>
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<td>6.13</td>
<td>2.83</td>
<td>6.40</td>
<td>6.24</td>
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<tr>
<td>Sex Ratio (M / F)</td>
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<td>1.03</td>
<td>1.02</td>
<td>1.04</td>
<td>1.04</td>
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<table>
<thead>
<tr>
<th>CNCV (m/s)</th>
<th></th>
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<th></th>
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</thead>
<tbody>
<tr>
<td>Male</td>
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<td>1.56</td>
<td>1.52</td>
<td>1.60</td>
<td>1.64</td>
</tr>
<tr>
<td>S.D.</td>
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<td>0.07</td>
<td>0.07</td>
<td>0.09</td>
</tr>
<tr>
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<td>1.51</td>
<td>1.57</td>
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<tr>
<td>S.D.</td>
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<td>0.07</td>
<td>0.10</td>
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<tr>
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<td>-0.05</td>
<td>0.03</td>
<td>0.07</td>
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<tr>
<td>Sex Ratio (M / F)</td>
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<td>0.97</td>
<td>1.02</td>
<td>1.04</td>
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Table 4. Mean neurophysiological data for each cohort and sex, including standard deviation (S.D), mean difference between males and females (M – F), and sex ratio (M / F). The sample size for each cohort (N) is given in parenthesis next to P100 means.

The small sample sizes obtained again for the 2nd and 3rd female cohorts, while the middle male cohort had one less VEP participant compared to the anthropomorphic
measures presented in the previous chapter. The middle, or 3rd, male cohort showed evidence of being relatively slower in terms of P100 and CNCV, and they had shorter head lengths in comparison to adjacent male cohorts or in comparison to the 3rd female cohort. These patterns were explicitly tested below.

**ANOVA**

Two-way independent ANOVA (analysis of variance) were performed on P100, head length, and Central Nerve Conduction Velocity (CNCV) (dependent variables) with sex and cohort entered as independent fixed factors along with sex by cohort interaction effects. Results of these analyses are shown in Table 5.

<table>
<thead>
<tr>
<th>Source</th>
<th>Dependent Variable</th>
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<th>Mean Square</th>
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</tbody>
</table>

Table 5. Two-way independent sample ANOVA for P100, head length, and Central Nerve Conduction Velocity (CNCV) with sex and cohort entered as fixed factors.

The only significant main effect of sex was for head length, where males had longer heads, while cohort was a significant main effect for P100 and CNCV but not head length. These findings reflect the above statement concerning male and female head length moving in relative lockstep with a higher male average across the lifespan. The fact that cohort was a significant main effect for CNCV but not head length indicates that
it was P100 values that drove this result since CNCV is derived from both measures (CNCV = head length/P100). The sex by cohort interaction was not significant for any measure. Figures 8a – 8c plotted cohort means by sex for each measure with 95% confidence intervals.

Figures 8a - 8c. Line plot of cohort means by sex for P100, head length, and Central Nerve Conduction Velocity (CNCV). Means bracketed by 95% confidence intervals. Note that the y-axis was inverted for P100 to give a more intuitive feel for ontogenetic changes.
The senescent trend was apparent for P100 and by extension CNCV, which was derived from P100 values for both sexes. Head length did not exhibit a secular trend although there was some fluctuation across the cohorts, and more so for males. The main effect of cohort for P100 and CNCV reflected the senescent changes that occurred across the cohorts. Although the cohort means for each sex fluctuated quite a bit the lack of a sex by cohort interaction effect indicated that these changes averaged out. The overall pattern for the 3rd male cohort was similar to the measurements already reported in terms of height, weight and head circumference in that the 3rd male cohort appeared compromised in all measures relative to adjacent cohorts.

Note that higher P100 latency values were indicative of slower visual processing and the converse is also true, that lower P100 latency values were reflective of faster visual processing. If we consider that a sex difference in the rate of senescence was responsible for the increase in the sex ratio for the first cohort, the sex ratio in the third cohort becomes more meaningful because both adjacent cohorts had lower sex ratios in the P100 measure; suggesting that males who initiated take-off growth during the contact period experienced compromises in neurophysiological development and function, in addition to the physical growth parameters already explored.

As expected, head length showed much the same pattern as head circumference presented in the preceding chapter. Note that smaller head size is, ceteris paribus, associated with shorter P100 latency values, because the nerve impulse has a shorter distance to propagate. This was noteworthy because the third male cohort, despite having smaller head sizes, did not have shorter P100 latencies. Their P100 latencies were, in fact, longer or slower than the P100 latencies of either adjacent cohort. The calculation of the
Central Nerve Conduction Velocity (CNCV) for this cohort was thus driven by smaller head length values in the numerator and greater P100 latencies in the denominator, which generated the markedly slower CNCV values observed in the 3rd cohort.

As shown below, males had a more pronounced senescence pattern in terms of P100 latency, particularly at older ages; while incorporating head length to calculate CNCV generated much faster CNCV values for males in the 5th, or youngest, cohort.

**Regression Analyses**

The main interest in using linear regression was to test the rate of senescence in the P100 measure and by extension CNCV. The approach here was the same as for the anthropomorphic measures where linear regression was applied to all the variables and in cases where another type of regression equation (quadratic, cubic, etc.) produced higher R-squared and significance values, that equation was also included in the figure. Linear regression equations for each measurement, R-squared and significance values can be found in Figures 9a through 9c.
Figures 9a – 9c. Linear regression fit lines, equations, R-Squared and significance values for age regressed on P100, head length, and Central Nerve Conduction Velocity (CNCV) for adults of each sex. Equation type, R-squared, and p-values are shown for figures with stronger relationship than linear regression.

The senescent trend in males was more pronounced for P100 and CNCV than in females as indicated by the steeper equation slopes and a slight age trend was found in males for head length. Age had a stronger relationship with P100 and CNCV in males than in females and in both cases quadratic equations fit the data better than linear regression.
equations with the largest improvements found in female P100 and CNCV. The majority of this improvement however was attributable to the last female data point at age 73 that was considerably further from the other data points for P100 and CNCV.

Regression analyses using a dummy-coded contact variable to test the effect of contact on the 3rd cohort failed to return significant values for any measure or sex although the beta coefficients for the 3rd male cohort were in the expected direction for P100 (0.639, p = 0.799), head length (-3.403, p = 0.188) and CNCV (0.042, p = 0.274) in terms of being longer (P100), shorter (head length), and slower (CNCV), respectively.

Conducting T-tests among cohorts within each sex would require the senescent trends just observed to be controlled for age. However, to further probe the male contact cohort, independent sample T-tests were performed comparing the middle male cohort (3rd) with the two adjacent cohorts (2nd and 4th). A statistically significant difference was found between the third and fourth cohorts for CNCV (T = 2.249, p = 0.041, 2-tailed) and head length (T = 2.714, p = 0.017) but not between the 3rd and 2nd cohorts for either measure. The CNCV means for the 2nd and 4th cohorts were very similar however, indicating that the small sample size in the 2nd cohort contributed to not finding a statistically significant difference in this case.

Moving Averages
A final approach for visualizing the changes in final adult body size across the lifespan was to repeat the LOWESS procedure for computing moving averages but to not discount, or give less weight to, values that were further from the moving average. We can also restrict the percentage or number of adjacent values that were used to compute
the average. In this way, the resulting line was more responsive to each individual measurement regardless of how far that individual was from the postulated average. For each of the following graphs, the bandwidth multiplier was reduced to 0.50, which incorporated 25% of the adjacent x-axis values and the kernel function was set to uniform, which gave equal weight to y-value measurements regardless of how far they were from the moving average.
Figures 10a – 10c. LOWESS smoother lines for P100, head length, and central nerve conduction velocity (CNCV) for Aché adults. Smoother parameters: kernel = uniform, bandwidth multiplier = 0.50. Arrow was set at the average age for the third cohort of males (average = 42.4) and females (average = 42.6), or 42.5 years.

Figures 10a – 10c were again marked with an arrow to indicate the average age of the contact, or third, cohort, at 42.5 years of age. A depression in the fit lines occurred across the middle cohort, about 39 to 45 years of age, as was found in the anthropomorphic measurements reported in the previous chapter. Shorter head length in combination with slower P100 latencies combined to produce the dramatic decrease in the derived measure of CNCV for the 3rd cohort. The wide swings in P100 latency and CNCV for younger females was again driven by the presence of only three females in the age range of 25 to 33.

d. Discussion

The overall pattern to emerge from the data presented here was generally consistent with the picture that emerged from the anthropomorphic data presented in the previous chapter although statistically significant findings were, by and large, not found with respect to the visual evoked potential data. A more pronounced senescence pattern was observed in P100 latency across the adult male lifespan in comparison to females and there was some evidence for a permanent, developmental perturbation with respect to neurovisual function being registered among those Aché males who initiated their take-off growth spurt during one of the severe contact years.

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5 The removal of one male from the sample for not having an identifiable P100 peak does not affect this age average.
There are several relevant issues with respect to the interpretation of the VEP data as supportive or not for the 3\textsuperscript{rd} cohort perturbation effect that was more obviously identified in the anthropomorphic data. First, P100 measurements are highly variable by nature and there exists poor understanding of variation that may arise from recording equipment, and test protocols and procedures (Odom et al., 2004). However, the ontogenetic patterns observed in P100 measurements for the Aché were in agreement with general findings from other VEP studies in that P100: showed a rapid maturation across childhood and adolescence and a slower senescence pattern across adulthood; showed that female P100 values were slightly lower, or faster, than male P100 values across the lifespan; and that males senesce more rapidly in comparison to females across the adult lifespan. These facts lend support to the contention that the VEP data collected here were accurately representative of the visual integrity and visual ontogeny of the Aché population. Yet, despite the high variation in P100 measurements, the mean P100 value for the 3\textsuperscript{rd} male cohort was nonetheless consistent in terms of direction and functional interpretation with the anthropomorphic measurements presented in the previous chapter.

Although the correlational T-test matrix was not presented in this chapter due to the senescent patterns observed in P100 measurements that need to be controlled for age, the head length data presented in this chapter, where there exists no obvious secular trend, indicated that the 3\textsuperscript{rd} male cohort had significantly shorter head lengths than the 4\textsuperscript{th} cohort (T = 2.714, p = 0.017, 2-tailed) in addition to slower CNCV (T = 2.249, p = 0.041, 2-tailed). The comparison of the 3\textsuperscript{rd} cohort with the 2\textsuperscript{nd} cohort did not return significant results for P100, head length, or CNCV. Head length among the 2\textsuperscript{nd} cohort was similar to

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the 3rd cohort, despite the 2nd cohort being significantly taller than the 3rd, which brought down CNCV values for the 2nd cohort, although they were still faster than the 3rd cohort (1.56 m/s versus 1.52 m/s).

With respect to the potential perturbational developmental effect, the longer P100 latencies observed in the contact cohort in combination with shorter head lengths, are persuasive in that, ceteris paribus, shorter head lengths are expected to associate with shorter (or faster) P100 latencies because the nerve pulse has less distance to propagate. The derived measure of Central Nerve Conduction Velocity (CNCV) reflected this pattern most abundantly, as shown in Figure 10c, where smaller head sizes and slower P100 values combined to produce the defined depression across the 3rd cohort.

Because head length is correlated with height (for adult males the Pearson correlation coefficient is 0.81), it could be argued that the P100 values were proportionately affected. In other words, height and head size represent similar indices. However, the Pearson correlation coefficient for P100 values and head length for the contact cohort is only -0.08; and, as was noted earlier and as can be seen in Table 3, in comparison to adjacent cohorts, the P100 latencies for the male contact cohort were actually longer in spite of them having shorter head lengths.

The non-significance of the regression analyses that coded the 3rd cohort as a dummy variable can be partly attributed to the fact that age accounted for much of the variance in the P100 measure—and by extension CNCV—across the adult male age range, owing to the strong senescence pattern observed in male P100 values. It is of note that the overall ontogenetic and senescence patterns in P100 and CNCV suggest that the Aché exhibit a somewhat delayed maturation and greater senescence across adulthood in
comparison to some other populations. The ontogenetic pattern evidenced by P100 was consistent with the indices of physical growth presented here and also by Walker et al. (2006). The more rapid senescence pattern across adulthood was also consistent with census data that show very few individuals surviving to advanced ages, in addition to other morbidity data that indicate the Aché experience more pronounced rates of disease (e.g., Hurtado et al., 2003).

The limitations of the current data are several. The small sample sizes across the adult cohorts and particularly the middle cohorts have already been mentioned but were unavoidable given the characteristics of the study population. The poorly understood variation in P100 measurements provides pause for over interpreting the patterns presented here. The correct interpretation of CNCV is also problematic due to numerous anatomical and conceptual issues that are not agreed upon by researchers in this area. For example, does adding head length to the numerator of P100 actually reflect nerve speed? Although these issues have not been satisfactorily worked out, for our purposes the combination of P100 and head length as a gestalt marker of neurophysiological and anatomical health appears less problematical.

Despite the unresolved issues of P100 variation, the generation of consistent and expected patterns in terms of ontogeny, senescence, sex differences and the potential effect of contact on the 3rd male cohort lends support that the data presented here was representative of a real pattern among the Aché. Although significant differences were

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6 See for example Allison et al. (1983) for a study using the same check size used here. However, VEP data cannot be formally compared with other VEP studies because of poor understanding of variability arising from recording equipment and techniques (Odom et al., 2004). On one hand, the large check sizes used in this study (60°) and clear age decline in P100 across adulthood suggest marked senescence. On the other hand, the relatively low luminance (30 cd/m2) of the checkerboard stimulus – also presented on a flat screen monitor which is different from virtually all earlier studies – would mitigate in the opposite direction by exaggerating senescent trends and is likely contributing to the relatively longer P100 latencies recorded here in comparison to other studies.
not found in the various statistical tests of the 3\textsuperscript{rd} cohort, in light of the anthropomorphic data where significant differences were found, the data here become more meaningful. A related conclusion is that, in conjunction with the strong anthropomorphic evidence for a permanent developmental perturbation in the male contact cohort, visual system development was less affected by the contact episode indicating perhaps that visual ontogeny—as measured by the VEP parameters of this study—was mostly complete by age of take-off growth for the male contact cohort. Relatedly, a disruption in visual system ontogeny may be correctable at later ages in ways that physical growth was not.

Overall, males exhibited a more pronounced senescence trend across adulthood in terms of visual system integrity and the middle cohort of males who initiated their adolescent growth spurt during one of the severe contact years exhibited limited evidence of a developmental perturbational effect with respect to neurovisual development and function. Evidence for a developmental perturbation in the male contact cohort gains further support from physiological measurements presented in the next chapter.
V. Bioelectric Impedance

a. Background

Bioelectric impedance analysis (BIA) is an increasingly popular technique for body composition assessment because it is low-cost, portable, non-invasive and safe (Barbosa-Silva and Barros, 2005a). BIA is based on the principle that various tissues in the human body have characteristic resistive and conductive properties, which can be measured when the body is subjected to a known alternating electrical currency. BIA does not directly measure body composition but uses the measures of whole body electrical conductivity and resistance to compute various predictive regression equations. However, these equations are population specific and are more profitably derived from post-hoc analysis in combination with an accepted means of measuring body composition, rather than attempting to apply regression equations a priori across populations (Kyle et al., 2004; Lupoli et al., 2004). Various regression equations available in the literature were used to calculate body composition using BIA parameters for the Aché. However, the values returned were extremely inaccurate; for example, giving body fat percentages upwards of 50% for both sexes. Thus, no further effort was made here to calculate body composition values from BIA parameters.

Investigators have found alternate applications for the electrical parameters generated from BIA (Barbosa-Silva and Barros, 2005a). The most intriguing of these measures is referred to as phase angle (PA) and will be described below following a description of the two component measures of impedance—resistance and reactance—from which it is derived.

The opposition of electric current as it flows through the body is referred to as
impedance, which consists of resistance (R) and reactance (Xc) components. Resistance, or R, is measured in Ohms and is inversely proportional to the fluid volume of the body following Ohm’s law (Foster and Lukaski, 1996). Lean tissues in humans such as muscle contain large amounts of water and conducting electrolytes and thus provide very little resistance to the flow of electricity, whereas lipid and bone tissue are poor conductors of electricity and provide higher resistance (Thomassett, 1962). Females typically have higher R values than males due to having higher proportions of body fat, which is a poorer conductor of electricity than muscle mass, which is typically higher in males (Chumlea et al., 1996; Dittmar, 2003). Resistance decreases from birth through early adulthood and is not typically affected by age throughout the lifespan, although it will decrease at older ages, likely associated with muscle loss and lower hydration, or in cases where severe illness marked by cachexia occurs.

Reactance (Xc), also known as capacitive reactance when describing biological tissues, is also measured in Ohms and represents the reciprocal of the opposition to the flow of electric current caused by the capacitance of cell membranes, tissue interfaces, and non-ionic tissues (Foster and Lukaski, 1996). A capacitor is a system capable of holding a charge or storing up electrons and typically consists of two or more conducting layers separated by an insulator or non-conductive substance. This insulated layer is called the dielectric and is a poor conductor of electricity but an efficient supporter of electrostatic fields. For example, the dielectric in a biological cell membrane consists of an inner layer of non-conducting lipid tissue surrounded by two highly conductive layers of protein-phosphate molecules, forming the so-called phospholipid bilayer. Xc increases with the volume of cell membrane capacitance and is an indirect measure of the
intracellular volume or body cell mass. Only cell membranes offer capacitive reactance and $X_c$ is therefore not affected by the quantity of body fat. Reactance reflects the ability of cells to store energy where a low reactance indicates a breakdown in cell membranes integrity and higher reactance is consistent with having healthier cells. $X_c$ values are typically higher in females although the strongest pattern occurs with age, where $X_c$ stabilizes during late adolescence before declining across the adult lifespan (Dittmar, 2003).

Resistance ($R$) and reactance ($X_c$) together represent the overall opposition to electric current in the body. Where $R$ is related to the amount of ionic substances in the body, mainly water and other electrolytes, $X_c$ represents the portion of the current that is stored by capacitors such as cell membranes. These properties create a phase shift in the current ($R$) and voltage ($X_c$) of the electrical circuit, which can be expressed quantitatively as phase angle. Phase angle ($PA$) is calculated as the arctan of $X_c/R$ and is usually expressed in degrees. A graphical representation of bioelectric impedance ($Z$), resistance ($R$), reactance ($X_c$), and phase angle ($PA$) is shown in Figure 10. Here we can see that $PA$ depends on both $R$ and $X_c$, where $PA$ is the angle between $Z$ and $R$. The higher the $X_c$ relative to $R$, the higher the $PA$. 
In human populations, phase angle (PA) shows an inverted-U shape across the lifespan, increasing up to early adulthood and then declining into old age (Bosy-Westphal et al., 2006). PA increases with increasing body cell mass (Dittmar, 2003) and is higher in males, which reflects their greater overall mass in comparison to females. While both R and Xc are influenced by the length of the conductor and can be standardized by dividing by height, for the calculation of PA the height term falls out and PA is given as a ratio typically expressed in degrees.

**Phase Angle as a Global Marker of Health**

PA has generated a significant amount of interest from health researchers because it is an extremely accurate predictor of health outcomes across a wide variety of clinical conditions. For example, of 40 trauma victims admitted to an intensive care unit for at least 5 days, PA predicted with 100% accuracy the survivors (PA>4) from those who perished (PA<3) (Mueller et al., 1998). PA has been found to be an accurate prognostic indicator for patients with advanced colorectal cancer (Gupta et al., 2004a), pancreatic
cancer (Gupta et al., 2004b), lung cancer (Toso et al., 2000), and patients with post-operative complications (Barbosa-Silva and Silva, 2005b) including severity of illness in pediatric patients after heart surgery (Shime et al., 2002). PA is an accurate predictor of survivability for patients with liver cirrhosis (Selberg, 2002) and patients on dialysis (Mushick et al., 2003). For HIV patients, PA is a significant prognostic marker of clinical progression and survivability (Ott et al, 1995; Schwenk et al., 2000). In patients with neuromuscular disease, PA was inversely correlated with disease progression and disease remission (Rutkove, 2002).

Phase angle has also been found to be a useful indicator of nutritional condition, showing positive correlations with body weight and arm muscle circumference among children (Nagano et al., 2000) and with weight and body-mass-index among anorexic patients (Scalfi et al., 1999). Phase angle and reactance have also been correlated with basal metabolic rate among obese patients (Marra et al., 2003) and a group of female patients with anorexia nervosa (Marra et al., 2005). Phase angle has also been found to be a significant indicator of sickle cell disease in Nigerian children, and correlated positively with levels of several omega-3 fatty acids that play important roles for cell membrane fluidity and function (VanderJagt et al., 2002).

The foregoing detail is provided to give the reader a sense of the broad applicability of phase angle across many health conditions, which indicates that PA is capturing some intrinsic quality of cellular and/or whole-body health. Although some reviewers correctly point out that capacitance results from properties of body tissues and cellular components other than cell membranes (Foster and Lukaski, 1996; Gabriel et al., 1996), most interpretations of reactance vis-à-vis phase angle rely on the dielectric
properties and integrity of cell membranes where membranes with greater functional integrity, it is argued, are associated with higher PA levels and vice-versa.

Although the integrity and function of cell membranes certainly contributes to the overall capacitance measures in the body, many researchers acknowledge that the biological meaning of PA is still poorly understood (e.g., Barbosa-Silva and Barros, 2005a). Dittmar’s (2003) investigation is notable for showing that physical fitness was associated with higher PA levels among older subjects, the only cohort that was investigated along these lines. Her finding suggests that other components are likely contributing to PA measurements beyond cell membrane function, such as cell size and the conductivity of the intracellular and extracellular media (Foster and Lukaski, 1996). In this view, Dittmar’s (2003) findings become more interpretable because, for instance, exercise is known to increase mitochondrial density in skeletal muscle tissue and may improve mitochondrial function as well (Holloszy and Coyle, 1984). It would seem unlikely that cell membrane integrity alone can account for the systematic correlation between PA and the broad range of health outcomes just reviewed. Further remarks and a path towards a possible resolution of this matter are outlined in the concluding chapter.

b. Methods

BIA measurements were collected from 150 Aché (82 males, age range 5-74, mean = 29.35, std. = 18.26; 68 females, age range 3-59, mean = 27.13, std. = 16.67).

BIA analysis was performed with an RJL quantum impedance analyzer (model 101; RJL Systems Inc., Mt. Clemens, MI), which applies an 800 µ-amp alternating current at a frequency of 50 KHz. Subjects were asked to void before measurements were taken. Subjects either did not have on or were asked to remove socks and shoes and were supine.
on a non-conducting surface (rice bags) with limbs abducted approximately 30° from the
body. Signal-introducing electrodes were placed on the first joint of the middle finger and
just below the middle toe. The detecting electrodes were placed with the upper edge of
the electrode bisecting the ulnar head of the wrist and the median malleolus of the foot.
Care was taken to ensure that the signal and detecting electrodes were not closer than 5
cm. Duplicate measurements of resistance (R) and reactance (Xc) were collected from
each subject. Phase angle was calculated as the angular transformation or arctan Xc/R.
The impedance analyzer was calibrated daily before data collection using a 500 Ω
resistor.

**Analytical Methods and Strategy**

The analytical approach pursued here was identical to that found in the previous chapter
concerning visual evoked potentials (VEP). There were again slight changes in the
sample sizes; in comparison to the original sample for anthropomorphic measurements,
there was one fewer male in each of the male cohorts except for the 3rd, and two fewer
females in the first cohort. Some subjects were not administered the BIA test in order to
conserve testing materials.

Sex differences were examined by evaluating relative aging trends in resistance
(R), reactance (Xc), and phase angle (PA) across the adult lifespan. Scatterplots of R, Xc
and PA across the entire lifespan were initially fit with non-parametric curve fitting (i.e.,
locally weighted scatterplot smoothing or LOWESS) to display subtle changes with age
that may not be captured in a parametric model. A final set of figures plotted just the
adult sample with the LOWESS parameters adjusted to allow for more individual variation
to be exhibited in the fit line.
Overall changes in the cohorts and between the sexes were analyzed with two-way independent ANOVA (analysis of variance), performed on the adult sample for each measure (dependent variable) to test for the main effects of sex and cohort (fixed factors), in addition to sex by cohort interaction effects. Means for each cohort and sex were plotted in line graphs with 95% confidence intervals for interpretation.

Similar to the P100 and CNCV measures presented in the previous chapter, BIA measures and particularly reactance and phase angle show age-specific changes across adulthood associated with senescence. Aging trends therefore complicated the detection of a secular trend in these measures as well as within-sex cohort comparisons (T-tests) because the effect of age needs to be controlled. Complete T-test cohort comparisons were therefore omitted from the present analysis.

Regression analyses were used to evaluate overall senescent trends in R, Xc, and PA as well as to test the contact cohort through the creation of a dummy-coded contact variable, which was regressed on each bioelectrical impedance measure along with age within each sex.

c. Results

Figures 11a through 11c depict across-lifespan measures of resistance (R), reactance (Xc), and phase angle (PA), plotted cross-sectionally by age.
Figures 11a – 11c. Cross-sectional scatter plots of resistance, reactance, and phase angle for males and females. Data were fit with LOWESS smoother, kernel = normal, bandwith multiplier = 1.0. Resistance and reactance reported in Ohms, while phase angle was reported as degrees (arctan R/Xc * 180/Π). Note that the y-axis was inverted for resistance (R) to give a more intuitive feel for ontogenetic changes.
Figures 11a – 11c provided the overall ontogeny patterns for R, Xc and PA. In comparison to females, males had lower R and lower Xc, and higher PA across the lifespan (note the inversion of the y-axis for resistance, or R). These findings were consistent with other studies (e.g., Barbarosa et al., 2005; Biasioli et al., 1993; Denti et al., 1997; Dittmar, 2003) and can be explained by females having higher relative proportions of body fat, which increases R, and males having more muscle mass, which decreases R. Xc had a much quicker developmental trajectory than R, reaching adult values around the age 10 before declining markedly across the lifespan in contrast to R, which showed minimal changes across the adult age range. PA increased throughout early adulthood, reaching peak values around age 25 before declining across the lifespan. Higher PA values in males relative to females can be explained by males having more body mass, which correlates with PA (Barbarosa et al., 2005). Changes across the adult lifespan are examined more in depth below.

Descriptive data for each age and sex cohort are presented in Table 6 along with sex differences and ratios.
Table 6. Mean bioelectric impedance data for each cohort and sex, including standard deviation (S.D), mean difference between males and females (M – F), and sex ratio (M / F). The sample size for each cohort (N) is given in parenthesis next to height means.

The limitations of sample sizes have already been mentioned and still obtained across the middle cohorts. The sex ratios for R and Xc did not appreciably change across the cohorts, although there was more variation in the sex ratio for PA. The middle, or 3rd, male cohort was similar to the 1st cohort in terms R and PA, and male Xc increased in moving from the 1st to 5th cohorts. Female trends were less pronounced. These patterns were examined further below.
ANOVA

Two-way independent ANOVA (analysis of variance) were performed on R, Xc, and PA (dependent variables) with sex and cohort entered as independent fixed factors along with sex by cohort interaction effects. Results of these analyses are shown in Table 7.

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Table 7. Two-way independent sample ANOVA for resistance, reactance, and phase angle with sex and cohort entered as fixed factors.

Sex was a highly significant main effect for resistance (p = 0.000), significant for phase angle (p = 0.011), and marginally significant for reactance (p = 0.077). Cohort was a significant main effect for reactance and phase angle, but not resistance, which was relatively stable across the adult lifespan for the Aché. The sex by cohort interaction was not significant for any measure. In general, the BIA measures showed marked sex differences and significant cohort effects emerged as reactance, and consequently phase angle, decreased across the lifespan, although these changes did not interact with sex.

Figures 12a – 12c plot cohort means by sex for each measure with 95% confidence intervals.
Figures 12a - 12c. Line plot of cohort means by sex for resistance, reactance, and phase angle. Means bracketed by 95% confidence intervals. Note that the y-axis was inverted for resistance to give a more intuitive feel for ontogenetic changes.

The significant main effect of cohort for reactance and phase angle was driven by the senescent trend in these measures. Within the 2\textsuperscript{nd} cohort, high resistance values were recorded for males and low reactance values were recorded for females, which produced
the separation in phase angle between the sexes for this cohort. However, recall that the 2\textsuperscript{nd} cohort for both males and females was limited in size (females = 3, males = 4) so over interpretation of this result was not warranted. The senescent trends in these measures were analyzed more explicitly with regression below.

Regression Analyses

The main objective for using linear regression was to compare the rate of senescence in resistance, reactance and phase angle between the sexes. The approach here was the same as for the anthropomorphic and visual measures where linear regression was applied to all the variables and in cases where another type of regression equation (quadratic, cubic, etc.) produced higher R-squared and significance values, that equation was included in the figure. Linear regression equations for each measurement, R-squared and significance values can be found in Figures 13a through 13c.
Figures 13a – 13c. Linear regression fit lines, equations, R-Squared and significance values for age regressed on resistance, reactance, and phase angle for adults of each sex. Equation type, R-squared, and p-values are shown for figures with stronger relationship than linear regression.

The senescent trend in males was more pronounced for reactance and phase angle than in females and a slight senescent trend in males was found for resistance. Age had a markedly stronger relationship with reactance and phase angle in males than in females.
and the only equation that was (slightly) improved was a cubic regression for female resistance.

Regression analyses were used to test the effect of contact on the 3rd cohort by creating a dummy-coded contact variable and regressing it along with age on the BIA parameters. These analyses failed to return significant values for any measure or sex although the beta coefficients for the 3rd male cohort were in the expected directions for resistance ($B = -23.861, p = 0.089$) and phase angle ($0.404, p = 0.158$) in terms of being higher (R) and lower (PA), respectively. In terms of reactance, the 3rd cohort fell almost precisely on the linear fit line ($B = 0.003, p = 0.999$).

Conducting T-tests among cohorts within each sex would require the senescent trends just observed to be controlled for age. However, to further probe the male contact cohort, independent-sample T-tests were performed comparing the middle male cohort (3rd) with the two adjacent cohorts (2nd, 4th). A statistically significant difference was found between the third and fourth cohorts for PA ($T = -2.164, p = 0.048, 2$-tailed) but not between the 3rd and 2nd cohorts ($T = -1.274, p = 0.235, 2$-tailed) although the means for the 2nd and 4th cohorts were virtually the same, indicating that the small sample size in the 2nd cohort contributed to a non-statistically significant finding in this case. With respect to resistance (R), the 3rd cohort was significantly different from the 2nd cohort ($T = 2.279, p = 0.049, 2$-tailed) in terms of having higher R, but very similar to the 4th cohort in terms of R. More will be said on this pattern in the discussion.

**Moving Averages**

A final approach for visualizing the changes in final adult body size across the lifespan was to repeat the LOWESS procedure for computing moving averages but to not
discount, or give less weight to, values that were further from the moving average. We can also restrict the percentage or number of adjacent values that were used to compute the average. In this way, the resulting line was more responsive to each individual measurement regardless of how far that individual was from the postulated average. For each of the following graphs, the bandwidth multiplier has been reduced to 0.50, which incorporated 25% of the adjacent x-axis values and the kernel function was set to uniform, which gave equal weight to y-value measurements regardless of how far they were from the moving average.
Figures 14a – 14c. LOWESS smoother lines for resistance, reactance and phase angle for Aché adults. Smoother parameters: kernel = uniform, bandwidth multiplier = 0.50. Arrow indicates the average age of the third cohort for males and females ~ 42.5 years.

Figures 14a – 14c were marked with an arrow to indicate the average age of the contact, or third, cohort, at 42.5 years of age. No obvious patterns emerged with respect to R and Xc showed a slight decrease in terms of the male contact, or 3rd cohort. Phase angle however showed a marked decrease across the contact cohort, or about 39 to 45 years of age; a pattern that was consistent with the anthropomorphic and neurophysiological information presented earlier. There were major fluctuations across the female sample and particularly in the age range of 25 to 33, where only three females were recorded. Because there were no previous patterns found among females with respect to the previous measures—as was the case with the middle male cohort—no further interpretation was attempted here.
d. Discussion

The pattern that emerged from the BIA measurements was consistent with the pattern observed in previous measurements with respect to more pronounced male senescence and the developmental perturbation in the male contact cohort, although statistically significant findings were, for the most part, not observed. Adult males showed more pronounced senescence in terms of reactance (Xc) and phase angle (PA) in comparison to females and some of this pattern can be attributed to young adult males who registered high values in these measures in comparison to older males. The pattern across the middle-male cohort was again consistent in terms of direction and functional interpretation with there being a permanent, developmental perturbation within the 3rd male cohort who initiated their take-off growth spurt during one of the severe contact years.

In general, the BIA data presented here appeared to collect meaningful information in that the expected population patterns were observed with respect to growth, senescence and sex differences. Males had lower resistance (R) values consistent with having lower body fat and higher muscle mass in comparison to females; males exhibited a more pronounced senescence trend across adulthood in terms of Xc and PA; and females had lower PA values across the lifespan consistent with their having overall lower body mass in comparison to males.

Similar to the visual evoked potential data, the non-significance of the regression analyses that used a dummy-coded variable to test for differences in the male contact cohort can be accounted for, in part, by the fact that age accounted for much of the variance in the BIA measures—particularly Xc and PA—across the adult age sample, and
especially for males (see Figures 13b and 13c). A novel finding of the present study was that adult males showed a much higher correlation of age with Xc and PA than was found among adult females. A cluster of young adult females recorded lower Xc and PA values than might be expected relative to the remainder of the adult female population.

Although pregnancy status was not recorded in the present study, the Aché are a natural fertility population and most reproductive-aged women cycle through pregnancy states continually throughout their reproductive careers (Hill and Hurtado, 1996). Changes in R and Xc have been observed across pregnancy where R and Xc decrease during gestation and return to normal post-partum (Lukaski et al., 1994; Lukaski et al., 2007). This pattern is associated with gains and losses in body weight and total body water that occur across the pregnancy and birth. With respect to phase angle (PA), Lukaski et al., (2007) reported a slight dip in PA during the first trimester that stayed consistent across gestation and an up to 8-10 weeks post-partum. However, among a rural population of Bangladeshi women, Shaikh et al., (2011) found that pregnancy also decreased R, Xc, and PA, and particularly during the third trimester, but PA values were not completely recovered even at 3 months post-partum. The authors suggest this pattern may have been related to the small stature, compromised nutritional status of the Bangladeshi women in addition to their experiencing first pregnancy at an early age; traits that are shared to some degree with the Aché population.

With respect to Aché growth and senescence patterns as indicated by BIA, it was not abundantly clear that the Aché exhibited either delayed maturation or greater senescence rates in comparison to other populations as might be expected according to data previously presented. Aché phase angle values fell in range with other population
reference values and the amount of decline across the lifespan was also comparable (e.g., Barbosa-Silva et al., 2005; Dittmar, 2003). Males did show more absolute decline in PA across the lifespan than females although some of this drop can be attributed to the younger Aché males having higher PA values.

Similar to the VEP data presented in the previous chapter, BIA data can also be highly variable although in this instance the variability is more readily understood and primarily concerns the hydration state of the subject. Although all efforts were made to achieve consistency in this factor as discussed in the methods section, there will arise some amount of variation around this factor that contributes to weakening the power of statistical tests. The data presented in this chapter are also once again limited by small sample sizes within the middle cohorts. However, given the inherent variation in BIA measurements in combination with the overall accurate depiction of the Aché population as a whole, lends support to the putative pattern within the male contact cohort despite the absence of statistically significant findings that, for the most part, did not emerge where p-values were evaluated as significant at 0.05 or lower. More will be said on this matter in the summary section of the concluding chapter (Chapter 7).

The general trend across the contact cohort was once again consistent with the direction and functional interpretation of the other measures already presented. Lower resistance (R) is associated with increased muscle mass (Thomasset, 1962), suggesting that the higher resistance recorded in the male contact cohort may be reflective of overall lower fat-free body mass (i.e., muscle) in comparison to adjacent cohorts. The difference between the 2nd and 3rd cohorts was significant in terms of R (T = 2.279, p = 0.049, 2-tailed). Considering the higher BMI values of the 2nd cohort, which were marginally
statistically higher than the 3rd cohort (T = 2.109, p = 0.061), we can deduce that the contact cohort was not only smaller, but had lower proportionate muscle mass in comparison to the 2nd cohort who did not experience the onset of take-off growth during one of the severe contact years. The 2nd cohort of males were taller, heavier and leaner in comparison to the contact cohort, who were more similar to the 4th cohort in terms of R and had similar BMI (from Chapter 2). This pattern suggests that the transition to reservation living added bulk to the younger, or 2nd cohort, but the additional pounds and calories were not expressed entirely as lean muscle mass; a result that may stem from the added nutritional inputs being derived from grain crops to the exclusion of wild foods, and possibly less physical activity related to the reduction in hunting and forest living during the developmental span of the younger cohorts (the 1st, or youngest, cohort had slightly lower R values than the 2nd cohort and their BMI values were slightly higher).

Evidence for a developmental perturbation occurring in the contact cohort comes from phase angle (PA), which was significantly lower in comparison to the 4th, or younger, cohort (T = 2.164, p = 0.048), but not in comparison to the 2nd, or older, cohort (T = 1.274, p = 0.235). The PA means of the 2nd and 4th cohort were, however, very similar indicating that the lower sample size in the 2nd cohort, in addition to higher variance, contributed to the non-significant finding in comparison to the contact cohort.

Although the biological significance of phase angle is not currently well understood (e.g., Barbosa-Silva and Barros, 2005), critical examination of the polyunsaturated fatty acids that make-up the interior of the phospholipid bi-layer has been linked to phase angle measurements in sickle cell disease (VanderJagt et al., 2002). This important work suggests that these lipids are involved in shaping the electrical
properties of cells, a view that is consistent with other work concerning the functional role of polyunsaturated fatty acids in neural and particularly visual function (Bloom et al., 1999). The connection between visual evoked potentials and phase angle may therefore have a tentative bridge in the form of essential fatty acids as these seem to play critical roles in both visual and cellular functions. These connections will be expanded on in the final chapter along with further discussion concerning the meaning of phase angle measurements.
VI. Variable Exploration

a. Background

This section explores potential relationships among the anthropomorphic, neurophysiological, and bioelectrical impedance variables. To this author’s knowledge, there are no studies that investigate bioelectrical impedance parameters in conjunction with visual evoked potentials. The relationship between bioelectrical impedance analysis (BIA) parameters and anthropomorphics is well known, particularly the influence of conductor length, or height, on resistance (R) and reactance (Xc). R is also influenced by fluid volume, while Xc is influenced by the capacitance of cell membranes, tissue interfaces, and non-ionic tissues (Foster and Lukaski, 1996), which correlate with body mass. Therefore, correlations among BIA parameters and body mass indices are to be expected and have been found repeatedly in addition to age and sex differences (e.g., Barbarosa-Silva et al., 2005; Dittmar, 2003).

Visual evoked potentials (VEP) have also been investigated in relation to anthropomorphics but primarily in terms of head size and head length, which in turn, tend to correlate with height and weight, and, by extension BMI. Sex differences in P100 latency tend to be small but consistent and are likely related to larger male head size or hormonal influences, which produce slightly longer P100 latencies for males (Allison et al., 1983; Celesia et al., 1987; Gregori et al., 2006). Age is a significant predictor of P100 latency, particularly during developmental years (Allison, et al., 1983), while evidence of a senescent trend will depend on parameters of the VEP test itself (reviewed in Chapter 4).
It was of interest therefore to examine whether significant relationships may exist among the anthropomorphic, neurophysiological, and bioelectrical impedance parameters collected in the current work, which have not been previously investigated in conjunction with one another. Of particular interest was to determine if there were correlations among phase angle (PA), P100 latency, and anthropomorphic measures such as height and weight, which might indicate some type of overall health relationship among these variables. To analyze these patterns, age and sex had to be taken into consideration in order to delineate whether these measures held a relationship within the Aché sample independent of age and sex effects, as described in the next section.

**Analytical Methods and Strategy**

In order to examine whether correlations existed among the variables collected in the previous chapters, it was necessary to control for age and sex because each variable exhibits a relationship with these two factors. Therefore, the analytical approach pursued here controlled for both age and sex by calculating residual values for each individual taken as the difference between each individual’s value and the LOWESS (locally weighted scatterplot smoothing) fit line within each sex and for each measure. In other words, separate LOWESS fit lines were generated for males and females and residuals were calculated from the sex-specific fit lines for each measure. Given the number of variables (10) and interrelated nature of some of the correlation tests, for example, head length will correlate with CNVC because it is in the denominator of the calculated CNCV value (CNCV = head length/P100), the bivariate correlational approach presented here provided expediency and transparency in the interpretation of the variable relations.
Residual values were computed in Minitab (Student Version 14) using the command line editor to create plot values of the LOWESS fit line for each subject’s age value. The LOWESS parameters in MINITAB were defined as fraction \( f = 0.5 \) and smoothing steps \( n = 2 \), where \( f \) and \( n \) are functionally equivalent to the bandwidth multiplier and kernel smoothing options for LOWESS available in SPSS.\(^7\) The LOWESS plot value was then subtracted from each subject’s actual measure to calculate the residual value for all subjects and variables explored below. The residual values were subsequently analyzed in SPSS (Version 15.0, 2006) using the bivariate correlation option. Note that in the interpretation of P100, the direction of the residual values was opposite of most other measures because higher, or positive, residual values are reflective of slower visual processing. For instance, if a subject had a short P100 latency, which is indicative of faster neural processing, they would fall below the LOWESS fit line to generate a negative residual, and this would be indicative of better functioning. To some extent the BIA measure of resistance can be interpreted the same way because higher resistance is associated with lower muscle mass, although it also interacts with fat deposition and total body water in terms of body composition.

The entire population sample was initially analyzed with bivariate correlations and then broken down by age and sex categories for further analysis. The age sample was split along adult and child categories, where child refers to all ages up until the cessation of growth as evidenced by the cross-sectional height and weight plots shown in Figures 2a and 2b, respectively, and discussed in the analytical methods and strategy section of Chapter 3. For females, growth was completed by roughly 17 years of age; therefore, the female child category included all females up to the 17th year, while the adult category

\(^7\) See footnote 3 in Chapter 3 for further details.
included females in their 18th year of age and older. For males, growth was completed by 22 years of age; therefore the male child category included all males up to the 22nd year, while the adult category included males in their 23rd year of age and older. The “child” category encompasses both childhood and adolescent growth phases. For simplicity, the label ‘child’ was used to signify the entire youth sample.

The entire adult and entire child samples were analyzed along with the entire female and male samples. An additional analysis examined all four age by sex categories: adult females and adult males, and female children and male children, respectively. Suggestive correlations among phase angle (PA) residuals and height and weight residuals were discovered among the child sample and the adult male sample. More detailed analyses of these correlations were therefore pursued and reported below.

c. Results

Bivariate Pearson Correlation coefficients with significance values and sample size (N) are provided in Table 8 for the entire sample and for all variables.
Nerve Conduction Velocity (CNCV), but that was to be expected given that P100 is in the denominator for the CNCV calculation (CNCV = head length/P100). Similarly, CNCV was significantly correlated with many of the anthropomorphic measurements but not with P100 or PA.

Correlations and significance values for the entire combined Aché sample can be found in Table 8. Note that the residual values were calculated from LOWESS fit lines specific to each sex to reflect the sex-specific nature of the variables being tested. The correlations that were of primary interest involve the P100 residual and the PA residual in combination with the various anthropomorphic measurements. Table 8 indicated that there weren’t any significant correlations of P100 or PA that did not involve a related measurement. For example, P100 was significantly inversely correlated with Central Nerve Conduction Velocity (CNCV), but that was to be expected given that P100 is in the denominator for the CNCV calculation (CNCV = head length/P100). Similarly, CNCV was significantly correlated with many of the anthropomorphic measurements but not with P100 or PA.
less so than head length; given that P100 was not significantly correlated with any anthropomorphic measurements, we can deduce that the correlation between CNCV and body size dimensions was driven by the correlation between head length and body size rather than P100.

Likewise, PA was significantly correlated with R and Xc, which was to be expected given that PA is derived from these measures; however, PA did not correlate with any other anthropomorphic measurement, while R and Xc were significantly correlated with the other anthropomorphic measurements. There was a significant correlation between Xc and CNCV; however, nearly the exact same correlation was found between Xc and head length, and no relationship between Xc and P100 latency, indicating that it was head length that drove this finding.

The absence of any relationship between P100 latency and either head length or circumference, suggests that P100 latency was minimally influenced by the physical size of the optical pathway vis-à-vis head size, although in terms of height, there was somewhat of a relationship between faster P100 latencies and shorter stature and, conversely, longer P100 latencies and taller stature (p = 0.107). For the entire population, PA does not appear to have any relationship to body size, although more will be said on this in a moment. There was also a strong, inverse correlation between Xc and body size for the entire sample consistent with smaller body size and higher Xc values. This pattern obtains in most of the correlation tests that follow below and will be addressed in the discussion section.
To examine if the residual variable relationships changed in subsets of the population, correlations were performed for the entire female and male sample separately (Table 9) and for the entire adult and child samples separately (Table 10).
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** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 9. Bivariate Pearson correlation coefficient matrix by sex for Aché females (top) and males (bottom). Coefficients, two-way significance values, and sample sizes (N) reported for each cell or correlation test. Individual residual values were calculated from LOWESS fit lines within each sex.
## Adults

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** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).
Table 10. Bivariate Pearson correlation coefficient matrix for Aché adults (top) and children (bottom). Coefficients, two-way significance values, and sample sizes (N) reported for each cell or correlation test. Individual residual values were calculated from LOWESS fit lines within each sex.

The expected correlational trends from Table 8 were repeated in the above analysis, although several slight or suggestive patterns emerged with respect to the other residual variables of interest. A relationship between faster P100 latency and increased height was more pronounced for the entire female sample (p = 0.074), whereas the entire male sample showed no relationship between P100 latency and height (p =0.397). Females also exhibited a slight relationship between increased height and lower PA (p = 0.082) that was not observed in the male sample (p = 0.775), and the correlation between female PA and P100 residuals was noticeably stronger (p = 0.145) than the correlation between male PA and P100 residuals (p = 0.681), although these results did not achieve statistical significance.

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**: Correlation is significant at the 0.01 level (2-tailed).
*: Correlation is significant at the 0.05 level (2-tailed).
Examination of the entire adult sample revealed only the suggestion of a slight inverse association between height residuals and P100 latency residuals (p = 0.123), where shorter stature was associated with longer P100 latencies and vice-versa. Among adults, height was more strongly related to P100 latency than either head length or head circumference. No evidence for a relationship between P100 latency residuals and height residuals emerged in the child sample. There was also no association found between PA residuals and height residuals in the combined adult sample, nor were there any noticeable patterns with respect to adult PA residuals for any other measure. However, the strongest pattern to emerge thus far concerned the correlations between child body size and PA residuals, where both residual height (p = 0.060) and residual weight (p = 0.061) were indicative of some relationship to PA such that smaller body sizes were correlated with larger PA values among male and female children. Recall the marginal association between smaller body size and higher PA values for the entire female sample as well; this pattern was explored in more detail below.

In sum, the relationship between P100 and height was stronger in women versus men and in adults versus children, although none of the correlational test achieved statistical significance. There was a stronger association between PA values and body size for the entire child-aged sample in comparison to the entire adult sample and this association was inversely proportional such that larger body size was correlated with lower PA values and smaller body size was correlated with higher PA values, a somewhat counterintuitive finding given that higher PA values are, ceteris paribus, expected to correlate with body mass. To further examine these patterns, the sample was
divided in terms of both age and sex for correlational analyses with the results shown in Tables 11 and 12.

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**. Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
### Table 11. Bivariate Pearson correlation coefficient matrix for adult females (top) and adult males (bottom). Coefficients, two-way significance values, and sample sizes (N) reported for each cell or correlation test. Individual residual values were calculated from LOWESS fit lines within each sex.

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<th>BMI Residual</th>
<th>Head Circ Residual</th>
<th>P100 Residual</th>
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<td><strong>Pearson Correlation</strong></td>
<td><strong>Sig. (2-tailed)</strong></td>
<td><strong>N</strong></td>
<td><strong>Pearson Correlation</strong></td>
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** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
### Female Children

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** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

### Male Children

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** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).

Table 12. Bivariate Pearson correlation coefficient matrix for female children (top) and male children (bottom). Coefficients, two-way significance values, and sample sizes (N)
reported for each cell or correlation test. Individual residual values were calculated from LOWESS fit lines within each sex.

From the previous analyses, the strongest correlation between P100 residuals and height residuals was expected to be observed among adult females; however, the correlation between P100 residuals and height residuals was slightly stronger in the female child sample than in the adult female sample, although none of these tests achieved statistical significance. In general, the smaller sample sizes in Tables 11 and 12 will contribute to lowering the statistical power of the correlation test to detect significance such that the correlation coefficients give us additional information about trends in the data.

Among children, the correlation between residual PA and residual body size (height and weight) was stronger in female children (p = 0.109 for height; p = 0.121 for weight) versus male children (p = 0.274 for height; p = 0.313 for weight) although the inverse relationship was consistent for both sexes and both measures.

Among adults, the only suggestive trend was between adult male PA residuals and body size (p = 0.124 for height; p = 0.122 for weight). However, in this case the direction of the trend was reversed from that of the child-aged sample, with larger adult male body size associated with higher PA values as might be expected given the positive association between PA and body size, all else being equal. Among adult females, the inverse correlation between PA residuals and height residuals remained in the same direction although the relationship was much weaker (p = 0.386), while the correlation between PA residuals and weight residuals virtually disappeared (p = 0.920).
Ad-Hoc Analysis of Phase Angle and Stature

The nearly significant inverse correlation between phase angle (PA) residuals and body size residuals (height and weight) among Aché children warranted further analysis given the variable nature of the human growth trajectory, which is marked by an extended period of slow, childhood growth followed by a shorter, more rapid period of adolescent growth. Also, in comparison with Western populations, the Aché exhibit different growth trajectories in terms of having slower childhood growth, a later onset of take-off growth spurt, lower peak-growth velocity, and extended growth into early adulthood (see Figures 1a and 1b). It was therefore of interest to examine whether the inverse correlation between PA residuals and body size residuals (height and weight) changed across the developmental age span, and if differences existed between the sexes or in terms of height and weight measurements. The adult male sample also revealed a mild association between PA residuals and body size, which was further explored below.

To probe for age and sex differences among the child-aged sample, the male and female samples were divided into childhood and adolescent growth periods separated by the age at which take-off growth initiated. From Figures 1a and 1b (Chapter 2), females initiated take-off growth at 10 years of age and males initiated take-off growth at 11 years of age. Figures 15a and 15b display scatterplots of PA residuals and height/weight residuals for each sex and age category.
Female

Height

Childhood

\[ R^2 = 0.4771 \]
\[ p = 0.128 \]
\[ N = 6 \]

Adolescence

\[ R^2 = 0.0123 \]
\[ p = 0.624 \]
\[ N = 22 \]

Weight

Childhood

\[ R^2 = 0.8249 \]
\[ p = 0.012^* \]
\[ N = 6 \]

Adolescence

\[ R^2 = 0.03 \]
\[ p = 0.438 \]
\[ N = 22 \]
Figures 15a – 15b. Scatterplots of phase angle (PA) residuals (y-axis) and height and weight residuals (x-axis). Plots are fit with linear regression lines with $R^2$, significance values, and sample size shown for each correlation. $N = $ sample size. (*) = Significant at the $p < 0.05$ level, (**) = Significant at the $p < 0.01$ level.

The strongest correlation between PA residuals and body size residuals occurred in female childhood, where weight residuals were more strongly correlated with PA residuals than were height residuals. Among male children, weight residuals were also more highly correlated with PA residuals than were height residuals, although neither correlation reached statistical significance. Height and weight residuals among the adolescent population showed no indication of being correlated with PA residuals for either females or males, although all of the trend lines were negative. Thus, weight and young age (childhood) emerged as more important than height or adolescence for being
associated with phase angle (PA), and this relationship was more pronounced in females versus males.

To probe further into the age dynamics of the potential relation between PA and anthropomorphic residuals, correlations were tested by truncating the age sample by one year from each end of the age spectrum. Table 13 presents PA residuals correlated with height and weight residuals with age truncated from eldest end of the age spectrum (increasingly younger sample), and with age truncated from the youngest end of the age spectrum (increasingly older sample).
Table 13a-b. Phase angle residuals correlated with height and weight residuals. Pearson correlation coefficients and two-way significance values for PA residuals (rows) correlated with and height and weight residuals (columns). 13a truncates the age sample from older to younger; 13b from younger to older. Note that the age range $> = 10$ was omitted from table 13b due to a lack of change in the sample size and test results. Individual residual values were calculated from LOWESS fit lines within each sex. N = sample size. (*) = Significant at the $p < 0.05$ level, (**) = Significant at the $p < 0.01$ level.
From Table 13a, the top left correlation among all children indicated that the overall relationship between PA residuals and height and weight residuals was stronger among females than males. Height and weight residuals were approximately equally correlated with PA residuals within each sex for the entire child age range, in that both the correlation coefficients and significance values were similar. The column to the right truncated the sample to all children less than or equal to 18 years of age (\(\leq 18\)). Because the adult female sample was demarcated at 18 years and older in accordance with the cessation of physical growth, there was no change in the sampling size among females, and hence, no change in the test results. For males, however, 7 participants were removed between the ages of 18 and 22, where age 23 was the lower cut off for the adult male sample, or the age at which physical growth ceased among males. The correlation between PA residuals and height and weight residuals for males did not appreciably change, although the significance values increased despite the smaller sample size, which indicated that the correlation became stronger with the removal of the oldest adolescent males. Removing another year of the age sample, such that only those 17 years of age or younger (\(\leq 17\)) were included in the correlational analysis, removed only one female and did not appreciably affect the relation between PA residuals and height or weight residuals among females.

Continuing to the right restricted the age sample to those 16 years of age or younger, which removed 5 children from the analysis (3 females and 2 males). Here the correlation between PA residuals and weight residuals became stronger in both sexes. The correlation coefficient increased in strength for females from -0.316 to -0.359 and in males from -0.180 to -0.250, and the significance values decreased despite there being
fewer subjects included in the analysis, which, in general, decreases the power of statistical tests. The correlational test for the combined sample achieved statistical significance at the $< 0.05$ level, ($p = 0.033$). There was no discernable change in the relation between PA residuals and height residuals for either sex in restricting the age sample to 16 years of age of younger.

Further truncation of the age range down to 14 and 13 years of age and younger, revealed a somewhat static relation between PA and weight residuals before increasing at the age of 12 and peaking at 10 years of age and younger for both sexes. Dropping the sample below the age of 9 and younger did not change the sample size and there were no significant correlations for the ages of 8 and younger for each sex or combined.

In combination with the previous analysis, it can be deduced that the strongest association between PA residuals and weight residuals was found among the very young and the effect remained strong up to the age of 12 before tapering off with increasing age. From Table 13b, restricting the age range to an increasingly older sample removed individuals that had the strongest relation between PA and weight residuals and did not return any significant values for either sex or the sexes combined.

The nearly significant correlation between PA and height residuals for the entire child sample ($p = 0.060$) did not show much variation across the age truncations in either direction although removing very young individuals up to the age of 8 years old increased the correlation to significance for the combined sample ($p = 0.021$) and the effect was approximately equal in each sex (from Table 13b). Truncating the age range down to 16 years of age and less revealed a combined $p$-value of 0.067 and coefficient of -0.282, which in comparison to the entire child sample $p$-value of 0.060 and coefficient of -0.239,
where there were 20 more subjects included in the test, indicated that the correlation between PA and height residuals was strongest between the ages of 8 and 15. Ad-hoc analysis of the age range 8-15 showed a p-value of 0.028 and coefficient of -0.394 with a combined 31 subjects. The effect was slightly stronger in males (-0.463, p = 0.095, N = 14) than females (-0.375, p = 0.138, N = 17).

Overall, PA residuals were more highly correlated with weight residuals than height residuals, and this effect was particularly strong at young ages and moreso in females versus males. The correlation between PA and height residuals was strongest in the 8-15 age range and was slightly stronger in males versus females.

The adult male sample showed some evidence of a positive association between PA residuals and body size residuals; from Table 11 the p-values for height and weight residuals were 0.124 and 0.122, respectively, and the correlation coefficients were also nearly identical at 0.230 for height, and 0.232 for weight. It was therefore of interest to examine whether this trend changed throughout the adult male lifespan.

Adjusting the sample to various age ranges revealed that the negative correlation between adult male PA residuals and height residuals remained until the mid-twenties age range and then became slightly positive for the remainder of the older, adult male age range although no age-range was statistically significant. The same relation obtained for male weight and PA residuals, and the positive correlation was stronger across the middle-age range where the strongest correlation occurred between the ages of 29 and 44 years (0.597, p = 0.011, N = 17). At older ages, the correlation direction reverted to the early life pattern such that higher PA residuals were once again associated with lower weight.
body mass, although none of the correlational tests of the various male age ranges showed statistically significant results.

d. Discussion

All 10 variables collected in this study were analyzed with bivariate correlational tests that controlled for age and sex by calculating residual values for each individual off of LOWESS lines fit for each variable and sex. The Aché sample was further tested along age and sex categories to reveal several suggestive patterns that extend beyond the expected variable correlations based on previous research. While the approach pursued here can be criticized for ad hoc adjustment of the sample to generate significant findings, given the exploratory and novel nature of examining the current variables in conjunction with one another, and particularly P100 latency and phase angle (PA), further probing of the tentative patterns that initially emerged in the analysis seemed warranted.

The strongest correlation among the variables of interest was found between child PA and body size ($p = 0.060$ for height; $p = 0.061$ for weight). However, the inverse relationship of this correlation was somewhat unanticipated based on previous research. For example, Nagano et al. (2000) found a positive association between PA and weight among a population of 71 well nourished children aged $3.36 \pm 3.12$ years ($R = 0.818$, $p < 0.001$), and that malnourished children had lower PA values than well nourished children. The age sample for Nagano’s study was considerably younger than the age range collected in the current study; however, analysis of the the 10 years and younger portion of the Aché population revealed a positive correlation between raw phase angle (PA) values and weight (12 males, 6 females, $R = 0.481$, $p = 0.043$). However, this analysis
and Nagano’s analysis do not control for the effect of age; using a partial correlation test that controls for age actually reverses the association between PA and weight for the Aché under 10 sample (R = -0.474, p = 0.055, N = 18) and the effect was stronger in females (R = -0.766, p = 0.131, N = 6) than in males (R = -0.344, p = 0.300, N = 12). Note that partial correlation analysis performs much the same work as the residual analysis already developed in this chapter and produced similar although statistically weaker results (compare the <= 10 column from Table 13a for PA and weight residuals).

A few studies have controlled for the effect of age across the lifespan in analyzing PA values (e.g., Baumgartner et al., 1988) or have controlled for age through age-matched samples to analyze another variable or health marker a (e.g., VanderJagt et al., 2003). However, no studies, to this author’s knowledge, have explicitly examined the relationship between PA and body mass in children across the developmental span while controlling for age. Although the results presented here were cross-sectional in nature and have lower explanatory power than a longitudinal design would, the apparent relationship between PA and weight for age may be of some significance. The inverse relationship between PA and body mass was stronger for weight than height, and stronger at younger ages before decreasing across the developmental span. The correlation was stronger in females than males and weight had an earlier and more significant relationship to PA than height. Whether these findings are particular to the Aché population versus other populations cannot be determined until further research in this area is conducted.

The relationship between higher PA and lower body weight for age may seem counter-intuitive given that higher PA values are generally indicative of better health.
status or condition (see background section of Chapter 5 for review of PA as a health status indicator). While studies have found positive associations between stature and health status (Leon et al., 1995; Peck and Vagero, 1989; Silventoinen et al., 1999), making this assumption among the Aché is problematic. As Walker and colleagues have demonstrated (2006b), there appears to be variation around the timing, rate and absolute growth of children in response to favorable or unfavorable ecological conditions. The later age of take-off growth and longer and slower growth velocity of the Aché in comparison to western populations is consistent with them enduring some degree of food shortage and this is also consistent with the author’s personal observations. Therefore, a strategy of staying small as a child for the Aché may be a more extreme representation of the general human pattern of slow childhood growth combined with an adolescent growth spurt, which is postulated to be an adaptation for offsetting energetic demands of multiple, closely spaced offspring that characterize the human reproductive career (Gurven and Walker, 2006). As such, the correlation of small body size and higher PA, presumably a marker of health status, would indicate that the healthiest Aché children are those who are small for their age. Or, if we assume that health status was relatively static among children and did not covary with PA, finding the opposite of the expected positive relationship between PA and body mass is a novel finding indicating that there is a connection between childhood growth effort and the PA measures.

Although the sample sizes for these bivariate correlational tests was limited, the strength of the correlation for children aged 10 years and younger (-0.674, p = 0.002, N = 18) cannot be in doubt as it actually exceeded the correlation between height and weight residuals (0.419, p = 0.052, N = 22) for this age range. Comparative data from other
populations would greatly assist for interpreting the inverse correlation between PA and weight-for-age observed among Aché children, which, based on the strength of the correlation between PA and weight residuals, was not a spurious result.

P100 latency residuals failed to correlate with either head length residuals or head circumference residuals within any subset of the Aché sample, indicating that P100 latency was minimally influenced by the physical size of the optical pathway vis-à-vis head size. In terms of height, there was somewhat of a relationship between faster P100 latencies and shorter stature among all females or, conversely, between slower P100 latencies and taller stature among all females (-0.207, p = 0.074, N = 75).

Although head circumference and head length residuals were significantly correlated with each other (p = 0.000 for all tests), they appear to relate differently to P100 values. A complete lack of a relationship was found between head circumference and P100 residuals among all children (0.000, p = 0.997, N = 72), compared to a somewhat stronger relationship among all adults (-0.105, p = 0.327, N = 90). Conversely, head length residuals were more correlated with P100 residuals among children (0.170, p = 0.154, N = 72) versus adults (-0.080, p = 0.452, N = 90). The strongest association was found in male children where P100 residuals were marginally correlated with head length (0.288, p = 0.075, N = 39) suggesting that head length was more important for affecting P100 latency during developmental years and that this factor became less important for determining P100 latency during adulthood. However, none of these trends reached statistical significance suggesting that other factors are involved for determining P100 latency at all ages.
The residuals of the BIA variables resistance (R) and reactance (Xc) were strongly and inversely correlated with body size residuals across much of the sample and particularly children. The inverse correlation between R and body size is interpretable in terms of total body water (TBW), which is a good conductor of electricity provided the water is an ionic solution, such that larger individuals contain more water and offer less resistance to the flow of electricity. Some researchers have identified extra-cellular water (ECW) as being more important than total body water, although ECW is highly correlated with TBW and FFM in healthy individuals at the 50kHz frequency, which was also used in the present study (Schoeller, 2000).

The strong, inverse correlation between Xc residuals and body size residuals across much of the sample is less readily interpretable. For height and weight residuals, the only non-significant correlations with Xc residuals were for height among adults of both sexes, and for height among female children. Weight residuals were strongly and inversely correlated with Xc residuals for the entire sample such that lower weight was associated with higher Xc. This is a noteworthy finding because Xc has been found to have a strong age effect such that it decreases across the adult life-span (Dittmar, 2003); a pattern also observed in the current data. However, the residual analyses conducted here controlled for age to reveal the inverse correlation between weight and Xc. To this author’s knowledge, there exist no other studies that have explicitly examined this phenomenon.

A major limitation of the current analyses lies in the variation in some of the measures and in the limitations in understanding what those measures indicate. The debate as to the meaning of the P100 and CNCV was discussed in Chapter 4, while the
limited understanding of what phase angle (PA) actually measures was covered in Chapter 5. To this sample we might now add reactance (Xc). Recall from Chapter 5 that Xc increases with the volume of cell membrane capacitance and only cell membranes offer capacitive reactance. Xc is therefore not affected by the quantity of body fat and is held to be more reflective of intracellular body water rather than extracellular body water (Dittmar, 2003). Further, Xc is believed to reflect the ability of cells to store energy such that lower Xc is consistent with a breakdown in cell membrane integrity and higher Xc is consistent with having healthier cells. Why females have higher Xc than males, a pattern that is found even at very young ages (Savino et al., 2004), remains unexplained on the basis of interpreting Xc to reflect cell membrane integrity or cell membrane volume unless we posit a sex difference in cell membrane integrity and/or higher cell membrane volume in females, both of which seem rather unlikely.

Other limitations of the current analyses involve the LOWESS parameters being arbitrarily set; adjusting these parameters to other values may change the patterns that emerged in the current analyses using the conventional settings. Further, there were gaps in the age sample that may bias the LOWESS fit line that was used to calculate individual residual values. As previously discussed, there existed limited numbers of Aché across the middle cohorts and also a gap that occurred when few births or successful offspring emerged during the contact experience. There were also few subjects at the much older and the much younger ends of the age range for both sexes, which will affect the LOWESS fit line and residual calculation at those ages.

As mentioned, the analytical approach can be criticized for adjusting the population into sample sizes for correlation tests in order to expose significant findings,
suggesting that a Bonferroni correction should be applied. However, the novel nature of examining P100 latency and phase angle (PA) in conjunction with each other and in relation to anthropomorphic measures, warranted the exploratory analysis performed here if for nothing else than to spur further research in this domain.

The data collected here strongly indicate that among the Aché, being smaller for one’s age was consistent with having higher Xc, and this pattern obtained at all ages and for both sexes. Although not as pronounced as Xc, phase angle (PA) was also consistent with this pattern in that it was found to be higher in individuals who were small for their age. This correlation was stronger in children versus adults, in females versus males, and in weight residuals versus height residuals. Further discussion of these results can be found in the following chapter.
VII. Summary and Conclusions

a. Summary

Overall, the results from this research indicate that, with respect to physical growth parameters, Aché males were more responsive to changing environmental conditions than Aché females. The secular increase in body size in making the transition from forest to settlement living among the Aché indicated that males out gained females for height (5.1% versus 2.4%) and weight (15.5% versus 13.7%), respectively. As discussed in the background chapter, the general pattern for hunter-gatherers making the transition to settled or agricultural living is for health indicators, such as stature, to decrease rather than increase (e.g., Larsen, 1995). However, the continual encroachment of civilization has more recently pushed extant foraging populations into less productive areas and it is this phenomenon that likely accounted for the nutritional and health improvements – at least as indicated by height and weight – being registered among the Aché in making the transition from forest to reservation, or settled, living. Thus, the initial environmental conditions of the forest period were reflected more obviously in Aché males via smaller adult body size; while the improved conditions of the reservation period were reflected by increased growth and larger body size in males relative to females.

This pattern was also generally consistent with the neuro- and physiological markers discussed shortly, although interpretation of these measures is more difficult than the anthropomorphic measures due to senescent patterns that obtain in the neuro- and physiological measures across adulthood. These measures corresponded more closely with the anthropomorphic measures in males, and particularly with respect to the
developmental perturbation across the middle male cohort discussed below; however, this should not be taken as evidence that males are more environmentally sensitive than females. Recall that there were very few surviving offspring born during the contact period, and presumably very few births in general, which is evidence that females adjusted their fertility schedules in response to the stress of the contact episode and is consistent with females responding to environmental cues in ways that most directly reflect their reproductive success, that is, fertility. That the neuro- and physiological variables were more consistent with growth parameters in males may be taken as evidence that these measures are more reflective of growth versus fertility indices and not as evidence that males are more environmentally sensitive than females. Rather, we expect that each sex will respond in accordance with physiological constraints that impact their relative fitness most directly; for males, physical growth and for females, fertility.

The other source of evidence that males have different developmental responses than females was found in the putative developmental perturbation that occurred among peri-pubertal Aché males, and, as suggested in this work, specifically those who initiated their adolescent growth spurt during one of the severe contact years (1971-1975). This cohort, referred to variously as the 3rd, middle, or contact cohort, evinced differences with respect to height, weight, head length and head circumference in comparison to either adjacent cohort. The adjacent cohorts experienced some amount of growth during the contact period, either during later adolescence (2nd cohort) or childhood (4th cohort). However, the defining characteristic of the 3rd cohort is that they initiated take-off growth during the severe contact period and this fact may explain the permanent character of
their growth perturbation versus the other cohorts, and is consistent with less catch-up growth occurring among adolescents who have their growth interrupted (Prader, 1978).

That Aché females did not show evidence of a similar pattern of growth across this age range may be construed as a weakness in this interpretation with two caveats: the female sample size across these cohorts was exceedingly small, and the effect that was shown was in the direction of increased growth as reflected by larger adult body size for all females who experienced some amount of growth during the contact period (2nd, 3rd, and 4th cohorts). This pattern, to the extent that it may exist, could have an adaptive interpretation as presented in the discussion section of Chapter 3 and discussed further below following a more in depth analysis of the proposed effect in males.

In agreement with the anthropomorphic measurements, measures of P100 latency, Central Nerve Conduction Velocity (CNCV), and phase angle (PA), were all consistent with the pattern of a permanent developmental perturbation being registered in the middle Aché male cohort. Figure 14 recapitulates the relevant variables for adult Aché males only.
Figure 16. LOWESS smoother lines for height, weight, P100, head length, CNCV and phase angle for Aché males. Smoother parameters: kernel = uniform, bandwidth multiplier = 0.50. Arrows are set at the average age for the third cohort of males ~ 42.5 years.
As Figure 16 shows, the middle male cohort—as indicated by the downward pointing arrows set to 42.5 years, or the average age of this cohort—showed a consistent negative pattern across these various measures. In contrast, females of this same cohort (not shown) did not show any evidence of growth impairments and appeared to have actually improved on most measures although, again, the sample sizes within these age ranges were too limited to draw firm conclusions in this respect. Note that patterns in BMI and head circumference were also consistent with this effect and might have been profitably added to Figure 16 and but were omitted due to redundancy.

The consistent pattern found for the male contact cohort across seemingly disparate measures was compelling in that the gross phenotypic measures (body size) were resonant with measures reflecting cellular health (PA) and visual function (P100, CNCV) within the contact cohort. Although no major trends emerged in the Aché population as a whole with respect to these measures—with the exception of Xc, PA and body size discussed below—these variables were consistent in direction and functional interpretation for the middle male cohort. While the statistical significance of the neuro- and physiological measures did not match the strength of the effect observed among the anthropomorphic variables, a number of factors warrant consideration.

As mentioned, the neuro- and physiological measures reflect senescent patterns across the adult lifespan and have higher inherent variation. Both of these effects decrease statistical power and contributed to the non-significant findings within these measures as defined by p < 0.05. However, there is debate and a growing consensus that arbitrarily setting significance p < 0.05 is not an ideal method for reaching appropriate conclusions because it implies specific critical effect sizes and the relative costs of Type I
and II errors (Mudge et al., 2012). The current practice of setting $p < 0.05$ creates a bias against accepting Type I error, or accepting a falsehood, and increases the bias against Type II error, or rejecting a truth. Convention, institutional inertia, and an appeal to “healthy skepticism” maintain the toleration for this bias, which assumes different costs for each type of error (Shrader-Frechette, 1991). There are, in fact, many instances where the failure to detect a real effect can be considered as important and serious as falsely detecting a non-existent effect (Mudge et al., 2012). For the current study, the limited and unavoidably small sample sizes contained within the middle cohorts increased the probability of Type II error, or failure to detect a real effect, where statistical significance was evaluated at $p < 0.05$.

For illustrative purposes, Figure 17 reproduces an original figure from Mudge et al., (2012) describing the optimal alpha, or p-value, that minimizes Type I and Type II error for an independent sample T-test for varying sample sizes.
Figure 17. Determination of optimal \( \alpha \) from the a priori combined probabilities of Type I and Type II error. \( \alpha \) and \( \omega \) (the average of Type I and Type II error) for independent, 2-tailed, 2-sample t-tests (\( n_1 = n_2 \)). Data are for 3 (dotted line), 10 (solid line), and 30 (double line) samples per group, with critical effect sizes of 1 SD of either group. Drop lines indicate the minimum average of Type I and Type II error and its associated value of \( \alpha \). (Reprinted with author’s permission; the only deviation is “Figure 17” reads “Figure 2” in the original publication).

Although post hoc adjustment of test parameters is strongly frowned upon in statistical analyses, for reference purposes recall that the comparisons between the 2\(^{nd}\) and 3\(^{rd}\) cohorts involved 11 subjects for VEP and BIA T-tests\(^8\), and 12 total subjects were involved for the anthropomorphic T-tests. From Figure 17, the appropriate p-value, or \( \alpha \), would fall roughly in the middle of the dotted line (6 total subjects) and solid line (20 total subjects), at approximately \( p = 0.25 \). For phase angle (PA), the mean difference between the 2\(^{nd}\) and 3\(^{rd}\) cohorts happened to be one standard deviation in terms of the 3\(^{rd}\) cohort (\( x = 6.97, \text{ S.D.} = 0.51 \)), compared to the 2\(^{nd}\) cohort that did have higher variation (\( x = 7.58, \text{ S.D.} = 1.12 \)). The 2-tailed significance test for this comparison was \( p = 0.235 \),

\(^8\) Note that independent T-tests were not explicitly performed for the neuro- and physiological measures because of senescent trends in these measures that need to be controlled.
which would fall below the p-value from above and would be consistent with there being a significant difference between these cohorts. In combination with the already significant mean difference—as defined by $p < 0.05$—that existed between the 3rd and 4th cohorts ($T = 2.164, p = 0.048, N = 16$), we can reasonably assume that the 3rd cohort was statistically different than either adjacent cohort in terms of PA.

With the exception of the comparison between the 3rd and 4th cohort for Central Nerve Conduction Velocity (CNCV), which was statistically significant at the $p < 0.05$ level ($T = 2.249, p = 0.041, N = 16$), the mean differences for the VEP measures of P100 and CNCV were less than one standard deviation for the comparisons among the 2nd, 3rd, and 4th cohorts and none were statistically significant as defined by $p < 0.05$. The difference between the 2nd and 3rd cohorts for CNCV was much weaker ($T = 0.667, p = 0.521, N = 11$) than the difference between the 3rd and 4th cohorts. Whereas, for P100, the difference between the 3rd and 4th cohorts was stronger ($T = 0.959, p = 0.354, N = 16$) than the difference between the the 2nd and 3rd cohorts ($T = 0.600, p = 0.563, N = 11$). P-values of this magnitude (0.3 – 0.5) are typically interpreted as evidence for a lack of an effect; however, the precautions just cited with respect to minimizing Type II error, or rejecting a truth, should be taken into consideration when dealing with such small sample sizes.

Overly large sample sizes will also increase Type I error, or accepting a falsehood such that, for mean comparisons, a minor difference between the groups will emerge as a statistically significant difference at the $p < 0.05$ level when the tests are conducted between very large samples. Ideally, studies should be designed a priori and take into account the cost of Type I and Type II errors and expected effect sizes in order to
determine the optimal sample size with which to detect a mean difference and minimize errors (Mudge, et al., 2012), although this is not always possible in practice.

For the current analyses, the effect of small sample sizes can be further evaluated by artificially increasing the sample sizes for the cohort comparisons as applied to the neuro- and physiological variables, while maintaining the same means and standard deviations within each cohort in order to achieve statistical significance as defined by $p < 0.05$. Comparing the three middle cohorts (2nd, 3rd, and 4th) with independent sample T-tests revealed that, for P100, an average sample size of 48 would have produced significant findings at $p < 0.05$ for the 2 comparisons; for phase angle (PA), an average sample size of 36 for the 2nd and 3rd cohort comparison; and for CNCV, an average sample size of 35 would have produced a significant finding for the comparison between the 2nd and 3rd cohorts. Recall that the comparison between the 3rd and 4th cohorts was marginally significant for CNCV using the original sample sizes ($T = 2.249, p = 0.041, N = 16$), which, in consideration of the above concerns about the appropriate value at which to evaluate significance, makes this finding more compelling.

The strength of evidence for the physical growth parameters, which indicated a permanent developmental effect was registered occur among the 3rd male cohort, bears on the subsequent interpretation, probability, and extent to which the neuro- and physiological markers also reflect this pattern within the contact cohort and for the general pattern across the middle cohorts. With respect to the proposed developmental effect on the male contact cohort, a hierarchy of effects may be formulated with respect to the variables used in this study in that physical growth appeared most susceptible to
the effects of stress, cellular health as indicated by phase angle was less susceptible, and visual ontogeny as indicated by VEP was minimally susceptible.

The fact that the differences observed in the anthropomorphic variables were highly significant at the p < 0.05 level in spite of the limited sample sizes, supports the argument that the pattern of developmental perturbation in the 3rd cohort was markedly strong in terms of physical growth, the ability to conduct catch-up growth, and the attainment of final adult body size. The developmental effect was less strong but still substantial in terms of phase angle (PA), where we can conclude that there was better evidence for a difference between the contact cohort and the adjacent cohorts, than there was for there not being a difference between the contact cohort and the adjacent cohorts. Further, that the developmental perturbation as registered by PA was less strong than the effect among the growth parameters.

Finally, we can conclude that the P100 measure was minimally affected by the contact experience on the 3rd cohort of males; a finding that sheds light on the susceptibility of the visual system to the effects of severe physical and and psychosocial stress. Although cognitive and neural systems are susceptible to stress throughout adolescence (Gunnar and Quevedo, 2007), the relatively early maturation of the VEP waveform in children (Brecelj, 2003) could account for the apparent immunity found in this measure with respect to the proposed effects of contact on the 3rd male cohort. More permanent effects to P100 latency might be sought among those Aché who were in postnatal stages during the time of contact; however, there were virtually no births among the Aché during this period. Alternately, any deficits in visual ontogeny may be overcome
more effectively than deficits in physical ontogeny as they relate to the male contact cohort.

In general, the overall consistent agreement among the anthropomorphic and neuro- and physiological variables in terms of direction and functional interpretation, lends strength to the argument that a developmental perturbation was, in fact, registered among the male contact cohort and that it was reflected to various degrees by each measurement. Additional evidence from another study of Aché physiology is given below.

**Walker and Hill (2003) – Growth and Senescence in Physical Performance Among the Aché of Eastern Paraguay**

Additional evidence for a developmental perturbation among the middle male cohort can be gleaned from the analysis of Walker and Hill (2003), who investigated physical strength and endurance measures among the Aché in order to interpret growth and senescence trends. As it were, five of the seven males in the middle cohort were included in Walker and Hill’s analysis. Four of these males were aged at 37 years old and an additional male at 39 years of age in their sample, which reflected data that collected about four years prior to the collection of the current data. Cursory examination of several of their graphs was suggestive for being consistent with a developmental perturbation among these males, particularly grip strength and, most revealing, an estimate of VO2 max, which was defined as the rate of oxygen intake during maximal aerobic exercise and is considered a valid measure of the functional capacity to perform work (see Walker and Hill, 2003, for methodological details). Figure 15 displays the original Figure for VO2 max on the left along with a graph of only adult males on the
right. Individuals that fall on the 37 year old reference line plus the one circled indicate those of the middle cohort that were included in the current population sample.

Figure 15. VO2 max values for Aché males and females from Walker and Hill (2003) shown to left. VO2 max values for adult Aché males shown to right. Reference line set at 37 years old and indicates four subjects from current study; fifth representative is circled and is 39 years old. Smoother parameters for Figure to the right are: kernel = normal, bandwidth multiplier = 1.00.

In both Figures, we see again the negative indentation across the middle male cohort, consistent with all the other measures previously discussed. Since VO2 max is calculated per unit body weight, a correlation between lung size and body size can be ruled out as the driving factor behind the pattern observed in this case. Rather, it would appear that here lies further physiological evidence of a permanent developmental perturbation being registered in the male contact cohort.

The picture of the Aché above and in the current analysis was drawn from data collected during the more recent reservation period. Interpreting what effects, if any, of the contact episode on growth trajectories and life history variables is difficult because the majority of data collected on the Aché took place after the contact period, excepting
post-hoc interviews that shed light on events in the forest and contact periods (Hill and Hurtado, 1996). However, in light of the extremely stressful and high-mortality conditions of the contact period, we might expect that there could have been short-term adjustments in growth rates in response to the contact experience. Potentially, this could account for the apparent pattern of larger adult body sizes among females that experienced their peri-pubescent phases during the severe contact years—in that they may have accelerated their growth rate in the face of extreme extrinsic mortality. The adaptive interpretation being that this adjustment would allow them to produce offspring before succumbing to the effects of contact.

It is well known that females increase reproductive rates following high mortality episodes such as wars, which we recognize as post-war baby booms (Glass, 1968). However, an increase in female growth—to the extent that it represents an increase in reproductive effort—in the actual face of ecological and psychical stress would represent a novel finding that is somewhat counterintuitive in light of other studies that show stress has the broad effect of decreasing reproductive effort through suppression of the hypothalamic-pituitary-ovarian axis (Vrekoussis et al., 2010). Again, the limited sample sizes among the female cohorts across the contact experience preclude drawing any firm conclusions on this matter.

**Bivariate Correlational Analysis of All Variables**

The bivariate correlational analyses of all variables controlled for age by computing residual values from LOWESS fit lines within each sex and revealed several noteworthy patterns although a direct correlation between the primary variables of interest—P100
and phase angle—did not emerge among the population as a whole. P100 residuals were only slightly and inversely correlated with height residuals among the entire sample (\(-0.127, \ p = 0.107, \ N = 162\)), and this effect was stronger for females (\(-0.207, \ p = 0.074, \ N = 75\)) versus males (\(-0.068, \ p = 0.531, \ N = 87\)) and stronger in female children (\(-0.263, \ p = 0.139, \ N = 33\)) versus female adults (\(-0.194, \ p = 0.219, \ N = 42\)).

More pronounced correlations were observed between body size and BIA variables. An inverse correlation occurred between phase angle (PA) and body size among children that was stronger for weight versus height, stronger for females versus males, and stronger at younger versus older childhood ages. Adults evinced little relationship between PA and body size, save for a correlation that emerged among adult males in the age range of 29 to 44 where PA residuals and weight residuals were significantly and positively correlated. Adult males in total had a modest and similar correlation for both height and weight residuals, whereas females showed little to no relationship in these measures.

The strongest correlation occurred between reactance (Xc) and body size, where a significant and inverse correlation obtained across the entire sample for weight and Xc residuals, such that being small for one’s age was correlated with higher Xc and, conversely, being heavier for one’s age was correlated with lower Xc. Height residuals held the same relationship with PA residuals for much of the sample as well but were less correlated than were weight residuals.

Interpreting these correlations remains problematical given the uncertainty as to what phase angle is measuring (Barbosa-Silva and Barros, 2005a) and, as might be suggested here, the equally difficult to interpret Xc measure. Consistent sex differences in
Xc and PA were found among the Aché and were consistent with a bevy of other studies (e.g., Dittmar, 2003) in terms of females having higher Xc at all ages, higher resistance (R) at all ages, and lower PA at all ages in comparison to males. A reexamination of Table 6 from Chapter 5, which shows data for the adult sample, reveals that females had higher Xc and R than males for all cohorts, but the sex difference was greater for R than Xc for the most part, which, in turn, generated the higher PA values for males versus females among the Aché.

The VEP and BIA variables did not appreciably correlate among the Aché although CNCV did show significant correlations with Xc for several subgroupings of the population. However, these correlations can be traced to the head length measure that was used in the calculation of CNCV rather than the P100 latency (CNCV = Head Length/P100) by comparing the stronger correlations of head length and Xc with the minimal correlation between P100 with Xc. Within the 3rd cohort, however, the VEP and BIA variables showed consistency with one another and particularly in relation to the anthropomorphic variables.

To this author’s knowledge, there is no basis for – and no previous findings of – correlations among stature, P100 latency and phase angle (PA). A potential connection between visual evoked potentials (VEP) and PA has been mentioned with respect to the role of essential fatty acids, which are critical for cellular membrane function, affect PA measurements (VanderJagt et al., 2002), and play key roles in visual function (Bloom et al., 1999). However, the connection between essential fatty acids and a permanent, adolescent growth disruption is not obvious.
There exists a wealth of data into the beneficial effects of long-chain essential fatty acids (EFA) for peri-natal brain growth and development (e.g., Koletzko et al., 2007) and females have been shown to have higher rates of long-chain EFA synthesis from shorter-chain EFA precursors than males (Burdge and Wootten, 2002). Given the critical role of long-chain EFA in cell membranes, and particularly brain and visual systems (Bloom et al., 1999; Uauy et al., 2001), this pattern could contribute to females having better cell membrane integrity, as reflected by higher Xc, than males. Although the effects of long-chain EFA have been primarily assigned to cognitive and visual roles, their role in improving cell membrane function and integrity appears to be global and is largely a function of dietary supply (Seo et al., 2006).

The role of long-chain EFA on perinatal physical growth is equivocal with some studies finding decreases in infant growth, and particularly among males, with increased access to long-chain EFA (Ryan et al., 1999). However, there is no obvious causal mechanism or explanation that would link physical growth deficits during adolescence with the neuro- and physiological measures examined here that show similar deficits within the male contact cohort. The lack of an obvious causal mechanism suggested that perhaps some type of gestalt effect arising from the tremendous psychosocial stress of the contact period (Hill and Hurtado, 1996) might have been involved. The poorly understood nature of PA (e.g., Barbosa-Silva and Barros, 2005a) in conjunction with its extreme accuracy as a health indicator would seem to warrant further attention.

The next section briefly summarizes the underappreciated role of bioelectricity in the structure and function of living organisms. In the course of seeking explanations that could potentially account for the current findings, and particularly with respect to the
apparent developmental perturbation observed in the middle male cohort, it became obvious that organisms are more electrically constituted and governed than is generally recognized by orthodox physiological models. Exploration of how and why this state of affairs emerged is beyond the scope of the current work. However, if the cell, organism and, indeed, all of life, are governed by electrical and magnetic forces at a fundamental level, we may yet arrive at a mechanism that could potentially account for the general agreement among the seemingly disparate measures observed in this research.

**Bioelectricity**

As discussed in Chapter 5, Foster and Lukaski (1996) point out that, in addition to the dielectric properties of the outer cell membrane, other factors will influence the PA measurement such as the size of cells and the conductivity of the extracellular and intracellular media; for example, the dielectric properties of intracellular mitochondrial membranes. The deeper question however is not how various bodily tissues interact with an outside generated source of electricity, as is the case with bioelectric impedance analysis (BIA) for example, but rather to what extent might the entire cellular apparatus be governed by electrical or energetic forces?

The underlying basis of bioelectric impedance analysis is admittedly not well understood (Barbosa-Silva and Barros, 2005a). In an effort to flesh out this area of knowledge, evidence emerged that cells contain micro-channels for the conduction of electrical current at the level of the photon. The work of Dr. Fritz-Albert Popp confirmed the existence of biophotons that transmit information within and between cells. Though originally discovered and described by Russian embryologist Alexander Gurwitsch in the 1920s, this work lay largely unappreciated until Popp reconfirmed his findings in the
1970s. A representative abstract of this work is given below, from Popp et al., Biophoton Emission: Experimental Background and Theoretical Approaches (1994):

Biophoton emission is a general phenomenon of living systems. It concerns a weak photon radiation from a few to some hundred photons per second, per square centimeter surface area, at least within the spectral region from 200 to 800 nm. The results indicate that biophoton emission can be assigned to a coherent field within living organisms, its functions being intra and intercellular regulation and communication.

Biophotons primarily emanate from and are reabsorbed by DNA in the nucleus before then being re-emitted (Popp et al., 1984). Popp’s work provides compelling evidence that electrical and subtle energetic forces are intimately involved in the workings of the organism.

Further evidence for the profound role of electricity in organisms can be found in the work of University of Michigan researchers Katherine Tyner, Raoul Kopelman, and Martin A. Philbert, who reported the discovery of tremendously powerful (15 million volts), albeit very low amperage, electric fields in the cytosol of cells (2007). These measurements were made possible by the use of nano-sized voltmeters that use light energy rather than electrons to measure electric fields. Previously, cytosol was not believed to hold any electric potential. The authors note that the:

“…resulting [electric] fields have profound effects on a variety of nonmembranous functions…the distances that such [electric] fields extend beyond the associated membrane and the range of their influence remain largely unmeasured and unknown. As a result, there are today wide gaps in the descriptions of cellular [electric] field profiles, profiles that could greatly enhance current knowledge of cellular organization and signaling.” (Tyner et al., 2007: p. 1163)
Thus, bioelectrical forces appear to be intimately involved, if not primal, to the functioning of cells and other life processes. The effects of bioelectricity on cellular regulation suggest a universal mechanism that could potentially integrate the findings observed across the seemingly disparate measures of this research, namely physical growth, visual evoked potentials (VEP), and phase angle (PA).

b. Conclusions

The current study provides a glimpse into a well controlled, natural experiment concerning the effects of an acute and global stressor as applied to an entire population, and the effects of transitioning from a traditional lifestyle to a more settled existence that occurred among the Aché across the contact period during the mid-1970s. By definition, it is not possible to manipulate the study design of natural experiments a priori or to repeat the same experiment exactly. It is therefore important to glean as much information as possible from natural experiments and, prima facie, this will tend to result in the violation of preferred research protocols that would otherwise be implemented in the more desirous a priori study design. For example, it would not have been possible to specify a particular sample or effect size with which to determine if there were a significant effect of the contact experience on a particular male cohort because the sample sizes were, for one, predetermined, and two, there was no previous knowledge that such a specific effect may have existed; rather, this was borne out by post hoc exploration of the data. Given the constraints and opportunities that natural experiments provide by design, the analytical approach pursued here seems warranted and of value for increasing our understanding of human ontogeny as it relates to ecology at the
diachronous scale, and in terms of the synchronous effect that contact visited on the Aché population at large.

Efforts to characterize the effects of stress on childhood growth, and/or to determine the nature and extent of potential sex differences in response to stressors, have been complicated by the existence of numerous confounding variables that tend to obtain in these types of studies including, but not limited to, the timing of the environmental insult, the duration and type of stressor, the effects on various body tissues, and the variety of methods employed across studies (Stinson, 1985). Examination of the Aché contact experience is valuable because the duration of the stressor was consistently applied to the entire population and thereby increases our knowledge about the impacts of a virgin soil epidemic within a traditional population; as the encroachment of civilization continues to eliminate traditional forms of subsistence across the globe, opportunities to investigate such occurrences will grow increasingly rare.

This study also adds further physiological and neurological detail to an already very well studied traditional society. It could be reasonably argued, in fact, that the Aché represent the most intensively studied foraging population in the anthropological domain, having been the focus of ethnographic and biocultural researchers for over 30 years (Hill and Hurtado, 1996; Hill et al., 2011). As such, the data collected and presented in the current work may have relevance for future investigators interested in examining the same or similar parameters in this or another traditional society. The neuro- and physiological information presented here also adds detail to human variation in response to ecological conditions in two dimensions; in the transition from forest to settled living,
and in the response to the extreme physical and psychosocial stressor that obtained in the form of the virgin soil contact episode.

The timing of the data collection was opportune because the age distribution of the extant Aché population provided sufficient numbers of subjects to flesh out the secular trends and patterns that obtained across the various cohorts. If the data presented here were collected 10 years prior, it would have been more difficult to assess secular trends because there would have been a much smaller group of adults who were born after the contact period who had completed growth at the time of data collection. Similarly, if the data presented here were collected 10 years in the future, the number of surviving Aché that completed growth before the contact period would likely be too small for analysis.

As such, the present study sheds light on sex differences in response to the environment across a number of parameters and suggests patterns of ontogenetic adjustment in relation to the environment. Recent advances in life history theory indicate that ontogenetic strategies are more subtle than previously appreciated. Whereas poor environmental conditions were typically understood to delay growth, post-pone reproduction in females and lead to overall smaller adult body sizes, new evidence suggests that other factors, and particularly high mortality, can accelerate growth and lead to earlier reproduction in combination with smaller adult body sizes (see Ellis et al., 2009 for review; Walker et al., 2006b). Out of 22 small-scale, hunter-gatherer societies investigated by Walker and colleagues (2006b), the Aché fell roughly in the middle in terms of adult body size and childhood growth velocity. They also did not deviate substantially from the regression line fit to all 22 societies for these two variables—
contrast to some African groups that have very fast growth rates combined with very small adult body sizes (Walker et al., 2006b).

The data provided by this study enhances our understanding of Aché ontogeny in that the slower, more extended periods of childhood growth, as marked by body size dimensions, and in comparison to Western populations, were not well reflected by the neuro- and physiological variables. Other counterintuitive findings were suggested by the data as well, including that women who were exposed to contact during any part of their adolescent growth period were as large or larger than women who were born after contact. The 4th female cohort, or those who experienced at least 2 years of adolescent growth during contact but had already reached take-off growth, were, in fact, taller and heavier than any other cohort, although, again the sample size was limited to 3 individuals so conclusions cannot be certain. That males of this same cohort were also taller and heavier than expected, and that it was only males in the 3rd cohort who showed evidence of a permanent developmental perturbation, or those who experienced the onset of the adolescent growth spurt during contact, is suggestive that there was a specific effect of stress on the onset of male take-off growth.

Additionally, the larger body size (height and weight) of the male and female cohorts that preceded the contact cohort, and the larger size of the 3rd, or contact, female cohort suggests there may have been an adaptive growth response to the contact experience. Interpreting larger body size resulting from increased growth rates in response to extreme environmental duress would represent a novel finding; alternatively, to the extent that a mortality bias may have operated against smaller individuals across

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9 The P100 measure did show signs of delayed maturity and increased senescence although there are caveats for comparing VEP data across laboratories, and for the use of various test parameters, which affect the interpretation of ontogenetic trends in visual maturation; see Chapter 4 discussion for further details.
the contact episode, would have had the effect of culling smaller males from the 4th cohort, thereby producing the relatively larger body sizes observed in the 4th cohort. Accordingly, this mortality bias against smaller individuals suggests that even smaller males were removed from the contact cohort. The current study provides evidence that the sexes may have differential growth responses to stress which interact with the age of take-off growth, also a potentially novel finding.

With respect to the putative developmental perturbation among the contact cohort, physical growth, VEP, and BIA parameters suggested a hierarchy of effects where permanent deficits were registered most significantly for the anthropomorphic variables, less so for the BIA variables and specifically phase angle (PA), and were only minimally reflected by the VEP variables. Why this hierarchy emerged is open to speculation; however, the combination of anthropomorphic with the neuro- and physiological variables examined in this study, represents a unique contribution to the field of human ecology. The patterns that emerged in the current Aché data point the way towards future investigations for unraveling several counter-intuitive correlations, for instance, between phase angle and small-for-age body size among children; between reactance (Xc) and small-for-age body size among the entire population; and the suggested pattern of a sex difference in peri-adolescent growth responses to stress, which interacts with age at take-off growth.

Although it was unavoidable given the size of the study population, it is a valid point that the sample sizes used to evaluate the presence of a permanent developmental perturbation may be too limited to draw satisfactory conclusions. Or, if one finds the evidence for the developmental perturbation convincing, they might argue that initiating
take-off growth during contact was not responsible for the effect and that some other variable may explain the pattern. However, it is still of interest that the seemingly disparate variables collected in this study moved together within the contact cohort. A sortie into the nascent and enigmatic field of bioelectricity was briefly pursued in an attempt to integrate the variables and findings among the contact cohort of Aché males.

We may then finally and tentatively arrive at a possible explanation for the pattern observed within the male contact cohort, that is, the extreme psychosocial and environmental stressors of the contact period may have in effect exhausted the electrical potential of the Aché population as a whole and, for those males who were on the cusp of initiating their growth spurt, this may have had the effect of permanently disrupting their growth spurt. The strong inverse correlation between reactance (Xc) and body-size for age, and/or the weaker but still present inverse correlation between phase angle (PA) and body-size for age among children, suggests that potential growth and exhibited growth may be related. That is, the less an individual had grown, their electrical capacitance was accordingly higher. Conversely, more growth for age was reflected by lower electrical capacitance. Although the exact mechanism of the growth spurt is not known, a model that incorporates bioelectricity suggests that the trauma of the contact experience produced an energetic drain which, in addition to impacting all somatic cells among the population as a whole, would have been most deleterious for those who had built up the largest energetic stores, that is, peri-adolescent males on the cusp of initiating the growth spurt. We might therefore expect this particular group to be most susceptible to the effects of having this energy siphoned off unexpectedly, which thereby produced the permanent developmental deficits that were observed in this cohort.
In aggregate, the evidence presented in this research indicates that Aché males and females had differential responses to environmental conditions as evidenced by body size measurements across the forest, contact, and reservation periods. And that peri-pubescent males and females responded differently to environmental perturbations that occurred across the initiation of the adolescent growth spurt.
VIII. Bibliography


