BIOLOGICAL BASIS OF SEX DIFFERENCES IN RISK AVERSION AND COMPETITIVENESS[*]

Anna Dreber[a] and Moshe Hoffman[b]

Abstract

We review the evidence regarding a biological basis for sex differences in risk aversion and competitiveness. We present the relevant literature in evolutionary theory, and animal behavior, endocrinology and neuroscience, as well as the literature relating human risk aversion and competitiveness to handedness, 2D:4D (a proxy of prenatal testosterone exposure), facial masculinity (a proxy of pubertal testosterone exposure), as well as circulating testosterone. While the results are thus far somewhat mixed, the evidence suggests that biological factors explain part of the sex gap in these economic preferences.

I. Introduction

Even though women in many countries are at least as likely as men to pursue higher education, women still earn considerably lower wages than men, work in very different occupations (e.g., Altonji and Blank 1999), and do not rise as high in the hierarchy (Bertrand and Hallock 2001). Recently, research has suggested that differences in preferences for risk and competition[1] may partly explain this sex[2] gap in the labor market (e.g., Gneezy et al. 2003; Niederle and Vesterlund 2007; Croson and Gneezy 2009). Risk preferences correlate with occupation and wages (Bonin et al. 2007; Dohmen et al. 2009; Barsky et al. 1997), as,

---

[*] We are thankful for comments from Juliette Caminade, Daniel Fessler, Uri Gneezy, Emir Kamenica, John List, and Andrew Mattarella Micke.

[a] Institute for Financial Research (SIFR), anna.dreber@sifr.org

[b] Rady School of Management, University of California, San Diego, mhoffman@chicagobooth.edu

[1] Competitiveness is typically measured as either the change in performance when comparing performance under a forced tournament vs. a piece-rate scheme, or as the choice of whether to compete or not when given the choice between a tournament and a piece-rate scheme.

[2] Throughout this chapter we use the term sex and not gender when we are arguing for a biological basis. However, in doing so we do not mean to rule out the role of socialization.

presumably, does competitiveness,[3] and women are generally more risk averse and less competitive than men (reviewed in Croson and Gneezy 2009, Bertrand 2010)[4].

Evidence shows that culture influences these sex differences. For instance, Gneezy et al. (2009) found that the sex gap in competitiveness found in many western countries as well as in the patriarchal Massai in Tanzania is reversed among the matrilineal Khasi in India. Another example comes from Dreber et al. (2009), who found that children in Sweden show no sex gap in performance when competing in running, unlike what Gneezy and Rustichini (2004) had found in Israel[5].

But does biology play a role as well?[6] Could part of the sex difference often observed be a product of evolutionary selection? Is part of the sex difference mediated by sex hormones and neural sex differences? This paper argues for a tentative yes[7]. For all the sex differences, the evolutionary theory, which we will call Triver's Theory for simplicity, behind these two sex differences is logically compelling and empirically supported[8], leading us to predict evolution will play an ultimate role in driving these sex differences. Given animal and human literature on sex hormones and sex differences in the brain, we predict these variables will play a proximate role in mediating these sex differences[9]. We review four lines of evidence for such mediating proximate roles, using handedness and footedness (a proxy for sex differences in the brain), 2D:4D (the ratio between the length of the index finger and ring finger, a proxy for prenatal hormones), facial masculinity (a proxy for pubertal hormones), and circulating testosterone. The

---

[3] However, we do not know of any empirical evidence for the relationship between competitiveness and occupational choice or wages.

[4] Eckel and Grossman (2008) review the literature on gender differences in risk aversion.

[5] See also Almlund et al. (2010) for a general discussion of the role of the role of personality on preferences.

[6] We have here omitted a discussion of the literature relating sex hormones to other preferences than those for risk and competition, as well as the literature on genes and economic behavior, where the latter obviously suggests a genetic component of many important behaviors. While these studies are of great interest, they are out of the scope for this paper, since they are perhaps not of direct relevance to sex differences. See also Pinker (2002) or Baron-Cohen for more discussion of the biological basis of sex differences in preferences.

[7] This paper does not argue for no role of socialization and thus the impact of culture. In fact, evolution is fully consistent with preferences adapting to the environment and socialization (see e.g. Belsky et al. 1991).

[8] Admittedly, there are many components of Triver's (1972) position that have been refuted, and other components that are still debated. However, these components are not essential for offering an ultimate explanation for human sex differences in risk and competitive preferences.

[9] Biologists often distinguish between two levels of causality—ultimate and proximate. An ultimate cause is a functional evolutionary explanation. A proximate cause is a physical pathway. Such a distinction prevents biologists from heated debates over the "true" cause of a behavior, when the debaters are merely looking at the same behavior at different levels.

results from this nascent literature are thus far inconclusive, but somewhat consistent with the hypothesized proximal and ultimate causes. We want to emphasize that the old dichotomy of nature versus nurture is not always that meaningful, since they often interact, thus nature via nurture might be a more important concept. For example, testosterone levels can be influenced by the presence of a woman (Roney et al. 2007). However, it is hard to see how socialization easily could explain in particular the first two lines of evidence. Combining these four lines of research with the animal literature we believe there is some support for biology in explaining part of the often observed sex differences in risk aversion and competitiveness.

Section II presents Triver's Theory as well as a plethora of evidence supporting the theory. Section III reviews evidence for the role of sex hormones in mediating animal sex differences. In section IV, we similarly review evidence for the brain's role in mediating animal sex differences. In section V, we present evidence for the brain's role, as proxied by handedness and footedness, in mediating sex differences in risk aversion and competitiveness. Section VI reviews the studies thus far on the role of prenatal androgen exposure, as proxied by 2D:4D. Section VII takes a look at the evidence for the role of pubertal hormone exposure, as proxied by facial masculinity, and section VIII reviews the studies on the role of circulating testosterone. In the final section, we conclude and propose some directions for future research.

II. Evolutionary Theory and Animal Evidence

There is a cogent evolutionary theory that predicts a sex difference in risk aversion and competitiveness: sexual selection. Trivers (1972) and Bateman (1948) argue that since males have a lower obligate investment in any given act of reproduction (termed parental investment), the maximum number of offspring a male can have is higher than the maximum number a female can have, even though the mean number is the same across sexes. Consequently, if a male can out-compete other males, he can achieve a larger maximum, so males have more of an incentive to invest in competing. Differences in the investment in competition between males and females should thus covary with the sex difference in the maximum number of offspring; for instance, if females all breed synchronously and in disparate locations, the maximum number of offspring a single male can have is close to the maximum number a single female can have, but if females breed asynchronously and a single male is able to control a large harem of females, the maximum number of offspring a male can have can be substantially larger than the maximum number a single female can have.[10]

Daly and Wilson (1985), Robson (1996) and Dekel (1999) extend this logic to include sex differences in risk aversion: since male parental investment is lower, the number of offspring a female is expected to have will be nearly linear in the amount of resources she has available to invest in parenting. Males, on the other hand, to the extent that they use their resources to compete over mates, are expected to have a convex mapping from resources to reproductive success, which will lead to the evolution of risk-seeking behavior in males but not females, and the sex difference is again expected to covary with the maximum number of offspring a single male can produce relative to a single female[11].

The theory is not merely logically sound; strong evidence from the animal kingdom also supports it[12]. As far back as Darwin,[13] zoologists have noticed that males are generally showier, more aggressive, more territorial, and have bigger weapons[14] (Andersson 1994), all traits

---

[10] Whether the ultimate cause is sex differences in variance in reproductive success, skew in reproductive success, or parental investment is still debatable. However, for our purposes, this distinction is not important since these variables covary and are certainly all sexually dimorphic (i.e. differs between men and women) and in the "right" direction in humans.

[11] Daly and Wilson (1985) present an informal argument. Robson (1996) and Dekel (1999) formalizes this argument. In fact, Dekel finds that the appropriate concept is not risk aversion but rather a preference for "tail dominance"—instead of a preference for higher variance it is a preference for lotteries that places higher probability on the right-hand tail. See also Robson and Samuelson (forthcoming) for further theoretical work on the evolutionary foundations of preferences.

[12] Admittedly, the animal evidence does not directly refer to the constructs in which we are interested: risk aversion—the tradeoff between expected value and variance—and competitiveness—preference for tournament vs. piece-rate payment scheme or performance change when comparing both payment schemes. Instead, the animal literature refers to aggression, weapons, ornaments, and the like. Nevertheless, these constructs all concern risk, and all are instrumental in competing for mates, and the theory would make the same prediction about our constructs as these, that nonhuman males are willing to accept more variance in exchange for the same expected value and have a stronger preference for tournament vs. piece rate-payment schemes remains to be shown. Moreover, whether risk aversion and competitiveness, per se, covary with degree of polygamy or relative parental investment remains to be shown.

[13] "Sexual differences . . . such as the greater size, strength, and pugnacity of the male, his weapons of offence or means of defense against rivals, his gaudy coloring and various ornaments, his power of song and other such characters" (Darwin 1871).

[14] The theory actually predicts a few exceptions, which will be discussed below. The theory, however, does not predict some other exceptions, but other theories unrelated to sexual selection can easily explain these. For example, in hyenas, females are more aggressive than males (Glickman et al. 1987). However, hyenas are also matrilineal—females control the troop. Thus females have a good reason to be more aggressive; namely, they have the potential to gain control of the troop through their aggression and pass this control on to their offspring. Note that this is unlikely to be the case in human societies.

relevant to risk-taking and competitiveness. For instance, among the 113 bird species with spurs—a sharp spike grown on the leg—both sexes have them in 16 species, males alone have them in 97, but females alone have them in none.

As parental investment theory predicts, sexual dimorphism covaries with the degree of polygamy. Among 65 species of birds within the *Fringillidae* and *Emberizidae* families, tail-length sexual dimorphism is larger among polygamous species than monogamous (Bjorklund 1990). Among 24 species of Cervidae deer, the antlers are longer when breeding groups are larger, even when controlling for body size (Clutton-Brock &Albon 1980). Spurs show up in seven different families of birds. Within these families, all but 2 of the 36 polygamous species have spurs, whereas 46 of 125 monogamous species lack them (Davison 1985)[15].

Exceptions exist to the general rule that males are more competitive, for example, among the phalarope, pipefish, and crickets. In these species, the females are more aggressive toward conspecifics (members of their own species), more territorial, and showier (reviewed in Andersson 1994). However, these exceptions support the theory of sexual selection—sex differences in parental investment drive sex differences in morphology and behavior. In phalaropes, the female drops the egg and departs to find another male with which to mate, while the male remains at the nest site and incubates the egg until it hatches (Hohn 1967). In pipefish, the male has a brood pouch—a sac that protects and nourishes the egg until it hatches (Svennson 1988). In crickets, the male gives a nuptial gift—a food transfer in exchange for copulation—that often constitutes a larger investment than the egg itself (Gwynne 1984). These behaviors are exactly what the theory predicts: since males are investing more than females, females stand to benefit more from competing over males.

Even exceptions to these exceptions exist that further support the theory. Among the 29 known species in which males contribute more to parental care, two—the three-spined stickleback and the Rhea americana—contain males that are more competitive (Clutton-Brock and Vincent 1991). This observation seems to contradict the claim that sex differences originate in differences in parental investment. However, upon closer inspection, it makes complete sense;

---

[15] Notice how in some animals the means for competition is in weaponry, while in others it is in ornaments. In still others, not mentioned here, it is speed, etc. The means of competition varies enormously across species and seems to be correlated with factors like whether the species resides in a two or three dimensional landscape. In humans several of these means of competition are thought to occur, such as ornaments and aggression (Puts 2010).

while the three-spined stickleback males build and maintain the nest, the nest can hold up to 10 or more clutches at once, whereas the females lay a clutch only once every three to five days (Clutton-Brock & Vincent 1991), and while the *Rhea americana* males incubate the eggs, they can incubate 20-30 at a time, which is a lot more than a single female can lay (Clutton-Brock & Vincent 1991). The males are doing more parenting on average, but the marginal cost of parenting an additional child is actually lower for a male, breaking the typical relationship between average parental investment and gains from competing. Therefore, in these cases, like in most species, the number of female clutches he can obtain constrains male reproduction.

More evidence supports the theory. In some cases, we can witness a sex role reversal *within* a species as a result of natural variation in which sex invests more in offspring. Among one population of Mormon crickets living in a high density, the females are bigger than the males, and two females will fight when they both detect the same male making a mating call. On the other hand, among another population of Mormon crickets living in low density, females are the same size as males, and males fight over females (Gwynne 1982). Why? For physiological reasons, Mormon crickets must give nuptial gifts of a fixed size. In high densities, corresponding resource scarcity means that fewer males can come up with such a precious gift, and the gift is even more valuable for females. Again, this observation fits the theory; in high densities, male parental investment is higher and females can benefit from competing over males who are on the mating market.

This sex role reversal can even be experimentally induced. Among a single population of bush crickets, researchers placed some crickets in high-pollen colonies and others in low-pollen colonies. In the low-pollen colonies, the females grappled with each other when they heard mating calls and disrupted other females attempting to mate, but in the high-pollen colonies it was the males who grappled with each other in the presence of females. The nuptial gifts were the same size in both colonies, and hence parental investment was estimated to be higher for females than males in the high-pollen colonies (where males could readily produce the nuptial gifts) but not the low-pollen colonies (where the reverse obtained) (Gwynne & Simmons 1990).

For the theory to apply to humans, we must verify that humans are not among the exceptional cases (e.g., the pipefish). In fact, if humans are exceptionally polygamous, the theory predicts a particularly strong sex difference in risk aversion and competitiveness. The evidence suggests humans are certainly not sex role reversed, nor is our species monogamous; rather, humans appear to be mildly polygamous. Based on measures of sexual dimorphism—for example, sexual dimorphism in body size, life expectancy, and age at sexual maturity, which all covary with degree of polygamy among primates (Daly and Wilson 1988; Plavcan 2001; Alexander et al. 1979)—we are middle of the road in terms of polygamy compared to other primates: we are less sexually dimorphic than baboons and gorillas, but more so than gibbons and marmosets (Daly and Wilson 1988). And human males do have more skewed reproductive success than females. For example, about 8 percent of Asian males are descended from a single male (likely, Genghis Kahn) (Zerjal et al. 2003). Solomon purportedly had 800 wives, FLDS leader Warren Jeffs had at least 80 wives, and Nigerian Mohammed BelloAbubakar had at least 170 children. When variance in reproductive success has been measured directly, it is consistently higher for males than females; the ratio of male variance to female variance is 3.1 for the Brazilian Xente Indians (Salzano et al. 1967), 1.77 for the Dobe !Kung (Howell 1979), between 2.02 and 4.69 for the Yanomoamo (Chagnon 1979), and 2.76 for the Aka pygmies (Hewlett 1988).

III. Animal Sex Hormones

Assuming a biological basis exists for human sex differences in risk aversion and competitiveness, we would expect androgens to mediate risk aversion and competitiveness, as strong evidence supports the thesis that androgens mediate sex differences across animals from snapping turtles, in which sex is not even genetically determined, to a closer relative such as the Rhesus monkey, and affect sex differences ranging from primary sexual characteristics, such as genitalia, to secondary sex characteristics, such as horn size and aggression.[16]

What is the evidence that androgens mediate primary sex characteristics? First, prenatal androgens influence the development of primary sexual characteristics. A pregnant guinea pig injected with testosterone throughout her 69-day gestation gives birth to females with external

---

[16] The animal literature includes many interesting studies that try to distinguish between prenatal, pubertal, and circulating androgens, or interesting interactions among these three. However, in our studies, we cannot parse these different mechanisms apart, nor will we attempt to do so in our literature review. See Nelson (2005) for a good review that does parse these apart in animals.

genitalia indistinguishable from their brothers (Phoenix et al. 1959). Ninety percent of female cattle, sheep, goats, or pigs who are twins with males end up as freemartins—sterile with malformed gonads that resemble testes (Lillie 1917).

Hormones affect not just sex organs and mating behavior but also secondary sexual characteristics—sexual differences that derive from sexual selection, such as horn size and aggression, as reviewed above. For instance, female mice are more aggressive if they shared the womb with two male siblings than if they shared the womb with one female and one male or two females (Nelson 2000). Female rhesus monkeys experimentally injected with androgens in utero later exhibit rough and tumble play at frequencies between those of normal males and normal females (Goy & Phoenix 1972). Castrated stags lose their antlers, and those who receive testosterone during the right season do not shed their antlers when others do, or, if they are already shed, the stags will rise in social rank without actually growing their antlers back, presumably because they behave more aggressively (Lincoln et al. 1972).

The role of androgens in mediating primary and secondary sexual characteristics is so evolutionarily ancient that we even see it in birds, where females and not males (opposite to mammals) have the heterozygous sex chromosomes. Female zebra finches that receive estradiol immediately after hatching and testosterone in adulthood will sing just like males (Gurney &Konishi 1980). In fact, as previously mentioned, the role of androgens extends to species where sex is not even genetically determined. Snapping turtles become male if the egg is sufficiently cold and female otherwise, regardless of their genes. However, if cold turtles receive estrogen, a female develops, and if hot turtles receive aromatase inhibitors, which prevent the production of estrogen, a male develops (Rhen& Lang 1994). Parrotfish do not maintain a single sex for the duration of their lives, as they are sequential hermaphrodites, turning from female to male as they age; parrotfish can be induced to become males earlier by experimentally injecting them with 11-ketotestosterone (Cardwell & Liley 1991). And when males split into two types, one more “masculine” and the other more “feminine,” the difference is again mediated by androgens. Male tree lizards, for instance, are either “spotted” or solid. Spotted males are territorial and have orange dewlaps with blue spots; solid males are non-territorial and have solid orange dewlaps. A solid male will grow to be spotted if given testosterone and progesterone while immature (Moore et al. 1998).

IV. Animal Brains

Assuming a biological basis for human sex differences in risk aversion and competitiveness, we would expect neural differences to mediate risk aversion and competitiveness, since, in a plethora of animals, male and female brains do differ considerably and these differences often mediate secondary sex characteristics, and sex hormones influence sex differences in the brain. This observation will lead us to expect that sex differences in human brains will likewise be mediated by sex hormones, and will correspond to sex differences in risk aversion and competitiveness.

Among the many neurological sex differences that have been found across a large variety of species, we list an assortment below. This list is not meant to be exhaustive but to give a flavor for the variety of different types of sex differences found as well as the variety of different species in which such sex differences are known: In the medial preoptic area of rats, females or males castrated before one day of age have more synapses in the dendritic spines, whereas males or females injected with testosterone before four days of age have more synapses in the dendritic shafts (Raisman and Field 1973). Looking at bullfrog arginine vasotocin receptors, we find sex differences in the distribution of such receptors throughout the brain, and estradiol and dihydrotestosterone modulate this distribution. Moreover, injections of AVT increase mating calls in males and phonotaxis—movement toward calling males—in females, indicating the distribution of AVT receptors allows the same chemical to lead to different traits in different sexes (Boyd 1997).

For the most well-understand example, we turn to the high vocal center (HVC) in singing birds. It is important to keep in mind that singing in many species, including the zebra finch, is used by males to send a signal to females of their genetic viability. Males therefore compete based on this secondary trait. In zebra finches, the HVC is three to six times larger in males (Nottobohm 1991). Researchers have good reason to believe this difference relates to sexually dimorphic behavior: in zebra finches, males sing and females do not, and when this brain region has lesions, song production is deficient (Nottobohm et al. 1976, 1982). Moreover, in canaries, where females also sing, though not as much as males, the sex differences in the size of this brain region is smaller (Brenowitz 1991). During the spring—the canary mating season—the sex differences in HVC is larger than during the fall (Nottobohm 1989). And in wrens, where females and males sing duets together, we see no sex difference in this brain region (Brenowitz 1991).

Can exogenous sex hormones change a male brain into a female brain? If estradiol is given to female zebra finches early enough, and testosterone later on, they will sing and their HVC will be the same size as a male's; if males are given DHT—a derivative of testosterone—early enough, they will never sing, and their HVC will not develop to be the same size as that of an untreated male (Gurney &Konishi 1980). In canaries, if females are injected with androgens, we can increase their HVC to the size of a male's (Nottebohm 1980). Additionally, using the same injections, male HVC size can be increased in the fall to equal that of males during the spring (Nottebohm 1989).

V. Left-handedness

In humans, handedness can be evaluated as a proxy for a "masculinized" brain (Geschwind and Galaburda 1987). Prenatal exposure to male sex hormones increases the frequency of left handedness (Smith and Hines 2000). In every culture studied, males are more likely to be left handed (Halpern 2000). A number of studies have found that left-handed individuals are more "masculinized" on various sexually dimorphic traits, ranging from spatial abilities (Porac and Coren 1981) to success in architectural school (Peterson and Lansky 1974). Handedness differences in the labor market also mimic sex differences, with left-handed individuals earning higher wages, tending toward the sciences, and being hugely overrepresented at the top of competitive industries, including the U.S. government (Porac and Coren 1981; Ruebeck et al. 2007). We should caution, however, that socialization could explain many handedness results. Many cultures, and their technologies, pressure left-handed individuals to perform tasks with their right hand, and substantial evidence suggests these pressures can influence handedness at the particular task for which there is pressure to switch (Porac et al. 1986; Teng et al. 1976; Annett 1970), Thus, if one measures handedness only in a particular task for which there is pressure to switch, one might merely be picking up on personality traits like obedience and not on underlying neural structure. However, carefully designed studies can rule out this explanation by collecting various measures of handedness, some of which have more or less social pressure for switching, since evidence also shows pressure to switch handedness does not generalize beyond the particular task (Porac et al. 1986; Teng et al. 1976; Annett 1970), so if the various measures work equally as well then it is unlikely to be explained by personality traits since such traits will covary more with one measure than the other.

Two studies to date relate handedness to risk aversion and competitiveness. Gneezy and Hoffman (2010) report both studies, which address the selection issue by collecting data on the foot with which the subject kicks, as well as the hand with which the subject writes, and find nearly identical results for the two measures. The first study relates competitiveness to handedness. The authors measured competitiveness among 1309 villagers in North East India by having subjects self-select into either a tournament or a piece-rate payment scheme. The task consisted of tossing 10 balls into a bucket 10 feet away. Before tossing the balls, subjects chose one of two different payment options for solving them. Option 1, the piece-rate scheme, gave the subject 20 rupees (approximately 1/5th of a day's wage) for every successful toss. Option 2 gave subjects 60 rupees for every successful toss if the subject had at least as many successes as an anonymous person who had previously completed the study. If the subject had fewer successes, he would get 0 rupees for this task. Option 2 was thus a tournament. This type of binary measure of competitiveness has previously revealed sex differences (Niederle & Vesterlund 2007; Gneezy et al. 2009).

The second study relates risk aversion to handedness. Two thousand U.S. residents participated using Amazon Mechanical Turk (AMT: mturk.com). AMT is a website which allows anyone to post "assignments" for anyone else to fulfill for a nominal fee, and has been a growing tool for researchers, since it enables large heterogeneous samples to be collected rapidly at low cost (see Horton et al. 2010 for more information). The risk task is adapted from Gneezy and Potters (1997). Subjects were given a balance of \$250 and told to choose an amount \$X between \$0 and \$250 to be invested in a risky investment. The remainder (\$250-\$X) was kept on the subject's balance. A coin flip determined the outcome of the risky investment. If the risky investment failed (tails), the participant lost \$X; if the risky investment succeeded (heads), the participant received \$X multiplied by 2.5, leading to a balance of \$250+\$1.5X. The fraction of the money invested, \$X, was the measure of an individual's risk taking. A risk-averse individual could thus choose to invest \$0 in order to get \$250 with certainty, whereas a risk-neutral or riskloving individual might invest all $250 and would then get $0 and $625 with equal probability, giving an expected value of $325. Investing is thus risky but offers high returns, so individuals make tradeoffs between risk and return. Subjects learned that only one random participant would receive what remained on the balance after the coin flip.

Congruent with prior findings, both studies report a higher frequency of left-handedness among men than among women. Both studies also report a relationship between left handedness and preferences, within each sex; the first study finds that left-handed individuals are more competitive, and the second study finds that they take more risks.

VI. 2D:4D

The ratio between the length of the second and fourth digits, or 2D:4D, also known as digit ratio, is thought to be a proxy of prenatal hormone exposure (Manning et al. 1998). Hand form and thus finger sizes are typically recorded using a digital scanner. Proponents argue that there is a negative correlation between the 2D:4D ratio and exposure to testosterone in utero. In accordance with this reasoning, the ratio is lower in men than in women in all populations investigated to date (Manning 2002). Actually measuring in utero hormone levels is fairly difficult to do on a large scale, but one study finds a negative correlation between 2D:4D and the amniotic testosterone-to-estradiol ratio (Lutchmaya et al. 2004).[17] A number of studies also find that 2D:4D correlates negatively with "masculinization" on a wide variety of sexually dimorphic traits, including competitiveness, athleticism, and performance in a mental rotation task (Hönekopp et al. 2006, Manning and Taylor 2001). Moreover, 2D:4D is fixed by birth and hence cannot easily be explained by socialization (McIntyre 2006).

It should also be noted that the behavioral literature on 2D:4D is fairly inconclusive thus far: one fourth of the published studies show mixed or negative results (Voracek and Loibl 2009). The true number of studies with mixed or negative results is likely to be higherif unpublished papers are included, since unpublished studies are more likely to contain null results. However, 2D:4D, unlike handedness, is a rather weak measure. The correlation with prenatal androgens is estimated at 20% (McIntyre 2006). Likewise, differences in 2D:4D

---

[17] Researchers have also observed that individuals with CAH (congenital adrenal hyperplasia), characterized by increased in-utero exposure to androgens, have lower digit ratios (Brown et al. 2002).

between ethnic groups, even between Caucasians from different European origins, is larger than variation between sexes within an ethnic group (Manning et al. 2004). Additionally, the overlap between the distribution of 2D:4D by sex is quite substantial, and the correlation with sexually dimorphic traits is rather weak (McIntyre 2006). Hence, unless studies are carefully designed with large sample sizes and ethnically homogenous heterosexual populations, we would expect many null results even if a true relationship exists between prenatal sex hormones and the sexually dimorphic trait under investigation. Additionally, even if the studies were all conducted with equal rigor, we ought to raise our priors for a relationship between risk aversion and 2D:4D as long as we have fewer than 19 null results for every significant result.

Dreber and Hoffman (2007) performed the first study on 2D:4D and risk taking. Participants were 147 undergraduate students at the Stockholm School of Economics and 125 undergraduate students at the University of Chicago. Dreber and Hoffman used the risk measure described in the above discussion of handedness, adapted from Gneezy and Potters (1997), the only difference being that in the Swedish sample the total was 1700 SEK instead of $250. Dreber and Hoffman found that, as predicted, risk aversion is positively correlated with 2D:4D in the sample of Caucasian men and women in Sweden. They also found that the sex difference in risk preferences diminished when they included 2D:4D as a control in their regression model. However, when examining the same relationship using a more ethnically heterogeneous sample of both men and women from Chicago, the authors did not find a relationship between 2D:4D and risk. In Chicago, however, no sex gap existed in the first place in risk preferences, potentially suggesting some uncommon selection in the men and women that participated.

Six other studies have subsequently explored the relationship between 2D:4D and risk taking. Apicella et al. (2008) studied a sample of 98 men aged 18–23 in Boston, using the same risk task as Dreber and Hoffman (2007). They found no evidence of 2D:4D being related to risk taking in this sample. This null result may be due to the ethnically heterogeneous sample: 2D:4D is known to vary between ethnic groups (Manning et al. 2004), and this sample contained 67% Caucasians, 10% East Asians, 4% Blacks, 4% Hispanics, and 15% “mixed or other.” Additionally, they could not analyze 17 individuals due to unclear images. They also analyzed the subsample of the 42 of participants who were heterosexual Caucasians with scans that could be properly coded, and found no relationship there either. Using a sample of 550 female and male University of Chicago MBA students, Sapienza et al. (2009) found no relationship between 2D:4D and risk taking in both the overall sample and the female and male samples analyzed separately. Sapienza et al. used the risk measure developed in Holt and Laury (2002)—subjects are given choices between lotteries and a certain amount. Pearson and Schipper (2009) explored how 2D:4D is correlated with bidding behavior in a repeated two-bidder first-price sealed-bid auction with symmetric independent private values where bids are negatively correlated with risk taking and profits are positively correlated with risk taking. Using a sample of 192 college students, they find no relationship between 2D:4D and either bidding or profits in both the overall sample and the female and male samples separately. Pearson and Schipper also looked at the two major ethnic groups in their sample, Caucasion and Asians, separately. They found an effect in the Caucasion sample, with 2D:4D correlating positively with profits, which is opposite what one would expect. Moreover, the men in the Caucasion sample drove the effect. Using a fairly unusual sample, Coates and Page (2009) looked at risk taking in 53 male traders on a London trading floor, finding that 2D:4D is negatively related to the amount of risk the traders take. Brañas Garza and Rustichini (2009) asked 189 men and women to perform two different risk tasks. One task measured risk via choices between different lotteries, and the other task was a version of the Holt and Laury task. They found 2D:4D to be negatively related to risk taking with one of the measures for men, but positively related risk taking in the other measure for women. Finally, using a version of the Holt and Laury risk task in an ethnically homogenous sample of 151 Caucasian female and male undergraduate students, Garbarino et al. (2010) found that 2D:4D is negatively related to risk taking among both men and women.

To conclude, there are several studies which find our predicted relationship between 2D:4D and risk aversion, albeit not all studies confirm this prediction. Some of these studies look at both hands either jointly or taking the average (Dreber and Hoffman 2007, Apicella et al. 2008, Sapienza et al. 2009, Garbarino et al. 2010), whereas others look only at the right hand (Coates and Page 2009, Pearson and Schipper 2009). To what extent hand choice explains some of the mixed findings remains for further exploration.

Only one study thus far has looked at competitiveness and 2D:4D. With the same 98 male subjects sampled in Apicella et al. (2008), Apicella et al. (2010) explored 2D:4D and competitiveness. They measured competitiveness using a similar measure to the one described in the handedness section, with the main exception being that the task was solving mazes instead of tossing balls into buckets. Each subject first received a practice maze and learned he would solve mazes similar to the practice one for five minutes. Before solving the mazes, subjects chose one of two different payment options for solving them. Option 1, the piece-rate scheme, gave the subject 25 cents for every maze solved. Option 2 gave subjects 75 cents for every maze they solved if the subject solved at least as many mazes at the person who had last completed the study. If the subject solved fewer mazes than the person who had last completed the study, he would get $0. Option 2 was thus a tournament. Apicella et al. (2010) found no relationship between 2D:4D and competitiveness in their sample of men.

VII. Facial masculinity

Facial masculinity is used as a marker for pubertal testosterone exposure since many masculine craniofacial features, such as jaw width, develop during puberty under the influence of testosterone (Johnston et al. 2001). In order to measure facial masculinity, experimenters typically take facial photos of the subjects looking into the camera with neutral expressions. They then standardize the faces on interpupillary distance, and then obtain four measures from marked face points with the software Psychomorph developed by David I. Perrett and Bernard P. Tiddeman (as in Penton-Voak et al. 2001, Little et al. 2008). Previous studies have found the identification of these features is reliable (Grammer & Thornhill 1994; Scheib et al. 1999). Facial masculinity is subsequently assessed by combining these four measures on the face, including cheekbone prominence, jaw height/lower face height, lower face height/face height, and face width/lower face height, after converting the measures to a z-score. A high score indicates increased facial masculinity. Although these four measurements are sexually dimorphic, exactly how much of facial masculinity is due to actual variation in pubertal testosterone levels is unclear. However, some evidence suggests testosterone during human male development facilitates the growth of bones by increasing outside bone diameter and bone mass (Vanderschueren & Bouillon 1995) and influences craniofacial growth and thus facial masculinity (as well as human male bone growth in general). Correspondingly, boys with delayed puberty and have delayed craniofacial development (such as shorter mandibular ramus length, upper anterior face height, and total cranial base length) compared to a control group;

likewise, delayed puberty boys who received low doses of testosterone over a period of one year demonstrated a significantly higher rate of growth in these features compared to the control group such that there is no difference between the delayed boys and the controls (Verdonck et al. 1999). Apicella et al. (2008, 2010), in the same study that looked at risk taking and competitiveness and 2D:4D, also attempted to correlate both risk taking and competitiveness with facial masculinity.[18] The authors obtained facial masculinity scores for the young men from photographs of the face in the manner described above. Apicella et al. (2008) found a positive and significant relationship between facial masculinity and risk taking, such that men with more masculine faces took more risk. When controlling for circulating testosterone and facial masculinity, Apicella et al. (2008) found that a man with a facial masculinity score one standard deviation higher than the mean invested 6 percent more than the average man. Apicella et al. (2010) found no relationship between facial masculinity and competitiveness. Thus, men with more pubertal testosterone exposure appear to take more risks also after puberty, indicating that testosterone exposure appears to play an organizational role in such preferences[19].

VIII. Circulating testosterone

Researchers have correlated circulating testosterone with a number of behaviors that are generally considered male-typical, namely aggression (Archer 2006), dominance, (Mazur and Booth 1998), status seeking (Mazur 1985, Kemper 1990, Mazur and Booth 1998), and sensation seeking (e.g. Campbell et al. 2010, see Roberti 2004 for an overview).

In the first study of the effects of testosterone on financial risk taking, using their sample of 98 men, Apicella et al. (2008) found that circulating testosterone, as measured from saliva collected with passive drooling when subjects arrived at the lab, correlates positively with risk taking.[20] Results indicate that a man with a testosterone level one standard deviation above the mean took 12% more risk than the average man.

---

[18] Note that Apicella et al. (2008, 2010) found no relationships between 2D:4D, facial masculinity, and circulating testosterone.

[19] Endocrinologists distinguish between organizational and activational role of hormones. Hormones exhibit an organizational role if they alter the brain or body's development. Hormones play an activational role if they merely activate certain traits while they are circulating in the brain and blood.

[20] The samples in Apicella et al. (2008) were collected around the same time of the day because testosterone fluctuates in a circadian fashion.

Zethraeus et al. (2009) administered either testosterone (40 mg/day), estrogen/estradiol (2 mg/day), or a placebo to 200 postmenopausal women during a four week period, finding no effect of exogenous hormones on economic preferences, including risk taking as measured by a version of the Holt and Laury task. Sapienza et al. (2009), when exploring variation in risk taking among MBA students, also measure circulating testosterone from saliva samples finding it to be positively correlated in women but not in men. However, two things are worth pointing out about these studies. Zethraeus et al. found exogenous testosterone and estrogen have little effect on the economic behavior of women, but whether we should expect exogenous testosterone to play the same role in women as in men is unclear. Male brains, unlike female brains, may be organized early in development to be more responsive to the activating effects of testosterone later in life. Thus, exploring the role of testosterone administration on risk taking in men would be interesting. Second, Sapienza et al. collected saliva samples at both the end and the beginning of the study and took the average as their independent testosterone variable. Since testosterone varies during the day, and factors in the actual study can also influence testosterone, making the first sample a better proxy of baseline testosterone than the second sample. Exploring the extent to which the unmanipulated beginning-of-the-study test correlated with risk taking by itself would thus be interesting.

Circulating testosterone also has a well-documented relationship with competition, albeit not with competitiveness, in the endocrinology literature. Testosterone (as well as cortisol) rises in anticipation of competition, and testosterone increases in winners more than in losers following competitions (see Archer 2006 for a review). Interestingly, most of the observed effects occur in men. However, up until recently, no study had looked at the correlation between baseline testosterone and competitiveness as measured by self-selection into a competition. The only study to test a correlation between testosterone and competitiveness as measured by a preference for competition is Apicella et al. (2010). As for 2D:4D and facial masculinity, no relationship exists between circulating testosterone and competitiveness in the sample of young men they explored.

IX. Future Research

But what is the relationship between human risk and competitive preferences and Triver's theory? There are several possibilities, which cannot be distinguished based on the evidence reported above. One possibility is that there was selection for psychological adaptations that are common to both small and large risky decisions. For instance, it is possible that males were selected to fear the worst case scenario less, or to feel greater hope in anticipation of the best case scenario. These adaptations may have been selected specifically for their implications on decision making under uncertainty. Alternatively, these adaptations may have been selected for their implications for aggressive behavior. Likewise for competitiveness, it could be that males were selected to gain a thrill from competing, or merely to overestimate their chance of winning a tournament.[21]

In addition to the evolutionary theory and animal evidence discussed in the first three sections of this paper, we also report four lines of evidence which all support the predictions made based on the three former sections. Admittedly, some of these lines of evidence, particularly the 2D:4D literature, but also the testosterone literature, are inconclusive. Much further research is warranted, but we believe that the results from the existing literature at least partly explain the often observed sex difference in preferences for risk and competition.

There are several directions where we see this emerging field heading. One direction for future research involves animal studies. Although the animal literature reviewed in Section II suggests sex differences in aggression, weapons, and ornaments, whether males are generally more risk seeking has yet to be shown using direct measures of this construct. Despite the fact that researchers have proven that many species are sensitive to the tradeoff between expected value and variance (e.g. Chen et al. 2006), and despite the fact that different species trade off expected value and variance at different rates, and despite the fact that laboratory measures have already been developed which successfully measure this rate, nevertheless, to our knowledge, no

---

[21] It may also be asked what we mean by competitiveness in this paper. We mean the likelihood of engaging in an activity where the payoffs depend on one's relative performance, defined along any dimension, as compared to engaging in an activity requiring the same behavior and having the same payoff distribution, but where payoffs are independent of other's performance. Note that our experimental measure of competitiveness confounds this concept of competitiveness with risk aversion since even though the task is the same the payoffs in one case have higher variance and a higher mean in the competitive payment scheme. It should also be noted that this definition of competitiveness does not distinguish between proximal motivations from engaging in competitive activities; this definition does not distinguish between engaging in competition because one believes one is better at the task or because one gains a thrill from engaging in competitive tasks. We refrain from drawing such a distinction because the theory is silent on the proximal mechanism.

researcher has investigated and demonstrated a sex difference in this rate. Likewise, finding a sex difference in how much animals care about position within a hierarchy compared to absolute resource availability would be interesting. However, we know of no laboratory measure that assesses this tradeoff.

If such sex differences are demonstrated in animals, future research could test additional predictions derived from parental investment theory, thereby further evaluating the evolutionary basis of these sex differences. For instance, the sex difference in risk aversion ought to correlate, across closely related species, with degree of polygamy. A simple study could compare this sex difference in prairie voles (who are monogamous) versus pine voles (who are polygamous). A more elaborate study would compare sex differences across a large group of closely related species and would employ more sophisticated statistical techniques. A third study could manipulate androgen levels, either by using early castration or testosterone injections, and witness the effect on risk aversion. Although such research cannot be done in humans, such research in animals would provide more direct evidence that androgens mediate the sex gap in risk aversion.

A second direction for future research involves relating risk aversion and competitiveness to direct measures of prenatal hormones. The evidence regarding 2D:4D and handedness reviewed in Section IV is indirect, relying on many auxiliary assumptions, and only providing lower bounds on the effect of biology on the sex gap in risk aversion and competitiveness. Thus, more direct measures of prenatal androgenization and corresponding brain morphology could prove useful. For instance, an ideal study would measure prenatal androgens in fetuses and then measure risk aversion and competitiveness many years later once the individual has passed puberty. Likewise, brain scans could provide direct evidence that brain morphology due to variation in androgenization correlates with risk aversion.

A third direction for future research involves theory. Although Dekel (1999) formalize the evolutionary basis for sex differences in risk aversion, no such model exists for competitiveness. Future research could extend Dekel's model to include competitiveness.

A fourth direction for future research involves the relationship between laboratory measures of competitiveness and labor market outcomes. Previous research has established such a relationship for risk preferences (Bonin et al. 2007; Dohmen et al. 2009; Barsky et al. 1997), but the same has yet to be done with regard to competitiveness.

A fifth direction would focus on replicating and extending the studies discussed in section V-VIII. The contradictory results on e.g. the relationship between risk preferences with 2D:4D and circulating testosterone need to be investigated.

A sixth direction would be to explore the role of the menstrual cycle further. The menstrual cycle, which lasts 28 days on average, can be divided into five different phases. The important hormones varying during the cycle are estradiol, progesterone and the luteinizing hormone. Previous studies on the menstrual cycle and risk aversion and competitiveness show inconclusive results (e.g. Buser 2009, Chen et al. 2009, Pearson and Schipper 2009, Wozniak et al. 2010). These studies rely on self-reported menstrual cycle data, which is likely to be error-prone since menstrual cycles vary substantially across individuals. Using over-the-counter tests to determine the timing of the luteinizing hormone surge that precedes ovulation would minimize type 2 errors that could arise from the use of somewhat unreliable cycle measurement methods. An interesting extension of this line of work would thus be to use e.g. over-the-counter tests to get more precise hormonal measurements. However, the theoretical prediction of how risk aversion and competitiveness should change over the cycle is not always clear, as, e.g., several hormones vary during the cycle and it is not obvious which is more important.

Finally, a seventh direction can take these findings and explore them further in the "field". There are already some interesting results suggesting that sex hormones influence choices related to risk outside of the laboratory. For example, White et al. (2006) find that testosterone levels among male MBA students are positively related to new venture creation. Sapienza et al. (2009), in their sample of MBA students, find that both individuals with higher testosterone and lower 2D:4D were more likely to choose careers in finance, which is considered to be risky. Coates and Herbert (2008) find that among a sample of traders, testosterone in the morning is positively correlated with their profitability during the day. Coates et al. (2009) find a similar relationship for 2D:4D.

X. Why should we care?

We argue that economists should care about the biological basis of the sex gap in risk and competitive preferences for two reasons: if economists ignore biology, they will 1) misunderstand the source of the sex gap in risk and competitive preferences and 2) such a misunderstanding can have serious consequences.[22]

In the social sciences, sex differences in outcomes are often interpreted as resulting from sex differences in social treatment—whether individuals are treated socially as male or female. While this is often the case, sex differences in biological treatment—whether individuals develop biologically as male or female—also exist[23]. Since we cannot assign individuals to biological treatments while fully controlling for social treatment, the experimental approach is ruled out and more novel designs are necessary to distinguish between the role of social and biological treatment. In this paper we have presented several novel techniques for parsing the biological treatment from the social treatment: in particular, we presented evidence from the animal literature where controlled experiments are possible, as well as evidence from two physiological variables—handedness and 2D:4D—which covary with the biological treatment but are not likely to covary with the social treatment.

In some cases, there are no good a priori reasons to presume that there should exist a relationship between the biological treatment and the outcome, such as the relation between sex and mathematical abilities, but there are other cases where there is good a priori reason to

---

[22] Others have also discussed the relevance of understanding the basis of the sex gap in economic preferences, but have reached slightly different conclusions than we have. Bertrand (2010) argue that finding that environmental factors play a bigger role than biological factors could lead policy to focus on educational reforms, whereas biological factors being more important could make policy makers focus affirmative action as a tool to reduce the gender gap in the labor market.

[23] The mere fact that social treatment coincides with outcome differences may simply be due to social treatment that reflects biological treatment. Taboos against incest provide a classic example; it seems more likely that taboos reflect an evolved psychology against incest than that taboos drive our aversion to incest. This is evident from the fact that when social treatment and biological treatment are in opposition, behavior is more consistent with biological treatment, as is the case with individuals who are raised communally in Israeli Kibbutzim and are not attracted to each other (Shepher 1983), and same thing for girls who are reared by their future husbands in Shim-pua marriages (Wolf 1995). Moreover, where the cultural and biological explanations make opposing predictions, the evidence falls squarely with the biological prediction, such as the presence of a relationship between views of third party incest and duration of co-residence with opposite sex partners, particularly for males, but the lack of a relationship between views of third party incest and liberal views of sexuality, parental views of incest, or degree of relatedness with opposite-sex partners (Lieberman, Tooby, and Cosmides 2003; Fessler and Navarette 2004).

presume that there is a relationship between some biological factors and some outcome. We argue that both sex differences in risk and competitive preferences are two such cases.

Now let's consider why it matters whether we assign the difference between men and women to the right treatment term. As with most empirical exercises in economics, the goal is to determine the causal link between an outcome variable of economic significance and an input variable that can be manipulated by a policymaker, firm, or individual so that we can better advise policymakers, firms or individuals. In our case, if we confuse biological treatment for social treatment, we are liable to proffer inefficient, and possibly counterproductive, advice.

On the one hand, if we falsely ascribe all of the biological treatment effect to the social treatment effect, we may be lead to the following erroneous conclusions: 1) Since discrimination is typically unprofitable (Becker, 1957), profit maximizing firms ought to treat males and females identically. 2) If society values gender equality, males and females ought to be educated identically.

However, once we recognize the biological treatment effect as distinct from the social treatment effect, we realize that not only are these conclusions non sequiturs, but they are also counterproductive. For instance, it would be unprofitable for the average principal to hire an equal ratio of male to female agents for jobs that require risk averse agents—such as risk management—as jobs that require risk neutral agents—such as day traders. Similarly, if teachers require either that all students compete to solve 100 two digit arithmetic problems or that no students compete, regardless of gender, then either male performance could be reduced, not gaining the added motivation from being in a competition, or female performance could be reduced, being encumbered by performance anxiety[24].

Moreover, if we ignore the biological treatment effect we might miss out on potentially productive interventions[25]. Importantly, biological perspective predicts many moderating

---

[24] To be fair, this follows from sex differences in performance under different competitive regimes, not strictly from different competitive preferences. Nevertheless, the underlying biological perspective treats both identically.

[25] It is often erroneously claimed that if a trait has a biological basis than that trait will be harder to alter via experience than if it has a social basis. For a good counterexample, allow us to return to incest avoidance. Despite a biological basis, those who we are not sexually aroused by is very much dependent on experience, namely who one grew up with. Moreover, understanding the biological basis for the trait can very much help us understand how to alter the trait. Thus, if we desire that individuals marry within their kibbutz, we are lead to the conclusion not that we should weaken our taboos against incest but that we should rear children separately until they are past some critical period.

variables, such as income inequality, hormones, and emotions, which can all be used for productive interventions. For instance, ironically, one way to reduce income inequality between men and women is to reduce income inequality within each sex. And one way to increase female employment in male dominated professions might be as simple as cleaning up the human resource department, literally instead of figuratively, since the evolutionary theory predicts and the evidence supports that male risk aversion is more sensitive than female risk aversion to the societal level income inequality (Daly and Wilson 2002) and less sensitive to current disgust levels (Fessler 2004); if in hiring decisions we measure competitiveness we might get a false impression of sex differences if the interview room is dirty, sex differences that won't generalize to actual on the job work if it is only the interview room and not the job candidate's future office that is dirty. Finally, if you are an individual who wishes to be at your most competitive during a negotiation, it could as a woman be useful to know what hormones peak during what phase of the cycle, and how this is likely to affect your risk aversion and competitiveness, (though the results on this topic are thus far inconclusive) whereas if you are a man, and we eventually will come to the conclusion that testosterone does influence economic behavior, it could be useful to know how your testosterone level is affected by having been around attractive women versus children (it rises with the former and decreases with the latter). We argue that hormone levels, and the factors that influence them, could relevant for risk aversion and competitiveness. More generally, good ultimate explanations are useful for coming up with candidates for proximate mechanisms, which are necessary if we want to moderate the effect. Without good candidates for proximal mechanisms, we are shooting in the dark, both with our research funds and with our choice of public policies.

Additionally, a good theory is required to generalize out of sample. Just noting a correlation between sex and risk preferences cannot tell us, for instance, if we should expect females to be more ambiguity averse, less overconfident, or have a stronger preference for gambles which are “right tail dominant,” holding constant variance (Dekel and Scotchmer 1999), but all of the above are predicted by the evolutionary theory. There are other testable predictions from evolutionary theory, such that the sex gap in behavior will be reduced through marriage, children, and old age (Daly and Wilson 2002).

Noting a correlation between sex and risk preferences is not enough to tell us whether or not we ought to reduce this correlation. A priori, it is not clear whether a single level of risk aversion or competitiveness is optimal, and if so whether this level is closer to the typical female or typical male level. A good theory is required for normative analysis. Only after understanding what causes a trait can we ask if the original source is currently present and worthy of being obeyed. Some traits evolved in response to circumstances still present, such as our blinking when a foreign object approaches our eyes, while other traits evolved due to circumstances that are no longer present, such as our fondness for sweet, salty, and fatty foods. Some traits that evolved in response to circumstances that are still present are not necessarily circumstances that we should care about, such as our proclivity to senesce rapidly after our reproductive capacity has dwindled. Only by understanding the biological foundation of sex differences in risk and competitive preferences can we ask if we should attempt to reduce the gap, or alter e.g. the distribution of men and women on top positions in other ways. For instance, if the only reason why males consistently perform risky behaviors like fist fights is because males evolved in an environment where the winner of the fight became the tribal leader, then this reduces the normative appeal of risky behavior, particularly since those who start bar fights in our society are more likely to find themselves in jail than leading a tribe. Likewise, if the only reason women avoid risk is because their genes were historically not rewarded for taking gambles, but in our current winner take all society, women as well as men's career prospects are rewarded by high risk ventures, then this increases the normative appeal of female risk taking in the labor market.

In sum, we argue that unlike for some differences in outcomes between the sexes, e.g. sex differences in mathematical abilities, the evolutionary argument here is quite compelling, being supported by cross species comparisons, animal experiments, as well as being somewhat supported by some nonstandard correlational evidence in humans. We argue that understanding the biological basis will prevent us from giving bad advice to policymakers, firms, parents, teachers, and individuals, and might lead us to fruitful ideas for good advice. Moreover, understanding the biological basis will enable us to see the generalizability of these sex differences, and guide our normative assessments of whether or not we should attempt to reduce the sex differences.

To conclude: In this paper we have reviewed the evidence concerning a biological basis for the sex gap in risk aversion and competitiveness. The human evidence is thus far inconclusive – this is a nascent field where more studies are needed. No matter what the robust results end up being, we believe that this type of research is important not only for increasing the understanding of sex differences in behavior, but also since it will help improve our understanding of the foundations of preferences in general. This latter is important not only for those interested in sex differences in preferences, but opens up the black box of preferences, something that in economics thus far mainly has been done by drawing on insights from psychology.

We want to finish by reminding the readers that we believe that much of the future research will emphasize the role nature via nurture, rather than the two things in opposition as explanations. We thus believe that further insights into the study of the foundations of preferences will come from collaborations between economists, biologists and psychologists.

References

Alexander, R.D., Hoogland, J.L., Howard, R.D., Noonan, K.M., and Sherman, P.W. 1979. “Sexual Dimorphism and Breeding Systems in Pinnipeds, Ungulates, Primates and Humans.” In *Evolutionary Biology and Human Social Behavior: An Anthropological Perspective*, N.A. Chagnon and W. Irons, (eds.). Scituate, M.A.: Duxbury Press: 402-435.

Altonji, J. G., and R.M Blank. 1999. “Race and Gender in the Labor Market.” In: O. Ashenfelter & D. Card (eds.), *Handbook of Labor Economics*, edition 1, volume 3, chapter 48: 3143-3259, Elsevier.

Almlund, Mathilde, Angela Lee Duckworth, James Heckman, and Tim Kautz. “Personality Psychology and Economics.” Mimeo.

Andersson, M. 1994. *Sexual Selection*. Princeton: Princeton University Press.

Apicella, Coren L., Anna Dreber, Benjamin Campbell, Peter B. Gray, Moshe Hoffman, and Anthony C. Little. 2008. “Testosterone and Financial Risk Preferences.” *Evolution and Human Behavior*. 29: 384−390.

Apicella, Coren L., Anna Dreber, Peter B. Gray, Moshe Hoffman, Anthony C. Little, and Benjamin C. Campbell. 2010. “Androgens and Competitiveness in Men.” Forthcoming in *Journal of Neuroscience, Psychology and Economics*.

Archer, J. 2006. “Testosterone and Human Aggression: An Evaluation of the Challenge Hypothesis.” I, 30: 319-345.

Baron-Cohen, Simon. 2003. *The essential difference: men, women and the extreme male brain*. Penguin/Basic Books.

Barsky, R. B., Juster, F. T., Kimball, M.S. and Shapiro, D. 1997. “Preference Parameters and Behavioral Heterogeneity: An Experimental Approach in the Health and Retirement Study.” *Quarterly Journal of Economics*. 112(2): 537-579.

Bateman, A.J. 1948. “Intra-Sexual Selection in Drosophila.” *Heredity*. 2(3): 349–368.

Belsky, J., Steinberg, L., and Draper P. 1991. “Childhood Experience, Interpersonal Development, and Reproductive Strategy: An Evolutionary Theory of Socialization.” *Child Developmen*t. 62(4):671-5.

Bertrand, Marianne. 2010. “New Perspectives on Gender.” In Handbook of Labor Economics, eds. Orley Ashenfelter and David Card, vol. 4, Elsevier.

Bertrand, Marianne, and Kevin Hallock. 2001. “The Gender Gap in Top Corporate Jobs.” *Industrial and Labor Relations Review*. 55(1): 3-21.

Bjorklund, M. 1990. “A Phylogenetic Interpretation of Sexual Dimorphism In Body Size and Ornament in Relation to Mating System In Birds.” *Journal of Evolutionary Biology*. 3: 171-183.

Boyd, S.K. 1997. “Brain, Vasotocin Pathways and the Control of Sexual Behavior in the Bullfrog.” *Brain Research Bulletin*. 44(4):345-350.

Brañas Garza, Pablo and Aldo Rustichini. 2009 “Organizing Effects of Testosterone and Economic Behavior: Not Just Risk Taking.” Unpublished manuscript.

Brenowitz, E. 1991. “Altered Perception of Species-Specific by Female Birds after Lesions of a Forebrain Nucleus. *Science*. 251(4991): 303–305.

Buser, Thomas. 2009. “The Impact of Female Sex Hormones on Competitiveness.” Mimeo
Pearson, Matthew, and Burkhard C. Schipper. 2009. “Menstrual Cycle and Competitive Bidding.” MPRA Paper 16784.

Chagnon, N.A. 1979. “Is Reproductive Success Equal in Egalitarian Societies?” In *Evolutionary Biology and Human Social Behavior*, Chagnon, N. & Irons, W (eds.). North Scituate: Duxbury

Cardwell, J. R. and Liley N.R. 1991. “Hormonal Control of Sex and Color Change in the Stoplight Parrotfish, Sparisoma Yiride.” *Gen. Comp. Endocrinol*. 81:7-20.

Chen, Keith, Venkat Lakshminarayanan, and Laurie Santos. 2006. “How Basic Are Behavioral Biases? Evidence from Capuchin Monkey Trading Behavior.” *Journal of Political Economy*. 114: 517-537.

Chen, Yan, Peter Katuscak, and Emre Ozdenoren. 2009. ”Why Can't a Woman Bid More Like a Man?” Mimeo.

Clutton-Brock, S.D. 1991. “Sexual Selection and the Potential Reproductive Rates of Males and Females.” *Nature*. 351: 58–60.

Clutton-Brock, S.D. Albon & F. E. 1985. “Guinness Parental Investment and Sex Differences in Juvenile Mortality in Birds and Mammals.” *Nature.* 313: 131–133.

Coates, John M., and Joe Herbert. 2008. “Endogenous Steroids and Financial Risk Taking on a London Trading Floor.” *Proceedings of the National Academy of Sciences*. 105: 6167–6172.

Croson, Rachel, and Uri Gneezy. 2009. “Gender Differences in Preferences.” *Journal of Economic Literature*.

Daly M., and Wilson, M. 1985. “Child Abuse and other Risks of not Living with Both Parents.” *Ethology & Sociobiology*. 6: 197-210.

Daly M, and Wilson, M. 1988. “Evolutionary Social Psychology and Family Homicide.” *Science*. 242: 519-524.

Davison, G.W.H. 1985. “Avian Spurs.” *Journal of Zoology*. 206(3): 353-366.

Dreber, Anna, Emma von Essen, and Eva Ranehill. 2009. “Outrunning the Gender Gap: Boys and Girls Compete Equally.” Stockholm School of Economics Working Paper.

Dreber, Anna, and Moshe Hoffman. 2007. “Risk Preferences are Partly Predetermined.” Mimeo, Stockholm School of Economics.

Garbarino, Ellen, Robert Slonim and Justin Sydnor. 2010. “Digit Ratios (2D:4D) as Predictors of Risky Decision Making.” Mimeo.

Geschwind, N., & Galaburda, A.M. 1987. *Cerebral Lateralization: biological mechanisms, associations and pathology*. MIT press: Cambridge, MA

Gneezy, Uri, and Moshe Hoffman. 2009. “Are Sex Differences In Risk Aversion And Competitiveness Partly Biological? Evidence From Handedness.” Mimeo.

Gneezy, Uri, Muriel Niederle, and Aldo Rustichini. 2003. “Performance in Competitive Environments: Gender Differences.” *Quarterly Journal of Economics*. 118(3): 1049-1074.

Gneezy, Uri, Kenneth Leonard, and John List. 2008. “Gender Differences in Competition: Evidence from a Matrilineal and a Patriarchal Society.” *Econometrica*. 77(5): 1637-1664.

Gneezy, Uri, and Jan Potters. 1997. “An Experiment on Risk Taking and Evaluation Periods.” *Quarterly Journal of Economics*. 112: 631-645.

Gneezy, Uri, and Aldo Rustichini. 2004. “Gender and Competition at a Young Age.” *American Economic Review*. 94(2): 377-381.

Gneezy, Uri, and Aldo Rustichini. 2005. “Executives verses Teachers: Gender, Competition and Self Selection.” Unpublished.

Goy, R. W. and Phoenix, C.H. 1972. “The Effects of Testosterone proportionate Administered Before Birth on Development of Behavior in genetic female Rheusus Monkeys.” UCLA *Forum Med. Science.* 15: 193-201.

Grammer, K., and Thornhill, R. 1994. “Human (Homo sapiens) facial attractiveness and sexual selection: the role of symmetry and averageness.” *Journal of Comparative Psychology*. 108: 233-242.

Gurney, M. and Konishi M. 1980. “Hormone Induced Sexual Differentiation in Brain and Behavior in Zebra Finches.” *Science.* 208: 1380-1382.

Gwynne, D.T. 1984. “Sexual Selection and Sexual Differences in Mormon Crickets.” (Orthoptera: Tettigoniidae, Anabrus simplex). *Evolution*. 38: 1011-1022.

Gwynne, D.T, Simmons L.W. 1990. “Experimental Reversal of Courtship Roles in an Insect.” *Nature*. 346:172–174.

Hewlett, B.S. 1988. “Sexual Selection and Paternal Investment among Aka Pygmies.” In *Human Reproductive Behavior: A Darwinian Perspective,* L Betzig, P Turke M Borgerhoff Mulder (eds.).

Holt, C.A. and Laury, S. K. 2002. "Risk Aversion and Incentive Effects." *American Economic Review*. 92(5): 1644-1655.

Hönekopp, J., Rudolph, U., Beier, L., Liebert, A., and Müller, C. 2006. “Physical Attractiveness of Face and Body as Indicators of Physical Fitness in Men.” *Evolution and Human Behavior*. 28(2): 106-111.

Hohn, E.H. 1967. “Observations of the Breeding Biology of the Wilson’s Phalarope (steganopus tricolor) in Central Alberta.” *AUK.* 84: 220-244.

Howell, N. 1979. *Demography of the Dobe !Kung*. New York: New York Academic Press.

Johnston, V.S., Hagel, R., Franklin, M., Fink, B., and Grammer, K. 2001. “Male Facial Attractiveness: Evidence for Hormone-Mediated Adaptive Design.” *Evolution and Human Behavior*. 22: 251-267.

Kemper, T. 1990. *Social Structure and Testosterone*.New Brunswick, NJ: Rutgers University Press.

Lillie, F.R. 1917. “The Free-Martin: A Study in the Action of Sex Hormones in the Fetal Life of Cattle.” *J. Exp. Zool*. 23: 371-452.

Lincoln, G.A. 1972. “The Role Of Antlers In The Behavior Of Red Deer.” *Journal of Experimental Zoology*. 182(2): 233 – 249.

Little A.C., Jones B.C., Waitt C., Tiddeman, B.P., Feinberg, D.R., Perrett, D.I., Apicella, C.L., Marlowe, F.W. 2008. “Symmetry is Related to Sexual Dimorphism in Faces: Data across Culture and Species.” *Public Library of Science One*. 3(5).

Lutchmaya, S., Baron-Cohen, S., Raggatt, P., Knickmeyer, R., and Manning, J. T. 2004. “2nd to 4th Digit Ratios, Fetal Testosterone and Estradiol.” *Early Human Development*. 77: 23-28.

Manning J.T. 2002. “Digit Ratio: A Pointer to Fertility,” *Behavior and Health*. NJ: Rutgers University Press.

Manning, J.T., Scutt, D., Wilson, J., and Lewis-Jones,D.I. 1998. “The Ratio of 2nd to 4th Digit Length: A Predictor of Sperm Numbers and Concentrations of Testosterone, Luteinizing Hormones and Oestrogen.” *Human Reproduction*. 13: 3000-3004.

Manning, J. T., Stewart, A., Bundred, P. E. and Trivers, R. L. 2004. “Sex and Ethnic Differences in 2nd to 4th Digit Ratio of Children.” *Early Human Development.* 80: 161-268.

Manning, J. T., & Taylor, R. P. 2001. “Second to Fourth Digit Ratio and Male Ability in Sport: Implications for Sexual Selection in Humans.” *Evolution and Human Behavior.* 22: 61-69.

Mazur, A. “Sex Difference in Testosterone Response to a Video Game Contest.” accessed at http://cogprints.org/630/1/pong.html

Mazur, A., and Booth, A. 1998. “Testosterone and Dominance in Men.” *Behavioral & Brain Sciences*. 21: 353-397.

McIntyre, Matthew H. 2006. “The Use of Digit Ratios as Markers for Perinatal Androgen Action.” *Reproductive Biology and Endocrinology*. 4: doi: 10.1186/1477-7827-4-10.

Moore, L.A., Broihier, H.T., Van Doren, M., Lunsford, L.B. and Lehmann, R. 1998. “Formation in Drosophila.” *Development.* 125(4): 667--678.

Nelson, R.J. 2005. *Introduction to Behavioral Endocrinology*. 3rd Ed. Sunderland, MA: Sinauer.

Niederle, Muriel, and Lise Vesterlund. 2007. “Do Women Shy away from Competition? Do Men Compete too Much?” *Quarterly Journal of Economics*. 122(3): 1067-1101.

Nottebohm, F. 1980. “A Continuum of Sexes Bedevils the Search for Sexual Differences?” *Behavioral and Brain Sciences*. 3: 245-246.

———. 1980. “Testosterone Triggers Growth Of Brain Vocal Control Nuclei In Adult Female Canaries.” *Brain Research*. 189(2): 12.

———. 1989 From bird song to neurogenesis. *Scientific American*. 260(2):74-9.

———. 1991 “Reassessing the Mechanisms and Origins of Vocal Learning in Birds.” *Trends in neurosciences*. 14(5):206-11.

Nottebohm F., Paton, J., Darcy B. and Kelley 1982. “Connections of Vocal Control Nuclei in the Canary Telencephalon.” *The Journal of Comparative Neurology*. 207(3): 344-357.

Nottebohm, F., Stokes, T.M., and Leonard, C.M. 1976. “Central Control of Song in the Canary, Serinus canarius.” *J Comp Neurol*. 165(4): 457.

Pearson, M. and Schipper, Burkhar C. 2009 “The Visible Hand: Finger Ratio (2D:4D) and Competitive Behavior.” Unpublished manuscript.

Penton-Voak, I. S., Jones, B. C., Little, A. C., Baker, S., and Tiddeman, B. 2001. “Symmetry, Sexual Dimorphism in Facial Proportions, and Male Facial Attractiveness.” *Proceedings of the Royal Society B*. 268: 1617-1623.

Peterson, J. and Lansky, L. 1974. “Left Handedness among Architects: Some Facts and Speculation.” *Perceptual and Motor Skills*. 38: 547-550.

Phoenix, C.H., Goy, R.W., Gerall, A.A. et al. 1959. “Organizing Action of Prenatally Administered Testosterone Propionate on the Tissues Mediating Mating Behavior in the Female Guinea Pig.” *Endocrinology*. 65: 369-382.

Pinker, Steven. 2002. *The Blank Slate: The Modern Denial of Human Nature*. Penguin.

Plavcan, J.M. 2001. “Sexual Dimorphism in Primate Evolution.” *American Journal of Physical anthropology*.

Porac, C. and Coren, S. 1981. *Lateral preferences and human behavior*. New York: Springer

Porac, C., Coren, S., and Searleman, A. 1986. “Environmental Factors in Hand Preference Formation: Evidence from Attempts to Switch the Preferred Hand.” *Behavior genetics*.

Raisman, G., and Field, P.M. 1973. “Sexual Dimorphism in the Neurophil of the Preoptic Area of the Rat and its Dependence on Neonatal Androgen.” *Brain Research*. 54(17): 1-29.

Rhen, T., and Lang, J.W. 1995. “Phenotypic Plasticity for Growth in the Common Snapping Turtle: Effects of Incubation Temperature, Clutch, and their Interaction.” *American Naturalist*. 146(5): 726-747.

Roberti, J. W. 2004. “A Review of Behavioral and Biological Correlates of Sensation Seeking.” *Journal of Research in Personality*. 38: 256-279.

Robson, Arthur. 1996. “The Evolution of Attitudes to Risk: Lottery Tickets and Relative Wealth.” *Games and Economic Behavior*. 14: 190-207.

Robson, Arthur, and Larry Samuelson. Forthcoming. “The Evolutionary Foundations of Preferences.” In *The Social Economics Handbook*, eds. Jess Benhabib, Alberto Bisin and Matthew Jackson.

Roney, James R., Lukaszewski, Aaron W. and Zachary L. Simmons. 2007. “Rapid Endocrine Responses of Young Men to Social Interactions with Young Women.” *Hormones and Behavior*. 52: 326-333.

Ruebeck, C.S., Harrington, JE Jr. and Moffit R 2007. “Handedness and Earnings.” *Laterality*. 12: 101-120.

Salzano, F.M., Weinstein, E.D., and Neel, J.V. 1967. “Further Studies on the Xavante Indians. The Physical Status of the Xavantes of Simões Lopes.” *Am J Hum Genet*. 19(4): 532–542.

Sapienza, Paola, Luigi Zingales, and Dario Maestripieri. (2009) “Gender Differences In Financial Risk Aversion And Career Choices Are Affected By Testosterone.” *Proceedings of the National Academy of Sciences*.

Scheib, J. E., Gangestad, S. W., and Thornhill, R. 1999. “Facial Attractiveness, Symmetry, and Cues to Good Genes.” *Proceedings of the Royal Society B*. 266: 1913-1917.

Smith, L and Hines M. 2000. “Language Lateralization and Handedness in Women Prenatally Exposed to Diethylstilbestrol (DES)”. *Psychoneuroendocrinology*. 25(5): 497-512.

Teng, E.L., Lee, P.H., Yang, K.S. and Chang, P.C. 1976. “Handedness in a Chinese population: biological, social, and pathological factors.” *Science*. 193(4258): 1148-1150

Trivers, Robert L. 1972. “Parental Investment and Sexual Selection.” In *Sexual selection and the descent of man*, B. Campbell (Ed.) 1871-1971 (pp 136–179). Chicago, Aldine.

Voracek M, and Loibl L.M. 2009. “Scientometric Analysis and Bibliography of Digit Ratio (2D:4D) Research, 1998-2008.” *Psychol Rep*. 104(3):922-56.

Verdonck, A., M. Gaethofs, C. Carels, and F. de Zegher. 1999. “Effect of Low-Dose Testosterone Treatment on Craniofacial Growth in Boys with Delayed Puberty.” *European Journal of Orthodontics*. 21: 137–143.

White, Roderick E., Stewart Thornhill, and Elizabeth Hampson. 2006. “Entrepreneurs and Evolutionary Biology: The Relationship between Testosterone and New Venture Creation.” *Organizational Behavior and Human Decision Processes*. 100: 21–34.

Wozniak, David, William Harbaugh, and Ulrich Mayr. 2010. “Choices About Competition: Differences by Gender and Hormonal Fluctuations, and the Role of Relative Performance Feedback.” MPRA Paper 21097

Zerjal, T., Yali Xue, Bertorelle, G., Spencer, R., Wells, Weidong Bao, et al. 2003. “The Genetic Legacy of the Mongols.” *The American Journal of Human Genetics*, 72(3): 717-721.

Zethraeus, Niklas, Ljiljana Kocoska-Maras, Tore Ellingsen, Bo von Schoultz, Angelica Lindén Hirschberg, and Magnus Johannesson. 2009. ”A Randomized Trial of the Effect of Estrogen and Testosterone on Economic Behavior.” *Proceedings of the National Academy of Sciences*. 106(16): 6535-6538.