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Mens Life History, Testosterone, and Health

Louis Alvarado

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MEN’S LIFE HISTORY, TESTOSTERONE, AND HEALTH

By

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DISSERTATION

Submitted in Partial Fulfillment of the Requirements for the Degree of

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Dedication

For Ma, Jess, and Gea
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ABSTRACT

Testosterone is hypothesized to mediate life history trade-offs between reproduction and survival in men, promoting mating effort over other forms of investment, which entails energetic and mortality costs. Sexually dimorphic musculature represents one form of somatic investment in mating. Favorable energy availability is posited to promote preferential investment in mating effort through upregulated testosterone production and augmented musculature, whereas nutritional constraint is predicted to downregulate testosterone to facilitate a diminished, thrifter phenotype. Furthermore, life history trajectories influencing men’s testosterone levels have important health implications for androgen-sensitive disease. Here, I examine broad features of men’s life history and health, and their association with testosterone.

Men’s reproductive ecology is characterized by distinctive features—sexual division of labor, decreased testosterone, and male provisioning—that may render a fixed relationship between testosterone and muscularity maladaptive. I collected demographic, life history, and anthropometric data from rural Polish men (at the Mogielica Human Ecology Study Site) to examine how variability in men’s testosterone levels interacts with marital and parental
status, workload, and musculature. Fatherhood jointly predicted decreased testosterone but increased workload, and positively predicted muscle mass and strength measures.

Next, longitudinal data were collected from the same community to examine seasonal fluctuation in men’s testosterone, workload, and anthropometry. Men had intensified work demands and decreased testosterone during the summer harvest, but also showed concomitant increase in arm circumference, chest and grip strength. Taken together, these data suggest the importance of provisioning and subsistence activities in determining skeletal muscle phenotype.

And lastly, androgenic hormones regulates growth and maintenance of the prostate gland. Although animal, clinical, and in vitro studies suggest that elevated testosterone increases prostate cancer risk, epidemiological investigations comparing testosterone levels of cancer cases with controls generally report an equivocal relationship. However, because testosterone levels are highest and most variable during early adulthood, I conducted a meta-analysis of studies reporting testosterone levels for population samples of younger men, in relation to prostate cancer incidence for the larger sampled populations. A positive association emerged between population differences in young men’s testosterone levels and prostate cancer disparities among older men, suggesting that testosterone exposure influences prostate cancer risk.
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Chapter 1: Introduction

Background

Evolutionary fitness can be defined as an organism’s ability to harvest energy from the environment to convert into viable offspring. However, energy within the environment is finite, and because energy allocated toward one area is spent and no longer available to be invested in another, selection favors strategic energy allocation across competing body systems in order maximize an organism’s reproductive success. Accordingly, energy allocations divided between growth, maintenance of soma, and reproduction (Gadgil and Bossert 1970; Kaplan et al., 2000; Stearns 1989). Mammals begin life dividing energy investment between growth and somatic maintenance. Reproduction is delayed until growth is completed, after which, the primary trade-off shifts between reproduction and maintenance (Hill 1993; Kaplan et al. 2000). Investment in reproduction can be further divided into parenting or mating effort, and in mammals, females support the energetic cost of reproduction in the form of internal gestation and lactation. Thus, females provide the greater minimum obligatory investment in offspring and invest preferentially in parenting effort. In contrast, males are able to preferentially invest in either parental or mating effort (Trivers, 1972), though direct paternal investment is rare among mammalian species (Clutton-Brock and Parker 1992). Spermatogenesis is energetically cheap and relatively insensitive to nutritional constraint (reviewed in Bribiescas 2001). In contrast, behavioral and secondary sexual characteristics are costly for males in terms of decreased increased energy expenditure, somatic maintenance, and mortality risks (Bribiescas 2001; Ketterson and Nolan 1992; Promislow et al., 1992; Redpath et al. 2006; Wilson and Daly 1985). Because crucial features of men’s reproductive biology are dependent upon testosterone (e.g., initiating
sexual differentiation, maintaining sexual function, and promoting the expression of secondary sexual characteristics (Bribiescas 2001; Krause 2006)), testosterone is thought to occupy a critical role in modulating male life history and reproductive strategies (e.g., Bribescas, 2001; Gray et al., 2002; Ketterson and Nolan 1992; Wingfield et al. 1990).

**Men’s Life History, Testosterone, Parenting, and Work**

Cross-cultural differences in energy availability have clear consequences on population variation in testosterone levels, such that well-nourished Westernized men exhibit significantly higher testosterone levels than men from subsistence groups (Bribiescas, 2001; Ellison et al., 1989). Bribiescas (1996, 2001) proposed that men living at subsistence level are subjected to chronic nutritional constraint, and are incapable of meeting the energetic demands of physiological processes associated with elevated testosterone. According to this perspective, males face a trade-off between somatic maintenance and reproduction that is modulated through testosterone’s management of sexually dimorphic muscle mass. The idea that diminution and augmentation of men’s musculature is mediated through this specific pathway remains an interesting but tentative hypothesis, because numerous studies have failed to substantiate that natural variation in men’s testosterone levels exerts somatic consequences on muscle mass (Ellison and Panter-Brick 1996; Campbell et al. 2003; Gettler et al. 2010). Other studies report only weak or indirect relationships (Campbell et al. 2007; Ellison and Panter-Brick 1996; Gettler et al. 2010; Lukas et al., 2004). Interestingly, testosterone and musculature appear to show stronger correlations in non-human primate males than in men, though data are somewhat sparse (reviewed in Alvarado et al., 2015). Chapters Two and Three develop a hypothesis to explain this divergence between humans and non-human primates.
Men’s Life History, Testosterone, and Prostate Cancer Risk

An accumulation of evidence from animal models, clinical research, and in vitro studies suggests that increased testosterone exposure is associated with prostate cancer risk (reviewed in Gronberg, 2003; Henderson et al. 1982; Hsing et al. 2008; Smith et al. 1994). Results of epidemiological investigations, however, have remained largely consistent (Roddam et al. 2008). An extensive meta-analysis of 18 prospective studies, which examined hormone levels in relation to prostate cancer incidence, reported a null association between men’s testosterone levels and prostate cancer risk. This influential study led many clinicians and researchers to conclude that no association exists between endogenous testosterone and prostate cancer (e.g., Carpenter et al., 2008; Morgentaler, 2008). However, men’s testosterone levels are highest and most variable during early adulthood (Ellison et al., 2002; Kehinde et al., 2006; reviewed in Alvarado, 2013). If cumulative exposure to testosterone across the lifespan determines prostate cancer risk, then age range which is routinely sampled in prospective case-control studies would provide little information regarding lifetime exposure (Alvarado, 2010, 2011). In contrast to previous meta-analyses (e.g., Roddam et al., 2008; Shaneyfelt et al., 2000), Chapter Four examines population variation in young men’s testosterone levels in relation to prostate cancer risk among older men. A novel meta-analytical design was developed to standardize, organize, and examine studies reporting testosterone levels for population samples of young men, in relation to prostate cancer rates from the large populations in which samples were drawn.
**Research Design**

The unifying theme connecting these chapters is interaction between testosterone and men’s life history and health. Chapters 2 and 3 rely on field observation, while Chapter 4 consists of an epidemiological analysis of existing literature.

**Description of study population, recruitment, and data collection.** Field observations were conducted at the Mogielica Human Ecology Study Site in Slopnice, Poland. Slopnice is a large village with a population of 6,198 (Statistical Office of Krakow, 2013), and is located in rural Sothern Poland within the Western Carpathian Mountains. Participants were healthy adult males, and were recruited through advertisements on community bulletins boards and referral sampling. Participants provided life history and demographic information including age, marital and parental status, occupational history, work schedules, and physicality ratings of labor. In addition, anthropometry (arm circumference, body fat percentage, height, and weight), physical performance measures (chest and grip strength), and morning and evening saliva samples were collected. Saliva samples were assayed for testosterone levels at the Hominoid Reproductive Ecology Laboratory, University of New Mexico. Study protocol is explained in further detail in Chapters 2 and 3.

**Description of literature search strategy and meta-analysis.** A comprehensive literature search was completed by using PubMed to identify relevant articles. Only study samples from larger populations with available incidences of prostate cancer were included in the analysis. Also, only study samples with a mean or median age of 39 years or less were included in the analysis, in order to evaluate the relationship between young men’s
testosterone levels and prostate cancer risk in older men. Data standardization and analysis is
described in Chapter 4.

Guide to Dissertation

This dissertation follows a hybrid format, in which Chapters 2, 3, and 4 represent
three separate manuscripts. Chapter 2 develops a novel perspective of men’s life history, the
Paternal Provisioning Hypothesis. Cross-sectional data were collected from 122 rural Polish
men to tests associations between: marital and parental status, testosterone, workload, and
upper-body musculature. This chapter was published in the American Journal of Physical
Anthropology (Alvarado et al., 2015). In Chapter 3, longitudinal seasonal data were collected
from the same rural population in order to conduct additional tests of the Paternal
Provisioning Hypothesis at the within-individual level. Chapter 3 examines seasonal
fluctuation in men’s testosterone, workload, and muscle mass. This chapter is currently in
preparation for manuscript submission. In Chapter 4, a meta-analysis of the existing literature
on men’s testosterone levels and prostate cancer incidence. This chapter was published in the
American Journal of Human Biology (Alvarado, 2010). Chapter 5 contextualizes and
summarizes key findings.
Literature Cited


Chapter 2: The Paternal Provisioning Hypothesis: Effects of Workload and Testosterone Production on Men’s Musculature

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Abstract

Objectives: Testosterone supports male reproduction through a broad range of behavioral and physiological effects, including the maintenance of sexually dimorphic muscle used in male-male competition. Although it is often assumed that a persistent relationship exists between men’s testosterone production and musculature, most studies either fail to find evidence for such a relationship, or document very weak associations. In non-human primates, by contrast, correlations between testosterone and muscle mass are higher. Here, we propose the ‘Paternal Provisioning Hypothesis,’ which predicts that men’s skeletal muscle is less dependent on the effects of androgens than that of other primates, and more sensitive to the physical demands of men’s work. This permits human fathers to downregulate testosterone, which has negative impacts on pair-bonding and parenting effort, but without sacrificing the strength and musculature necessary to provision mates and offspring.

Methods: We tested predictions of the Paternal Provisioning Hypothesis by assessing parental status, salivary testosterone levels, anthropometry, and strength among 122 men (ages 18-78) at the Mogielica Human Ecology Study Site in rural Poland. We chose this population because men practice subsistence agriculture, regularly engaging in physically demanding labor. Grip and chest strength were assessed using a dynamometer, and upper-body musculature was estimated from arm muscle circumference.

Results: In this population, testosterone showed no association with measures of strength and musculature, and was lower in older men and pair-bonded fathers. Marital and parental status and workload, by contrast, were positive predictors of muscle mass and strength measures.
**Discussion:** These findings offer support for the Paternal Provisioning Hypothesis.

The steroid hormone testosterone supports male reproduction through a broad range of morphological, physiological, and behavioral effects (Ketterson and Nolan, 1992, 1999; Krause, 2006; Wingfield et al., 1990). Accumulating evidence suggests that testosterone’s primary function is to promote male mating effort (Ketterson and Nolan, 1992), with implications for two fundamental life history trade-offs: that between current and future reproduction, and that between quantity and quality of offspring (Trivers, 1972, Hill 1993). Across vertebrates, elevated testosterone is associated with investment in male-male competition and mate-seeking behaviors (Bribiescas, 2001; Creel et al., 1997; Fox, 1983; Ketterson and Nolan, 1992; Wingfield et al., 1990). Consequently, in a range of species, testosterone reduces both paternal care and provisioning (i.e. investment in offspring quality: Wingfield et al. 1990, Smale et al., 2005; Lynn, 2008). Testosterone can also increase mortality from extrinsic and intrinsic causes, adversely affecting future reproduction (Ketterson and Nolan, 1992; Kruger and Nesse, 2006; Marler and Moore, 1988; Redpath et al., 2006; Wilson and Daly, 1985).

Sexually dimorphic traits - both ornaments and armaments - are supported by testosterone in many vertebrates (e.g. Bókony et al., 2008; Whiting et al., 2003; Lincoln, 1971, 1992). Such traits can increase mating success, but also incur energetic costs, and sometimes decrease survival (Promislow, 1992, Promislow et al., 1992). In humans, muscle mass and strength are highly sexually dimorphic, and skeletal muscle hypertrophy is thought to have reproductive benefits via male-male competition and female choice (Dixson, 2009). However, muscle tissue is also metabolically active and energetically expensive, accounting for approximately 20% of men’s basal metabolic rate (Bribiescas, 2001). Accordingly,
testosterone has been hypothesized to directly mediate competing energy allocations between reproduction and survival in men, through the management of sexually dimorphic muscle mass (Bribiescas, 1996, 2001; Bribiescas et al., 2012).

This ‘Somatic Allocation’ model has been used to explain both inter- and intra-population variation in men’s testosterone levels (Bribiescas, 1996, 2001; Bentley et al., 1993; Campbell et al., 2003, 2007; Ellison and Panter-Brick 1996; Gettler et al., 2010). The model hypothesizes that increased energy availability promotes elevated testosterone, which supports muscular hypertrophy. Nutritional constraint, on the other hand, downregulates testosterone, producing a diminished, thriftier phenotype (Bribiescas, 1996, 2001).

Consistent with this hypothesis, men’s testosterone levels fall on an urbanization gradient across populations (Bribiescas, 2001; Gray et al., 2006). Well-nourished, Westernized men exhibit the highest levels of testosterone, and the largest body sizes, while men involved in subsistence food production generally have suboptimal nutrition, reduced testosterone levels, and shorter stature (e.g., Bentley et al., 1993; Bribiescas 1996, 2001; Gray et al., 2006; Ellison et al., 1989; Stini, 1979). Nutritional constraints appear to have a similar effect on chimpanzees, as males living in the wild maintain smaller body sizes and lower levels of urinary testosterone than their well-fed, captive counterparts (Muller and Wrangham, 2005). The model has been less successful in predicting intra-population variation in testosterone, particularly among subsistence groups, in which men show little testosterone response to changes in food availability (e.g., Bentley et al., 1993; Ellison and Panter-Brick 1996; for chimpanzees see Muller & Wrangham 2005).

Implicit in the Somatic Allocation Hypothesis is an assumption that a direct, persistent link is maintained between testosterone and men’s musculature, and that any
effects of energy availability on muscle mass are mediated by testosterone. Evidence for this assumption, however, is weak. Whether variation in men’s testosterone levels predicts variation in musculature, across populations, has rarely been examined. One exception is a study of Ariaal pastoralists, comparing salivary testosterone and anthropometry among sub-populations of settled and nomadic men (Campbell et al., 2006). Interestingly, settled men had higher morning and evening testosterone than nomads. Fat free mass was not appreciably different between the two groups, however, suggesting that inter-population variation in men’s testosterone did not produce differential expression of skeletal muscle phenotype. Within populations, some studies report relationships between measures of testosterone and muscle mass, but also find that testosterone predicts little of the variance in men’s musculature (e.g., Campbell et al., 2007; Lassek and Gaulin, 2009; Lukas et al., 2004). Men’s testosterone levels predicted only three percent of the variance in fat free mass within a large cohort of American men (Lassek and Gaulin, 2011; N = 1,048), and in a study of Zimbabwean men, testosterone levels accounted for less than one percent of the variance in fat free mass (Lukas et al., 2004; N = 109).

Other large- and small-scale studies have generally failed, within populations, to find any direct relationship between men’s testosterone levels and their strength or musculature (Kenyan Ariaal and Turkana men, Nepalese Taman and Kami men, and Filipino men: Campbell et al. 2003, 2006, 2006a; Ellison and Panter-Brick 1996; Gettler et al. 2010). By contrast, although data are somewhat sparse in non-human primates, measures of testosterone and muscle mass appear to be correlated in chimpanzee males (Emery Thompson et al., 2012), baboons males (Muehlenbein et al., 2001), and in male pig-tailed and rhesus macaques (Muehlenbein et al., 2002). In relatively small cross-sectional samples of captive
baboons (N = 21), pig-tailed macaques (N = 41) and rhesus macaques (N = 53), moderate correlations were found between serum testosterone and upper-arm circumference (muscle mass estimate) after accounting for age (Muehlenbein et al., 2001, 2002). Despite small samples, testosterone levels explained 18% of the variance in baboon arm circumference, and 15% and 9% of the variance, respectively, in pig-tailed and rhesus macaque arm circumference. In wild chimpanzees, Emery Thompson and colleagues (2012) examined longitudinal data consisting of 60 accumulated observation-years for males in the community. Mean annual urinary testosterone in males showed a strong correlation with a measure of muscle mass from urinary creatinine, with testosterone explaining 35% of the variance. Why is a relationship between testosterone and musculature apparently so much easier to detect in non-human primate males than in men?

Although it makes intuitive sense that investment in sexually dimorphic muscle should be mediated by testosterone, specific features of human life history call the logic of this hypothesis into question. Distinct from most mammals, humans exhibit a sexual division of labor, with male provisioning of mates and offspring (Kaplan et al., 2000; Lancaster and Lancaster, 1983; Wood and Marlowe, 2013). In foraging societies, men specialize in hunting large game, a difficult, dangerous activity that requires strength and endurance (Apicella, 2014; Murdock and Provost, 1973; Lancaster and Kaplan, 2009; Gurven and Hill, 2009). In traditional, preindustrial, and industrial societies, a clear trend emerges in which men’s physical work, on average, involves greater reliance on strength tasks, particularly episodic bursts of upper-body strength and force (Murdock and Provost, 1973; Wood and Eagly, 2002). This trend is manifested more prominently among societies, such as foragers and agriculturalists, in which a pronounced sexual division of labor is associated with greater
reliance on strength tasks in male work roles (Ibid.). Although substantial variability in men’s parenting effort appears within the cross cultural record (Gray and Anderson, 2010), evidence from a number of studies shows that men increase their workload according to the number and age of their dependents (Hooper, 2011; Knoester and Eggebeen, 2006; Lee et al., 2007; Marlowe, 2003; Quinlan, 2000; Wood and Marlowe, 2013), which, in an evolutionary context, suggests that fathers in particular need to maintain physical strength and musculature to support their provisioning efforts.

At the same time, considerable evidence suggests that testosterone has a negative impact on male parenting effort and pair bonding in humans, just as it does in many other species (reviewed in Gettler, 2014). For example, in various studies men with elevated testosterone were found to be less responsive and less sympathetic to infant cries (Fleming et al., 2002), less interested in infants or infant stimuli (Roney et al. 2006, Storey et al. 2000; Weisman et al., 2014), less involved with families and parenting (Alvergne et al., 2009; Mascaro et al., 2013), less committed to their current partner (Caldwell Hooper et al., 2011), and more interested in extra-pair mating (McIntyre et al., 2006). And numerous cross-cultural studies have reported that investing fathers maintain lower testosterone levels than single childless men, including among American, Canadian, Chinese, Filipino, Hadza, Jamaican, Senegalese, and Swiss men (Alvergne et al., 2009; Fleming et al., 2002; Gettler et al., 2011; Gray et al., 2002, 2006, 2007; Kuzawa et al., 2009; Muller et al., 2009; Perini et al., 2012; Storey et al., 2000). Furthermore, more pronounced diurnal declines in testosterone have been observed in relation to indices of paternal involvement, providing further evidence for a suppressive effect of male parenting on testosterone production (Gettler et al., 2012; Muller et al., 2009). Lower testosterone levels during fatherhood have been interpreted as a
physiological shift toward investment in a current partner and shared offspring, and away from male status competition and mate-seeking behavior (Gettler et al., 2011; Gray et al., 2002; Kuzawa et al., 2009; Muller et al., 2009). Accordingly, in societies where men have little direct involvement with offspring, or maintain non-exclusive pair bonds, marriage and fatherhood may not be associated with reduced testosterone (Gray, 2003; Muller et al., 2009).

The Somatic Allocation model posits that testosterone determines physiological investment in mating effort by increasing muscle mass, and thus, effectiveness in reproductive competition (Bribiescas, 1996, 2001; Bribiescas et al., 2012). The model assumes, however, that men’s activity patterns remain constant (Bribiescas, 1996), whereas recent evidence indicates that men’s work demands change substantially across the lifespan, and particularly with fatherhood (Hooper, 2011; Marlowe, 2003; Wood and Marlowe, 2013). This suggests an important puzzle: if men’s muscle mass is predominantly supported by testosterone, which declines sharply during fatherhood, then wouldn’t men’s physical capabilities be compromised at the precise time when their provisioning responsibilities intensify?

We propose the Paternal Provisioning Hypothesis, which predicts that the lack of evidence for a clear relationship between men’s testosterone levels and their musculature reflects a real phenomenon in which men’s skeletal muscle is less dependent on the effects of androgens than that of other primates. This permits men’s musculature and strength to be augmented during fatherhood, despite suppressed testosterone levels, through the physical demands of increased provisioning. We tested predictions of this ‘Paternal Provisioning’ hypothesis in the Mogielica Human Ecology Study Site in southern Poland. We chose this
population because men often practice subsistence agriculture, and regularly engage in physically demanding labor.

Mutually exclusive predictions can be derived from the Paternal Provisioning and Somatic Allocation Hypotheses. For the Paternal Provisioning Hypothesis to be tenable, the following predictions must be supported. Pair-bonded, involved fathers should show lower testosterone levels than childless men (P1). Fathers should have increased workloads compared to childless men (P2). Despite lower testosterone levels, however, fatherhood and intensified workload will positively affect both musculature (P3) and strength (P4). In contrast, the Somatic Allocation Hypothesis proposes that testosterone is the primary driver of somatic allocation toward musculature. According to this competing perspective, higher testosterone levels should have a positive impact on muscle mass (CP1) and strength (CP2). Furthermore, if men experience a reduction in testosterone levels during fatherhood, then pair-bonded fathers should show decreased musculature (CP3) and strength (CP4).

Methods

Study site and participants. In summer 2011, research was conducted at the Mogielica Human Ecology Study Site, in the rural village of Slopnice, southern Poland. Slopnice sprawls across 56.74 square kilometers of high mountain terrain and has a total population of 6,198 (Statistical Office of Krakow, 2013). Seasonal and labor-intensive work has been documented in villages from this mountain region (Jasienska, 2013; Jasienska and Ellison, 2004). Mean body mass index of the sample was 26.56 (S.D. = 3.76), not unlike that of other Western populations (e.g., Sassi, 2010), which indicates that food availability was not limited, and is consistent with previous research conducted in this region (Jasienska, 2013; Jasienska and Ellison, 2004). And although the populace is rapidly transitioning to a
Westernized lifestyle, it retains several characteristics that distinguish it from more developed regions of Europe, such as greater reliance on subsistence agriculture, physically-demanding labor that is concentrated during the summer harvest, and higher completed fertility (Colleran, 2013; Jasienska, 2013; Jasienska and Ellison, 2004).

One hundred and twenty-two men participated in this study. Participants were healthy adult males residing in Slopnice, whose ages ranged from 18-78 years (mean: 38.93 years, S.D. = 16.97 years). In this sample, men’s primary occupations included: 36% employed in building trades (e.g., bricklayer, carpenter, construction worker, framing), 17% worked as physical laborers in a variety of occupations (e.g., butcher, plumber, metal worker, mechanic, etc.), 15% were retired, 15% were white-collar workers (e.g., accountant, shop keep, soldier, supervisor, etc.), eight percent were farmers, and eight percent were students. Many participants practiced subsistence agriculture as a secondary occupation, particularly among retirees and students, such that 54% of the sample farmed as either a primary or secondary occupation. Table 1 provides further descriptive statistics for the study sample.

**Data collection.** Men were recruited by advertisements posted on community bulletin boards and by referral sampling. We visited households using structured interviews to collect demographic, physical activity, and work data from men. Participants provided information regarding their age, marital and parental status, children’s ages, and occupational status. Participants provided further information regarding work patterns: their profession, hours worked in a week, and physicality of their work. Participants were asked about additional sources of physical exertion, such as sports participation, weightlifting, or other exercise, as well as injuries or illness that interfered with their work.
After the interview, height, weight, body fat percentage, and flexed arm circumference were collected. Body fat percentage was estimated from triceps skinfold using standard procedures (Donoghue, 2009). Following Sell and colleagues (2009), upper-body muscle mass was estimated using flexed arm circumference, which was measured at the widest point of the upper-arm with biceps maximally contracted. We adapted this method, because Sell et al. did not account for participants’ body fat percentage, which also contributes to arm circumference. Accordingly, we use arm muscle circumference [upper-arm circumference - (triceps skinfold * 3.14)] of participants’ flexed biceps for a more precise measure of muscularity (McWhirter and Pennington, 1994). Upper-body strength was estimated using a portable dynamometer to measure grip and chest strength. For grip strength, the participant’s elbow was flexed at 90° with the forearm in neutral position, and participants used their dominant hand to squeeze the dynamometer with maximum effort (Mathiowetz et al., 1985). For chest strength, participants used both hands to grasp the dynamometer at the sternum and pressed hands together with maximum effort (Sell et al., 2009). Each strength test was repeated in triplicate, with the mean used for statistical analyses.

Circulating testosterone exhibits a diurnal rhythm in which secretion peaks in the morning and declines steadily throughout the day, until reaching an evening nadir (van Cauter, 1990). To account for diurnal fluctuation, participants provided morning and evening saliva samples. We also calculated proportional diurnal decline for morning to evening testosterone levels (AM T/PM T), such that higher positive values indicate more precipitous proportional decline. Morning samples were collected immediately after waking, and evening samples shortly before bedtime. Participants provided 4 ml of saliva via passive drool into a
collection tube. Salivary testosterone levels correlate well with serum free testosterone, the fraction of circulating hormone available to target tissues (Goncharov et al., 2006; Wang et al., 1981). Participants were asked to refrain from eating, drinking alcoholic or caffeinated beverages, brushing teeth, engaging in sexual activity, and smoking for at least thirty minutes prior to providing samples, because these activities can influence salivary hormone measurements (Salimetrics, 2013).

Some participants were unable to complete chest strength or grip strength tests due to existing injuries (N = 4 and N = 2, respectively), such as shoulder problems or missing fingers. One participant did not provide morning and evening saliva samples. This study was approved by the University of New Mexico Human Research Review Committee, and participants provided written informed consent.

**Statistical analysis.** Before evaluating predictions, we constructed a correlation matrix of independent and dependent variables to assess bivariate relationships. Multiple regression analysis was used to evaluate predictions. Regression models employed a backward elimination procedure in which all independent variables were entered into the regression equation and were sequentially removed based on their partial correlation with the dependent variable. Age was correlated with several behavioral, demographic, and physiological traits examined in this study (Table 2), and was included as a predictor variable in all regression models. Because energetic factors can potentially affect testosterone levels (Ellison et al., 1989; Pritchard et al., 1998), as well as development of skeletal muscle tissue (Stini, 1979), body fat percentage was used as an indication of energy status in regression models predicting testosterone levels, muscle mass and strength.
Some of the questionnaire data employed categorical responses (e.g., marital and paternal status, physicality of workload, and medical conditions that interfere with work). These responses were dichotomized and converted into binary variables. For purposes of this analysis, marital and parental status were combined into a single binary variable (single men and childless men = 0, pair-bonded father =1). This partitioning is justified, because although reductions in men’s testosterone levels have been reported in the contexts of both pair bonds and parenting (Alvergne et al., 2009; Burnham et al. 2003, Gettler et al., 2011; Gray et al., 2002, 2006, 2007a; Muller et al., 2009), the combination of pair bonding and fatherhood appears to produce the most suppressive effects on testosterone production (Kuzawa et al., 2009). Accordingly, inclusion in the pair-bonded father group was limited to men who were jointly fathers and married. A small portion of our sample consisted of newlyweds who were nulliparous (N = 4), and widowers who were fathers but not currently partnered (N = 6; two of the widowers’ had a youngest child who was an adolescent, whereas the other widowers had adult children all older than 30 years of age). Married men without children as well as widowers were assigned to the “single men and childless men” group, while all other participants in this grouping were neither married nor had children. Two men in the sample were unwed fathers. One had had no interaction with his child or the mother of his child for nearly a decade, and he was assigned to the single and childless group. The other resided with his partner and newborn infant, provided childcare, and was saving money for a wedding. This participant was assigned to the “pair-bonded father” group. Taken together, marital and parental status, age, and body fat percentage were used to predict variation in men’s morning and evening testosterone levels, as well as variation in diurnal testosterone decline (P1).
Participants reported number of days and hours normally worked in a week during the summer season when the survey was administered. Men’s workload was examined as a function of marital and parental status (P2). Because injuries interfering with work were fairly common in this community (Table 1), participants’ injuries were included in regression models predicting workload (no injuries interfering with work = 0, injuries interfering with work = 1). Total number of work hours was regressed on marital and parental status, age, and injuries. As a subsequent test of men’s commitment to family provisioning, a similar regression analysis was conducted in which total work hours were regressed on age, injuries, and total number of children (instead of the binary variable representing marital and parental status).

Although most of the study population participated in physically demanding work, we were particularly interested in the effects of manual labor on phenotype. Accordingly, another measure of workload was created, which consisted of men’s time spent in heavy manual labor. Participants provided ratings of the physicality of their work: sedentary, light, fairly heavy, or very heavy. Fairly and very heavy work hours were summed for a more direct assessment of physical work, and used as a predictor variable of muscle mass and strength measures. Many participants helped friends and family with their farm work during the summer harvest, but work contributed toward others’ farms was sporadic and much less predictable than participants’ own work schedule. To accommodate for weekly variability in participants’ work toward others’ farms, which was considerable, whether or not participants helped friends or family with farm work was converted to a binary variable (do not contribute work to others’ farms = 0, contribute work to others’ farms = 1). Finally, information about weightlifting and sporting activities was collected, which was also
converted into a binary variable (no sports/weightlifting involvement = 0, sports/weightlifting involvement = 1). Because our analysis concentrated on upper-body strength, we limited inclusion of sporting activities to those involving more rigorous upper-body exercise. By this criterion, the most common forms of recreational physical activity in this sample were: weightlifting 6%, volleyball 6%, and swimming 5%. However, a broader definition of sporting activities, which also included activities with less direct upper-body involvement, such as soccer 25%, running 4%, and bicycling 4%, had a negligible impact on subsequent analyses.

Taken together, age, body fat percentage, injuries interfering with work, marital and parental status, morning or evening testosterone levels, heavy work hours, weightlifting/sports participation, and work contributed to others’ farms were used to predict arm muscle circumference (P3), chest strength, and grip strength (P4), in separate regression models. Morning and evening testosterone were entered individually into models.

Predictions derived from the competing hypotheses described earlier can be evaluated in regression analyses of muscle mass and strength measures. If the Somatic Allocation Hypothesis is accurate, men’s testosterone levels are expected to positively predict arm muscle circumference (CP1), and both chest and grip strength (CP2), while fatherhood is expected to be negatively associated with these variables (CP3,4). All non-binary variables were cube root transformed before analysis to better adhere to the assumptions of parametric statistics.

**Hormone analysis.** Saliva was collected via passive drool in polypropylene tubes and frozen within eight hours of collection. Samples were analyzed for testosterone levels using an established enzyme immunoassay protocol (Salimetrics, State College, PA; Kit No. 1-
at the Hominoid Reproductive Ecology Laboratory, University of New Mexico. The assay manufacturers report a correlation of saliva and serum total testosterone of 0.96 and a limit of detection of ~ 1 pg/ml. Frozen samples were thawed, vortexed, and centrifuged for 15 min prior to dispensing into the assay in order to break up and precipitate mucins. Inter-assay coefficients of variation (CVs) were 5.1% for high and 9.9% for low salivary control. Intra-assay CV for duplicate determinations averaged 5.7%.

Results

Table 2 presents a correlation matrix to illustrate bivariate relationships between the analyzed variables. Regression analysis was used to examine the effect of relationship and fertility status on men’s testosterone levels (P1). As predicted, pair-bonded fathers exhibited lower evening testosterone as well as more pronounced diurnal decline in testosterone, although marital and parental status did not predict lower morning testosterone. Regressing morning testosterone levels on age, body fat percentage, and marital and parental status produced a significant model, adj. $R^2 = 0.21$, $F(1, 120) = 31.96$, $p < 0.001$. However, body fat percentage and marital and parental status were non-significant, while age negatively predicted morning testosterone levels ($p < 0.001$). For evening testosterone levels, body fat percentage was not a significant predictor, and was eliminated from the model. Age and marital and parental status were negative predictors of evening testosterone ($p < 0.001$ and $p = 0.022$, respectively), resulting in a significant model, adj. $R^2 = 0.23$, $F(2, 120) = 18.53$, $p < 0.001$. Greater diurnal decline in testosterone levels was predicted by marital and parental status ($p = 0.001$), resulting in a significant model, adj. $R^2 = 0.08$, $F(1, 120) = 10.76$, $p = 0.001$. Neither age nor body fat percentage were significant predictors in this model (See Table 3.)
To examine the effects of men’s workload (P2), participants’ total work hours were regressed on age, injuries interfering with work, and marital and parental status. As predicted, pair-bonded fathers worked more than single and childless men. This generated a significant model, adj. $R^2 = 0.08$, $F(2, 121) = 6.44$, $p = 0.002$, in which total work hours were negatively predicted by age ($p = 0.039$) and positively predicted by marital and parental status ($p < 0.001$). Injuries interfering with work were eliminated as a variable. Participants’ total work hours was then regressed on age, injuries interfering with work, and number of children. This model was also significant, but less robust, adj. $R^2 = 0.06$, $F(2, 121) = 4.60$, $p = 0.012$. Total work hours was negatively predicted by age ($p = 0.016$) and positively predicted by number of children ($p = 0.003$). Injuries interfering with work were again eliminated. See Table 4 for analyses of men’s workload.

Finally, we examined muscle mass and strength measures (P3, 4 and CP1-4). As predicted, marriage and fatherhood and workload had substantive effects on muscularity and strength. We regressed arm muscle circumference on: age, body fat percentage, heavy work hours, injuries interfering with work, marital and parental status, morning or evening testosterone levels, sports and weight lifting involvement, and work on others’ farms. The overall model was significant, adj. $R^2 = 0.32$, $F(4, 120) = 15.32$, $p < 0.001$. Arm muscle circumference was negatively predicted by age ($p < 0.001$) and injuries interfering with work ($p = 0.008$), and positively predicted by heavy work hours ($p < 0.001$), and marital and parental status ($p = 0.009$). All other predictors were non-significant. Chest and grip strength were regressed on the same predictor variables, which produced significant models; chest strength: adj. $R^2 = 0.42$, $F(5, 116) = 18.03$, $p < 0.001$; and grip strength: adj. $R^2 = 0.40$, $F(5, 118) = 16.59$, $p < 0.001$. Chest strength was negatively predicted by age ($p < 0.001$) and
injuries interfering with work \((p = 0.013)\), but positively predicted by body fat percentage \((p = 0.028)\), heavy work hours \((p < 0.001)\), and marital and parental status \((p = 0.001)\). The remaining predictors were non-significant and eliminated from the model. Grip strength was negatively predicted by age \((p < 0.001)\) and injuries interfering with work \((p = 0.006)\), and positively predicted by body fat percentage \((p = 0.013)\), heavy work hours \((p = 0.011)\), and marital and parental status \((p = 0.019)\). (See Table 5.) All other predictors were non-significant and eliminated.

Although we found strong support for the Paternal Provisioning Hypothesis, no support was found for the Somatic Allocation Hypothesis. Neither morning nor evening testosterone predicted muscle mass \((CP1)\) or strength measures \((CP2)\). No interaction effects of testosterone and workload were evident either on arm muscle circumference or chest and grip strength. Although pair-bonded fathers demonstrated lower evening testosterone levels, and greater diurnal decline in testosterone, than single and childless men, marriage and fatherhood did not produce a decrease in either musculature \((CP3)\) or strength \((CP4)\) (Table 5). Instead, and consistent with the provisioning model of men’s life history, pair-bonding and parenting had a positive impact on musculature and strength, despite being associated with lower testosterone. See the Data Supplement for further details of regression analyses in which significant predictors were entered separately into models, so that the individual contributions of these variables could be observed.

**Discussion**

We contrasted competing models of men’s life history by testing associations between parental status, workload, testosterone, and muscle mass in a rural Polish population. The Somatic Allocation Hypothesis has emphasized the role of testosterone in
mediating trade-offs between survival and reproduction primarily through the maintenance of skeletal muscle (Bribiescas, 1996, 2001; Bribiescas et al., 2012). However, several studies have failed to show that natural variation in men’s testosterone levels produces somatic effects on muscle mass, both between- and within-populations (Campbell et al. 2003, 2006, 2006a; Ellison and Panter-Brick 1996; Gettler et al. 2010). Other studies report weak, inconsistent, or indirect relationships (Campbell et al. 2007; Ellison and Panter-Brick 1996; Gettler et al. 2010; Lassek and Gaulin, 2009; Lukas et al., 2004). We proposed an alternative model, the Paternal Provisioning Hypothesis, which recognizes the importance of men’s provisioning responsibilities, and associated changes in the testosterone production and physical activity patterns of involved fathers. We suggest that muscle mass and strength are augmented during fatherhood, despite suppressed testosterone levels, in response to the physical demands of intensified provisioning. We found support for this hypothesis. Pair-bonded fathers in our sample exhibited lower evening testosterone levels, along with a more precipitous decline in morning to evening testosterone, than single and childless men. And even within this community in which men’s work normally involves demanding manual labor, lower testosterone among pair-bonded fathers did not negatively impact productivity. On the contrary, fathers calibrated their work patterns to family need, increasing labor with additional children.

No associations were found between marital and paternal status and morning testosterone levels. Many studies have reported stronger associations between evening testosterone levels and pair-bonding and parenting (e.g., Berg and Wynne-Edwards, 2001; Gettler et al., 2012; Gray et al., 2002, 2004, 2004a; Muller et al., 2009). One potential explanation is that evening testosterone reflects cumulative social interactions experienced
throughout the day, whereas morning testosterone may be more reflective of baseline, dispositional differences between individuals (Muller and Wrangham 2004; Gray et al., 2002, 2004; Muller et al., 2009). If this is the case, then it is sensible to expect stronger relationships with evening testosterone, though this does not fully explain why some studies detect an influence of pair-bonding and parenting on morning testosterone while others do not.

In many small scale societies, men’s provisioning is dependent on physical labor (Apicella et al., 2014; Lancaster and Kaplan, 2009; Gurven and Hill, 2009; Wood and Marlowe, 2013). In Slopnice, work often requires manual labor, and a strong, positive correlation exists between men’s total work hours and heavy work hours (Table 2). The level of physical labor practiced among Slopnice men allows us to parse relative contributions of men’s provisioning activities and testosterone levels in the maintenance of muscle mass. Indicative of male parenting effort, fathers exhibited both lower evening testosterone as well as higher productivity to accommodate family need. Fathers also maintained more upper-body muscle mass and strength than their childless counterparts. (See Figs. 1 and 2)

These findings are of interest because male secondary sexual characteristics, such as dimorphic musculature, are thought to represent investment in mating effort—supported by testosterone (Bribiescas, 1996, 2001). It seems likely that elevated testosterone during sexual maturation is associated with increased musculature in later adolescence, when males are investing heavily in mating effort. This close relationship is not evident, however, after fatherhood and during men’s prime reproductive years. We observed a divergent relationship, in which enhanced musculature and strength was associated with men’s parenting effort and decreased testosterone production. Among primates, this pattern may be
unique to humans. The distinct reproductive ecology of humans, which places a premium on biparental care of altricial young and the sexual division of labor (Kaplan et al., 2000; Wood and Marlowe, 2013; Marlowe, 2003), likely renders a constant relationship between men’s testosterone and muscle mass maladaptive. If men’s muscle mass were primarily supported by testosterone, then physical capabilities would be compromised when testosterone levels decline during fatherhood—precisely when men need to increase productivity because of provisioning demands. Instead, paternal investment disrupts putative associations between mating effort and testosterone in relation to sexually dimorphic muscle, such that the labor demands of parenting effort determine musculature.

It is not our contention that men’s circulating testosterone has no effect on muscle anabolism. Indeed, there is evidence for stronger associations between testosterone levels and measures of upper-body musculature among adolescent males, in both Western and non-Western populations (Danish boys: Hansen et al., 1999; Dogon boys: Beverly Strassmann, personal communication). These findings are consistent with some portion of the Somatic Allocation Hypothesis, but this relationship is weaker in adulthood, when men begin their reproductive careers. It is plausible that testosterone levels during puberty may have a lasting—potentially lifelong—impact on the development and maintenance of muscularity, though longitudinal data are not available to speak to this point. However, it is clear that substantial plasticity exists in determination of adult musculature (Bhasin et al., 2000, 2001), and in this sample of rural Polish men, pair-bonded fathers allocated more time and effort toward work than single and childless men, along with greater somatic investment in strength (Figs 1 and 2). We propose that the influence of endogenous testosterone on men’s skeletal muscle is secondary to, and superseded by, subsistence and provisioning activities.
The lack of associations between men’s testosterone levels and their muscle mass and strength reported here is not altogether unexpected. Several studies examining such relationships report null findings, or find indirect, inconsistent, or weak relationships, particularly among non-Western groups (e.g., Campbell et al., 2003, 2006, 2006a; Ellison and Panter-Brick, 1996; Gettler et al., 2010; Lukas et al., 2004). A cohort study, using a large sample of young Filipino men, found that salivary testosterone levels were not predictive of lean mass, arm muscle area, or grip strength (Gettler et al., 2010). However, an interaction effect emerged in which morning salivary testosterone among physically active sports participants predicted lean body mass, arm muscle area, and grip strength. And in a population sample of Zimbabwean men, a significant but weak relationship (explaining less than one percent of the variance) was reported between afternoon salivary testosterone and fat-free mass (Lukas et al., 2004). Among Ariaal pastoralists of Northern Kenya, no relationship was found between salivary testosterone and lean body mass (Campbell et al., 2003, 2006). However, a subsequent study of the same population reported that arm muscle area and lean body mass were predicted by evening salivary testosterone after accounting for androgen receptor sensitivity, which was evaluated by genotyping for CAG repeats in the androgen receptor gene (Campbell et al., 2007). It is worth noting that the extent to which CAG repeats modulate transcriptional activity of the androgen receptor gene remains inconclusive, because experimental research has shown dose dependent effects of exogenous testosterone on men’s anabolic response without interaction from CAG repeat length (Woodhouse et al., 2003). Thus, it would appear that a key component of the Somatic Allocation Hypothesis has not been supported empirically.
Experimental research investigating testosterone’s effects on musculature within laboratory settings, although far removed from naturalistic conditions, has yielded mixed results. Although men receiving supraphysiologic doses of testosterone consistently demonstrate anabolic effects on skeletal muscle tissue (Bhasin et al., 1996, 2001), doses within a more normative physiological range generally do not produce lean mass accretion in eugonadal men (e.g., Bower and Reardon, 1972; Casner et al., 1971; Crist et al., 1983; Fahey and Brown, 1973; Fowler et al., 1965; Golding et al., 1974; Johnson et al., 1972; Loughton and Ruhling, 1977; Friedl et al., 1991; see Hartgens and Kuipers, 2004, for a comprehensive review).

A separate line of research investigating hormonal and somatic changes during prolonged bed rest has implications for discriminating between the effects of testosterone and physical activity on muscle tissue conservation. One study examined how exercise regimens influence men’s body composition, strength, and hormone levels during 30 days of bed rest (Wade et al., 2005). Healthy men were restricted to bed rest and assigned to one of three conditions. The control group avoided all exercise. A second group followed an exercise regimen intended to preserve lower body strength. The final group followed a regimen intended to preserve aerobic capacity. Exercise groups completed rigorous, 30-minute programs twice a day for five days a week. Relative to pre-bed-rest baselines, plasma testosterone was decreased in exercise groups but not the control group. Although exercise groups exhibited decreased testosterone levels, they maintained aerobic and muscular work capacities; the control group did not, despite unaltered steroid concentrations. In a second study (Zachwieja et al., 1999), men were restricted to 28 days of bed rest without exercise, but some received testosterone injections at supraphysiologic doses while others were given
placebo. Men receiving testosterone gained lean body mass, but, nevertheless, showed reduction in lower- and upper-body strength comparable to controls, such that testosterone administration produced no appreciable benefit toward strength preservation in the absence of physical activity.

Experimental data have demonstrated relationships between testosterone administration and muscular development. However, these data also highlight inconsistencies in the relationship between testosterone and muscularity, in which physical workload appears to be a mediating factor. Physical activity has a protective effect on skeletal muscle tissue, even under adverse health conditions and suppressed testosterone production (e.g., AIDS: Bhasin et al., 2000; sarcopenia: Roth et al., 2000). Accordingly, observational and experimental data call into question assumptions that are present within existing models of male life history, particularly any model that hold men’s activity level constant while emphasizing the effect of testosterone.

Recently, Trumble and colleagues (2013) documented acute increases in salivary testosterone after tree chopping in the Tsimane, a group of forager horticulturalists. They proposed that such increases might function to augment skeletal muscle capabilities during work, thus supporting male parenting effort. They further suggested that decreased testosterone would compromise men’s parenting effort by impeding their provisioning abilities: “While many have argued that decreases in testosterone with fatherhood would increase investment in current offspring (Gray, Kahlenberg, Barrett, Lipson, & Ellison, 2002; Gettler et al., 2011), in subsistence populations dependents necessitate increased food production, and thus a diminished testosterone response during physical activity could have negative effects on physically intensive food production strategies.” (355). Our data indicate
that this second conjecture is incorrect. Pair-bonded fathers exhibited, concomitantly, lower evening testosterone levels, greater diurnal decline in testosterone, and increased strength and productivity.

There are also several reasons to doubt the functional interpretation for exercise-induced testosterone increases favored by Trumble and colleagues. First, although transient elevations in testosterone with exercise are well documented, these generally result not from increased hormone production, but from (1) decreased clearance (because steroid hormones are cleared by the liver, their concentration increases with physical activity, as blood is shunted toward exercising muscles: Cadoux-Hudson et al., 1985; Terjung, 1979) and (2) decreased blood volume (during exercise water is absorbed into interstitial spaces, increasing blood concentration: Raastad et al., 2000). These effects can be observed in both men and women, and in a wide range of hormones, including estradiol, progesterone, prolactin, leptin, cortisol, DHEA, and DHEAS (e.g. Bonen and Keizer, 1987; Bonen et al., 1979; Fisher et al., 2001; Jurkowski et al., 1978; Keizer et al., 1980, 1987; Kraemer et al. 2001). A specific functional role for testosterone in this context is thus unlikely. Second, although steroid hormones exhibit an acute increase during exercise (reviewed in McMurray and Hackney, 2000), this is followed by prolonged suppression (Hakkinen and Pakarinen, 1993), particularly after sustained activity.

Trumble and colleagues (2013: 354) acknowledge that seasonal wood chopping among the Tsimane occurs over a truncated timeframe, and is likely not substantial enough to promote muscular development. However, they suggest that acute testosterone increases might amplify muscular performance in short-term contexts, and contribute to muscle hypertrophy with sustained work effort over more extended periods. A substantial body of
research in exercise science, however, has failed to support these ideas. Fraysse et al. (2014), for example, found that in vivo androgen treatments had no immediate effect on the maximal force, power or fatigue resistance of muscles in mice, nor on their evoked calcium transient. They concluded that “androgens have no major rapid action on either intact fast skeletal muscle or isolated muscle fibres” (Fraysse et al. 2014: 11). In humans, a recent series of careful studies (which were precise enough to detect upregulated gene expression of striated muscle hypertrophy in response to consumption of 25 g of protein after exercise) demonstrated that exercise-induced increases in men’s testosterone levels had no apparent influence on muscular performance, growth, or strength, either during or post-exercise (West and Phillips, 2012; West et al., 2009, 2010, 2012). Wilkinson et al. (2006) demonstrated that muscular hypertrophy occurs in response to resistance exercise without acute increases in androgen concentration. Finally, a number of studies that are frequently cited (including by Trumble and colleagues) to support an effect of post-exercise testosterone increases on musculature (e.g. Ronnestad et al. 2011) have been shown to be seriously flawed, in both their methods and interpretation (Phillips, 2012; Schroeder et al., 2013).

To be clear, we are not disputing the fact that transient elevations in circulating testosterone are biologically meaningful. An extensive literature has found strong associations between acute testosterone increases and both mating and competitive motivation (reviewed in Archer, 2006). Such increases are generally due to increased steroid production rather than reduced clearance, being observed in response to competition and courtship displays that do not involve physical exertion (Mazur et al., 1992; Cohen et al., 1996; Roney et al., 2003; Steiner et al., 2010). Testosterone surges in response to sexual stimuli are associated with elevated LH levels, further suggesting that endocrinological
fluctuation encouraging mating effort is specifically due to increased testosterone production (LaFerla et al., 1978; Stoleru et al., 1993). Given the experimental evidence that exercise-induced rises in testosterone have no effect on muscle performance, and the lack of evidence that such spikes represent investment in parenting effort, as opposed to a nonspecific physiological response to physical activity, we question the relevance of such increases to men’s work.

In sum, we developed a model of men’s life history, the Paternal Provisioning Hypothesis, which is consistent with observed changes in men’s testosterone levels and workload across the life course. We find support for our hypothesis in a sample of rural Polish men, whose subsistence often relies on demanding physical labor. Although fatherhood predicted lower evening testosterone and more pronounced diurnal testosterone decline, indicative of parenting effort, fathers did not show muscle atrophy or diminished strength. Instead, fathers augmented strength and musculature in response to increased provisioning demands. We situate our findings within the larger evolutionary context of how human males apportion mating and parenting effort, and the adaptive effect on men’s phenotypes. Although enhanced muscularity, presumably supported by elevated testosterone, is believed to represent investment in mating effort in younger men, this relationship appears to change for married fathers. Human males may represent an outlier among primates, in which paternal provisioning disrupts the relationship between testosterone and muscle mass, becoming the primary driver of investment in dimorphic musculature.

Because of distinctive features of the human life course, it is difficult to position our results within a broader pattern of primate life history. Pair-bonding and biparental care among primates is rare, but convergent evolution of male parenting has been observed most
extensively in the family Callitrichidae (Cleveland and Snowdon, 1984; Fernandez-Duque et al., 2009; Rutberg, 1983). Similar to humans, tamarin and marmoset males have shown reductions in circulating and urinary androgens in response to expectant mates, paternal experience, and olfactory cues of their infants (Nunes et al., 2001; Prudom et al., 2008; Ziegler et al., 2004). The sexual division of labor found in humans, however, is unprecedented among extant primates (Kaplan et al., 2000). Moreover, relative to other primate species in which males participate in offspring care, the slow life history of humans may result in an even more prolonged period of suppressed testosterone across the life course (Gettler et al., 2011). Human reproduction is exceptional in many respects, and characterized by remarkably altricial young, short interbirth intervals, lengthened juvenile periods, and multiple dependents of overlapping ages (Lancaster and Lancaster, 1983; Kaplan et al., 2000). We hypothesized that because men’s life history places a premium on parenting effort and division of labor, men must augment their productivity and physical capabilities under conditions of prolonged, downregulated investment in mating effort and testosterone production.

Future research includes supplementing our cross-sectional analysis with longitudinal data to test additional predictions of our model. We do not suggest that the manner in which testosterone exposure affects pair-bonded fathers is fundamentally different from that of young single men. Rather, we propose that muscularity only becomes less dependent on androgenic stimulation because of a unique confluence of life history traits specific to human males. And although the convergence of these traits—reduced testosterone production, increased productivity, and augmented muscularity—is most pronounced during fatherhood, we expect a similar pattern in other contexts in which these coalesce at some level, not only
between fathers and non-fathers but also, potentially, within individuals based on fluctuation in workload.

Toward this end, we have begun collecting and analyzing longitudinal seasonal data on Slopnice men (e.g., Alvarado et al., 2014; Klimek et al., 2014). Our analyses are preliminary, but agree with our overarching hypothesis (Alvarado et al., 2014). Men’s workload intensified during the summer harvest, leading to reductions in body fat and testosterone production, but increases in strength and musculature, compared to a period of lower physical activity during the winter. Although humans are not seasonal breeders, it is instructive to draw comparative reference from seasonally-breeding mammals in which seasonal rise in testosterone levels promotes heightened expression of secondary sexual characteristics, including dimorphic musculature, while returning to non-breeding testosterone baseline results in degeneration of these traits (Asher and Peterson, 1991; Ben Saad and Bayle, 1985; Field et al., 1985; Forager and Breedlove, 1987; Lincoln, 1971). This contrasts with the pattern predicted by the Paternal Provisioning Hypothesis for humans, and the one identified in our longitudinal study of rural Polish men.

Potential physiological differences between humans and other primates underlying the link between testosterone and musculature remain a black box, and warrant further investigation. The peptide hormones human growth hormone (hGH) and insulin-like growth factor (IGF-1) exert intrinsically coupled mechanistic actions (Florini et al., 1996), which have been proposed to regulate muscle function and performance (reviewed in Rennie, 2003), and some researchers have employed an evolutionary perspective to posit regulatory adaptive effects of hGH on skeletal muscle phenotype (e.g., Bribiescas, 1996). However, a comprehensive meta-analysis that aggregated five decades of hGH research into a single
dataset, and carefully calculated effect sizes across 44 studies, found that hGH administration
did not enhance muscular performance, and actually had a degenerative influence on exercise
capacity (Liu et al., 2008). Nor does IGF-1 administration have any appreciable effect on
physical capabilities (Doessing et al., 2010). Furthermore, women maintain significantly
higher levels of circulating hGH than men, making it improbable as a primary regulator of
men’s dimorphic musculature (e.g. Engstrom et al., 1998).

Repetitive bouts of exercise produce acute increases in the rate of muscle protein
synthesis (Biolo et al., 1995; Phillips et al., 1997) and small net accretions that produce a
chronic hypertrophic response from local intramuscular mechanisms (Rennie et al., 2004).
Local mechanisms within skeletal muscle tissue that affect the rate of muscle protein
synthesis include the p70S6K, JAK, STAT, and mTOR intracellular signaling pathways,
which generate a synthetic response to muscle force production (Biolo et al., 1995; Phillips et
al., 1997; West et al., 2009, 2010, 2012). These pathways represent promising areas for
future research on differences between humans and non-human primates in muscle
maintenance.

Future research will also involve more detailed comparisons of our dataset with other
Polish samples, in order to enrich the existing ethnographic work on parenting and
fatherhood among Polish men. An earlier study of urban Polish men found no significant
difference in morning or evening salivary testosterone between fathers and non-fathers
(Jasienska et al., 2012), though a relatively small number of non-fathers were included in the
sample (N = 18). Interestingly, fathers’ testosterone levels interacted with educational
achievement and number of children. Although our current analysis was not situated at
examining these issues, these relationships warrant further investigation, and data collected
from our sample of rural Polish men may help to further elucidate relationships between 
fathers’ testosterone levels, educational and socioeconomic status, and parity.

Finally, our data provide valuable insight into the expression of men’s testosterone 
levels across the life course, which may have important health implications. Development 
and maintenance of the prostate gland is regulated by androgenic hormones (O’Malley, 1971; 
Platz and Giovannucci, 2004), and rapidly rising rates of prostate cancer, though 
concentrated among Western nations, have become a global concern (Kamangar et al., 2006). 
Although large-scale epidemiological studies examining men’s hormonal profiles near the 
time prostate cancer develops often report null associations between prostate cancer cases 
and controls (Roddam et al., 2008), testosterone levels are most variable and highest during 
early adulthood, so that variation in men’s testosterone levels is diminished and difficult to 
detect at older ages (Ellison et al., 2002; Kehinde et al., 2006). However, there is evidence 
that testosterone exposure across the lifespan is associated with prostate cancer risk 
(Alvarado, 2010, 2011). Accordingly, it is reasonable that allostatic diminution of men’s 
testosterone production would have a protective effect on prostate cancer risk (reviewed in 
Alvarado, 2013). Investigating how socioecological factors interact with men’s parenting 
effort and reproductive physiology can only work to elucidate observed trends in androgen-
sensitive disease.

Limitations

Our findings are subject to several limitations, most notably the cross-sectional nature 
of the study design. Hormone measures were based on two (one morning and one evening) 
saliva samples from each participant. Because of the variability inherent in hormonal data, 
potential relationships between testosterone, muscle mass, and strength may have been
obscured beyond detection. However, we were able to detect clear relationships between testosterone and other variables, such as age and marital and parental status. Thus, it seems unlikely that measurement error was responsible for null relationships between testosterone and muscle mass and strength, which are absent in many studies.

Phenotypic correlation represents another potential confound (Stearns, 1992). Specifically, an alternative explanation for the positive associations between testosterone and muscularity among non-human primates is that robust phenotypes can incur the costs of both elevated testosterone and augmented muscle mass, without these traits being causally linked. However, in the context of our analysis, phenotypic correlation would imply that more robust phenotypes can (1) afford higher testosterone levels and greater muscularity, (2) are more attractive to potential mates, and (3) have higher probability of fertility. Instead, we found that although fathers work more and have greater muscularity and strength than childless men, they also had lower testosterone. More importantly, it is unlikely that phenotypic correlation can explain, concomitantly, why the relationship between testosterone and skeletal muscle appears much easier to detect in non-human primates than in humans. Phenotypic correlation is also inconsistent with our preliminary seasonal analyses, which indicate that, within individuals, relaxed work effort during the winter was associated with seasonal testosterone elevation, but muscular atrophy and strength loss, whereas heavy work during the summer harvest was associated with enhanced musculature and decreased salivary testosterone.

Our estimates of duration and physicality of work relied on participant appraisal, which may be susceptible to reporting error. Such error was likely minimal, however, given that self-reported work data were associated with anthropometric and physical performance
measures (i.e. men reporting longer hours and heavier work exhibited greater upper-body musculature and strength).

Lastly, our analysis lacked a measure of paternal involvement. Consequently, we cannot examine the importance of direct care as a predictor of testosterone suppression in fathers.

Acknowledgments

We thank Jane B. Lancaster for her helpful comments on an earlier draft of this manuscript as well as two anonymous reviewers for their suggestions. We are grateful to our colleagues Andrzej Galbarczyk, Ludwik Odrzywolek, and Mariusz Rogozik for their assistance in the field. Also, a special thanks to the Slopnice men who participated in our study. Alvarado was supported by the National Science Foundation Graduate Research Fellowship Program (2008-2011), along with graduate research fellowships from the Program for Interdisciplinary Biological Biomedical Sciences (2011-2013) and the Robert Wood Johnson Foundation Center for Health Policy at the University of New Mexico (2011-2013). Alvarado was also supported by a Ford Foundation Dissertation Fellowship (2013-2014), and an Andrew W. Mellon Foundation Dissertation Fellowship at the University of New Mexico (2014-2015).
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Crist DM, Stackpole PJ, Peake GT. 1983. Effects of androgenic anabolic steroids on
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Table 1. Descriptive Statistics of Study Sample

<table>
<thead>
<tr>
<th></th>
<th>Entire Study Sample</th>
<th>Single and Childless Men</th>
<th>Pair-Bonded Fathers</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean Age (yrs.):</strong></td>
<td>38.93, S.D. = 16.97</td>
<td>31.38, S.D. = 17.83</td>
<td>45.13, S.D. = 13.45</td>
</tr>
<tr>
<td><strong>Age Distribution:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-29</td>
<td>38%</td>
<td>71%</td>
<td>10%</td>
</tr>
<tr>
<td>30-39</td>
<td>20%</td>
<td>9%</td>
<td>28%</td>
</tr>
<tr>
<td>40-49</td>
<td>16%</td>
<td>2%</td>
<td>28%</td>
</tr>
<tr>
<td>50-59</td>
<td>12%</td>
<td>5%</td>
<td>18%</td>
</tr>
<tr>
<td>60-69</td>
<td>5%</td>
<td>4%</td>
<td>6%</td>
</tr>
<tr>
<td>70-79</td>
<td>9%</td>
<td>7%</td>
<td>9%</td>
</tr>
<tr>
<td><strong>Mean Height (cm.)</strong></td>
<td>175.05, S.D. = 7.31</td>
<td>176.60, S.D. = 7.44</td>
<td>173.79, S.D. = 7.00</td>
</tr>
<tr>
<td><strong>Mean Weight (kg.)</strong></td>
<td>81.30, S.D. = 11.99</td>
<td>77.39, S.D. = 12.23</td>
<td>84.51, S.D. = 10.87</td>
</tr>
<tr>
<td><strong>Mean Body Mass Index</strong></td>
<td>26.56, S.D. = 3.76</td>
<td>24.86, S.D. = 3.94</td>
<td>27.96, S.D. = 2.97</td>
</tr>
<tr>
<td><strong>Sports and/or Weightlifting</strong></td>
<td>17%</td>
<td>25%</td>
<td>10%</td>
</tr>
<tr>
<td><strong>Injuries Interfering with Work</strong></td>
<td>20%</td>
<td>11%</td>
<td>27%</td>
</tr>
</tbody>
</table>
Table 2. *Pearson Correlation Matrix of Variables Used in Regression Analysis*

<table>
<thead>
<tr>
<th></th>
<th>Age</th>
<th>Arm Muscle Circum.</th>
<th>Body Fat (%)</th>
<th>Chest Strength</th>
<th>Grip Strength</th>
<th>Injuries</th>
<th>Marital/Parental Status</th>
<th>Morning Salivary T</th>
<th>Evening Salivary T</th>
<th>Diurnal Decline in T</th>
<th>Sports/Weightlifting</th>
<th>Work Hrs., Heavy</th>
<th>Work Hrs., Total</th>
<th>Work on Others’ Farm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arm Muscle Circum.</td>
<td>-0.42**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>0.70**</td>
<td>-0.16</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chest Strength</td>
<td>-0.44**</td>
<td>0.69**</td>
<td>-0.13</td>
<td>1</td>
<td>-0.18</td>
<td>0.76**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grip Strength</td>
<td>-0.50**</td>
<td>0.63**</td>
<td>-0.18</td>
<td>0.76**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Injuries</td>
<td>0.46**</td>
<td>-0.35**</td>
<td>0.34**</td>
<td>-0.29**</td>
<td>-0.39**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marital/Parental Status</td>
<td>0.47**</td>
<td>0.04</td>
<td>0.48**</td>
<td>0.09</td>
<td>0.01</td>
<td>0.20**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morning Salivary T</td>
<td>-0.46**</td>
<td>0.18</td>
<td>-0.31**</td>
<td>0.17</td>
<td>0.27**</td>
<td>-0.32**</td>
<td>-0.16</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Evening Salivary T</td>
<td>-0.45**</td>
<td>0.16</td>
<td>-0.36**</td>
<td>0.10</td>
<td>0.11</td>
<td>-0.24**</td>
<td>-0.38**</td>
<td>0.64**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diurnal Decline in T</td>
<td>0.03</td>
<td>0.04</td>
<td>0.09</td>
<td>0.11</td>
<td>0.17</td>
<td>-0.07</td>
<td>0.29**</td>
<td>0.28**</td>
<td>-0.55**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sports/Weightlifting</td>
<td>-0.40**</td>
<td>0.20**</td>
<td>-0.39**</td>
<td>0.17</td>
<td>0.06</td>
<td>-0.23**</td>
<td>-0.20**</td>
<td>0.15</td>
<td>0.29**</td>
<td>-0.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Work Hrs., Heavy</td>
<td>0.05</td>
<td>0.26**</td>
<td>0.09</td>
<td>0.35**</td>
<td>0.19</td>
<td>0.14</td>
<td>0.22**</td>
<td>-0.16</td>
<td>-0.12</td>
<td>-0.05</td>
<td>0.09</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Work Hrs., Total</td>
<td>-0.04</td>
<td>0.22**</td>
<td>0.10</td>
<td>0.35**</td>
<td>0.22**</td>
<td>0.04</td>
<td>0.26**</td>
<td>-0.00</td>
<td>-0.15</td>
<td>0.16</td>
<td>-0.16</td>
<td>0.68**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Work on Others’ Farm</td>
<td>-0.24**</td>
<td>0.08</td>
<td>-0.28**</td>
<td>-0.05</td>
<td>0.01</td>
<td>-0.22**</td>
<td>-0.13</td>
<td>0.17</td>
<td>0.10</td>
<td>0.06</td>
<td>0.14</td>
<td>-0.06</td>
<td>-0.00</td>
<td>1</td>
</tr>
</tbody>
</table>

**p < .01
*p < .05
See Statistical Analysis for description of variables; see Table 2 for summary of variables. Data were cube root transformed. Tests were two tailed.
<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.47</td>
<td>0.12</td>
<td>-0.35</td>
<td>-3.89</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Marital and parental status</td>
<td>-0.27</td>
<td>0.12</td>
<td>-0.21</td>
<td>-2.32</td>
<td>0.022</td>
</tr>
</tbody>
</table>

**Table 3b. Multiple regression model for diurnal decline in salivary testosterone**

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marital and parental status</td>
<td>0.08</td>
<td>0.03</td>
<td>0.29</td>
<td>3.28</td>
<td>0.001</td>
</tr>
</tbody>
</table>
Table 4. Summary of Regression Analyses Predicting Workload

Table 4a. Multiple regression model for total work hours

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.54</td>
<td>0.26</td>
<td>-0.21</td>
<td>-2.09</td>
<td>0.039</td>
</tr>
<tr>
<td>Marital and parental status</td>
<td>0.88</td>
<td>0.25</td>
<td>0.35</td>
<td>3.56</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Table 4b. Multiple regression model for total work hours

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.79</td>
<td>0.32</td>
<td>-0.30</td>
<td>-2.44</td>
<td>0.016</td>
</tr>
<tr>
<td>Number of children</td>
<td>0.65</td>
<td>0.22</td>
<td>0.37</td>
<td>3.00</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Marital/parental status and number of children were entered separately into models.
Table 5. Summary of Regression Analyses Predicting Chest Strength, Grip Strength, and Arm Muscle Circumference

Table 5a. Multiple regression model for arm muscle circumference

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.09</td>
<td>0.02</td>
<td>-0.44</td>
<td>-4.66</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Heavy work hours</td>
<td>0.02</td>
<td>0.01</td>
<td>0.27</td>
<td>3.43</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Injuries interfering with work</td>
<td>-0.06</td>
<td>0.02</td>
<td>-0.23</td>
<td>-2.72</td>
<td>0.008</td>
</tr>
<tr>
<td>Marital and parental status</td>
<td>0.05</td>
<td>0.02</td>
<td>0.23</td>
<td>2.65</td>
<td>0.009</td>
</tr>
</tbody>
</table>

Table 5b. Multiple regression model for chest strength

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.56</td>
<td>0.09</td>
<td>-0.66</td>
<td>-6.39</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Body fat percentage</td>
<td>0.30</td>
<td>0.13</td>
<td>0.22</td>
<td>2.32</td>
<td>0.028</td>
</tr>
<tr>
<td>Heavy work hours</td>
<td>0.08</td>
<td>0.02</td>
<td>0.32</td>
<td>4.41</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Injuries interfering with work</td>
<td>-0.20</td>
<td>0.08</td>
<td>-0.20</td>
<td>-2.53</td>
<td>0.013</td>
</tr>
<tr>
<td>Marital and parental status</td>
<td>0.22</td>
<td>0.07</td>
<td>0.28</td>
<td>3.30</td>
<td>0.001</td>
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</table>
### Table 5c. Multiple regression model for grip strength

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE(B)</th>
<th>β</th>
<th>T</th>
<th>Sig. (p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.45</td>
<td>0.07</td>
<td>-0.68</td>
<td>-6.19</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Body fat percentage</td>
<td>0.28</td>
<td>0.11</td>
<td>0.26</td>
<td>2.51</td>
<td>0.013</td>
</tr>
<tr>
<td>Heavy work hours</td>
<td>0.04</td>
<td>0.01</td>
<td>0.19</td>
<td>2.59</td>
<td>0.011</td>
</tr>
<tr>
<td>Injuries interfering with work</td>
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<td>0.07</td>
<td>-0.23</td>
<td>-2.80</td>
<td>0.006</td>
</tr>
<tr>
<td>Marital and parental status</td>
<td>0.13</td>
<td>0.06</td>
<td>0.20</td>
<td>2.39</td>
<td>0.019</td>
</tr>
</tbody>
</table>

1. Morning and evening testosterone were entered separately into models.
Figures

1a) Morning Salivary T (pg/ml) vs. Age (years)

1b) Evening Salivary T (pg/ml) vs. Age (years)

1c) Total Weekly Work Hours vs. Age (years)

1d) Arm Muscle Circum. (cm) vs. Age (years)

1e) Chest Strength (kg) vs. Age (years)

1f) Grip Strength (kg) vs. Age (years)

Figure 1. Age Decline in Salivary Morning and Evening Testosterone Levels, Workload, Arm Muscle Circumference, Chest and Grip Strength Among Single and Childless Men and Pair-Bonded Fathers
Figure 2. Mean and 95% Confidence Intervals for Morning and Evening Salivary Testosterone, Workload, Arm Muscle Circumference, Chest and Grip Strength Among Single and Childless Men and Pair-Bonded Fathers. Values were adjusted for age as well as injuries interfering with work, and cube root transformed. All t-tests were two-tailed. A Satterthwaite approximation t-test was used for comparisons of morning testosterone means and workload means because of marginally unequal and significantly unequal sample variances, respectively.
Chapter 3: Seasonal Fluctuation in Workload, Testosterone Levels, and Anthropometry among Rural Polish Men: Testing Models of Men’s Life History

Louis Calistro Alvarado¹, Martin N. Muller¹, Melissa Emery Thompson¹, Magdalena Klimek², Ilona Nenko², Grazyna Jasienska²

¹Department of Anthropology, University of New Mexico; ²Department of Environmental Health, Jagiellonian University

Abstract

Testosterone has been hypothesized to mediate tradeoffs between reproduction and survival through the management of sexually dimorphic muscle mass, such that a persistent link is maintained between men’s testosterone levels and musculature. However, previous research has found that pair-bonded fathers exhibit lower testosterone levels than single and childless men, but also greater muscularity and strength from increased labor demands. Here, we extend this research beyond between-individual comparisons of fathers and non-fathers by examining within-individual seasonality in men’s workload, testosterone levels, body composition, and physical performance. Life history, anthropometric, and hormonal data were collected from 103 rural Polish men in a rural agricultural community (at the Mogielica Human Ecology Study Site) during the summer harvest and in winter. Wilcoxon signed-rank tests were used to examine within-individual seasonal variation. During the winter when work demands were relaxed, men exhibited increased body fat percentage and elevated levels of morning testosterone, but concomitant decrease in flexed biceps circumference, and chest and grip strength. These data contribute to a growing literature which suggests that muscle mass maintenance in human males is comparatively less dependent on androgenic
stimulation than has been observed in other mammals and primates. These findings are also consistent with recent evidence suggesting that men’s provisioning and subsistence activities are a primary determinant of their skeletal muscle phenotype.

**Introduction**

Previous models of men’s life history have emphasized testosterone’s role in determining muscular development and performance (e.g., Bribiescas, 1996, 2001, 2010; Trumble et al., 2013). In contrast, we recently proposed that maintenance of men’s skeletal muscle phenotype is relatively insensitive to natural variation in testosterone levels, and that men’s physical activities are the primary determinant of muscularity (Alvarado et al., 2015). Here, we extend this line of research by testing predictions from competing life history models to analyze seasonal variability in men’s workload, testosterone levels, body composition, and physicality.

Testosterone supports male reproductive function and behavior in vertebrates, as well as promoting the development of secondary sexual characteristics (e.g., Brockman et al., 1998; Ketterson and Nolan, 1992, 1999; Krause, 2006; Muller and Wrangham, 2004; Wingfield et al., 1990). Higher testosterone levels and greater expression of secondary sexual characteristics may benefit males in mating competition but can also entail energetic costs and mortality risks (Kruger and Nesse, 2006; Marler and Moore, 1988; Mestler and Hamilton, 1969; Promislow, 1992, Promislow et al., 1992). Accordingly, testosterone’s involvement in the regulation of skeletal muscle phenotype has been proposed to represent a trade-off between reproduction and survival in men, such that favorable energy availability promotes elevated testosterone and subsequent muscular hypertrophy, while nutritional constraint suppresses testosterone to facilitate a diminished, thriftier phenotype (Bribiescas,
This conceptual framework, which we term the Somatic Allocation Hypothesis, has generally proven effective in explaining variability in men’s testosterone levels between populations (e.g., Bribiescas, 1996, 2001a, b; Gray et al., 2006a; Ellison et al., 1989; for chimpanzees, see Muller and Wrangham, 2005). It has shown less success in predicting intra-population variation, particularly among non-Western groups (e.g., Bentley et al., 1993; Ellison and Panter-Brick, 1996; for chimpanzees, see Muller and Wrangham, 2005). In fact, the prediction that testosterone is a robust mechanism determining somatic investment in musculature does not appear to be well-supported in studies of adult men, either at the between- or within-population level (reviewed in Alvarado et al., 2015).

Interestingly, in other mammals and primates, positive associations are generally found between testosterone and dimorphic musculature in cross section and longitudinally (e.g., Asher and Peterson, 1991; Emery Thompson et al., 2012; Field et al., 1985; Forger and Breedlove, 1987; Lincoln, 1971; Muehlenbein et al., 2002).

Unlike other primates, humans demonstrate a sexual division of labor (Kaplan et al., 2000; Murdock and Provost, 1973; Lancaster and Lancaster, 1983), and male parenting effort is a critical feature in the evolutionary ecology of human reproduction (Kaplan et al., 2000; Marlowe, 2003; Lancaster and Lancaster, 1983; Wood and Marlowe, 2013). Among subsistence populations, men’s parenting effort entails physical labor to provision mates and offspring (Apicella, 2014; Kaplan et al., 2000; Lancaster and Lancaster, 1983; Wood and Marlowe, 2013), and pronounced sexual division of labor places a greater premium on strength tasks in men’s work roles, particularly episodic bursts of strength and force (Harris, 1993; Murdock and Provost, 1973). Additionally, there is evidence from subsistence groups that men adjust their workload to accommodate family need, and increase productivity.
according to their number of dependents (Quinlan, 2000; Marlowe, 2003; Hooper, 2011; Wood and Marlowe, 2013), which may suggest selective pressure on fathers, in particular, to maintain their physical capabilities in order to support provisioning responsibilities.

During fatherhood, however, men also experience a significant decrease in testosterone levels (reviewed in Gettler, 2014). Lower testosterone among involved fathers is believed to represent investment in parenting effort, while elevated testosterone among single men is indicative of greater investment in mating effort (Gray et al., 2002; Kuzawa et al., 2009; Muller et al., 2009; Gettler et al., 2011). Consistent with this interpretation, men with higher testosterone are less interested and responsive to infants (Fleming et al., 2002; Storey et al., 2000), less committed to their partners with more interest in extra-pair mating (Caldwell Hooper et al., 2011; McIntyre et al., 2006), have less familial and parenting involvement (Alvergne et al., 2009), and generally direct increased courtship effort toward multiple partners with greater cumulative mating success (Peters et al., 2008; van Anders et al., 2007). Accordingly, a plethora of cross-cultural studies, using cross-sectional and longitudinal data, have reported that pair-bonded fathers exhibit lower testosterone levels than single childless men (e.g., American: Gray et al., 2002; Canadian: Fleming et al., 2002; Chinese: Gray et al., 2006b; Filipino: Gettler et al., 2011; Hadza: Muller et al., 2009; Jamaican: Gray et al., 2007; Polish: Alvarado et al., 2015; Senegalese: Alvergne et al., 2009; Swiss: Perini et al., 2012). However, if a relatively fixed relationship exists between endogenous testosterone and muscle mass, then it would appear that fathers’ physical capabilities would be compromised at the exact time when provisioning responsibilities intensify.
To address this incongruity, we developed the Paternal Provisioning Hypothesis which predicts that men’s musculature and strength capabilities are augmented during fatherhood, despite suppressed testosterone levels, from the physical demands of increased work responsibilities (Alvarado et al., 2015). This hypothesis was tested in a sample of rural Polish men whose work often involves demanding manual labor. We found that pair-bonded fathers had lower levels of evening salivary testosterone as well as more pronounced diurnal decline in testosterone than did single and childless men. However, decreased testosterone among fathers did not inhibit their productivity. On the contrary, fathers spent more time working, and their family size predicted total workload. And as predicted, although fathers showed lower testosterone levels, they possessed greater upper-body musculature and strength.

According to the Paternal Provisioning Hypothesis, the manner in which testosterone exposure affects muscle tissue is not fundamentally different for pair-bonded fathers than it is for single and childless men. We argue that because of the unique reproductive ecology of human males, men’s skeletal muscle is relatively buffered from the effects of androgens, but sensitive to the physical demands of their work. If this model is accurate, although the convergence of these traits—reduced testosterone production, increased productivity, and augmented muscularity—is most pronounced during fatherhood, a similar pattern would be expected, at some level, in other contexts in which these traits align. The current study expands on previous research by employing a longitudinal design to examine within-individual variation in workload, testosterone levels, and body composition among rural men in Slopnice, Poland (see Description of Study Site).
In some respects, Slopnice may represent an intermediate between Westernized and non-Western societies, which was a motivating force behind the current study. Westernized men, in general, are fairly well-nourished and exhibit elevated testosterone levels (Bribiescas, 2001; Ellison et al., 1989; Kehinde et al., 2006; Muller and Wrangham, 2009), but are more susceptible to suppressed testosterone production in response to acute bouts of energetic stress (e.g., Strauss et al., 1985; Trumble et al., 2010 cf. Bently et al., 1993; Ellison and Panter-Brick, 1996). As a community, Slopnice typically has favorable energy availability, but, unlike Westerners, residents tend to rely on physically demanding and seasonal labor for subsistence (Alvarado et al., 2015; Jasienska, 2013). This provides an excellent opportunity to parse the relative contributions of testosterone and workload on skeletal muscle phenotype.

The Paternal Provisioning Hypothesis makes the following predictions: Men will experience intensified workload during the summer harvest compared to winter (P1). Men will have higher testosterone levels during the winter when workload is relaxed (P2), which would also be predicted by the Somatic Allocation Model (CP1). However, despite elevated testosterone in winter, men will show a concomitant decrease in upper-body musculature (P3) and strength (P4), because of lower workloads. Competing predictions can be derived from the Somatic Allocation model. Men’s muscle mass will decrease with seasonal decline in testosterone levels (CP2), as will their strength (CP3).

Methods

**Study site and participants.** Research was conducted in Slopnice, Poland at the Mogielica Human Ecology Study Site during summer 2011 and winter 2011-2012. Slopnice is a rural village located within the Western Carpathians in southern Poland, and has an
approximate population of 6,198 inhabitants (Statistical Office of Krakow, 2013). Labor-intensive seasonal work has been documented in villages across this mountain region, with more demanding work and higher energy expenditure during the summer harvest (Alvarado et al., 2015; Jasienska, 2013). Food availability is generally not limited in this community. And although the populace is rapidly transitioning to a Westernized lifestyle, it retains several characteristics that distinguish it from more developed regions of Europe, such as greater reliance on subsistence agriculture and manual labor and high completed fertility averaging nearly four children per woman (Colleran, 2013; Jasienska, 2013).

One hundred and three men participated in both the summer and winter portions of this study. One participant became a father between the summer and winter of data collection. This participant was excluded from the sample, because the primary objective of the current study was to examine the influence of seasonality on men’s workload, hormone levels, and body composition, whereas fatherhood has been shown to affect many of the same variables under investigation here, and represents a potential confound within the context of this seasonal analysis (e.g., Alvarado et al., 2015; Gettler et al., 2011; Wood and Marlowe, 2013). Participants were healthy adult males residing in Slopnice, Poland whose ages ranged from 18-78 years (N = 102; mean age = 41.05 years, S.D. = 17.56 years).

Seventy eight percent of participants’ occupations involved fairly heavy or very heavy work, while 22% of participants’ described their work as light or sedentary (see Data Collection). Two men whose occupations normally include physically demanding work were temporarily unemployed, and were placed in the light and sedentary group since they were not currently involved in manual labor. Men’s professions included: builder 37%, trade specialists (i.e., electrician, plumber, stone mason, welder) 16%, farmer 14%, student 12%,
retired 12%, office worker 4%, policeman or enlisted military 4%, butcher 2%. Some participants had secondary occupations, particularly among students and retired men, and 37% of participants listed subsistence farming as a secondary profession.

Participants were also asked about additional sources of physical exertion, such as sports participation, weightlifting, or other exercise. In the summer, the most common forms of recreational activity in this study sample were soccer (20%), jogging/hiking (7%), and weightlifting (7%). Similarly, weightlifting (9%), jogging/hiking (7%), and soccer (6%) were the most common forms of recreation during winter. See Table 1 for further descriptive statistics of the study sample.

Data collection. Participants were recruited by advertisements posted on community bulletin boards and referral sampling. Participants provided information regarding their work patterns, such as their profession, hours normally worked in a day and days worked during a week, and physicality ratings of their work (sedentary, light, fairly heavy, or very heavy). Although it was not uncommon for participants to help friends or family with farm work, work contributed toward others’ farms was much more sporadic than their own work schedule. Because labor contributed to others’ farms tended to be an inconsistent measure of work patterns, many participants were unable to provide a specific estimate of weekly hours spent working on others farms, such that this measure was excluded from the analysis.

After interviews were conducted, participants’ anthropometry was collected: height, weight, body fat percentage, and flexed biceps circumference. Body fat percentage and body weight were collected with a Tanita scale (model UM-028). Body fat percentage was only available for 61 participants at both time points (see Limitations). Upper-body musculature was estimated from flexed biceps circumference, which was measured at the widest point of
the upper-arm while biceps was maximally contracted (Sell et al., 2009). Upper-body strength was estimated from chest and grip strength, which were collected with a portable dynamometer. For grip strength, the participant’s elbow was at flexed 90° with the forearm in neutral position, and participants used their dominant hand to squeeze the dynamometer with maximum effort (Mathiowetz et al., 1985). For chest strength, participants used both hands to grasp the dynamometer at their sternum and pressed hands together with maximum effort (Sell et al., 2009). Strength tests were repeated in triplicate, and the mean of these attempts was used in statistical analyses. Some participants were unable to complete chest strength (N = 5) or grip strength (N = 1) tests because of existing injuries, such as shoulder problems or missing fingers. In addition, one participant’s morning saliva sample from winter did not contain enough saliva for hormone analysis. This study was conducted with the approval of the University of New Mexico Human Research Review Committee. Participants provided written informed consent.

**Hormone analysis.** Participants provided 4 ml of saliva via passive drool into a collection tube, which was assayed for testosterone. Salivary testosterone has been shown to correlate well with serum free testosterone, the fraction of circulating hormone available to target tissues (Goncharov et al., 2006; Wang et al., 1981). To take into account diurnal rhythm in testosterone levels (van Cauter, 1990), an evening saliva sample was collected shortly before sleeping, and the morning sample was collected immediately after waking. Participants were asked to refrain from eating, drinking alcoholic or caffeinated beverages, brushing teeth, sexual activity, and smoking for at least thirty minutes prior to providing samples, because these activities can influence salivary hormone measurements (Salimetrics,
Inter-assay coefficients of variation (CVs) were 3.0% for high and 13.0% for low salivary control. Intra-assay CV for duplicate determinations averaged 3.5%.

**Statistical analysis.** We used a within-individual design to test whether natural fluctuation in men’s testosterone levels and work patterns had functional consequences on skeletal muscle mass. Wilcoxon signed-rank test were used to evaluate seasonal differences in workload, recreational activity, body fat percentage, morning and evening testosterone levels, chest and grip strength, and flexed biceps circumference. Tests were two-tailed.

Although most of the men in this sample participated in demanding physical labor, we divided the sample according to the physicality of men’s labor for a more fine-grained analysis. Participants were divided into men whose work was fairly or very heavy (N = 78) and those involved in sedentary and light work (N = 24). The rationale for this apportionment was that if work-related seasonality affects testosterone levels and body composition, then participants in light or sedentary jobs should be less prone to these effects, and can thus be used as a control. In contrast, we expect men whose professions entail seasonal fluctuation in manual labor will exhibit amplified hormonal and anthropometric differences between summer and winter.

**Results**

In winter, relative to summer, men had decreased workload (Z = 55.50, p < 0.001), higher body weight and body fat percentage (Z = 3,958.50, p < 0.001, and Z = 1,470.00, p < 0.001, respectively), and higher levels of morning salivary testosterone (Z = 3,281.00, p = 0.017). No significant seasonal difference was observed for evening testosterone (Z = 2,788.00, p = 0.472). Despite elevated morning testosterone in winter, men’s flexed biceps circumference and grip strength decreased (Z = 245.00, p < 0.001, and Z = 1,332.00, p <
0.001, respectively), and chest strength showed a marginally significant decrease (Z = 1,838.50, p = 0.053). Men spent more time in recreational activities during the summer, but this difference was not significant (Z = 178.00, p = 0.094). See Figure 1.

Next, the study sample was divided into men whose work was sedentary or light versus those involved in fairly heavy and very heavy work. Men involved in heavy labor worked less during the winter than in the summer (Z = 97.45, p < 0.001), had higher body weight and body fat percentage (Z = 2,133.50, p = 0.001, and Z = 901.00, p = 0.001, respectively), and higher morning testosterone levels (Z = 2,096.00, p = 0.003). Additionally, in winter, these men exhibited declines in flexed biceps circumference (Z = 93.50, p < 0.001), chest strength (Z = 968.00, p = .035), and grip strength (Z = 658.00, p < 0.001). No significant seasonal change was evident in evening testosterone levels (Z = 1,652.00, p = 0.445), nor for time spent in recreation (Z = 81.00, p = 0.232).

Light and sedentary working men also had decreased workload in the winter (Z = 10.00, p = 0.023), but showed no significant difference in either morning or evening testosterone (Z = 140.00, p = 0.775, and Z = 157.00, p = 0.841, respectively). In winter, these participants had higher body weight and body fat percentage (Z = 286.00, p < 0.001, and Z = 74.00, p = 0.046, respectively), but lower flexed biceps circumference (Z = 27.00, p = 0.001). However, significant seasonal differences were not observed for either chest or grip strength (Z = 139.00, p = 0.753, and Z = 114.00, p = 0.303, respectively). See Figure 2. Nor was there a significant difference for recreation (Z = 20.00, p = 0.310).

**Discussion**

We contrasted competing models of men’s life history by testing mutually exclusive predictions with seasonal data from a rural Polish population. Predictions of the Paternal
Provisioning Hypothesis were generally supported, whereas little support was found for the Somatic Allocation Hypothesis. During summer, men reported heavier workloads and also showed reduced body weight, body fat percentage, and morning testosterone. Despite decreased testosterone in summer, men showed greater flexed arm circumference and higher chest and grip strength, although seasonal difference in chest strength was only marginally significant ($p < 0.10$). Moreover, relative to sedentary or light workers, heavy working men demonstrated a more clearly defined pattern of seasonal differences in anthropometry, hormone levels, and strength capabilities. Heavy workers exhibited decreased body weight, body fat percentage, and morning testosterone in summer, but increased flexed biceps circumference, chest and grip strength during the same period. Altogether, this suggests that variability in men’s physical labor produced seasonal alterations in men’s phenotype.

A similar pattern of seasonality, though comparatively diminished, was found among non-physical workers, which likely indicates that seasonal change in men’s energetic status and physical demands was not entirely captured by changes in participants’ work schedules. However, as noted earlier, while light and sedentary workers exhibited seasonal change in body weight, body fat percentage, and flexed biceps circumference, they did not show seasonality in other measures that manual laborers did, such as chest and grip strength or morning testosterone.

These findings support our contention that an enduring and relatively constant relationship between endogenous testosterone and muscle phenotype would not be adaptive for human males (Alvarado et al., 2015). We previously hypothesized that because of the important roles of biparental care and division of labor in the evolution of human reproduction, men must augment their productivity and physical capabilities under conditions
of prolonged testosterone suppression, which we found evidence of in an earlier analysis within this same rural population (Ibid.). However, an analogous relationship would be expected, not only between fathers and non-fathers, but in other ecological contexts in which this specific sequence of traits may emerge. Accordingly, we find that in contrast to positive associations observed between men’s workload and their muscularity and strength, seasonality in work demands was inversely associated with seasonal change in morning testosterone. Together, these complementary lines of cross-sectional and longitudinal evidence suggest that men’s subsistence and provisioning activities are fundamental in regulating somatic investment in muscular development and performance.

In sum, these seasonal data add to a growing body of literature which suggests that within the broad range of normative human variation, men’s musculature is comparatively insensitive to hormonal regulation. This divergence between humans and non-human primates becomes evident when evaluating between-individual variance in testosterone and musculature, in which associations are generally more robust in primate males than in men (reviewed in Alvarado et al., 2015). Here, we can further expand on this comparative perspective beyond cross-sectional comparisons to contextualize the results of our seasonal analysis.

Although humans are not seasonal breeders, it is informative to draw comparative reference from seasonally breeding mammals. During the breeding season, elevated testosterone increases expression of secondary sexual characteristics, including dimorphic musculature (Asher and Peterson, 1991; Ben Saad and Bayle, 1985; Field et al., 1985; Forager and Breedlove, 1987). Although elevated testosterone provides fitness benefits by promoting mating competition, but can impose concomitant increase in mortality risk,
energetic and immunological costs, as well as suppression of paternal behavior (Ketterson and Nolan, 1992; Robertson et al., 2004; Promislow, 1992; Promislow et al., 1992; Wingfield et al., 1990). Accordingly, seasonally breeding males generally show marked reduction in testosterone levels following the breeding season, inhibiting the expression of costly behavioral and morphological traits used in mating competition (Asher and Peterson, 1991; Ben Saad and Bayle, 1985; Field et al., 1985; Forager and Breedlove, 1987; Wingfield et al., 1990). In contrast, our seasonal data accord with previous findings that men augment musculature in response to the physical demands of subsistence and provisioning activities, even under conditions of suppressed testosterone production (Alvarado et al., 2015). In this sample, although testosterone suppression did not conserve energy through diminished musculature, intensified work demands were nonetheless associated with suppress testosterone production, such that it would appear that increased energy expenditure may generally compete with investment in mating effort.

Taken altogether these findings provide preliminary evidence, at multiple levels of analysis, that men’s life history may represent a departure from that of other primates, and mammals in general, in which maintenance of sexually dimorphic musculature is indicative of mating effort and testosterone-dependent.

Limitations

Our study relied on participant estimates of their work schedules and physicality of labor, which is susceptible to reporting error. However, it seems unlikely that measurement error had a significant impact on analyses, given that self-reported data were in general agreement with anthropometric measures. Men who reported seasonal fluctuation in heavy
work exhibited the most pronounced pattern of seasonal alterations in body composition, physical performance, and hormone measures.

Another limitation is that seasonal shifts in body fat percentage at both summer and winter time points were only available for 61 of 102 participants. In the initial summer portion of our study, we relied primarily on skinfold caliper measurements to estimate body fat percentage. In winter, it was difficult to accurately measure triceps skinfolds, because men often wore longer sleeved shirts. And although men rolled up their sleeves for caliper measurements, this also tended to stretch the triceps fat depot so that it became difficult to obtain a reliable reading. As such, we chose to use bioelectrical impedance to estimate seasonal variation in body fat percentage, though again these data were only available for 61 participants. Although body fat data were not available for the entire sample, the available data were consistent with both participants’ self-report and anthropometric data, such that decreased body fat values were associated with reports of intensified workload as well as lower body weight.

Finally, because we were unable to collect a reliable measure of participants’ triceps skinfolds in winter, we were also unable to calculate upper-arm muscle circumference (upper-arm circumference – [triceps skinfold * 3.14]; McWhirter and Pennington, 1994) for seasonal comparison, and, instead, used flexed biceps circumference to estimate upper-body muscularity (after Sell et al., 2009). Although less precise, any inherent measurement error only impeded the ability to support our predictions, considering that body fat percentage, which also contributes to arm circumference, fluctuated in the opposite direction as our prediction for flexed biceps.
Literature Cited


## Tables

Table 1. *Descriptive Statistics of the Study Sample*

<table>
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<tr>
<th></th>
<th>Entire Study Sample</th>
<th>Heavy Working Men</th>
<th>Sedentary Working Men</th>
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<tr>
<td></td>
<td>N = 102</td>
<td>N = 78</td>
<td>N = 24</td>
</tr>
<tr>
<td><strong>Mean Age (yrs.):</strong></td>
<td>41.05, S.D. = 17.56</td>
<td>40.79, S.D. = 15.97</td>
<td>41.85, S.D. = 22.36</td>
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<tr>
<td>Age Distribution</td>
<td></td>
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<tr>
<td>18-29:</td>
<td>32%</td>
<td>29%</td>
<td>42%</td>
</tr>
<tr>
<td>30-39:</td>
<td>20%</td>
<td>19%</td>
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<tr>
<td>40-49:</td>
<td>18%</td>
<td>22%</td>
<td>4%</td>
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<tr>
<td>50-59:</td>
<td>13%</td>
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<tr>
<td>60-69:</td>
<td>7%</td>
<td>8%</td>
<td>4%</td>
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<tr>
<td>70-79:</td>
<td>11%</td>
<td>6%</td>
<td>25%</td>
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<tr>
<td><strong>Mean Height (cm):</strong></td>
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<td>174.52, S.D. = 7.10</td>
<td>176.19, S.D. = 7.49</td>
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<tr>
<td><strong>Mean Weight (kg)</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Summer:</td>
<td>80.92, S.D. = 12.30</td>
<td>81.82, S.D. = 12.84</td>
<td>77.38, S.D. = 9.68</td>
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<tr>
<td><strong>Mean Body Mass Index</strong></td>
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<td></td>
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<td><strong>Weekly Work Hours</strong></td>
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<tr>
<td>Summer:</td>
<td>52.22, S.D. = 19.82</td>
<td>58.76, S.D. = 13.03</td>
<td>30.96, S.D. = 23.21</td>
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<td>Winter:</td>
<td>36.84, S.D. = 22.40</td>
<td>42.86, S.D. = 19.12</td>
<td>17.29, S.D. = 21.42</td>
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<td><strong>Weekly Sports/Weights Hours</strong></td>
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<td>Summer:</td>
<td>1.67, S.D. = 4.32</td>
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<td>1.12, S.D. = 3.00</td>
<td>1.33, S.D. = 3.35</td>
</tr>
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</table>
Figures

1a) Median and 95% Confidence Intervals for Seasonal Fluctuation in Men’s Workload, Testosterone Levels, Anthropometry, and Physicality

1b) Morning Salivary T (pg/ml), N = 101

1c) Body Fat Percentage, N = 61

1d) Flexed Biceps Circum. (cm), N = 102

1e) Chest Strength (kg), N = 97

1f) Grip Strength (kg), N = 101

*All seasonal analyses were non-parametric and two-tailed.

Figure 1. Median and 95% Confidence Intervals for Seasonal Fluctuation in Men’s Workload, Testosterone Levels, Anthropometry, and Physicality

*Figures 1a, 1b, 1c, 1d, 1e, 1f show the median and 95% confidence intervals for various parameters such as weekly work hours, morning salivary testosterone levels, body fat percentage, flexed biceps circumference, chest strength, and grip strength, comparing summer and winter seasons. The p-values (p < 0.01, p = 0.02, p < 0.01, p < 0.01, p = 0.05, p < 0.01) indicate statistical significance for these comparisons.*
Figure 2. Median and 95% Confidence Intervals for Seasonal Fluctuation in Workload, Testosterone Levels, Anthropometry, and Physicality among Heavy Working and Sedentary/Light Working Men
Chapter 4: Population Differences in the Testosterone Levels of Young Men are Associated with Prostate Cancer Disparities in Older Men

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This manuscript has been accepted for publication:


Abstract

Although there is evidence that greater exposure to testosterone is associated with an increased risk of prostate cancer, a recent analysis of 18 prospective studies found no relationship between levels of endogenous sex hormones and prostate cancer development. However, the reviewed studies were subject to methodological constraints that would obscure any potential relationship between prostate cancer and androgenic hormones. If prostate cancer risk is mediated by lifetime exposure to testosterone, then case-control studies that concentrate on endogenous sex hormones near the ages that prostate cancer is diagnosed would provide limited information on cumulative testosterone exposure across the lifespan. Alternately, early adulthood has been suggested as the most salient period to
evaluate the influence of steroid physiology on prostate carcinogenesis. As such, an exhaustive literature search was completed to obtain testosterone values reported for study samples of younger men, along with prostate cancer incidences for the larger populations from which the study populations were sampled. A novel analytical method was developed to standardize, organize, and examine 12 studies reporting testosterone levels for 28 population samples. Study populations were generally apportioned according to ethnicity and geographic residence: Americans of African, Asian, Caucasian, and Hispanic ancestry from several different regions within the United States as well as men from China, Germany, Japan, Kuwait, New Zealand, South Korea, and Sweden. Population differences in the testosterone levels of young men were significantly associated with population disparities in the prostate cancer incidence of older men (Spearman’s rho = .634, \( p = .002 \)).

Growth and maintenance of the prostate gland is supported by androgenic hormones (O’Malley, 1971), and there is evidence that greater exposure to testosterone is associated with an increased risk of prostate cancer (Platz and Giovannucci, 2004; Shaneyfelt et al., 2000). However, an extensive meta-analysis of eighteen prospective case-control studies conducted by the Endogenous Hormones and Prostate Cancer Collaborative Group (2008) found no association between endogenous sex hormones and the development of prostate cancer within a pooled sample of 3886 men with incident prostate cancer and 6438 control subjects. This finding was not altogether unexpected. Although circulating androgen levels have been hypothesized to influence prostate cancer risk, case-control studies in which men developing prostate pathologies are compared to controls with healthy prostate function have provided inconsistent results. Some studies have suggested that androgenic stimulation has an effect on carcinogenesis of the prostate (Henderson et al., 1982; Parsons et al., 2005;
Shaneyfelt et al., 2000), whereas others report an equivocal relationship (Carter et al., 1995; Sofikerim et al., 2007). Since the comprehensive meta-analysis by the Endogenous Hormones and Prostate Cancer Collaborative Group, several researchers have denounced any relationship between elevated testosterone levels and prostatic malignancy (e.g., Carpenter et al., 2008; Morgentaler, 2008), with few dissenting voices (e.g., Hsing et al., 2008). Here, I challenge the position that no relationship exists between concentrations of endogenous sex hormones and prostate cancer, and advance an alternative hypothesis that testosterone’s influence on prostate cancer risk is observable in the steroid hormone physiology of young men.

Men from modern Western cultures possess testosterone levels significantly higher than men from small-scale and preindustrial societies (Ellison et al., 1989; Kehinde et al., 2006). Furthermore, men’s testosterone secretion appears to conform to an urbanization gradient (Bribiescas, 2001; Gray et al., 2006). In a study of reproductive endocrinology among social groups within the larger developing population of South Africa, men’s testosterone levels varied as a function of modernity and socioeconomic status (Gray et al., 2006). South African men in subsistence communities had lower testosterone levels, while affluence and urban living were associated with increased testosterone secretion. Indeed, men involved in subsistence food production are often subjected to chronic energetic stress and generally incapable of supporting the metabolic cost of physiological processes affiliated with elevated testosterone levels, particularly the energetic demands of muscle anabolism (Bribiescas, 1996).

Along with elevated testosterone values, men from developed regions of the world also demonstrate the greatest incident rate of prostate cancer (Kamangar et al., 2006).
Comparisons between Western and non-Western populations are instructive in this regard. The difference in testosterone levels between these groups is primarily among young men (Bribiescas, 2001; Ellison et al., 2002; Kehinde et al., 2006). Western men exhibit a robust decline in testosterone levels following early adulthood, while men from traditional populations show less of a precipitous age-related decline, so that no appreciable difference exists between Western and non-Western men in later life (Bribiescas, 2001; Ellison et al., 2002). Because young men exhibit higher and more variable androgen production, early adulthood has been suggested as the most relevant time to evaluate the potential influence of steroid physiology on the prostate gland since a better representation of lifetime hormone exposure is provided during this period (Grönberg, 2003; Platz and Giovannucci, 2004). Similarly, it has been suggested that women’s risk of breast cancer is influenced by lifetime exposure to ovarian steroid hormones (Jasieńska et al., 2000). Premenopausal Western women have elevated levels of ovarian steroids and also experience a higher number of menstrual cycles during their reproductive years that further increases cumulative hormone exposure (Jasieńska et al., 2000; Eaton et al., 1994). And similar to the population distribution for hormone-dependent cancer in men, Western women have shown the highest incidence of breast cancer (Kamangar et al., 2006). Jasieńska and Thune (2001) found evidence of the relationship between young-adult hormone concentrations and cancer risk among population samples from pre-industrialized and developed nations in which young women who were from populations with an increased incidence of breast cancer demonstrated higher luteal progesterone levels, with US women having the highest progesterone levels and breast cancer incidence.
Comparisons of steroid physiology between modern populations are equally informative. In healthy men from modern populations, testosterone secretion peaks during early adulthood (Gapstur et al., 2002; Jankowska et al., 2000; Uchida et al., 2006), and subsequently declines with age in a manner that diminishes interpopulational variation (Bribiescas, 2001; Ellis and Nyborg, 1992; Ellison et al., 2002; Kehinde et al., 2006). For instance, African-American men have repeatedly shown higher testosterone levels than other Western ethnic groups (Ellis and Nyborg, 1992; Kehinde et al., 2006; Ross et al., 1986), though this relationship becomes inconsistent at later ages (Ellis and Nyborg, 1992; Pettaway, 1999). And accordingly, African-American men exhibit the highest incidence of prostate cancer, an incident rate which is nearly twice that of their Caucasian counterparts in some areas of the United States (Curado et al., 2007). This disparity is noticeable as early as age 45, suggesting that prostate cancer risk is determined at a relatively young age (Ross et al., 1986). Moreover, autopsy examinations have revealed fairly high rates of latent prostate carcinoma among younger men (Sánchez-Chapado et al., 2003), which is also suggestive that etiological factors during early adulthood affect prostate cancer development in later life.

If cumulative exposure to testosterone across the lifespan determines prostate cancer risk, then the steroid physiologies of late-middle aged and elderly men, the typical age range sampled in prospective case-control studies, would offer limited information on lifetime androgen exposure. As such, it is predicted that population differences in the testosterone levels of young men will be positively associated with population disparities in prostate cancer among older men.
Methods

Study design and data collection. Some epidemiological investigations have compared sex steroids of young men sampled from populations that exhibit substantial disparity in prostate cancer risk (e.g., Jakobsson et al., 2006; Kehinde et al., 2006; Ross et al., 1986, 1992; Winters et al., 2001). And although this cross sectional approach has its limitations, it does provide a richer understanding of population differences in testosterone production. A thorough literature review was completed for studies reporting testosterone values of young men from population samples with a mean or median age of less than 39 years. Ideally, a younger sample would be better suited at detecting between-population variation, but the paucity of these studies necessitated the inclusion of a broader age group. Total testosterone levels were reported more frequently than free testosterone, and so, total testosterone values were used in analyses.

I searched PubMed for articles using the terms: androgen, epidemiology, prostate cancer, sex hormones, steroids, testosterone, ethnicity, and race. In addition, a manual search of the bibliographies from all retrieved articles was completed. Some datasets were reproduced within multiple publications, but only one representation of each was included in this analysis. Each study compared two or more populations. Only study samples from larger populations that had available incidences of prostate cancer were included in analyses. This requisite was particularly important for study samples in the United States, given the overall diversity of the American populace. A total of 12 studies reporting testosterone values for 28 population samples were gleaned from the published literature (Table 1). Population samples were apportioned according to ethnicity and geographic residence: Americans of African, Asian, Caucasian, Hispanic, and Mexican ancestry from several
different regions within the United States as well as men from China, Germany, Japan, Kuwait, New Zealand, South Korea, and Sweden. When studies reported longitudinal data, testosterone values for the earliest age were used in analyses. Age-standardized rates of prostate cancer incidence were gathered from the regional population in which study samples were recruited. Prostate cancer rates were obtained from a worldwide collection of cancer registries compiled within the Cancer Incidence in Five Continents Vol. IX database (CI5 IX) (Curado et al., 2007). The State Cancer Profiles database (CDC, 2008; SEER, 2007) was used to supplement CI5 IX data when a more precise measure of local cancer incidence for American ethnicities was obtainable. Cancer incidences for ethnic groups in Bernalillo County, New Mexico and Allegheny County, Pennsylvania were collected from the State Cancer Profiles database, while all other incidence data were collected from CI5 IX.

Of the retrieved studies that reported testosterone levels of young men, Ross and colleagues (1992) was excluded from analyses for two reasons. First, newly acquired serum samples were compared with older specimens that were reanalyzed after being stored for an extended period of time, which is methodologically problematic (e.g., Bolelli et al., 1995). Also, the older specimens from Ross et al. (1992) were previously reported in Ross et al. (1986).

**Statistical analyses.** Immunoassay procedures and laboratories used to analyze hormone samples differed across many studies so that the diversity of assays utilized and lack of comparative data on their performance made direct comparisons of testosterone levels between studies impossible. This concern is especially salient given the high degree of measurement variability among competing testosterone assays (Boots et al., 1998). Standardization of these data was crucial in order to construct an aggregate dataset. Toward
this end, a comparison of ratios was computed in which the proportional difference of mean testosterone levels from population samples within a study was compared to the proportional disparity in prostate cancer incidence for the larger populations from which the study populations were sampled. This comparison can be formally expressed as:

\[
\frac{\text{Study}_x \text{ population sample}_1 \text{ testosterone}}{\text{Study}_x \text{ population sample}_2 \text{ testosterone}} \text{ in relation to } \frac{\text{Population}_1 \text{ prostate cancer}}{\text{Population}_2 \text{ prostate cancer}}
\]

For instance, a study reporting testosterone levels for two population samples would produce a single value within this dataset. If this value were graphically represented, the data point would reference the proportional dissimilarity in testosterone between population samples within a study on the X-Axis, with respect to the proportional disparity of prostate cancer on the Y-Axis. This provided a measure of standardization so that population samples from different studies could be aggregated into a single dataset (Table 2).

Although comparisons were made among 28 separate population samples, the methodological design of using within-study comparisons as the basic unit of analysis produced a dataset consisting of 21 values. These data were not normally distributed, and non-parametric methods were used for statistical analysis. Spearman's rank correlation was used to test for a relationship between population differences in testosterone levels and population disparities in prostate cancer. Tests were two-tailed.

**Results**

The proportional difference in testosterone levels between population samples within a study was significantly associated with proportional disparities in prostate cancer incidence within the larger populations from which study participants were sampled \( (r_s = .634, N = 21, \)
This analysis had many comparisons between African-Americans and Caucasian-Americans, albeit from separate regional populations. Consequently, there was potential of generating a biased outcome. However, removing population comparisons of African-Americans to Caucasian-Americans from the analysis provided a similar result ($r_s = .662, N = 14, p = .010$), and removing population comparisons that contained either African-Americans or Caucasian-Americans also produced a significant result, ($r_s = .829, N = 6, p = .042$). See Figure 1 for a graphical representation of these relationships.

This meta-analysis focused on within-study contrasts in an effort to control for methodological variability across datasets from different studies. Yet, as more variable testosterone levels were expected in younger men, it is feasible that greater testosterone disparity was present in studies with the youngest population samples. This was the case. Using an average of the reported sample ages for each population comparison (or an average of midpoints for studies that reported age ranges), studies with younger samples had greater disparity in testosterone levels ($r_s = -.697, N = 21, p < .001$). See Table 2 for the ages of population samples. However, this effect did not drive the observed association between testosterone and prostate cancer. Population comparison values were log transformed, and after controlling for the age of men sampled for testosterone, a significant correlation remained in the proportional disparity between population comparisons of testosterone levels and prostate cancer incidence (partial $r = .780, df = 18, p < 0.001$).

**Discussion**

Population samples used for analyses were generally divided according to participants’ ethnicity, but this does not necessarily imply that androgen production or prostate cancer susceptibility is a function of ancestral lineage. To the contrary, Santner et al.
(1998) found that a Chinese study sample of Beijing men had lower testosterone levels than Chinese-Americans living in Pennsylvania, while no significant difference existed between Chinese-Americans and their Caucasian counterparts from the same locality. Moreover, recognized differences in testosterone levels among some American ethnic groups become inconsistent once anthropometry and lifestyle factors are accounted for (Rohrmann et al., 2007). The incidence of prostate cancer also varies greatly between countries and ethnic groups as well as between populations of shared ancestry that inhabit different regions (Figure 2). Comparisons of migrant groups with their ancestral populations illustrate the role of ecological variability in the expression of cancer disparities (e.g., Jin et al., 1999; Kovi and Heshmat, 1973; Marks et al., 2004). For instance, Chinese- and Japanese-Americans demonstrate substantially higher incidences of prostate cancer than Chinese and Japanese nationals (Curado et al., 2007). Similarly, African-Americans have an extraordinarily high prostate cancer incidence when compared to African regions for which data exist, including West Africa (Curado et al., 2007; Kovi and Heshmat, 1973; but see Odedina et al., 2006). Altogether, this suggests that environmental correlates of ethnicity within a specific local ecology affect steroid physiology as well as prostate cancer etiology, and this should be considered in future epidemiological investigations.

Rohrmann and colleagues (2007) found no significant difference in the testosterone levels of younger Caucasian- and African-American men after accounting for adiposity, age, alcohol and tobacco use, and physical activity. Thus, it appears unlikely that the difference in testosterone secretion normally observed between these groups is a direct function of ancestry. However, analyses that control for environmental correlates of ethnicity discount meaningful anthropometric and ecological dissimilarities that may very well influence steroid
physiology and prostate cancer risk. Indeed, the relationship between young men’s testosterone levels and prostate cancer disparities is strengthened when the adjusted testosterone values reported by Rohrmann et al. are removed from the current analysis (all population comparisons: $r_s = .634$, $N = 21$, $p = .002$ vs. population comparisons without values reported by Rohrmann et al.: $r_s = .721$, $N = 18$, $p = .001$).

In summary, the findings of this meta-analysis are consistent with the androgen-dependent nature of prostate disease, and furthermore demonstrate a dose-dependent relationship in which proportional differences in testosterone are associated with proportional disparities in prostate cancer incidence. This suggests an alternative explanation for the null relationship found by the Endogenous Hormones and Prostate Cancer Collaborative Group: Androgen production among older men is often diminished to the extent that meaningful differences between prostate cancer cases and controls are no longer apparent. Comparisons of early age testosterone production more appropriately capture relative differences in cumulative androgen exposure, and the positive association found here is consistent with the observed influence of testosterone on prostate cell proliferation.

**Acknowledgments**

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Literature Cited


SEER. 2005. Surveillance, Epidemiology, and End Results Program. NIH Publication No. 05-4772.


Tables

Table 1. Characteristics of studies used in analyses.

<table>
<thead>
<tr>
<th>Study</th>
<th>Ethnicity</th>
<th>Population</th>
<th>Prostate Cancer Incidence: Region/Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jakobsson et al. (2006)</td>
<td>Swedish, South Korean, German, Kuwaiti Chinese</td>
<td>Gothenburg, Sweden, Incheon, South Korea, Kuwait, Shanghai, China</td>
<td>Sweden, Incheon, South Korea, Saarland, Germany, Kuwait, Shanghai, China</td>
</tr>
<tr>
<td>Kehinde et al. (2006)</td>
<td>Japanese, New Zealander,</td>
<td>Japan, New Zealand</td>
<td>Miyagi Prefecture, Japan, New Zealand</td>
</tr>
<tr>
<td>Santner et al. (1998)</td>
<td>Caucasian-American, Chinese</td>
<td>Pennsylvania, USA, Beijing, China</td>
<td>Pennsylvania, USA, Shanghai, China</td>
</tr>
</tbody>
</table>

1 When multiple ethnic populations inhabit a single region, the region is italicized.
2 Ettinger et al. reported testosterone values for urban centers in a multi-regional United States sample. To approximate this population, national incidences of prostate cancer were obtained from SEER registries in CI5 IX, which are also more urban than a representative sample of the US populace (SEER, 2005).
3 A nation-level incident rate of prostate cancer for Japan was not available from CI5 IX, though cancer incidences were available for some Japanese prefectures. Using incident rates from different reporting prefectures, or an average value from all prefectures, had no appreciable effect on analyses.
4 Prostate cancer incidence for Beijing was not available; Shanghai was used to approximate an urban Chinese population.
5 The Asian-American study sample in Wang et al. consisted of Los Angeles men of Chinese, Japanese, and Korean descent. Prostate cancer incidences for Chinese, Japanese, and Korean men from Los Angeles County were averaged, and this value was used in analyses.
Table 2. Descriptive characteristics and calculations for population samples.

<table>
<thead>
<tr>
<th>Study</th>
<th>Ethnicity</th>
<th>Age¹</th>
<th>Sample Size</th>
<th>Proportional Difference in Testosterone Levels²</th>
<th>Proportional Disparity in Prostate Cancer Incidence (per 100,000)</th>
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</table>

¹ Unless age range is indicated, mean age is reported for study samples, with the exception of Rohrmann et al. and Tsai et al. for which median age is reported.

² The original units of measurement as reported in studies were retained so that testosterone values could be referenced.

³ Rohrmann et al. reported testosterone values after controlling for anthropometry and lifestyle factors.

⁴ Tsai et al. reported longitudinal data for study samples, which were divided into prostate cancer cases and controls as well as by ancestry. Testosterone values for the earliest age reported were averaged between case and control groups for each ancestral group, and this value was used in analyses.
Figure 1. Rank ordered proportional difference in testosterone levels with respect to ranked proportional disparity in prostate cancer incidence. Abbreviations for population samples: AA African-American, AsA Asian-American, CA Caucasian-American, HA Hispanic-American, MA Mexican-American, NA Native-American, CHN Chinese, GER German, JPN Japanese, KOR South Korean, NZ New Zealander, and SWE Swedish. Data points were labeled according to the ethnicity and region of study samples. Linear relationship for all population comparisons, $y = 0.63x + 4.03$. Holding all ranks constant, linear relationships for population comparisons without AA/CA comparisons, $y = 0.78x + 2.53$, and without comparisons containing either AA or CA samples, $y = 0.86x + 1.88$. 
Figure 2. Age-standardized incident rates of prostate cancer. Incidence data collected from Cancer Incidence in Five Continents, Vol. IX (Curado et al., 2007).
Chapter 5: Summary and Conclusions

This dissertation examined the role of testosterone in men’s life history and health. Chapters 2 and 3 contrasted competing models of men’s life history. The model developed in this dissertation, the Paternal Provisioning Hypothesis, is consistent with observed changes across life course, in which men experience suppressed testosterone production but increase workload during their prime reproductive years. In contrast to prevailing models predicting that testosterone has a pivotal role in men’s muscular development and performance, we find that somatic investment in muscularity and strength is predicted by parenting effort and workload, whereas testosterone levels produced a null effect. These data suggests that distinct features of men’s reproductive ecology, particularly the sexual division of labor and paternal provisioning, may disrupt putative relationships between testosterone and musculature. The data further suggest that any influence of endogenous testosterone on men’s skeletal muscle phenotype is secondary to, and superseded by, subsistence and provisioning activities. This pattern is distinct from that of other mammals and primates in which muscular hypertrophy is indicative of investment in mating effort and directly supported by testosterone, such that male mammals and primates generally show positive associations between testosterone levels and muscle mass measures. An analogous relationship is difficult to detect in human males.

In Chapter 4, a meta-analysis was conducted to examine the existing literature for relationships between population samples reporting testosterone values for young men, in relation to prostate cancer rates for the larger populations from which study populations were sampled. By focusing the analysis on young men’s testosterone levels, who exhibit the highest and most variable testosterone values, a strong association emerged between
population differences in testosterone levels of young men was positively associated with prostate cancer disparities in older men. These data suggests that cumulative hormone exposure, as measured in early adulthood, affects prostate cancer risk in later life.

**Limitations**

Limitations were noted at the conclusion of Chapters 2-4, but it is worth reiterating the most salient points. The hypothesis proposed in Chapters 2 and 3 is novel, and has only been formally tested within a single population. Future research will include supplementing these Polish data with cross-cultural analyses examining determinants of men’s testosterone production and skeletal muscle phenotype.

The epidemiological meta-analysis in Chapter 4 necessitated an ecological study design, because longitudinal hormonal data spanning from young adulthood to older age are not yet available. However, large-scale projects collecting anthropometric, health, and hormone data are currently underway in Western and non-Western populations (e.g., Cebu Project in the Philippines: Adair et al., 1993; NHANES in the US: Center for Disease Control, n.d.; Yetley and Johnson, 1987), such that the influence of cumulative hormone exposure across the lifespan on prostate cancer risk should be testable within the foreseeable future.
Literature Cited

