Sex Hormones and Choice under Risk*

Burkhard C. Schipper†

This Version: December 5, 2022; First Version: October 9, 2011

Abstract

We study the correlation of choice under risk in Holt-Laury lotteries for gains and losses with gender, the use of hormonal contraceptives, menstrual cycle information, salivary testosterone, estradiol, progesterone, and cortisol as well as the digit ratio (2D:4D; length of the index finger to the ring finger of the right hand) in more than 200 subjects (45% females). In males, salivary testosterone is negatively correlated with risk aversion for gains only. In females, salivary cortisol is positively correlated with risk aversion for gains only. No other significant correlations between risk preferences and salivary hormones are observed. No significant correlations between risk preferences and the menstrual cycle are observed in naturally cycling females. No significant correlations between risk preferences and the digit ratio are observed in either gender and/or race.

Keywords: Testosterone, Estradiol, Cortisol, Progesterone, Menstrual cycle, Contraceptives, Digit ratio, 2D:4D, Gender, Holt-Laury lottery task, Risk aversion, Risk seeking, Endocrinological economics.

JEL-Classifications: C91, C92, D81, D87.

*I am grateful to the editors of the special issue and two anonymous reviewers for their helpful suggestions. I thank Gabriel Mathy, Wen Yong Tang, and Nick Zolas for excellent research assistance. Moreover, I thank Coren Apicella, David Cesarini, Anna Dreber, Nikolaos Georgantzis, and audiences at NYU, Hong Kong University of Science and Technology, the University of Heidelberg, the 2013 Xiamen University International Workshop on Experimental Economics, the conference on "New Challenges for Economic Decision-Making: Biology, Neuroscience, and Economic Models" at the University of Southern California 2011, and the MOVE Workshop on Gender Differences in Competitiveness and Risk Aversion, Barcelona 2010, for helpful comments. Financial support through the UC Davis Hellman Fellowship is gratefully acknowledged. An earlier version of paper has been circulated on SSRN and RePEc since 2012. The data and code are available from `https://faculty.econ.ucdavis.edu/faculty/schipper/research.htm`.

†Department of Economics, University of California, Davis. Email: bcschipper@ucdavis.edu

1 Introduction

Decisions in the face of risk and uncertainty have been at least as pervasive in the past of human mankind as they are in present. While today a decision maker's economic success or overall success in life is affected by her risk preferences, it is conceivable that in the past also reproductive success was influenced by risk preferences. Thus, evolutionary selection has probably acted on traits that affect how a decision maker takes decisions under risk and uncertainty (see Robson 1996, Dekel and Scotchmer, 1999, and Hamo and Heifetz, 2002, for theoretical models). These traits should have biological markers. In this paper, we study rather comprehensively to what extent some biological factors like hormones are associated with risk preferences. Hormones are chemical messengers that transport signals from one cell to another. Finding hormones that are associated with risk preferences allow us to learn about the biological mechanisms involved in decision making under risk. For instance, it would be important for explaining why risk aversion seems to increase with age (Dohmen et al., 2011), which is relevant for savings, pension schemes, and long run investments in general.

The empirical literature on risk preferences studied the association with gender, which is strongly positively correlated with sex, a biological factor relevant to hormones. In summarizing the sizeable literature, Croson and Gneezy (2009, p. 2) write that "(t)he robust finding is that men are more risk-prone than are women." Similarly, Eckel and Grossman (2008) state that "in most studies, women are found to be more averse to risk than men. Studies with contextual frames show less consistent results".[1] Filippin and Crosetto (2016) strike a more cautionary note in concluding that "gender differences are less ubiquitous than usually depicted." From an evolutionary point of view, gender differences in risk preferences would not be too surprising because males and females play different roles in human reproduction. Males and females differ also in circulating levels of various sex hormones like testosterone, estradiol, and progesterone. Thus, gender differences in risk preferences suggest that risk preferences may also vary by sex hormones.

In this paper, we study the association of risk preferences for both gains and losses with salivary testosterone, estradiol, progesterone, and cortisol. Testosterone is a steroid hormone that has anabolic effects such as stimulating the bone density and muscle mass as well as androgenic effects such as the maturation of sex organs and secondary sex characteristics especially in males. Since it is observed in most vertebrates, it must have had a long evolutionary history (Mechoulam et al., 1984). In humans, testosterone is associated with aggression (e.g. Archer, 1991, Dabbs and Hargrove, 1997) and dominance (e.g. Mazur and Booth, 1998, Mehta and Josephs, 2006) especially in males. More importantly for our study, testosterone has been previously associated with risk preferences. Apicella et al. (2008) find a significant negative correlation between basal

[1] See also Byrnes, Miller, and Schafer (1999) for any earlier review of the literature.

salivary testosterone and risk aversion in choice under risk in a sample of males. Sapienza et al. (2009) do not find a significant association between salivary testosterone and risk aversion in males but a significant negative relationship in females. Stanton et al. (2011a) find a negative association for both genders in a gambling task that had not been incentivized. Stanton et al. (2011b) report an inverse u-shaped relationship between risk attitudes and salivary testosterone in both genders, where individuals with high or low testosterone are approximately risk neutral and individuals with intermediate levels of testosterone are risk averse. Apicella et al. (2015) review the literature including the present study. Kurath and Mata (2018) present a meta-study that includes the present study and argue for a small negative correlation between testosterone and risk aversion. Recently, Stanton et al. (2021) reported no significant association of endogenous testosterone with risk taking in men and women. The evidence is even more mixed if we go beyond correlation studies. Zethraeus et al. (2009) did not find a significant association between randomly administered testosterone and risk-taking in a placebo-controlled experiment with 200 postmenopausal women. Boksem et al. (2014) find significant negative association between randomly administered testosterone and risk aversion in a study with 54 females. Buskens et al. (2016) find no effect of testosterone administration on risk aversion in a study with 84 participants. The literature on causal evidence is reviewed by Dreber and Johannesson (2018). Recently, Schaefer et al. (2022) find no evidence for a causal effect of exogenous testosterone administration on risk aversion in sample of 80 females. In a sample of 40 men, Votinov et al. (2022) find evidence for a negative effect of exogenous testosterone on risk aversion in the gain domain and a smaller positive effect in the loss domain. Stanton et al. (2021) do not find any causal effect of exogenous testosterone in risk taking in two studies involving 30 and 117 male participants, respectively. Nadler et al. (2021) find no effect of exogenous testosterone and risk taking in a sample of 332 men. In our current study, we replicate the finding by Apicella et al. (2008) with a different lottery experiment. That is, we find that in males, endogenous testosterone is significantly positively associated with risk taking in the gain domain of the Holt-Laury lottery tasks. We do not observe an association in the loss domain, which was new. Moreover, we observe null results for females both gains and losses.

We measure risk preferences using a lottery task due to Holt and Laury (2002), which consists of presenting each subject of our experiment with a list of pairs of lotteries. The subject has to make choices between the lotteries for each pair of lotteries in the list (see Section 2.1). The probability of outcomes varies systematically across the list of lottery pairs. While this design has been originally applied to lotteries involving monetary gains only, Laury and Holt (2008) extend it also to lotteries involving monetary losses. This allows for studying postulates of prospect theory (see Kahneman and Tversky, 1979, and Tversky and Kahneman, 1992) such as the reflection effect, i.e., risk aversion for gains and risk seeking for losses.[2]

[2]See Harrison and Ruthström (2008) for a survey on experiments on risk preferences.

While salivary testosterone has been previously studied, we also include salivary estradiol, progesterone, and cortisol in our study. Estradiol is also a steroid hormone that has anabolic effects on the bone structure and androgenic effects on the maturation of female sex organs and secondary sex characteristics. Progesterone is a steroid hormone as well. It is secreted in the ovaries, especially the corpus luteum, the adrenal glands, and during pregnancy in the placenta. There is evidence that increases in salivary progesterone are positively associated with women's avoidance of "social risks" such as infections as reflected in behavior in public bathrooms (Fleischman and Fessler, 2011), increased attention to social stimuli (Maner and Miller, 2014) as well as non-conscious needs to have close, friendly relationships with others (Schultheiss, Wirth, and Stanton, 2004, Wirth and Schultheiss, 2006, Brown et al., 2009). In a companion study, Schipper (2015) finds that salivary progesterone is significantly positively associated with bidding of females in first-price auctions, which in theory is consistent with a positive association of progesterone with risk aversion. Finally, we include in our study the steroid hormone cortisol. Cortisol is correlated with stress (Dickerson and Kemeny, 2004, Hellhammer, Wuest, and Kudielka, 2009) and thus we find it intuitive that it may be correlated with risk preferences as well. Nofsinger et al. (2018) find a negative association between cortisol and risk taking in a simulated portfolio allocation task. Cortisol has been also hypothesized to jointly affect risk taking with testosterone. Mehta and Josephs (2006) and Mehta et al. (2015) suggest what they call a dual hormone hypothesis according to which the effect of testosterone on risk taking should be moderated by the inhibitory function of high cortisol. Several studies on the causal effect of cortisol on risk preferences have recently appeared. Using a double-blind placebo-controlled experiment, Kandasamy et al. (2013) find that exogenous cortisol administration makes subjects more risk averse. Chumbley et al. (2014) find no significant correlation between chronic exposure to endogenous cortisol as measured in hair samples and risk aversion in 53 males.[3] Buckert et al. (2014) show that stress-induction raised risk-taking significantly in subjects who also responded with higher salivary cortisol. Kluen et al. (2017) report that placebo-controlled administration hydrocortisone decreases risk aversion in men but not in women. In our study, we observe that cortisol is significantly positively associated with risk aversion for gains in females only but it is insignificant when controlling for multiple comparison of four hormones. We observe null results for cortisol and risk aversion in the loss domain and for males both in the gain and loss domains. We also observe null results for both estradiol and progesterone and risk aversion. Kurath and Mata (2018) present a meta study of the literature related to testosterone, estradiol, and cortisol and risk taking.[4] They conclude a small correlation between estradiol and risk taking but not for cortisol.

Some sex hormones like progesterone and estradiol vary over the menstrual cycle. Estradiol

[3]They do report a significant effect on loss aversion.

[4]While Kurath and Mata (2018) include results on testosterone of our study, for unexplained reasons they do not consider our null findings for estradiol and cortisol.

peaks during the midcycle and again in the second half of the cycle. Progesterone rises after ovulation. There is some mixed evidence of the association of bidding behavior in first-price auctions with menstrual cycle phases. Recall that in the theory of first-price auctions, bids are positively associated with risk aversion. Pearson and Schipper (2013) show that in first-price auctions women bid similarly to men during ovulation but significantly higher in other phases of their menstrual cycle. Chen, Katuščák, and Ozdenoren (2013) report that women bid higher than men in all phases of their menstrual cycle in a symmetric independent private value first-price auction but not in a second-price auction. In our study, we also elicited menstrual cycle information from female subjects and observe no association of risk preferences for gains and losses with menstrual cycle phases. This is consistent with a study by Drichoutis and Nayga (2015), who also do not find an association of risk preferences with menstrual cycle phases.

Some females in our sample use hormonal contraceptives. All hormonal contraceptives contain a progestin, a synthetic version of progesterone. We find that females who use hormonal contraceptives do not have risk preferences different from naturally cycling females. There is emerging small literature on behavioral effects of hormonal contraceptives. Pearson and Schipper (2013) and Schipper (2015) show that the use of hormonal contraceptives is positively associated with bidding in first-price auctions. Buser (2012a) reports that the use of hormonal contraceptives is negatively associated with selection into competitive environments and that this is not due to risk preferences. Alvergege and Lummaa (2009) survey evidence on how hormonal contraceptives affect mate choice behavior. Women on hormonal contraceptives seem to prefer men with less masculine faces. Any of these correlational observations could be due to hormonal contraceptives or to a selection effect. Ranehill et al. (2018) study causal evidence in a placebo-controlled study with 340 women involving the administration of oral contraceptives. They find no effect of progestin on risk aversion.

Behavior may not just be affected by circulating hormones levels but also by prenatal exposure to certain hormones. We are interested in what sense risk preferences may be influenced by biological events before birth. We use as a putative biological marker the ratio between the length of the 2nd (index) finger and the 4th (ring) finger of the subjects' right hand (so called "digit ratio" or 2D:4D). (See Manning, 2002, for an introduction.) The digit ratio is thought to be positively correlated with prenatal exposure to estrogen and negatively correlated to prenatal exposure to testosterone (Manning et al., 1998, Lutchmaya et al., 2004, Hönekopp et al., 2007, Zheng and Cohn, 2011, Ventura et al. 2013), a hypothesis recently questioned by Hollier et al. (2015) and Kaltwasser et al. (2017). On average, men have lower 2D:4D than women. 2D:4D is to a large extent genetically determined (Paul et al., 2006), but it may also be affected by the environment *in utero*. In any case, 2D:4D is determined before birth and thus before common economic, social, and cultural factors could shape risk preferences of the individual directly. Results on 2D:4D and risk taking is mixed: Some studies find a positive correlation between 2D:4D and risk aversion (Dreber and Hoffman, 2007, Garbarino et al., 2011, Stenstrom et al.,

2011, Brañas-Garza and Rustichini, 2011, Sytsma, 2014, Bönte et al., 2016, Brañas-Garza et al., 2018, Barel, 2019) although such finding are often restricted to subsamples or often would not be considered significant when controlling for multiple of comparison. There are also null results (Apicella et al., 2008, Sapienza et al., 2009, Stenstrom et al., 2011, Aycinena et al., 2014, Sytsma, 2014, Drichoutis and Nayga, 2015, Bönte et al., 2016, Chicaiza-Becerra and Garcia-Molina, 2017, Dalton and Goshal, 2018, Brañas-Garza et al., 2018, Lima de Miranda et al., 2018, Alonso et al., 2018, Barel, 2019, Parslow et al., 2019, Neyse et al., 2020, 2021). Hoffman, Jordan, and Yoeli (2013) present an early meta study of the literature (that includes the present study). Overall they report a significant positive correlation between risk aversion and 2D:4D. A more comprehensive review is provided by Apicella et al. (2015) who conclude that the evidence is ambiguous and discuss potential reasons for that including publication bias. We replicate the null results in the literature using the Holt-Laury lottery tasks both for gains and losses and for both males and females including white and asian subsamples.

Finally, we also study plain gender effects on risk preferences in gains and losses. Surprisingly, there is no gender effect on risk preferences for gains in our sample. Yet, in the loss domain, females are significantly more risk averse than males but this finding is not robust. Moreover, males have significantly more often a unique switch-point in the Holt-Laury lottery task and respect dominant choices.

The paper is organized as follows: The next section introduces the experimental design and hypotheses. The results are presented in Section 3. Section 4 concludes with a discussion. Further details on the methodology and instructions for subjects are relegated to an appendix.

2 Experimental Design

2.1 Holt-Laury Lottery Tasks

We study choice under risk using lottery tasks introduced by Holt and Laury (2002) for the gain domain and by Laury and Holt (2008) for the loss domain. Each lottery task consists of a menu of 10 decisions between pairs of lotteries. Table 1 shows the lottery choices for the gain domain. The first column numbers the decisions. The second and third columns present the pairs of lotteries, named "option A" and "option B", respectively. For each of the 10 choices, each subject had to decide between option A and B, and mark her choice in the fourth column. The fifth and last column is not shown to subjects in the experiment but printed here for convenience of the reader. It shows for each decision the difference between expected payoffs of options A and B.

In Decision No. 1, the choice is between a gain of \$3.20 (option A) and a gain of \$0.20 (option B). A subject respecting dominance should chose option A. Observe that the two payoffs

Table 1: Holt-Laury Lottery Task for the Gain Domain

Decision No.	Option A	Option B	Your Choice	Exp. Payoff A - Exp. Payoff B (not shown)
1	\$3.20 if throw of die is 1 to 10	\$0.20 if throw of die is 1 to 10		\$3.00
2	\$4.00 if throw of die is 1 \$3.20 if throw of die is 2 to 10	\$7.70 if throw of die is 1 \$0.20 if throw of die is 2 to 10		\$2.33
3	\$4.00 if throw of die is 1 or 2 \$3.20 if throw of die is 3 to 10	\$7.70 if throw of die is 1 or 2 \$0.20 if throw of die is 3 to 10		\$1.66
4	\$4.00 if throw of die is 1 to 3 \$3.20 if throw of die is 4 to 10	\$7.70 if throw of die is 1 to 3 \$0.20 if throw of die is 4 to 10		\$0.99
5	\$4.00 if throw of die is 1 to 4 \$3.20 if throw of die is 5 to 10	\$7.70 if throw of die is 1 to 4 \$0.20 if throw of die is 5 to 10		\$0.32
6	\$4.00 if throw of die is 1 to 5 \$3.20 if throw of die is 6 to 10	\$7.70 if throw of die is 1 to 5 \$0.20 if throw of die is 6 to 10		-\$0.35
7	\$4.00 if throw of die is 1 to 6 \$3.20 if throw of die is 7 to 10	\$7.70 if throw of die is 1 to 6 \$0.20 if throw of die is 7 to 10		-\$1.02
8	\$4.00 if throw of die is 1 to 7 \$3.20 if throw of die is 8 to 10	\$7.70 if throw of die is 1 to 7 \$0.20 if throw of die is 8 to 10		-\$1.69
9	\$4.00 if throw of die is 1 to 8 \$3.20 if throw of die is 9 or 10	\$7.70 if throw of die is 1 to 8 \$0.20 if throw of die is 9 or 10		-\$2.36
10	\$4.00 if throw of die is 1 to 9 \$3.20 if throw of die is 10	\$7.70 if throw of die is 1 to 9 \$0.20 if throw of die is 10		-\$3.03

in lotteries under option A have roughly the same magnitude. Thus, lotteries under option A are relatively "safe". The lower the decision in Table 1, the higher becomes the probability for the best outcome \$4.00 for option A and \$7.70 for option B. The optimal choice of a risk neutral individual is to choose option A for the first five decisions and then switch to option B

for decisions 6 to 10 as the expected value is higher for A than B in the first five decisions, while the expected value for B is higher than A for decisions 6 to 10 (see last column). A sufficiently risk averse individual tends to switch to option B after Decision No. 6, while a sufficiently risk seeking individual switches to option B before Decision No. 6.

The lottery task for the loss domain is analogous to Table 1 except that gains are replaced with corresponding losses (see Appendix F) and thus the signs of differences in expected payoffs of the last column are reversed. A risk neutral individual will start out in Decision No. 1 with option B and switch to option A from Decision No. 6 onward. A sufficiently risk averse individual will switch to option A after choosing option B for less than the first five decisions, while a sufficiently risk seeking individual will switch to option A after Decision No. 6.

In both, the gain and loss domains, risk neutrality implies choosing option A five times, sufficient risk aversion implies choosing option A more than five times, while sufficient risk seeking implies choosing option A less than five times. Thus, as a matter of terminology, we say that an individual is *risk averse* if she chooses option A more than five time, *risk neutral* if she chooses option A exactly five times, and *risk seeking* if she chooses option A less than five times. We say that a group X of subjects is *more risk averse (resp. more risk seeking)* than a group Y if on average it chooses option A more often (resp. less often) than group Y.

It is possible to fit for each domain the corresponding interval of risk parameters for popular utility functions such as constant relative risk aversion (see Holt and Laury, 2002, Laury and Holt, 2008, Harrison and Ruthström, 2008). But we believe that for this correlation study it would add nothing beyond our behavioral definitions of risk aversion and risk seeking behavior above.

Kahneman and Tversky (1979) and Tversky and Kahneman (1992) claim that frequently individuals are risk averse in the gain domain while risk seeking in the loss domain. This is called the reflection effect. In our experiment, we can study the reflection effect because we conduct lottery tasks both in the gain and loss domains. In our setting, a subject is said to display the *reflection effect* if she chooses option A more than five times in gain domain and less than five times in the loss domain.

Not all subjects may display a unique cut-off for switching between the options but may switch several times between options A and B. Moreover, a subject may not respect dominance and thus may not choose option A and B in Decision No. 1 in the gain and loss domain, respectively. If we observe a subject switching several times, we should not dismiss the preferences too quickly as being "inconsistent". We simply do not know why it switches several times. It could be that the subject is just indifferent and that is why when forced to make a choice may switch between options several times. It could also be that the subject is not indifferent but simply makes a mistake in writing down the wrong choice. Finally, it could be that the subject "happily violates" the assumptions underlying the maximization of expected utility. In any case, the

information obtained from subjects who switch several times or choose the dominated option is limited. That's why we call a subject's preference *accessible* if the subject has an unique cut-off for switching between options and respects dominance. Otherwise, we call the subject's preferences *inaccessible*.

Appendix F contains the instructions for the lottery task provided to subjects of our experiment.

2.2 Experimental Design

Subjects were recruited from the campus of the University of California, Davis, using the ORSEE recruitment system by Greiner (2004). Since our experiment also included auction games, it was advertised as a "market game" mostly via announcements in big classes, in advertisements on Facebook, and through the distribution of leaflets. All sessions were run between February 8 and March 16, 2010, at 16:00. Upon arrival at the lab, subjects were seated randomly at one of nine desks with computer terminals separated by dividers. Each subject faced the wall of the room. Subjects were given a consent form to read and sign. At every session, the same male native-English speaking experimenter was present to explain the instructions and supervise the experiment. Every session of the experiment was divided into eight phases:

1. First Saliva Sample: Subjects received written instructions for saliva sampling (see Appendix B.2) and a styrofoam cup that contained a 4.5 ml sterile Nunc Cryo Tube© vial. The cup functioned simply as a container to prevent the vial from falling over. Each vial had been labeled prior to the experiment with the session and subject number. Subjects also received one piece of chewing gum - Trident© Original Flavor - to stimulate saliva (see Dabbs, 1991) as well as a sterilized plastic straw through which to drool about 2.5 ml saliva into the vial. After depositing the saliva, each subject closed the vial by screwing the top and placed it back into the cup. The cups with the vials were collected by the experimenter and immediately frozen. Further details of the salivary hormone methodology are contained in Appendix B.

2. Holt-Laury Lottery Task: Subjects received written instructions on the Holt-Laury lottery task (see Appendix F). Subject had five minutes to read the instructions. Then the experimenter publicly explained the task to all subjects, after which any questions were answered also in public. The task was conducted on paper-sheets for both gains and losses. All subjects made decisions in private first for the gain domain and only then for the loss domain.[5] In order to eliminate as much as possible any wealth effect on the following tasks, the lotteries were not played out immediately after completing the tasks. After all subjects completed their choices,

[5]Laury and Holt (2008, p. 9) claim that the order of these tasks do not matter. However, their experiment differs from ours in that their tasks were separated by the play of a matching pennies game and additional Holt-Laury lottery tasks with varying payoffs were included.

the paper sheets were collected by the experimenter.[6]

3. Auction Game: Each subject received printed instructions for the auction game. The auction game itself was computerized. The correlation between bidding and profits in those auctions and salivary hormones is analyzed in detail in a companion paper, Schipper (2015), where the instructions can be found as well. Since the auction game could not affect behavior in the prior lottery task (except for expectations about future earnings in the auction game), it will not be discussed here any further.

4. Questionnaire: After the auction task, subjects completed a computerized questionnaire (see Appendix G). This questionnaire elicited demographic information, menstrual cycle information, the use of hormonal contraceptives, information relevant for assessing the quality of saliva, information on sexual orientation and sexual behavior, social lifestyle, personality characteristics, emotions during the experiment, dietary preferences, academic grades, etc. some of which are used as controls in our analysis. See Appendix B.5 for an analysis of some of those factors with salivary hormones.[7]

5. Playing out the Holt-Laury Lottery Task: Once subjects finished the questionnaire, the previously completed paper-sheets on the Holt-Laury lottery tasks were played out in front of the subjects. For each subject, a ten-sided die was rolled four times. The first roll decided which binary choice in the gain domain is selected. The second roll played out this lottery in the gain domain. The third roll decided which binary choice in the loss domain is selected. The final fourth roll played out this lottery in the loss domain. Payoffs for each subject were noted on the decision sheet of each subject.

6. Hand Scan: After playing out the lottery tasks, each subject's right hand (and the right hand only) was scanned with a conventional office image scanner. The purpose of the hand scan is to measure the length of the 2nd and 4th finger and analyze the digit ratio (2D:4D). The second and fourth digits were later measured independently by two separate researchers from the center of the flexion crease proximal to the palm to the top of the digit using the measurement tools in Adobe Photoshop and Gimp. When measuring the fingers, the researchers did not know whether the hand belong to a male or female subject or how this subject behaved in the experiment. The measures used here are based on the averages of both measurements for each finger of each subject respectively. In Appendix D, we present the intraclass correlations between the two measurements.

7. Second Saliva Sample: About 20–30 minutes after the auction task, subjects were asked for a second saliva sample in the same manner as for the first saliva sample. Since it takes

[6]In a companion paper, Schipper (2015) uses the data on the lottery task to control for risk aversion in estimations of bidding behavior in auctions.

[7]The extensive questionnaire was also used to generate a sufficiently long waiting period between the auction task and the collection of the second saliva sample.

about 15 to 30 minutes before effects on hormones become measurable in saliva (see for instance Schultheiss et al., 2005, Kivlighan, Granger, and Booth, 2005, Edwards and O'Neal, 2009, Saad and Vongas, 2009) and only less time had passed between playing out the Holt-Laury lottery tasks and the second saliva sample, this data will not be analyzed here.[8]

8. Payment: At the end of the experiment, subject received in private their total cash payment from the show up fee, the auction task, and the lottery tasks. The average total earning was US$ 19.03 with a maximum of US$ 48.39 and a minimum of US$ 5.00. The entire procedure took about 1 hour and 20-30 minutes. The average earning is above what a typical student job would earn in Davis for about the same time. Our lottery experiment may involve losses as well. Losses can typically not be collected from subjects. Yet, subjects knew that they can earn also money in the gain domain of the lottery task as well as from the auctions.

2.3 Hypotheses

Gender Differences: Robson (1996) shows in an evolutionary model that some males may gamble and females behave strictly risk averse. Moreover, as discussed in the introduction, the received view in the prior literature is that females are more risk averse than males. Thus, we hypothesize that females are more risk averse than males in both the gain and loss domains.

Hormonal Contraceptives: All hormonal contraceptives contain a progestine, a synthetic version of the sex hormone progesterone. Some contain also a version of estradiol, another sex hormone. Progesterone may have a sedating effect (see Pluchino et al., 2006, van Broeckhoven et al., 2006).[9] Throughout the human brain, there are neurons secreting the neurotransmitter GABA. When GABA interacts with other neurons, they are inhibited which means that they are less likely to "fire". Well-known benzodiazepines drugs like Valium, Librium, and Xanax enhance the inhibitory effect of GABA, thus reducing anxiety and having a calming and sedating effect. The same has been observed for some metabolites of progesterone, i.e., substances converted from progesterone in the body. We hypothesize that the calming effect of progestines in hormonal contraceptives may make females more risk averse in both the gain and loss domain. Moreover, because of the sedating effect we hypothesize that they have less accessible risk preferences. Note however that we cannot claim a causal effect since any effect may be due to selection. In particular, women who decide to take hormonal contraceptives may also differ systematically in their risk preferences from women who decide not to take any hormonal contraceptives. It is not clear whether a priori more risk averse women are more likely to use hormonal contraceptives or

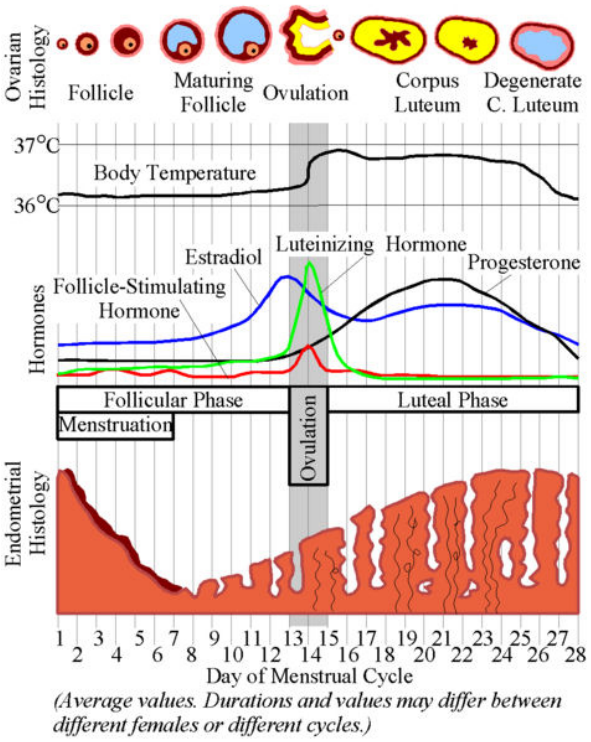
[8]The purpose of including a second saliva sample into the experiment was to study salivary hormone responses to profits in the auctions; see Schipper (2015). We can use the second saliva sample to access measurement errors for estradiol and progesterone; see Appendix B.6.

[9]We thank Coren Apicella (private communication) for drawing our attention to the connection between progesterone and $GABA_A$.

whether women with more risky sexual behavior are more likely to take hormonal contraceptives. Conclusive evidence could be obtained in an experiment in which oral contraceptives or a placebo are double-blindly and randomly assigned to women. Obviously, such an experiment is rather difficult to conduct. Moreover, women who would agree to participate in such a "risky" experiment may systematically differ in their risk preferences from the rest of the population. See Ranehill et al. (2018) for such a double-blind placebo-controlled study reporting a null result.

Menstrual Cycle: Fecundity varies over the menstrual cycle. It is not clear from an evolutionary point of view why risk aversion should stay constant throughout the menstrual cycle since gains and losses of risky behavior vary of the cycle. On one hand, risky behavior may lead to a higher probability of conception, genetic diversity and higher quality offspring through extrapair mating. This may be especially successful in monogamous societies where some females must be matched with genetically substandard males. Thus females with risky behavior near ovulation may have a higher reproductive success. On the other hand, extrapair mating involves the risk of losses because if discovered it is punished severely in most societies and may lead to a loss of the long term mating partner who supports child rearing.[10] This is significant because males of higher genetic quality tend to have poorer parental qualities (Gangestad and Simpson, 2000). These arguments suggest that in order to maximize fitness over the menstrual cycle, a female should have the highest propensity for extrapair mating during her fecund period. Bressan and Stranieri (2008) show that partnered women favor single men with more masculine features during their fecund phase, while they prefer attached men during their

Figure 1: Menstrual Cycle



http://en.wikipedia.org/wiki/Image:MenstrualCycle2.png

[10]There is some evidence for greater mate guarding near ovulation (see Gangestad, Thorndill, and Garver, 2002, and Haselton and Gangestad, 2006), which may be a long-term male mate's best response to riskier behavior of the female during her fecund window and may in turn require more risky behavior of females to escape the guard. Yet, in humans, ovulation is concealed (to some extent even to the female herself) and thus time-targeted mate guarding must be rather limited.

low-fecundity phase.[11] Wilcox et al. (2004) show that the frequency of intercourse increases during the fecund period.[12] We hypothesize that females may behave more risky during their fecund period of the menstrual cycle, the peri-ovulatory phase.

The hypothesis that women may seek more risks during their fecund phase of their cycle seems to contradict Bröder and Hohmann (2003), who report that women avoid taking risks near ovulation in order to reduce the chance of being raped (see also Chavanne and Gallup, 1998, for an earlier study).[13] In contrast, Fessler (2003) argues that rape is not less frequent during the ovulatory phase. The experiment by Bröder and Hohmann (2003) did not discriminate between risks in the gain and loss domains. Differences in risk preferences between the gain and loss domains have been discussed since the emergence of prospect theory (Kahneman and Tversky, 1979). Such risk preferences may be relevant in an evolutionary context (see Hamo and Heifetz, 2002, for some evolutionary arguments for such s-shaped utility functions). It is conceivable that women are more likely to avoid the risk of losses during the fecund phase but are also more likely to embrace risk of gains.

Some hormones such as progesterone vary across the menstrual cycle (see Figure 1) (e.g., Chatterton et al., 2005). For instance, progesterone rises during the luteal phase after ovulation and declines before menstruation. As discussed above, there is evidence that increases in progesterone during the luteal phase are positively associated with women's avoidance of infections as reflected in behavior in public bathrooms (Fleischman and Fessler, 2011). Increases in progesterone during the luteal phase are also associated with increased accuracy in decoding facial expressions and increased attention to social stimuli (Maner and Miller, 2014) as well as non-conscious needs to have close, friendly relationships with others (Schultheiss, Wirth, and Stanton, 2004, Wirth and Schultheiss, 2006, Brown et al., 2009). Such dispositions could reduce social risks. Since increases in progesterone in the luteal phase prepare the body for pregnancy. Minimizing the risk to a potential fetus at the same time could yield an evolutionary advantage. Thus, we hypothesize that the luteal phase is positively associated with risk aversion.

Salivary Hormones: We use hormone measurements of the first saliva sample to analyze associations with risk preferences. In the introduction, we discussed the somewhat mixed prior evidence on testosterone and risk preferences in the literature. Nevertheless, we hypothesize a negative correlation between salivary testosterone and risk aversion.

With regard to estradiol, we hypothesize that if there is any association, then it is similar to testosterone but mainly in naturally cycling females.

[11] For related evidence, see Gangestad, Thornhill, and Garver-Apgar (2006), Penton-Voak et al. (1999), and Penton-Voak and Perrett (2000).

[12] In Wilcox et al. (2004), evidence is provided only for women in stable relationships.

[13] I am grateful to Arndt Bröder who privately communicated to me that in a later study he could not replicate the results of Bröder and Hohmann (2003).

We hypothesize that progesterone is positively correlated with risk aversion in naturally cycling females only. The hypothesis is based on two alternative explanations. First, as we discussed earlier, progesterone may have a sedating or calming effect (see Pluchino et al., 2006, van Broeckhoven et al., 2006) because metabolites of progesterone enhance the inhibitory effect of GABA. Thus analogous to progestins in females using hormonal contraceptives discussed earlier, we believe that progesterone may make naturally cycling females more risk averse in both the gain and loss domains. The second explanation is based on the association between the luteal phase and risk to a potential fetus that we referred to in the hypotheses on the menstrual cycle. As discussed above, there is evidence that females try to minimize risk to a potential fetus in the second half of the menstrual cycle (Fleischman and Fessler, 2011, Maner and Miller, 2014, Schultheiss, Wirth, and Stanton, 2004, Wirth and Schultheiss, 2006, Brown et al., 2009). This is the phase when progesterone peaks (see Figure 1). Thus, progesterone should be positively associated with risk aversion in naturally cycling females. Moreover, because of the sedating effect of progesterone, we hypothesize that progesterone is negatively associated with the accessibility of risk preferences in naturally cycling females.

Cortisol responds positively to stress (see Dickerson and Kemeny, 2004, and Hellhammer, Wuest, and Kudielka, 2009) and concentrates the body's functions on dealing with the stressor. Thus, additional stressors like a risk should be avoided. Therefore, we hypothesize that risk aversion is positively correlated with stress and cortisol.

Digit Ratio: Based on the mixed evidence in the literature discussed in the introduction, we hypothesize that if there is a relationship, then the digit ratio should be positively correlated with risk aversion in both males and females.

Table 2: Basic Demographics

Variable	Number	Mean	Std. Dev.
Subjects	208		
Female	93	0.45	
Age	208	20.36	2.24
White	79	0.38	
Asian	116	0.55	
Hispanic	13	0.06	
Black	5	0.02	
Others	8	0.04	
GPA	202	3.17	0.52
Math	5	0.02	
All Sciences	61	0.29	
Economics	103	0.50	
Other Social Sciences	65	0.31	
Humanities	20	0.10	
Pregnant	1		
Homo- or Bisexual	14	0.07	

3 Results

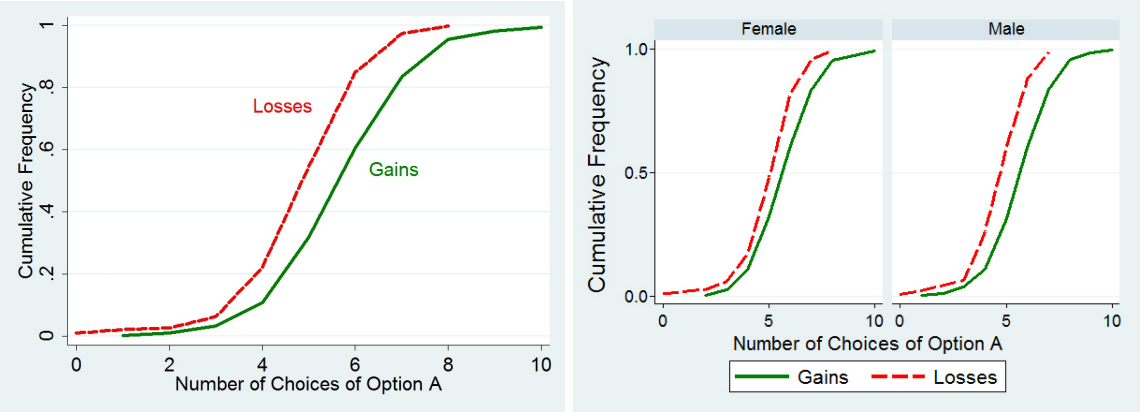
The datasets and Stata do-file that reproduce the entire analysis reported here and additional analysis are available from `https://faculty.econ.ucdavis.edu/faculty/schipper/`.

Table 2 presents the demographics of the

data as elicited with the questionnaire (see Appendix G).[14] We had 208 subjects in sessions of 8 subjects each. Out of the 208 subjects, 93 (45%) are female. Most of the subjects are Asian-Americans (55%) followed by Whites (38%).[15] Six subjects (three females and three males) did not provide their GPA. One woman reported that she is pregnant. Since circulating levels of various steroids change during pregnancy, we exclude her from our analysis of salivary hormones and the menstrual cycle information but not in our analysis of gender differences and the digit ratio.

The left panel of Figure 2 shows the cumulative frequency of option A for both the gain and the loss domain. Recall that both in the gain and loss domain, a risk neutral individual would choose option A exactly five times. If all subjects were risk neutral, the cumulative frequency would be zero up to five and one thereafter. However, we see that both for gains and losses some subjects choose less than five times option A while others choose more. This suggest that some subjects may be risk seeking while others are risk averse. The cumulative frequency for the gain domain is below the one for the loss domain indicating that subjects' choices may be relatively more risk averse in the gain domain as compared to the loss domain. This finding is consistent with Laury and Holt (2008).

Figure 2: Cumulative Frequency for the Entire Sample (left panel) and By Gender (right panel)



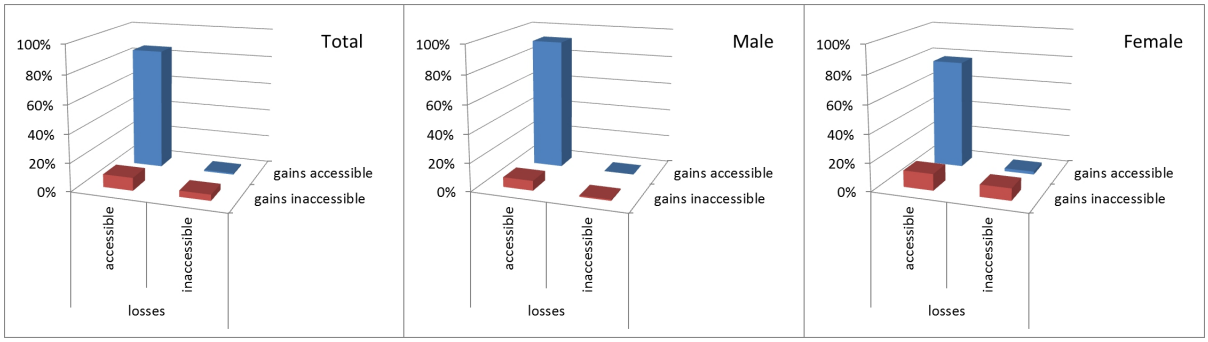
The left panel of Figure 2 considers choices by all subjects including subjects whose preferences are inaccessible. There are a few inaccessible subjects who do not have an unique cut-off for

[14]Subjects were allowed to select multiple majors and ethnic backgrounds. Thus, the means do not add up to unity. In our sample all math majors happened to be male.

[15]For comparison, the distribution of races among all UC Davis students at 2010 was 42% White, 38% Asian, 3% Black, 14% Hispanic, and 3% Other. We do not know why we have a larger fraction of Asians in our sample. It could be that relative more asians are enrolled in majors that we reached with our advertisement. In particular, about 59% of economics students at UC Davis are asian. Another reason could be that Asians were more attracted to our experiments. For instance, Loo et al. (2008) surveying the literature on Chinese gambling find that gambling is widespread preferred form of entertainment among Chinese.

switching between options A and B or do not respect dominance. The left panel in Figure 3 shows that most of our subjects, 178 out of 208 (86%), have accessible risk preferences both in the gain and loss domains. However, slightly more subjects are accessible in the loss domain than in the gain domain ($t = -3.8296$, $p < 0.001$, one-sided). This holds both for men ($t = -2.9195$, $p = 0.002$, one-sided) and women ($t = -2.5703$, $p = 0.006$, one-sided). We do not know whether this is due to the fact that subjects had to choose among the loss lotteries *after* they chose among the gain lotteries and thus had more experience in thinking through the problem or whether subjects are simply more careful in making choices when it comes to losses. As a comparison, Laury and Holt (2008) observe that 72% of their subjects have accessible risk preferences.

Figure 3: Fraction of Subjects with (In-)accessible Preferences



We can classify all accessible subjects approximately into risk averse, risk seeking, and risk neutral according to whether they choose option A more, less or exactly five times, respectively (see Section 2.1). The left panel in Figure 4 shows the risk attitudes for both the gain and the loss domain. The modal subjects (34) are risk neutral both in the gain and loss domains followed closely by 33 subjects who are risk averse in the gain domain and risk seeking in the loss domain. Latter subjects display the reflection effect (see Section 2.1). These findings are somewhat different from Laury and Holt (2008). In their treatment corresponding to ours, they observe that the modal subject exhibits risk aversion both in the gain and loss domains.

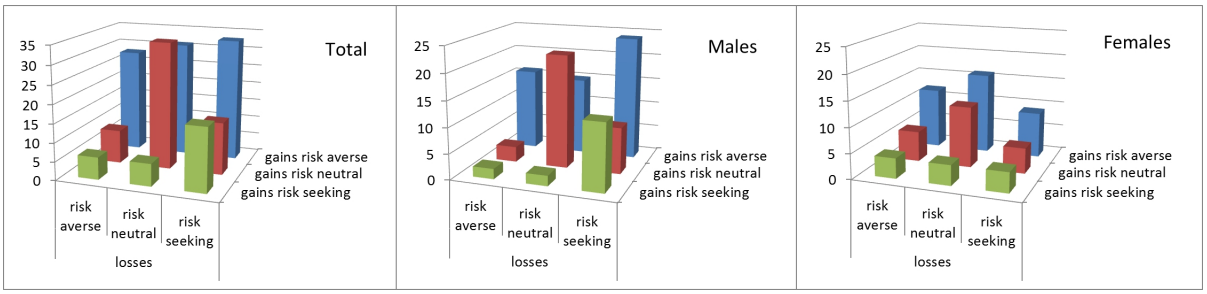
3.1 Gender Effects

We will estimate versions of the following parametric model

$$r_i = \beta_0 + \beta_1 g_i + \gamma Q_i + \varepsilon_i \tag{1}$$

where i is the index of the subject, r_i is the number of choices of option A by subject i (in either the gain domain or the loss domain), β_0 is a constant, g_i is a dummy for the gender of individual i taking on 1 if i is female and 0 otherwise, Q_i is a subset of questionnaire variables

Figure 4: Number of Subjects by Risk Attitudes



including age, race, gpa, and ε_i is an unobserved error term of subject i. With regard to race, we leave out white as the omitted category. This model will be estimated with ordinary least squares method (OLS). To account for heteroscedasticity, we use robust standard errors in all regressions.[16] We also estimate specifications analogous to equation (1) where we replace the number of choices of option A by either a binary variable for reflection or a binary variable for accessibility as dependent variable. Because of the binary dependent variable, we will then estimate the model with logit.

The middle and right panels of Figure 4 suggest that relatively fewer women than men are risk seeking in the loss domain while in the gain domain women and men behave quite similar. This observation is confirmed in Table 3. In specification G0A, we regress the number of choices of option A for subjects with accessible preferences in the gain domain on gender and a subset of demographic variables using OLS. We do not find a significant gender effect ($p = 0.617$). This is in contrast to the analogous specification L0A for the loss domain. Here being female is significantly positively correlated with risk aversion ($p = 0.019$).

Table 3: Gender Effects for Gains & Losses

	(G0A)	(G0)	(G1A)	(L0A)	(L1)	(L1A)
Female	0.1153	0.1988	−0.0246	0.4290*	0.4058*	0.3139
	(0.2304)	(0.2179)	(0.2138)	(0.1812)	(0.1804)	(0.1850)
Demographics	Yes	Yes	Yes	Yes	Yes	Yes
Majors of Study	Yes	Yes	No	Yes	Yes	No
Number of Observations	174	202	174	191	202	191
R^2	0.0550	0.0357	0.0208	0.1059	0.0995	0.0434

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

[16]For our regression specifications we use the following name conventions: "G" (resp. "L") signifies that the dependent variable is the number of choices of option A in the *gain* (resp. *loss*) domain. "R" stands for (the binary variable) *reflection*, while "A" stands for (the binary variable) *accessibility*. "F" will indicate the *female* subsample and "M" the *male* subsample.

Specifications G0A and L0A just consider choices of accessible subjects in the gain and loss domain, respectively. The middle and right panels of Figure 3 reveal that preferences of men are slightly more accessible than preferences of women. About 77% of females have accessible risk preferences, while for males it is 92%. Specification A0 in Table 4 confirms that on average preferences of women are significantly less accessible ($p = 0.012$). In this specification, we regress the binary variable for accessibility of risk preferences on gender and a subset of demographic variables using logit. As we explained in Section 2.1, we do not know why subjects' preferences are inaccessible. It could be that the subject is not maximizing expected utility. But it could also be that a subject is indifferent or just makes a mistake in filling out the decision sheet. In the last two cases, we would "misclassify" subjects if we consider them as inconsistent although they actually have consistent risk attitudes. Moreover, it could be that such misclassification is correlated with a particular risk attitude and that this misclassification is especially prevalent in females. Thus, when we dropped inaccessible subjects in specifications G0A and L0A, we may have introduced a selection bias. To check for such a bias, we estimate analogous specifications G0 and L0 in which we consider as dependent variable the number of choices of option A of all subjects no matter whether their risk preferences are accessible or inaccessible. This increases the number of observations but presumably also adds some noise. Our results remain qualitatively unchanged.

Another reason for why we may not find a gender effect in specifications G0A and G0 is that regressors are correlated. In particular, females may select on average into different majors than males. For instance, all of our five math majors are male and 68% of our econ majors are male. Therefore in specifications G1A and L1A we drop the major of study. There is still no significant gender effect for gains ($p = 0.909$). Moreover, the gender effect for losses ceases to be significant ($p = 0.091$), showing that the findings in specifications L0A and L0 are not robust.

Table 4: Gender Effects for Reflection & Accessibility

	(R0)	(R1)	(A0)	(A1)
Female	−0.7108	−0.8193	−1.1744*	−1.1380**
	(0.5119)	(0.4560)	(0.4666)	(0.4319)
Demographics	Yes	Yes	Yes	Yes
Majors of Study	Yes	No	Yes	No
Number of Observations	172	172	197	202
Pseudo R^2	0.0912	0.0421	0.0813	0.0757

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

The right panel of Figure 2 shows that females' risk preferences in both the gain and loss domains are slightly more similar to each other than males' risk preferences. The middle and right panels of Figure 4 seem to suggest that relatively fewer women display the reflection effect. Yet, when we regress for accessible subjects the binary variable for the reflection effect

on gender and a subset of demographic variables using logit in specification R0 (see Table 4), we do not find a significant gender effect ($p = 0.165$). Again, it could be that variables for the major of study absorb some of the gender effect since some majors are more popular for men than for women. When we drop the dummy variables for major of study in specification R1 in Table 4, the coefficient for female remains insignificant ($p = 0.072$). We summarize our findings as follows:

Observation 1 (Gender effects) *In contrast to our hypothesis, we do not find significant gender effects for risk aversion in the gain domain. In the loss domain, females are significantly more risk averse than males but only when we control for majors of study. Preferences of females are significantly less accessible than males.*

3.2 Hormonal Contraceptives

Roughly 23% of women in our sample administer hormonal contraceptives. This number is reasonable given the age of women and their ethnic background.[17]

Table 5: Contraceptives and Gains & Losses

	(G2F)	(L2F)	(R2F)	(A2F)
Contraceptives	−0.4312	0.2357	−0.9121	1.5390*
	(0.3124)	(0.2918)	(0.9053)	(0.7020)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	71	80	69	90
R^2	0.0717	0.0646		
Pseudo R^2			0.1219	0.1036

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

We amend our earlier regression approach (equation (1)) by including also a dummy for the use of hormonal contraceptives and restrict to the subsample of females with accessible risk preferences only. In Table 5 we present results from regressions of the number of choices of option A on hormonal contraceptives and a subset of demographic variables using OLS. Specification G2F applies to the gain domain while specification L2F is on the loss domain. The use of hormonal contraceptives is not significant ($p = 0.172$ and $p = 0.422$ for G2F and L2F, respectively).

The logit specification R2F in Table 5 reveals that the use of contraceptives is not significantly

[17]The United States Department of Health and Human Services (2010) estimates that in the US roughly over 11% of asian, hispanic, and black women between 15 to 44 years of age use oral contraceptives compared to over 21% of white women. The use of oral contraceptives varies also with age. In the age group 15 to 19, it is slightly over 15%, while it increases to 26% in the age group 20 to 24. Note that the mean age of women in our sample is 20.1 years.

correlated with the reflection effect in the female subsample ($p = 0.314$). In contrast to our hypothesis, the logit specification A2F in Table 5 shows that women who use hormonal contraceptives are significantly more likely to have accessible risk preferences than women who do not take hormonal contraceptives ($p = 0.028$). Again, we like to emphasize that we are unable to disentangle a causal effect from a selection effect.

Observation 2 (Hormonal Contraceptives) *Contrary to our hypothesis, females who use hormonal contraceptives do not show risk attitudes differently from naturally cycling females. However, in contrast to our hypothesis, risk preferences of females on hormonal contraceptives are significantly more accessible than risk preferences of naturally cycling females.*

Table 6: Empirical Distribution of Menstrual Cycle Phases and Contraceptive Use

Menstrual Cycle Phases	Days	28-Days Stand.		Uniform Adj.		Fol. Phase Adj.		Expected
		Number	Mean	Number	Mean	Number	Mean	Frequency
Menstrual Phase	Days 1 - 5	17	0.18	16	0.17	17	0.18	0.14
Follicular Phase	Days 6 - 13	12	0.13	16	0.17	16	0.17	0.22
Peri-Ovulatory Phase	Days 14 - 15	6	0.07	6	0.07	5	0.05	0.05
Luteal Phase	Days 16 - 23	19	0.21	19	0.21	19	0.21	0.22
Pre-Menstrual Phase	Days 24 - 28	17	0.18	14	0.15	15	0.15	0.14
Total		71	0.77	71	0.77	71	0.77	0.77
Hormonal Contraceptives		21	0.23	21	0.23	21	0.23	0.23

3.3 Menstrual Cycle

Women differ from men in circulating levels of certain hormones, and some of those hormones change across the menstrual cycle. Estradiol, progesterone, the lutenizing hormone, and the follicular stimulating hormone all change over the menstrual cycle (see Figure 1).[18] Thus menstrual cycle information provides relatively easy to observe within-female measures of some hormones.

From all female subjects, we obtained information about their menstrual cycle. Table 6 presents the distribution across menstrual cycle phases for naturally cycling women. Women who take hormonal contraceptives do not have a natural menstrual cycle, and their circulating

[18]The lutenizing hormone and the follicular stimulating hormone are glycoproteins that cannot be measured in saliva.

levels of hormones may differ from naturally cycling women.[19] Therefore we consider for the classification of women into menstrual cycle phases only women who *do not* take hormonal contraceptives.

For the 28-days standardized menstrual cycle phases (third and fourth columns in Table 6), we distinguish between the menstrual phase (days 1 to 5), the follicular phase (days 6 to 13), the peri-ovulatory phase (days 14 to 15), the luteal phase (days 16 to 23), and the premenstrual phase (days 24 to 28). One major implicit assumption behind the standardized 28-day menstrual cycle is that *all* women follow a *menstrual cycle of exactly 28 days*. Yet, we find substantial variation in usual cycle length in our sample. All 72 naturally cycling women reported their usual cycle length. The average is 29.5 days with a standard deviation of 3.24.[20] Further noise may be introduced through intrapersonal variability in cycle length. The length of the menstrual cycle may vary from cycle to cycle even within the same woman, and the woman can not know the exact length of her *current* menstrual cycle. Finally, there is measurement error due to imperfect recall. Self-reports may be inaccurate (Small, Manatunga, and Marcus, 2007) and this inaccuracy may depend on the day of the menstrual cycle. Menstruating women usually know that they are menstruating while later in the cycle women may not remember exactly their first day of their menstrual cycle. This raises the question whether estimation results will be robust to slight changes in the definitions of the menstrual phases.

Fortunately, we can use the information collected on the usual length of the menstrual cycle to construct more individualized menstrual cycle measures as in Pearson and Schipper (2013). *Individualized phases* are constructed in two ways: uniformly adjusted phases and follicular adjusted phases.

Uniformly Adjusted Phases: We uniformly adjust the phases by the individual length of the menstrual cycle. Let

$$x_i := \frac{\text{Subject } i's \text{ number of days since the first day of the last menstruation period}}{\text{Length of subject } i's \text{ typical menstruation cycle}}.$$

We define the female subject i to be in the

1. *Uniformly Adjusted Menstrual Phase* if and only if $x_i \leq \frac{5.5}{28}$,
2. *Uniformly Adjusted Follicular Phase* if and only if $\frac{5.5}{28} < x_i \leq \frac{13.5}{28}$,

[19] See Briggs and Briggs (1972), Kjeld et al. (1976), Wiegratz et al. (1995), Coenen et al. (1996), Spona et al. (1996), Kirschbaum, et al. (1999), Schultheiss et al. (2003), Edwards and O'Neal (2009), and Liening et al. (2010). The withdrawal bleeding during the "pill break" should not be confused with the menstrual phase; see Fritz and Speroff (2011).

[20] Regarding the "Length Menstrual Cycle", answers of "> 35 days" have been normalized to 37 days. Answers "< 25 days" have been normalized to 24 days. Our estimations are robust to small changes of those upper and lower bounds.

3. *Uniformly Adjusted Peri-ovulatory Phase* if and only if $\frac{13.5}{28} < x_i \leq \frac{16.5}{28}$,

4. *Uniformly Adjusted Luteal Phase* if and only if $\frac{16.5}{28} < x_i \leq \frac{23.5}{28}$,

5. *Uniformly Adjusted Premenstrual Phase* if and only if $\frac{23.5}{28} < x_i$.

Follicular Adjusted Phases: Hampson and Young (2008) write "The length of the luteal phase is relatively fixed at 13 to 15 days. Therefore, most of the variation in cycle length from woman to woman is attributable to differences in the length of the follicular phase." Thus, we consider adjusting the length of the follicular phase only. As in Pearson and Schipper (2013), we start by redefining recursively the last three phases starting with the last phase. Let y_i be subject i's the number of days since the first day of the last menstrual cycle, and d_i the average duration of i's menstrual cycles. Female subject i is in the

1. *Follicular Adjusted Premenstrual Phase* if and only if $y_i > d_i - 5$,

2. *Follicular Adjusted Luteal Phase* if and only if $y_i > d_i - 13$ and i is not in the Follicular Adjusted Premenstrual Phase,

3. *Follicular Adjusted Peri-ovulatory Phase* if and only if $y_i > d_i - 16$ and i is not in the Follicular Adjusted Premenstrual Phase or the Follicular Adjusted Luteal Phase.

Next, female subject i is in the

4. *Follicular Adjusted Menstrual Phase* if and only if i is in the Menstrual Phase.

Finally, female subject i is in the

5. *Follicular Adjusted Follicular Phase* if and only if i is not in the Follicular Adjusted Menstrual Phase, Follicular Adjusted Peri-ovulatory Phase, Follicular Adjusted Luteal Phase or Follicular Adjusted Premenstrual Phase.

Table 8, columns 5 to 8, show the empirical distribution of uniformly adjusted and follicular adjusted menstrual cycle phases in our naturally cycling females, respectively. The distributions differ slightly. For comparison, we report in the last column of Table 8 the expected frequency of natural menstrual cycle phases assuming an uniform probability to participate in the experiment at any day of a 28 days standard menstrual cycle conditional on 23% of the female population taking hormonal contraceptives.

In Figure 5 we plot the female/male differences in the number of choices of option A for the gain and the loss domain, respectively. The left panel for the gain domain seem to suggest that women become less risk averse towards the midcycle when fecundity is highest. But the

Figure 5: Female/Male Difference in Risk Aversion over the Menstrual Cycle

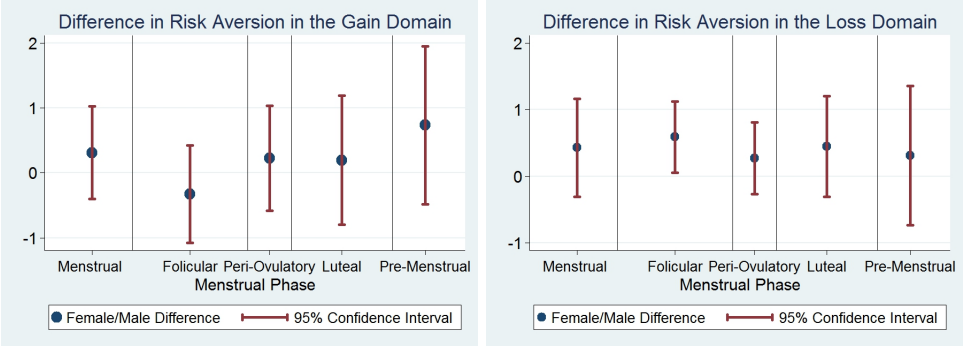


Table 7: Menstrual Cycle Phases

	(G5)	(L5)	(R5)	(A5)
Fol. Adj. Follicular Phase	−0.7711	−0.0932	0.3975	−0.1305
	(0.5008)	(0.4314)	(1.1085)	(0.8339)
Fol. Adj. Peri-ovular Phase	−0.6558	−0.6395	0.0000	−1.6947
	(0.5775)	(0.4676)	(.)	(1.2659)
Fol. Adj. Luteal Phase	−0.3012	−0.1002	0.0000	−0.2827
	(0.6288)	(0.5487)	(.)	(0.7942)
Fol. Adj. Premenstrual Phase	0.2321	−0.3714	1.2640	0.0625
	(0.6670)	(0.6504)	(1.2306)	(0.8852)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	51	60	34	68
R^2	0.1178	0.0803		
Pseudo R^2			0.1590	0.0611

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

95% confidence intervals make also clear that noise is quite high. No such cyclic tendency is observed for losses.

We amend our earlier regression approach (equation (1)) further by including also dummies for menstrual cycle phases and restricting to the subsample of naturally cycling females only. We leave out the menstrual phase as omitted category. Our analysis involves *multiple comparison* of four menstrual cycle phases (in addition to the omitted menstrual phase). The chance of falsely observing one phase to be significantly correlated with risk preferences is much higher when four phases are analyzed as compared to when, from the beginning, just one variable is analyzed. Thus, the use of p-values may lead to errors of inference, and in particular to the underestimation of false positives. We will point out in the text whether or not results are significant when we use Bonferroni correction, which is a conservative method to correct for multiple testing. If the desired significance level is $\alpha = 5\%$, then the Bonferroni corrected significance level for each menstrual cycle phase is $\frac{\alpha}{4} = 1.25\%$ (since there are four phases in

addition to the omitted menstrual phase). Thus, any phase that is significant at the 1.25% level is Bonferroni corrected significant at the 5% level.

In Table 7 we report coefficients and standard errors for regressions on dummies for follicular adjusted menstrual phases. We obtain clear null results for risk preferences in the gain and loss domains (G5 and L5, respectively), for the accessibility of risk preferences (A5), and the reflection effect (R5).[21] Similar results obtain when using the other two definitions of menstrual cycle phases (not reported). We do not find support for the hypothesis that naturally cycling females are more risk seeking in the peri-ovulatory phase or more risk averse in the luteal phase. Moreover, we do not find support for the hypothesis that naturally cycling females are less accessible in the luteal phase.

Observation 3 (Menstrual Cycle) *Contrary to our hypotheses, we do not find any significant correlations between menstrual cycle phases and risk preferences for gains or losses, the reflection effect, or accessibility of risk preferences.*

3.4 Salivary Hormones

From each subject we collected saliva after they arrived for the experiment about 5 to 8 minutes before they made decisions in the Holt-Laury lottery tasks. For one male subject, the amount of saliva we collected was not sufficient to assay progesterone and cortisol, so he is excluded from the analysis of salivary hormones. Table 8 provides summary statistics for salivary hormones by gender and hormonal contraceptive use. Figure 6 displays histograms and kernel distributions by gender. Gender differences are notable especially for testosterone and progesterone.

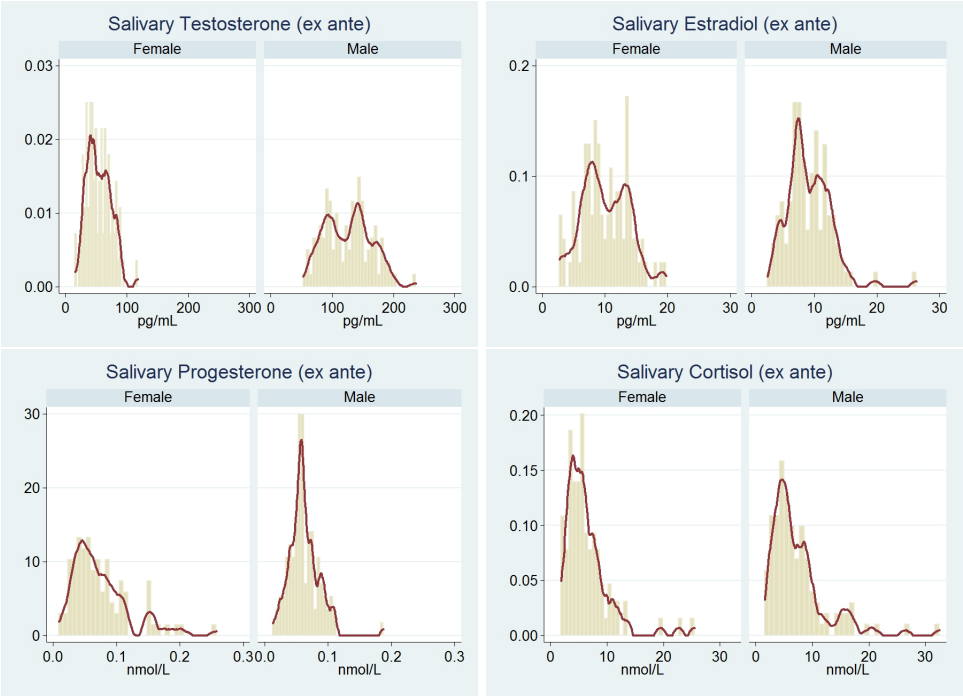
Table 8: Summary Statistics of Salivary Hormones by Gender and Hormonal Contraceptive Use

	Natur. Cycling Females		Fem. on Contraceptives		Male	
Salivary Hormone	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Testosterone (pg/mL)	57.9536	19.0284	42.0580	17.5637	125.7049	37.9870
Estradiol (pg/mL)	10.1028	3.8498	9.9486	3.6519	9.1036	3.4890
Progesterone (nmol/L)	0.0779	0.0481	0.0648	0.0382	0.0623	0.0242
Cortisol (nmol/L)	6.8084	4.3430	5.1743	2.2237	7.4268	5.0665

The relationships between ex ante salivary hormones and risk aversion in both the gain and loss domains are preliminarily explored in Figure 7 in which we print by gender for each hormone a scatter plot and fit a linear regression between the hormone level and the number of choices of option A.

[21] In the logit specification R5, failure of reflection is predicted perfectly when the (accessible) women is in the

Figure 6: Densities of Salivary Hormones by Gender



In the upper two panels of Figure 7 we observe that higher testosterone is negatively correlated with risk aversion in males but not in females. This is more pronounced in the gain (upper left panel) than in the loss (upper right panel) domain. That is, testosterone is negatively correlated with risk aversion in males. A positive correlation is observed between estradiol and risk aversion for both males and females in the loss domain (second upper right panel) but not in the gain domain (second upper left panel). Similarly, a positive correlation is observed between progesterone and risk aversion for both males and females in both the gain and loss domains but the correlation is more pronounced for females and in the gain domain. Finally, a positive correlation is observed between cortisol and risk aversion for both males and females in the gain domain but not in the loss domain.

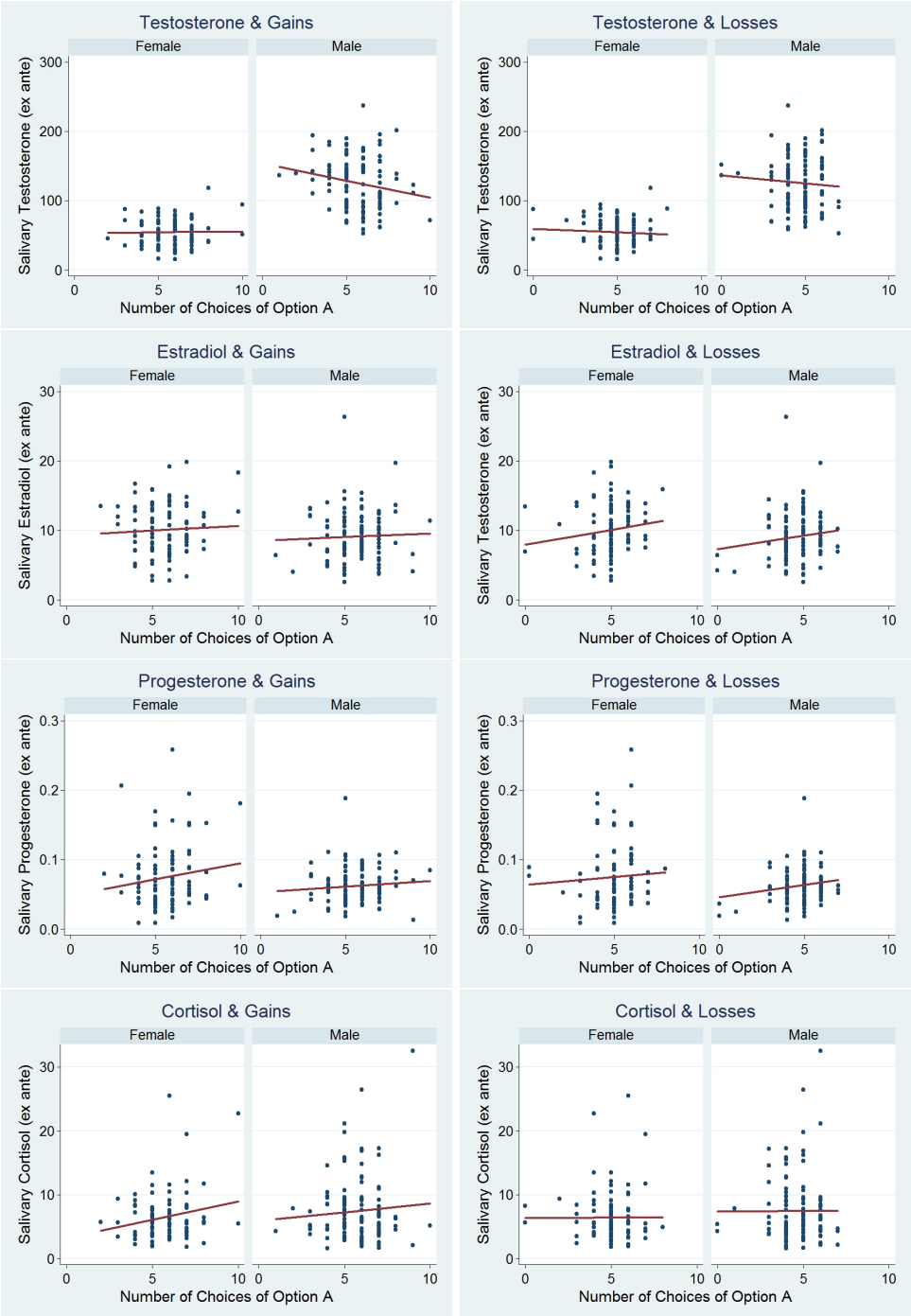
We seek to corroborate these preliminary observations with multivariate regressions. In particular, we will estimate versions of the following parametric model

$$r_i = \beta_0 + \beta_1 H_i + \gamma Q_i + \varepsilon_i \tag{2}$$

where i is the index of the subject, r_i is the number of choices of option A by subject i (in either the gain domain or the loss domain), β_0 is a constant, H_i is the vector of salivary hormone

peri-ovulatory or luteal phases.

Figure 7: Salivary Hormones and Risk Aversion by Gender



measures, Q_i is a subset of questionnaire variables including age, race, and gpa, and ε_i is an unobserved error term of subject i. We exclude White from race as omitted category. The model will be estimated with ordinary least squares method. We also estimate specifications

analogous to equation (2), in which we replace the number of choices of option A by either a binary variable for reflection or a binary variable for accessibility as dependent variable. These will be estimated with logit.

Some hormones like salivary testosterone or estradiol are measured in pg/ml while others like progesterone or cortisol are measured in nmol/L. Moreover, we see in Table 8 that their absolute levels differ quite a bit. To be better able to interpret the regression results, we use standard scores or z-scores by centering hormones measures to their mean and dividing them by their standard deviation. Since sex hormones differ by gender, this normalization could affect gender specific results. To circumvent this problem, we compute z-scores for each gender separately. That is, we normalize each hormone by

$$z_i := \frac{h_i - \mu_{g(i)}}{\sigma_{g(i)}},$$

where h_i is subject i's salivary hormone level, g is a function that assigns to each subject i its gender $g(i) \in \{\text{male}, \text{female}\}$, and $\mu_{g(i)}$ and $\sigma_{g(i)}$ are the mean and the standard deviation, respectively, of salivary hormone levels of subjects with gender $g(i)$. For instance, in OLS regressions, the coefficient for any hormone measures the effect on the number of choices of A in the Holt-Laury lottery task when the hormone level increases by one standard deviation (keeping everything else constant). Since z-scores have gender-specific definitions, we estimate all specifications separately for the subsamples of males, naturally cycling females, and females using hormonal contraceptives.

To account for heteroscedasticity, we use robust standard errors in all regressions. Moreover, we account for multiple comparison of four hormones using Bonferroni correction. Thus, if the desired significance level is $\alpha = 5\%$, then the Bonferroni-corrected significance level for each hormone is $\frac{\alpha}{4} = 1.25\%$ (since there are four hormones). Any hormone that is significant at the 1.25% level is Bonferroni-corrected significant at the 5% level. In the regression tables, we report plain p-values but we point out in the text whether or not an estimate is considered significant after Bonferroni correction.

Table 9 presents regression results for males. Specifications G3M and L3M show results for the gain and loss domains, respectively. We observe null results except for testosterone in the gain domain. Testosterone is significantly negatively associated with risk aversion in males ($p = 0.007$). Roughly, an increase of testosterone by two standard deviations would let the male individual switch from A to B almost one lottery earlier in Table 1. The association is robust to dropping demographic variables, adding demographic variables like body-mass-index, number of siblings, physical activity, living situation, dating, sexual preferences, and the digit ratio, dietary preferences, adding the major of study, including data from males with inaccessible preference, controlling for factors that could potentially affect the quality of saliva samples, dropping all

Table 9: Salivary Hormones and Risk Preferences in Males

	(G3M)	(L3M)	(R3M)	(A3M)
Testosterone	−0.4211**	−0.0859	−0.3236	0.0593
	(0.1520)	(0.1191)	(0.3492)	(0.3368)
Estradiol	−0.0232	0.1050	−0.4474	−0.8856
	(0.1420)	(0.1077)	(0.3602)	(0.5318)
Progesterone	0.1252	0.1202	0.0610	−0.1706
	(0.1767)	(0.1198)	(0.2620)	(0.4136)
Cortisol	0.2507	0.0228	0.0328	0.1491
	(0.1568)	(0.1138)	(0.2554)	(0.4227)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	102	110	102	111
R^2	0.1042	0.1340		
Pseudo R^2			0.1220	0.1728

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

salivary hormones except salivary testosterone, or using session fixed-effects instead OLS. It is also significant when controlling for multiple comparison of four hormones using Bonferroni correction.

Recall that Stanton et al. (2011b) observed an u-shaped relationship between testosterone and risk taking. We do not find such an association when we include as an additional regressor the square of the testosterone measure in specifications analogous G3M and L3M (not reported).

The logit specifications R3M and A3M are on the reflection effect and the accessibility of risk preferences, respectively. We observe null results.

Observation 4 (Males) *In males, more risk-taking for gains is significantly positively correlated with salivary testosterone. This association is also significant when accounting for multiple comparisons for four hormones using Bonferroni correction. No other significant associations with salivary hormones are observed in males.*

Table 10 shows regression results when we restrict data to the subsample of naturally cycling females. For risk aversion in the gain and loss domains (G3F and L3F, respectively), we observe null results except for a significant positive association of risk preferences in the gain domain and cortisol ($p = 0.025$). This is in line with our hypothesis although it is not clear why we observe it in naturally cycling females only. The association is robust to dropping subsets of demographic variables, adding demographic variables like body-mass-index, number of siblings, physical activity, living situation, dating, sexual preferences, and the digit ratio, adding dietary preferences, adding the major of study, including data from naturally cycling females with inaccessible preference, dropping all salivary hormones except salivary testosterone, or using session fixed-effects instead OLS. It becomes insignificant if we control for factors that could

potentially affect the quality of saliva samples. In Appendix B.5 we show that both the time last eaten and the switch to daylight saving time affected salivary cortisol in females. Our cortisol finding becomes insignificant when controlling for multiple comparison using Bonferroni correction.

Table 10: Salivary Hormones and Risk Preferences in Naturally Cycling Females

	(G3F)	(L3F)	(R3F)	(A3F)
Testosterone	0.1081	−0.1764	0.7582	−0.7749
	(0.3155)	(0.3116)	(0.4635)	(0.4193)
Estradiol	−0.0910	0.0375	−0.2517	0.2670
	(0.2662)	(0.1839)	(0.5923)	(0.3555)
Progesterone	0.2057	0.1052	0.8069	0.3016
	(0.2293)	(0.1465)	(0.4788)	(0.3182)
Cortisol	0.4452*	0.1970	0.3381	0.5765
	(0.1915)	(0.1718)	(0.3429)	(0.3460)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	51	60	50	69
R^2	0.1946	0.0957		
Pseudo R^2			0.2225	0.1214

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Motivated by Stanton et al. (2011b), we also run a specification analogous to G3F and L3F in which included the square of the testosterone measure as an additional regressor. Both the coefficient for the testosterone measure and the coefficient for the squared testosterone measure are insignificant (not reported).

Observation 5 (Naturally Cycling Females) *In naturally cycling females, risk aversion is significantly positively correlated with salivary cortisol. However, this association is insignificant when controlling for multiple comparisons using Bonferroni method. No other significant associations with salivary hormones are observed in naturally cycling females.*

Ideally we would like to present an analogous analysis for females on hormonal contraceptives. Unfortunately, we just have 21 women in our sample who take hormonal contraceptives of which only 19 have accessible risk preferences. We fear that this sample is too small to draw meaningful inferences with respect to covariation of hormones and risk preferences. Thus the following analysis should be considered as extremely preliminary. Table 11 shows regression results females on hormonal contraceptives. Because of the lack of observations, we also include data on females using hormonal contraceptives whose risk preferences are not accessible. Moreover, we drop any demographic variables and only regress on the four salivary hormones and a constant.

Specification G3P shows that risk aversion in the gain domain is significantly positively correlated with salivary progesterone in females using hormonal contraceptives ($p = 0.002$).

Table 11: Salivary Hormones in Females on Hormonal Contraceptives

	(G3P)	(L3P)	(A3P)
Testosterone	−0.3888	−0.3508*	−0.8545
	(0.3371)	(0.1500)	(1.6470)
Estradiol	−0.3307	0.7606**	5.9286
	(0.2437)	(0.1365)	(4.6948)
Progesterone	0.6600**	−0.4538**	−0.8473
	(0.1731)	(0.0942)	(1.1127)
Cortisol	0.0373	0.0146	6.2060**
	(0.3597)	(0.1939)	(2.1199)
Number of Observations	21	21	21
R^2	0.4286	0.5855	
Pseudo R^2			0.4123

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

It remains significant if we add demographic variables. It is also significant when correcting for multiple comparison for four hormones using Bonferroni method. In the loss domain, risk aversion is significantly negatively correlated both with salivary testosterone ($p = 0.033$) and progesterone ($p < 0.001$) and significantly positively correlated with salivary estradiol ($p < 0.001$). The association with estradiol remains significant if we add demographic variables, while the association with progesterone remains weakly significant. Note that associations with progesterone in the gain and loss domains have different signs. This suggest that reflection should be correlated with progesterone. This is indeed the case. Salivary progesterone slightly above the female mean perfectly predicts reflection. That is why this logit specification on the reflection effect is missing from Table 11. Finally, salivary cortisol is significantly positively associated with accessibility of risk preferences in females using hormonal contraceptives ($p = 0.003$). Yet, demographic variables such as gpa would predict accessibility of risk preferences perfectly. Altogether, this preliminary data suggests that it may be interesting to examine risk preferences and steroid hormones in a larger data set on females who use hormonal contraceptives.

3.5 Digit Ratio

We scanned each subject's right hand from which we measured and calculated the digit ratio (2D:4D), the ratio between the lengthes of the index finger to the ring finger. The summary statistics are presented in Table 12. For one subject we accidently scanned the left hand instead the right hand. We measured the fingers nevertheless and include this observation in our analysis. Our results do not change when we drop this subject. As it is well-known in the literature, females have on average a larger digit ratio than males. The female digit ration is significantly larger than the male digit ratio for Total ($t = -3.0161$, $p = 0.001$, one-sided), White ($t = -2.9397$, $p = 0.002$, one-side), and Asian ($t = -1.6930$, $p = 0.047$, one-sided) but

not for Other Ethn. ($t = -1.5783$, $p = 0.064$, one-sided).

Table 12: Summary Statistics of the Digit Ratio by Gender and Race

	Females		Males	
Ethnicity	Mean	Std. Dev.	Mean	Std. Dev.
White	0.970	0.0275	0.952	0.0267
Asian	0.957	0.0274	0.948	0.0296
Other Ethn.	0.968	0.0384	0.944	0.0360
Total	0.962	0.0294	0.949	0.0290

In Figure 8, we present scatter plots for the correlation between the digit ratio and the number of choices of option A by gender for both gains and losses. The upper two panels are for the full sample by gender. We also fit linear regressions. These regression lines indicate a positive relationship between digit ratio and risk aversion. This appears to be more pronounced in females than males. However, when we try to corroborate these preliminary observations with multivariate regressions, we do not find significant results. In particular, we estimate the following model

$$r_i = \beta_0 + \beta_1 d_i + \gamma Q_i + \varepsilon_i \tag{3}$$

where i is the index of the subject, r_i is the number of choices of option A by subject i (in either the gain domain or the loss domain), β_0 is a constant, d_i is individual i's digit ratio, Q_i is a subset of questionnaire variables including age, race, and gpa, and ε_i is an unobserved error term of subject i. This model will be estimated with ordinary least squares method (OLS). We also estimate specifications analogous to equation (3) where we replace the number of choices of option A by either a binary variable for reflection or a binary variable for accessibility of preferences as dependent variable. Because of the binary dependent variable, we estimate the model with logit. Since the digit ratio is known to differ by gender, we estimate all specifications separately for both the male and female subsamples.

Table 13 presents results for males. We observe null results for risk aversion in the gain domain (specification GDRM), loss domain (specification LDRM), the reflection effect (specification RDRM) as well as accessibility of risk preferences (specification ADRM). We also observe null results for females, see Table 14.

The digit ratio is known to vary substantially with race (Manning et al. 2003, 2004). In the previous literature, a significant association of risk aversion with the digit ratio has been reported in a homogeneous white population while such an association was absent in samples with racial heterogeneity (see Dreber and Hoffman, 2007, and Garbarino et al., 2011, versus

Figure 8: Digit Ratio and Risk Aversion by Gender and Ethnicity

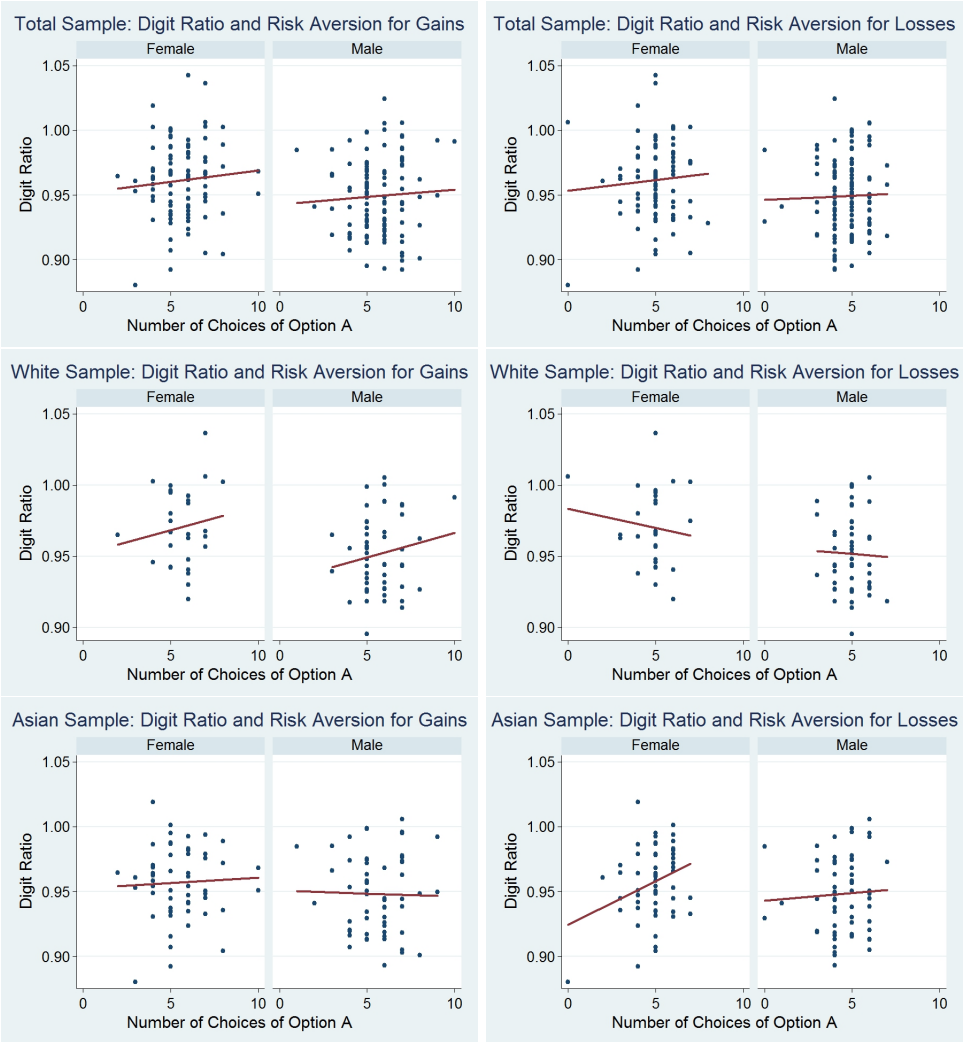


Table 13: Male Subsample: Digit Ratio and Risk Aversion

	(GDRM)	(LDRM)	(RDRM)	(ADRM)
Digit Ratio	3.9619	1.6611	−13.8094	12.8730
	(5.3825)	(4.1649)	(9.4024)	(12.2703)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	103	111	103	112
R^2	0.0142	0.1028		
Pseudo R^2			0.1106	0.0376

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Table 14: Female Subsample: Digit Ratio and Risk Aversion

	(GDRF)	(LDRF)	(RDRF)	(ADRF)
Digit Ratio	4.0044	5.6613	−4.5873	−1.9258
	(5.6461)	(8.0630)	(9.9518)	(10.2484)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	71	80	69	90
R^2	0.0615	0.0738		
Pseudo R^2			0.1071	0.0618

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Table 15: White Male Subsample: Digit Ratio and Risk Aversion

	(GDRWM)	(LDRWM)	(RDRWM)	(ADRWM)
Digit Ratio	8.8903	−0.4056	−7.0561	26.9925
	(6.7151)	(4.8567)	(18.4838)	(23.9602)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	47	50	47	50
R^2	0.1096	0.0136		
Pseudo R^2			0.0768	0.2014

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Table 16: White Female Subsample: Digit Ratio and Risk Aversion

	(GDRWF)	(LDRWF)	(RDRWF)	(ADRWF)
Digit Ratio	1.4099	−8.9336	−9.9750	−23.3457
	(8.0587)	(11.0921)	(27.1597)	(16.2836)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	25	27	25	28
R^2	0.1085	0.0780		
Pseudo R^2			0.1253	0.1308

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Table 17: Asian Male Subsample: Digit Ratio and Risk Aversion

	(GDRAM)	(LDRAM)	(RDRAM)	(ADRAM)
Digit Ratio	−0.0148	3.5655	−26.9841*	17.3519
	(8.1505)	(6.5435)	(11.7804)	(18.4137)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	52	56	52	56
R^2	0.0352	0.1072		
Pseudo R^2			0.0968	0.0932

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Table 18: Asian Female Subsample: Digit Ratio and Risk Aversion

	(GDRAF)	(LDRAF)	(RDRAF)	(ADRAF)
Digit Ratio	6.6254	15.6264	−2.1721	10.9579
	(8.3323)	(10.0170)	(8.7443)	(10.7891)
Demographics	Yes	Yes	Yes	Yes
Number of Observations	41	48	39	55
R^2	0.0274	0.1279		
Pseudo R^2			0.0902	0.0811

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

Apicella et al., 2008). Controlling linearly for race (with White as the omitted category), as we do in Tables 13 and 14, may not be appropriate as the digit ratio could have different associations in different races. That's why we split the samples further and analyze separately the white and asian subsamples by gender. Tables 15 and 16 show results for the white subsample by gender, respectively.[22] Again, we observe null results for both genders. Similarly, Tables 17 and 18 show null results for the asian subsample by gender, respectively, except for the reflection effect for asian males. It appears that the propensity to display the reflection effect is negatively correlated with the digit ratio in asian males ($p = 0.022$). This observation is robust to dropping demographic variables or adding major of study or using probit instead of logit. It becomes insignificant when demographic variables are dropped and also asian males with inaccessible risk preferences are considered. We do not have a hypothesis for this finding. It may simply be a false positive given that this is the only significant finding in our 24 regressions on the digit ratio. It would require a replication with larger samples to clarify the status of this observation.

Observation 6 (Digit Ratio) *No significant correlation between the digit ratio and risk preferences in the gain or loss domains are observed in either males, females, white males, white females, asian males and asian females.*

4 Discussion

We present a comprehensive study of correlations between risk preferences and sex hormones. While salivary testosterone has been studied previously for choice under risk in the gain domain, we are not aware of any previous study of choice under risk for *both gains and losses* and *all of salivary testosterone, estradiol, progesterone, and cortisol* in both *males and females*. We show null results except for testosterone and cortisol. With regard to testosterone, we replicate the observation of Apicella et al. (2008) that testosterone in males has a positive association with risk taking in the gain domain using a measure of risk different from Apicella et al. (2008).

[22]This time, we include just gpa and age as demographic variables.

The findings on testosterone have potentially broader implications for economics. Attitudes towards risk play a role many market settings. For instance, Nadler et al. (2018) report a positive association between testosterone administration and trading behavior in experimental asset markets. Testosterone seems to cause larger and longer-lasting bubbles. In such double auctions, bidding is affected by risk attitudes. In a real-life setting, Coates and Herbert (2008) concluded from a sample of 17 professional traders that their morning levels of testosterone predicted daily trading profits and their cortisol rises with market volatility. These findings are somewhat in contrast to a null finding between circulating testosterone and bidding in first-price auctions by Schipper (2015) even though the theoretical prediction in first-price auctions is that bids are increasing in risk aversion.

We also observe that risk aversion for gains is positively associated with cortisol in females. This association is insignificant if we control for multiple comparison using Bonferroni correction. Bonferroni correction is typically used when we worry about false positives due to multiple comparison. However, in the case of cortisol, we should be more worried about false negatives as the finding has potentially important methodological implications for experimental studies of risk preferences. The reason is that cortisol follows a circadian cycle. It is usually higher in the morning and then falls in the afternoon. It is also known to be affected by stress such as taking exams etc. If risk aversion is correlated with cortisol, then it matters when we run experiments on choice under risk. A prudent experimentalist should run the sessions at the same time of the day or at least control for potential session effects due to the circadian cycle of cortisol.

A reviewer asked us to discuss the dual hormone hypothesis of Mehta and Josephs (2006) and Mehta et al. (2015) according to which the effect of testosterone on risk taking should be moderated by the inhibitory function of high cortisol (see also Nofsinger et al., 2018). Testosterone is hypothesized to exert only an effect on risk taking if cortisol is low because of the inhibitory effects of cortisol. We conducted an post hoc analysis of our data. In particular, we investigate specifications analogous to above but with interaction terms for testosterone and cortisol. We also investigated specifications with interactions of testosterone with a dummy for low cortisol (i.e., below the mean). Finally, we considered specifications with the ratio of testosterone to cortisol. We found no evidence for the dual hormone hypothesis (not reported).

Both estradiol and progesterone vary over the menstrual cycle. Consistent with our null results on the correlation of risk preferences with salivary estradiol and progesterone, we also observe null results on the correlation of risk preferences with menstrual cycle phases. Our null finding with respect to hormonal contraceptives is consistent with the null result on salivary progesterone too since hormonal contraceptives contain an synthetic version of progesterone. Nevertheless, the null-finding for progesterone is puzzling because in our companion study, Schipper (2015), we report a robust positive association between basal progesterone and bidding of naturally cycling females in first-price auctions. The effects of risk aversion in first-price

auctions with symmetric independent private values are well established in theory (see Krishna, 2002, Chapter 4.1). Risk aversion increases equilibrium bids above risk-neutral Nash equilibrium. A higher bid translates into a higher probability of winning the auction, but it also leads to a lower profit conditional on winning the auction.[23] The experimental evidence for risk aversion in first-price auctions with symmetric independent private values is at best mixed (see for a survey, Kagel, 1995). Indeed, Schipper (2015) finds that the addition of our measures of risk preferences from our Holt-Laury lottery tasks to regressions of bidding on salivary progesterone does not diminish the positive association of bidding with progesterone. Thus, the association of progesterone on bidding does not seem to be mediated through risk preferences as elicited with Holt-Laury lotteries. This is somehow at odds with the literature showing that progesterone seems to mitigate "social risk" (Fleischman and Fessler, 2011, Maner and Miller, 2014, Schultheiss, Wirth, and Stanton, 2004, Wirth and Schultheiss, 2006, Brown et al., 2009). One explanation for reconciling the evidence is that risk preferences are context-specific rather than universal. Individual behavior in Holt-Laury lottery tasks may not be well-suited to measure risk attitudes in strategic contexts like auctions. The context-specificity of risk attitudes may also explain to some extent poor correlations between behavior in the Holt-Laury lottery task and other elicitation methods (see Deck et al., 2013, and Lönnqvist et al., 2015).

Our null-results on the digit ratio are largely consistent with the mixed results in literature on the digit ratio and risk preferences. Such null findings are relevant given empirical findings on the association between the digit ratio and relevant economic outcomes. For instance, Coates, Gurnell, and Rustichini (2009) find that lower 2D:4D predicts the 20-month average profitability of 44 male high-frequency traders in London. Guiso and Rustichini (2018) report that entrepreneurs with lower digit ratio manage larger firms and experience faster average growth.

References

[1] Alonso, J., Di Paolo, R., Ponti, G., Sartarelli, M. (2018). Facts and misconceptions about 2d:4D, social and risk preferences, Frontiers in Behavioral Neuroscience 12, 22.

[2] Alvergne, A., Lummaa, V. (2009). Does the contraceptive pill alter mate choice in humans?, Trends in Ecology and Evolution 25, 171–179.

[3] Apicella, C.L., Carré, J.M., Dreber, A. (2015). Testosterone and economic risk taking: A review, Adaptive Human Behavior and Physiology 1: 358–385.

[4] Apicella, C.L., Dreber, A., Campbell, B., Gray, P.B., Hoffman, M., Little, A.C. (2008). Testosterone and financial risk preferences, Evolution and Human Behavior 29, 384-390.

[23] Various dispositions towards uncertainty, like anticipated regret from losing the auction (see Filiz and Ozbay, 2007), overconfidence in the winning probability of a bid, ambiguity aversion, etc. lead to similar behavioral predictions in first-price auctions with independent private values.

[5] Apicella, C.L., Dreber, A., Gray, P.B., Hoffman, M., Little, A.C., Campbell, B.C. (2011). Androgens and competitiveness in men, Journal of Neuroscience, Psychology, and Economics 4, 54–62.

[6] Archer, J. (1991). The influence of testosterone on human aggression, British Journal of Psychology 82, 1–28.

[7] Aycinena, D., Baltaduonis, R., Rentschler, L. (2014). Risk preferences and prenatal exposure to sex hormones for ladinos, PLOSone 9, e103332.

[8] Barel, E. (2019). 2D:4D, optimism, and risk taking, Current Psychology 38, 204–212.

[9] Bönte, W., Procher, V.D., Urbig, D. (2016). Biology and selection into entrepreneurship - the relevance of prenatal testosterone exposure, Entrepreneurship Theory and Practice 40, 1121–1148.

[10] Boksem, M.A.S., Mehta, P.H., Van den Bergh, B., van Son, V., Trautmann, S.T., Roelofs, K., Smidts, A., Sanfey, A.G. (2013). Testosterone inhibits trust but promotes reciprocity, Psychological Science 24, 2306–2314.

[11] Brañas Garza, P., Galizzi, M.M., Nieboer, J. (2018). Experimental and self-reported measures of risk taking and digit ratio (2d:4d): Evidence from a large, systematic study, International Economic Review 59, 1131–1157.

[12] Brañas Garza, P., Rustichini, A. (2011). Organizing effects of testosterone and economic behavior: Not just risk taking, PLoS ONE 6, e29842.

[13] Bressan, P., Stranieri, D. (2000). The best men are (not always) already taken, Psychological Science 19, 145-151.

[14] Briggs, M., Briggs, M. (1972). Plasma hormone concentration in women receiving steroid contraceptives, Journal of Obstetrics and Gynaecology 79, 946–950.

[15] van Broekhoven, F., Bäckström, T., Verkes, R.J. (2006). Oral progesterone decreases saccadic eye velocity and increases sedation in women, Psychoneuroendocrinology 31, 1190–1199.

[16] Bröder, A., Hohmann, N. (2003). Variations in risk taking behavior over the menstrual cycle: An improved replication, Evolution and Human Behavior 24, 391–398.

[17] Brown, S.L., Fredrickson, B.L., Wirth, M.M., Poulin, M.J., Meier, E.A., Heaphy, E.D., Cohen, M.D., Schultheiss, O.C. (2009). Social closeness increases salivary progesterone in humans, Hormones and Behavior 56, 108-111.

[18] Buckert, M., Schwieren, C., Kudielka, B.M., Fiebach, C.J. (2014). Acute stress affects risk taking but not ambiguity aversion, Frontiers in Neuroscience 8, Article 82.

[19] Byrnes, J., Miller, D.C., Schafer, W.D. (1999). Gender differences in risk taking: A meta analysis, Psychological Bulletin 125, 367-383.

[20] Chatterton Jr., R.T., Mateo, E.T, Hou, N.J., Rademaker, A.W., Acharya, S., Jordan, V.C., Morrow, M. (2005). Characteristics of salivary profiles of oestradiol and progesterone in premenopausal women, Journal of Endocrinology 186, 77-84.

[21] Chavanne, T.J., Gordon G. Gallup, G.G. (1998). Variation in risk taking behavior among female college students as a function of the menstrual cycle, Evolution and Human Behavior 19, 27–32.

[22] Chen Y., Katuščák, P., Ozdenoren, E. (2013). Why can't a woman bid more like a man?, Games and Economic Behavior 77, 181–213.

[23] Chicaiza-Becarra, L.A., Garcia-Molina, M. (2017). Prenatal testosterone predicts financial risk taking: Evidence from Latin America, Personality and Individual Differences 116, 32–37.

[24] Chumbley, J.R., Krajbich, I., Engelmann, J.B., Russell, E., Van Uum, S., Koren, G., Fehr, E. (2014). Endogenous cortisol predicts decreased loss aversion in young men, Psychological Science 25, 2102–2015.

[25] Coates, J.M., Gurnell, M., Rustichini, A. (2009). Second-to-fourth digit ratio predict success among high-frequency financial traders, Proceedings of the National Academy of Sciences 106, 623–628.

[26] Coates, J.M., Herbert, J. (2013). Endogenous steroids and financial risk taking on a London trading floor, Proceedings of the National Academy of Sciences 104, 6167–6172.

[27] Coenen, C.M.H., Thomas, C.M.G., Borm, G.F., Hollanders, J.M.G., Rolland, R. (1995). Changes in androgens during treatment with low-dose contraceptives, Contraception 53, 171–176.

[28] Croson, R., Gneezy, U. (2009). Gender differences in preferences, Journal of Economic Literature 47, 1-27.

[29] Dabbs, J.M. (1991). Salivary testosterone measurements: Collecting, storing, and mailing saliva samples, Physiology and Behavior 49, 815–817.

[30] Dabbs, J.M., Hargrove, M.F. (1997). Age, testosterone, and behavior among female prison inmates, Psychosomatic Medicine 59, 477-480.

[31] Dalton, P.S., Ghosal, S. (2018). Self-confidence, overconfidence and prenatal testosterone exposure: Evidence from the lab, Frontiers in Behavioral Neuroscience 12, 5.

[32] Deck, C., Lee, J., Reyes, J., Rosen, C. (2013). A Failed Attempt to Explain Within Subject Variation in Risk Taking Behavior Using Domain Specific Risk Attitudes, Journal of Economic Behavior and Organization87, 1-24.

[33] Dekel, E., Scotchmer, S. (1999). On the evolution of attitudes towards risk in winner-take-all games, Journal of Economic Theory 87, 125-143.

[34] Dickerson, S.S., Kemeny, M.E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research, Psychological Bulletin 130, 355–391.

[35] Dreber, A., Hoffman, M. (2007). Portfolio selection in utero, Stockholm School of Economics.

[36] Drichoutis, A.C., Nayga, R.M.Jr. (2015). Do risk and time preferences have biological roots? Southern Economic Journal 82, 235-256.

[37] Eckel, C.C., Grossman, P.J. (2008). Men, women, and risk aversion: Experimental evidence, in: Plott, C. and Smith, V. (eds.), Handbook of Experimental Economics Results, Vol. 1, New York, Elsevier, Chapter 113, 1061–1073.

[38] Edwards, D.A., O'Neal, J.L. (2009). Oral contraceptives decrease salive testosterone but do not affect the rise in testosterone associated with athletic competition, Hormones and Behavior 56, 195–198.

[39] Fessler, D.M.T. (2003). Rape is not less frequent during the ovulatory phase of the menstrual cycle, Sexualities, Evolution and Gender 5.3, 127-147.

[40] Filippin, A., Crosetto, P. (2016). Reconsideration of gender differences in risk attitudes, Management Science 62, 3138–3160.

[41] Filiz, E., Ozbay, E. (2007). Auctions with anticipated regret: Theory and experiment, American Economic Review 97, 1407-1418.

[42] Fleischman, D.S., Fessler, D.M.T. (2011). Progesterone's effects on the psychology of disease avoidance: Support for the compensatory behavioral prophylaxis hypothesis, Hormones and Behavior 59, 271–275.

[43] Fritz, M.A., Speroff, L. (2011). Clinical gynecologic endocrinology and infertility, Lippincott Williams & Wilkins.

[44] Gangestad, S.W., Simpson, J. A. (2000). The evolution of human mating: Trade-offs and strategic pluralism, Behavioral and Brain Sciences 23, 573-644.

[45] Gangestad, S.W., Thorndill, R., Garver-Apgar, C.E. (2006). Adaptations to ovulation, Current Directions in Psychological Research 14, 312-316.

[46] Gangestad, S.W., Thorndill, R., Garver, C.E. (2002). Changes in women's sexual interests and their partners' mate-retention tactics across the menstrual cycle: Evidence for shifting conflicts of interest, Proceedings of the Royal Society of London B, Biological Sciences, 269, 975-982.

[47] Garbarino, E., Slonim, R., Sydnor, J. (2011). Digit ratios (2D:4D) as predictors of risky decision making for both sexes, Journal of Risk and Uncertainty 42, 1–26.

[48] Greiner, B. (2004). An online recruitment system for economic experiments, in: Kremer, K., Macho, V. (eds.), Forschung und wissenschaftliches Rechnen 2003. GWDG Bericht 63, Göttingen: Ges. für Wiss. Datenverarbeitung, 79-93.

[49] Guiso, L., Rustichini, A. (2018). Understanding the size and profitability of firms: The role of a biological factor, Research in Economics 72, 65–85.

[50] Hamilton, L. D., Meston, C.M. (2011). The role of salivary cortisol and DHEA-S in response to sexual, humorous, and anxiety-inducing stimuli, Hormones and Behavior 59, 765–771.

[51] Hampson, E., Young, E.A. (2008). Methodological issues in the study of hormone-behavior relations in humans: Understanding and monitoring the menstrual cycle, in: Becker. J.B. et al. (Eds.), Sex differences in the brain. From genes to behavior, Oxford University Press, 63-78.

[52] Hamo, Y., Heifetz, A. (2002). An evolutionary perspective on goal seeking and S-shaped utility, mimeo., Tel Aviv University.

[53] Harrison, G.W., Ruthström, E. (2008). Risk aversion in the laboratory, in: Cox, J. and Harrison, G.W. (eds.), Risk aversion in experiments, Vol. 12, Bingley, UK: Emerald, Research in Experimental Economics, 41-196.

[54] Haselton, M.G., Gangstad, S.W. (2006). Conditional expression of women's desires and men's mate guarding across the ovulatory cycle, Hormones and Behavior 49, 509-518.

[55] Hellhammer, D.H., Wuest, S., Kudielka, B.M. (2009). Salivary cortisol as a biomarker in stress research, Psychoneuroendocrinology 34, 163–171.

[56] Hönekopp, J., Bartholdt, L., Beier, L., Liebert, A. (2007). Second to fourth digit length ratio (2D:4D) and adult sex hormone levels: New data and a meta-analytical review, Psychoneuroendocrinology 32, 313-321.

[57] Hoffman, M., Jordan, J., Yoeli, E. (2013). The evolutionary basis of sex differences in risk preferences, University of California, San Diego.

[58] Hollier, L., Keelan, J., Jamnadass, E., Maybery, M., Hickey, M., Whitehouse, A. (2015). Adult digit ratio (2d:4d) is not related to umbilical cord androgen or estrogen concentrations, their ratios or net bioactivity, Early Human Development 91, 111–117.

[59] Holt, C., Laury, S. (2002). Risk aversion and incentive effects, American Economic Review 92, 1644-1655.

[60] Johnson, D.D.P, McDermott, R., Barrett, E.S., Cowden, J., Wrangham, R., McIntyre, M.H., Rosen, S.P. (2006). Overconfidence in wargames: experimental evidence on expectations, aggression, gender and testosterone, Proceedings of the Royal Society B 273, 2513–2520.

[61] Kagel, J.H. (1995). Auctions: A survey of experimental research, in: Kagel, J.H. and Roth, A.E. (eds.), The handbook of experimental economics, Princeton University Press, Chapter 7, 501–585.

[62] Kahneman, D., Tversky, A. (1979). Prospect theory: An analysis of decision under risk, Econometrica 67, 263-291.

[63] Kaltwasser, L., Mikac, U., Busko, V., Hildebrandt, A., (2017). No robust association between static markets of testosterone and facts of social-economic decision making, Frontiers in Behavioral Neuroscience 11, 250.

[64] Kandasamy, N., Hardy, B., Page, L., Schaffner, M., Gragaber, J., Powlson, A.S., Fletcher, P.C., Gurnell, M., Coates, J. (2013). Cortisol shifts financial risk preferences, Proceedings of the National Academy of Sciences 111, 3608–3616.

[65] Kirschbaum, C., Kudielka, B.M., Gaab, J., Schommer, N.C., and Hellhammer, D.H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis, Psychosomatic Medicine 61, 154–162.

[66] Kivlighan, K.T., Granger, D.A., Booth, A. (2005). Gender differences in testosterone and cortisol response to competition, Psychoneuroendocrinology 30, 58-71

[67] Kjeld, J.M., Puah, C.M., Joplin, G.F. (1976). Changed levels of endogenous sex steroids in women on oral contraceptives, British Medical Journal 2, 1354–1356.

[68] Kluen, L.M., Agorastos, A., Wiedemann, K., Schwabe, L. (2017). Cortisol boosts risk decision-making behavior in men but not women, Psychoneuroendocrinology 84, 181–189.

[69] Krishna, V. (2002). Auction theory, Academic Press.

[70] Kurath, J., Mata, R. (2018). Individual differences in risk taking and endogenous levels of testosterone, estradiol, and cortisol: A systemic literature search and three independent meta-analysis, Neuroscience and Biobehavioral Reviews 90, 428–446.

[71] Laury, S. K., Holt, C. A. (2008). Further reflections on prospect theory, in: Cox, J. and Harrison, G. W. (eds.), Risk aversion in experiments, Vol. 12, Bingley, UK: Emerald, Research in Experimental Economics.

[72] Liening, S.H., Stanton, S.J., Saini, E.K., O.C. Schultheiss (2010). Salivary testosterone, cortisol, and progesterone: Two-week stability, interhormone correlations, and effects of time of day, menstrual cycle, and oral contraceptive use on steroid hormone level, Physiology & Behavior 99, 8–16.

[73] Lima de Miranda, K., Neyse, L., Schmidt, U. (2018). Risk preferences and predictions about others: No association with 2d:4d ratio, Frontiers in Behavioral Neuroscience 12, 9.

[74] Lönnqvist, J.-E., Verkasalo, M., Walkowitz, G., Wichardt, P.C. (2015). Measuring individual risk attitudes in the lab: Task or ask? An empirical comparison, Journal of Economic Behavior & Organization 119, 254–266.

[75] Loo, J.M.Y., Raylu, N., Oei, T.P.S. (2008). Gambling among the Chinese: A comprehensive review, Clinical Psychological Review 28, 1152–1166.

[76] Lutchmaya, S., Raggatt, P., Knickmeyer, R., Manning, J.T. (2004). 2nd to 4th digit ratios, fetal testosterone and estradiol, Early Human Development 77, 23–28.

[77] Maner, J.K., Miller, S.L. (2014) Hormones and social monitoring: Menstrual cycle shifts in progesterone underlie women's sensitivity to social information, Evolution and Human Behavior 35, 9–16.

[78] Manning, J.T. (2002). Digit ratio. A pioneer to fertility, behavior and health, Rutgers University Press.

[79] Manning, J. T., Henzi, P., Venkatramana, P. (2003). 2nd to 4th digit ratio: ethnic differences and family size in English, Indian and South African populations, Annals of Human Biology 30, 579–588.

[80] Manning, J.T., Stewart, A, Bundred, P.E., Trivers, R.L. (2004). Sex and ethnic differences in 2nd to 4th digit ratio of children, Early Human Development 80, 161-168.

[81] Mazur, A., Booth, A. (1998). Testosterone and dominance in men, Behavioral and Brain Sciences 21, 353-397.

[82] Mehta, P.H., Josephs, R.A. (2006). Testosterone change after losing predicts the decision to compete again, Hormones and Behavior 50, 684-692.

[83] Mehta, P.H., Welker, K.M., Zilioli, S., Carr, J.M. (2015). Testosterone and cortisol jointly modulate risk-taking, Psychoneuroendocrinology 56, 88–99.

[84] Mechoulam R., Brueggemeier. R.W., Denlinger D.L. (1984). Estrogens in insects, Journal Cellular and Molecular Life Sciences 40, 942–944.

[85] Nadler, A., Wibral, M., Dohmen, T., Falk, A, Previtero, A., Weber, B., Camerer, C., Dreber, A., Nave, G. (2021). Does testosterone increase willingness to compete, confidence, and risk-taking in men? Evidence from two randomized placebo-controlled experiments and a meta-analysis, `https://psyarxiv.com/62af7`.

[86] Nadler, A., Jiao P., Johnson, C.J., Alexander, V., Zak, P. (2018). The bull of wall street: Experimental analysis of testosterone and asset trading, Management Science 64, 4032–4051.

[87] Nelson, R.J. (2011). An introduction to behavioral endocrinology, 4th edition, Sinauer Assoc.

[88] Neyse, L., Johannesson, M., Dreber, A. (2021). 2d:4d does not predict economic preferences: Evidence from a large, representative sample, Journal of Economic Behavior and Organization 185, 390–401.

[89] Neyse, L., Vieider, F.M., Ring, P., Probst, C., Kaernbach, C., van Eimeren, T., Schmidt, U. (2020). Risk attidues and digit ratio (2d:4d): Evidence from prospect theory, Journal of Risk and Uncertainty 60, 29–51.

[90] Nofzinger, J.R., Patterson, F.M., Shank, C.A. (2018). Decision-making, financial risk aversion, and behavioral bias: The role of testosterone and stress, Economics & Human Biology 29, 1–16.

[91] Parslow, E., Ranehill, E., Zethraeus, N., Blomberg, L., van Schoultz, B., Lindén Hirschberg, A., Johannesson, M., Dreber, A. (2019). The digit ratio (2d:4d) and economic preferences: No robust associations in a sample of 330 women, Journal of the Economic Science Association 5, 149–169.

[92] Paul, S.N., Kato, B.S., Cherkas, L.F., Andrew, T., Spector, T.D. (2006). Heritability of the second to fourth digit ratio (2d:4d): A twin study, Twin Research and Human Genetics 9, 215–219.

[93] Pearson, M., Schipper, B.C. (2012). The Visible Hand: Finger ratio (2D:4D) and competitive bidding, Experimental Economics 15, 510–529.

[94] Pearson, M., Schipper, B.C. (2013). Menstrual cycle and competitive bidding, Games and Economic Behavior 78, 1–20.

[95] Penton-Voak, I.S., Perrett D.I. (2000). Female preference for male faces changes cyclically: Further evidence, Evolution and Human Behavior 21, 39-48.

[96] Penton-Voak, I.S., Perrett, D.I., Castles, D.L., Kobayashi, T., Burt, D.M., Murray, L.K., Minamisawa, R. (1999). Female preferences for male faces changes cyclically, Nature 399, 741-742.

[97] Pluchino, N., Cubeddu, A., Giannini, A., Merlini, S., Cela, V., Angioni, S., Genazzani, A.R. (2009). Progestogens and brain: An update, Maturitas 62, 349–355.

[98] Pluchino, N., Luisi, M., Lenzi, E., Centofanti, M., Begliuomini, S., Freschi, L., Ninni, F., Genazzani, A.R. (2006). Progesterone and progestins: Effects on brain, allopregnanolone and β-endorphin, Journal of Steroid Biochemistry and Molecular Biology 102, 205–213.

[99] Ranehill, E., Zethraeus, N., Blomberg, L., von Schoultz, B., Hirschberg, A.L., Johannesson, M., Dreber, A. (2018). Hormonal contraceptives do not impact economic preferences: Evidence from a randomized trail, Management Science 64, 4515–4532.

[100] Robson, A. (1996). The evolution of attitudes to risk: Lottery tickets and relative wealth, Games and Economic Behavior 14, 190-207.

[101] Saad, G., Vongas, J.G. (2009). The effect of conspicuous consumption on men's testosterone levels, Organizational Behavior and Human Decision Processes 110, 80–92.

[102] Sapienza, P., Zingales, L., Maestripieri, D. (2009). Gender differences in financial risk aversion and career choices are affected by testosterone, Proceedings of the National Academy of Sciences 106, 15268–15273.

[103] Schaefer, L., Ikink, I., Woyke, I., Heuvelmans, V., Roelofs, K., Figner, B. (2022). No evidence for a causal effect of exogenous testosterone on risk decision-making in women: An experiment and meta-analysis, Decision 9, 347–365.

[104] Schipper, B.C. (2015). Sex hormones and competitive bidding, Management Science 61, 249–266.

[105] Schultheiss, O.C., Dargel, A., Rohde, W. (2003). Implicit motives and gonadal steroid hormones: Effects of menstrual cycle phase, oral contraceptive use, and relationship status, Hormones and Behavior 43, 293–301.

[106] Schultheiss, O.C., Wirth, M.M., Stanton, S.J. (2004). Effects of affiliation and power motivation arousal on salivary progesterone and testosterone, Hormones and Behavior 46, 592–599.

[107] Schultheiss, O.C., Wirth, M.M., Torges, C.M., Pang, J.S., Villacorta, M.A., Welsh, K.M. (2005). Effects of implicit power motivation on men's and women's implicit learning and testosterone changes after social victory or defeat, Journal of Personality and Social Psycholology 88, 174-188.

[108] Small, C.M., Manatunga, A.K., Marcus, M. (2007). Validity of self-reported menstrual cycle length, Annals of Epidemiology 17, 163–170.

[109] Spona, J., Feichtinger, W., Kinderman, Ch., Wünsch, C., Brill, K. (1996). Inhibition of ovulation by an oral contraceptive containing 100 μg levonorgestrel in combination with 20 μg ethinylestradiol, Contraception 54, 299–304.

[110] Stanton, S., Liening, S.H., Schultheiss, O. (2011a). Testosterone is positively associated with risk taking in the Iowa Gambling Task, Hormones and Behavior 59, 252–256.

[111] Stanton, S., Mulette-Gillman, O.A., McLaurin, R.E., Kuhn, C.M., LaBar, K.S., Platt, M.L., Huettel, S.A. (2011b). Low- and high-testosterone inidivuals exhibit decreasing aversion to economic risk, Psychological Science, Psychological Science 22, 447–453.

[112] Stanton, S.J., Welker, K.M., Bonin, P.L., Goldfarb, B., Carré, J.M. (2021). The effect of testosterone on economic risk-taking: A multi-study, multi-method investigation, Hormones and Behavior 134, 105014.

[113] Stenstrom, E., Saad, G., Nepomuceno, M.V., Mendenhall, Z. (2011). Testosterone and domain-specific risk: Digit ratios (2D:4D and rel2) as predictors of recreational, financial, and social risk-taking behaviors, Personality and Individual Differences 51, 412–416.

[114] Sytsma, T. (2014). Handling risk: Testosterone and risk preference, evidence from Dhaka, Bangladesh, Master Thesis, University of San Francisco, `http://repository.usfca.edu/thes/85`.

[115] Tversky, A., Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of Uncertainty, Journal of Risk and Uncertainty 5, 297–323.

[116] U.S. Department of Health and Human Services (2010). Use of contraception in the United States: 1982 – 2008, Vital and Health Statistics Series 23, No. 29.

[117] Votinov, M., Knyazeva, I., Habel, U., Konrad, K., Puiu, A.A. (2022). A Bayesian modelling approach to examine the role of testosterone administration on the endowment effect and risk-taking, Frontiers in Neuroscience 16, 858168.

[118] Wiegratz, I., Jung-Hoffmann, C., Kuhl, H. (1995). Effect of two oral contraceptives containing ethinylestradiol and gestodene or nogestimate upon androgen parameters and serum binding proteins, Contraception 51, 341–346.

[119] Wilcox, A.J., Baird, D. D., Dunson, D.B., McConnaughey, D.R., Kesner, J.S., Weinberg, C.R. (2004). On the frequency of intercourse around ovulation: Evidence for biological influences, Human Reproduction 19, 1539-1543.

[120] Wirth, M.M., Schultheiss, O.C. (2006). Effects of affiliation arousal (hope of closeness) and affiliation stress (fear of rejection) on progesterone and cortisol Hormones and Behavior 50, 786–795.

[121] Zethraeus, N., Kocoska-Maras, L., Ellingsen, T., von Schoultz, B., Lindén Hirschberg, A., Johannesson, M. (2009). A randomized trial of the effect of estrogen and testosterone on economic behavior, Proceedings of the National Academy of Sciences 106, 6535–6538

[122] Zheng, Z., Cohn, M.J. (2011). Developmental basis of sexually dimorphic digit ratios, Proceedings of the National Academy of Sciences 108, 16289–16294.

Supplementary Appendix for "Sex Hormones and Choice under Risk" by Burkhard C. Schipper

A Instruction for Saliva Collection

Instructions for Saliva Collection Terminal: __

In this experiment we are collecting saliva from the participants (you). The saliva is analyzed for the hormones it contains. You have received a collection tube. We need it about half full. Please do not eat, drink or chew any chewing gum other than provided by the experimenter during the experiment, as this will affect your saliva.

How to collect saliva?

1. Chew one piece of Trident original sugarless chewing gum to stimulate saliva.
2. After half a minute, spit the gum out into a tissue.
3. Uncap the collection tube.
4. A short straw is provided for you. Please place it in the tube.
5. Drool saliva through the straw into the tube until it is approximately half full.
6. Remove the straw onto a tissue.
7. Recap the tube.

The experimenters will collect the tubes during the experiment.

The used chewing, straws and tissues should be deposited into the rubbish bin at the end of the experiment.

If you have any questions, please raise your hand and an experimenter will attend to your question.

B Salivary Hormone Methodology

B.1 Steroid Hormones

Sex differences in brain and behavior are to a large extent influenced by sex steroids. According to the classic organizational-activational hypothesis of sexual differentiation (see Phoenix et al, 1959), a transient rise in testosterone during prenatal or early postnatal development masculinizes the brain in males, while the absence of testosterone leads to female neural structures. During puberty, testicular and ovarian hormones act on previously sexually differentiated circuits to facilitate expression of sex-typical behaviors in particular social contexts (see Schultz et al., 2009, Arnold and Breedlove, 1985, and Kelly et

al., 1999). Thus, sex steroids are thought to have both organizational effects and activational effects. Organizational effects refer to more permanent influences that often occur prenatally, early after birth, or during puberty. An example is the prenatal organization of tissues of male adult reproductive behavior. Activational effects are temporary effects and often they depend on prior organizational effects. For instance, a male does not display mating behavior facilitated by the prenatally organized tissues unless adequate sex hormones are produced at puberty (see Baron-Cohen et al. 2006).

Often, exogenous hormones are distinguished from endogenous hormones. The latter are hormones released by glands in the body, while exogenous hormones are hormones administered orally, transdermally, intranasally, or by various types of injections. Exogenous hormones may have effects different from endogenous hormones of the same type depending on absorbtion, metabolism, and the presence of prior organizational effects mentioned above. For instance, orally administered steroids are subject to the first-pass effect, i.e., the metabolic reduction of the steroid in the liver, and not all of the administered dose becomes available in the blood for binding on receptors.

We focus on four endogenous steroid hormones: testosterone, estradiol, progesterone, and cortisol (for overviews, see Nelson, 2011). Testosterone, $C_{19}H_{28}O_2$, belongs to the androgen group. It is derived via some intermediated steps from cholesterol and secreted in the testis, ovaries, and adrenal gland. Some of it is aromatized into estradiol. Since it is observed in most vertebrates, it must have had a long evolutionary history (Mechoulam et al., 1984). Testosterone has anabolic effects such as stimulating the bone density and muscle mass as well as androgenic effects such as the maturation of sex organs and secondary sex characteristics especially in males. It is necessary for sperm development. In humans, various behavioral correlations with testosterone have been reported mostly pertaining to aggression (e.g. Archer, 1991) and dominance (e.g. Mazur and Booth, 1998, Mehta and Josephs, 2006).

Estradiol, $C_{18}H_{24}O_2$, sometimes also named E2 or 17β-estradiol, is a member of the estrogen group. It is also derived via some intermediated steps from cholesterol and secreted in the testis, ovaries, and the adrenal cortex. It changes over the menstrual cycle, peaking shortly before ovulation and again in the second half of the cycle (see Fritz and Speroff, 2011). However, in blood plasma, estradiol is bound to globulin and albumin, and only a small fraction is free and biologically active. This fraction is constant over the menstrual cycle (Wu et al., 1976). Estradiol enters cells relatively freely. Its anabolic effects include effects on the bone structure and its androgenic effects are on the maturation of female sex organs and secondary sex characteristics. Wu et al. (2009) suggest that the aromatization of testosterone into estradiol is important for the organization and activation of neural circuits that control male territorial behaviors in mice.

Progesterone, $C_{201}H_{300}O_{200}$, sometimes denoted by P4, belongs to the progesten group. It is derived from cholesterol, secreted in the ovaries, especially the corpus luteum, the adrenal glands, and during pregnancy in the placenta. It is also contained in milk. Progesterone is stored in fat tissue. It can be metabolized (via some intermediate steps) into cortisol, testosterone, and estradiol. Progesterone changes over the menstrual cycle, rising after ovulation and declining before menstruation (see Fritz and Speroff, 2011). As its name suggests, it plays a prominent role during pregnancy ("pro-gestation"). Progesterone is a neurosteroid that can be synthesized within the central nervous system. There is a surprisingly small literature on behavioral effects in humans; see Fleischman and Fessler (2011), Maner and Miller (2014), Schultheiss, Wirth, and Stanton (2004), Wirth and Schultheiss (2006), and Brown et al. (2009).

Cortisol, $C_{21}H_{30}O_5$, is a steroid hormone belonging to glucocorticoid group. It is secreted in adrenal

glands and controlled by the hypothalamus. It is considered to be the "stress hormone" since it is released in response to stress (Hellhammer, Wuest, and Kudielka, 2009). It increases blood sugar, suppresses the immune system, and is aiding fat, protein, and carbohydrate metabolism. As the other steroid hormones, it is derived from cholesterol via some intermediated steps. Massage (Field et al. 2005), intimacy (Ditzen et al., 2007, 2008), and sexual arousal (Hamilton and Meston, 2011) reduce cortisol levels. Caffeine (Lovallo et al., 2006) and sleep deprivation (Leproult et al, 1997) can increase cortisol levels. Cortisol follows a typical circadian cycle. On average it is lowest at 4:00 am and peaks at 8:00 am. In terms of behavioral effects, cortisol may interact with testosterone. For instance, Mehta and Josephs (2010) report that testosterone is positively correlated with dominance in low cortisol males while negatively correlated in high cortisol males.

B.2 Instruction for Saliva Collection

Instructions for Saliva Collection Terminal: __

In this experiment we are collecting saliva from the participants (you). The saliva is analyzed for the hormones it contains. You have received a collection tube. We need it about half full. Please do not eat, drink or chew any chewing gum other than provided by the experimenter during the experiment, as this will affect your saliva.

How to collect saliva?

1. Chew one piece of Trident original sugarless chewing gum to stimulate saliva.
2. After half a minute, spit the gum out into a tissue.
3. Uncap the collection tube.
4. A short straw is provided for you. Please place it in the tube.
5. Drool saliva through the straw into the tube until it is approximately half full.
6. Remove the straw onto a tissue.
7. Recap the tube.

The experimenters will collect the tubes during the experiment.

The used chewing, straws and tissues should be deposited into the rubbish bin at the end of the experiment.

If you have any questions, please raise your hand and an experimenter will attend to your question.

B.3 Further Details on Saliva Collection and Storage

All sessions were run between February 8 and March 16, 2010, at the same time of the day in the afternoon. This is important as some hormones such as cortisol follow a circadian cycle (Van Cauter and Turek, 1995). The starting time of each session, 16:00, was scheduled such as to have sufficient time passed after lunch and to complete the session before dinner time. This is because salivary testosterone or cortisol may respond to meals 30 to 60 minutes before saliva collection (e.g. Al-Dujaili and Bryant,

2005). For testosterone, late-afternoon collections represented samples with physiologically relevant "low" hormone concentrations (Granger et al., 2004).

We must mention that the switch to Daylight Saving Time occurred on March 14, 2010. Although we were not able to find studies analyzing the effect of Daylight Saving Time on cortisol or other steroid hormones, it is know from Valdez et al. (2003) and Kantermann et al. (2007) that the switch to Daylight Saving Time may affect the circadian cycle. Thus, salivary hormones from subjects in sessions on March 15 and 16 may be affected by Daylight Saving Time. We will analyze this issue below.

Saliva samples were stored immediately after collection at -20°C till the end of March 2010 and then at -80°C till May 2010 when they were assayed. Granger et al. (2004) study testosterone concentration in stored saliva samples. They found no associations between testosterone levels and storage duration for samples stored at -80°C over a period of 36 months. The same applies for samples collected in the late afternoon and stored at -20°C over a period of 24 months.

B.4 Assays

Assays were conducted by the Endocrine Core Laboratory of the California National Primate Research Center at the University of California, Davis. Prior to assay of cortisol, progesterone, estradiol and testosterone, saliva samples were centrifuged at 3000 rpm for 20 min to separate the aqueous component from mucins and other suspended particles.

Salivary concentrations of testosterone were estimated in duplicate using the salivary testosterone enzyme immunoassay kit (Salimetrics LLC, State College, PA). Assay procedures were run in accordance to manufacturer's protocol salivary testosterone enzyme immunoassay kit insert revision 2-2010. The salivary testosterone assay has a least detectable dose of 1.0 pg/mL, and intra- and inter-assay coefficients of variation were 4.44 and 7.96, respectively.

Salivary concentrations of estradiol were estimated in duplicate using the high sensitivity salivary 17β–estradiol enzyme immunoassay kit (Salimetrics LLC, State College, PA). Assay procedures were run in accordance to manufacturer's protocol HS Salivary 17β-Estradiol EIA Kit Insert, revision date 2-22-10. The salivary estradiol assay has a least detectable dose of 0.1 pg/mL, and intra- and inter-assay coefficients of variation were 3.43 and 6.01, respectively.

Salivary concentrations of progesterone were estimated in duplicate using commercial radioimmunoassay kits (Siemens Healthcare Diagnostics, Inc., Los Angeles, CA). Assay procedures were modified to accommodate overall lower levels of progesterone in human saliva relative to plasma as follows: (1) standards were diluted to concentrations ranging from 0.05–4.0 ng/mL, and (2) sample volume was increased to 200 μl. The salivary progesterone assay has a least detectable dose of 0.00914 ng/ml, and intra- and inter-assay coefficients of variation were 4.15 and 5.84, respectively.

Salivary concentrations of cortisol were estimated in duplicate using commercial radioimmunoassay kits (Siemens Healthcare Diagnostics, Inc., Los Angeles, CA). Assay procedures were modified to accommodate overall lower levels of cortisol in human saliva relative to plasma as follows: (1) standards were diluted to concentrations ranging from 2.76 to 345 nmol/L, (2) sample volume was increased to 200 μl, and (3) incubation times were extended to 3 h. Serial dilution of samples indicates that the modified assay displays a linearity of 0.98 and a least detectable dose of 1.3854 nmol/L. Intra- and inter-assay

coefficients of variation are 5.44 and 6.12, respectively.

B.5 Factors Affecting Salivary Hormones

As mentioned above, the quality of saliva samples may be compromised by food intake prior to collection. To control for such effects, we asked subjects in the questionnaire to report whether or not they had lunch today, when they had lunch today, about the time they ate last, what they ate last, about the time they drank last, and what they drank last. From this we construct variables "When lunch today" that is zero if lunch was skipped and monotonically increases with the lunch time of the day. Similarly, we construct variables "Time last eaten" and "Time last drank" that monotonically increase with the time since last eaten (resp. drank).

Granger et al. (2004) and Kivlighan et al. (2004) show that salivary testosterone may be increased by blood contamination through microinjuries in the mouth or teeth brushing. Similarly, Kivlighan, Granger, and Schwartz (2005) observed decreased levels of salivary estradiol and increased levels of salivary progesterone due to microinjuries in the mouth or teeth brushing. Kivlighan et al. (2004) found that cortisol is irresponsive to microinjuries in the mouth or teeth brushing. To control for potential blood contamination, we asked subjects in the questionnaire to report on their daily dental care, the last time they brushed their tooth and whether they know of any injuries in their mouth. From this information we construct a dummy variable for "Mouth injuries", and variables "Freq. toothbrush." and "Time last toothbrush.", respectively.

Smoking may impact the endocrine system (Kapoor and Jones, 2005) but the evidence is mixed. Zumoff et al. (1990) show an association of smoking on serum levels of progesterone and estradiol but Thomas et al. (1993) were unable to find significant effects of smoking on salivary progesterone, plasma testosterone, and urinary estradiol. The use of tobacco can affect salivary testosterone levels (Attia et al., 1989). We don't know whether smoking could change the endocrine system or just measurable levels of salivary hormones. Anyway, we asked in the questionnaire to self-report the frequency of smoking and created a variable "Smoking" that is monotonically increasing in the frequency of smoking.

As mentioned above the switch to Daylight Saving Time on March 14, 2010, may affect our data collected on March 15 and 16. Although, we were not able to find studies analyzing the effect of Daylight Saving Time on cortisol or other steroid hormones, it is know from Valdez et al. (2003) and Kantermann et al. (2007) that the switch to Daylight Saving Time may affect the circadian cycle. We created a dummy variable "Daylight Sav. Time" that is one for sessions March 15 and 16 and zero otherwise.

In the questionnaire (see Appendix G) we collected further information on factors that may affect salivary hormones. Ellison and Lager (1986) report that moderate recreational running may be associated with lower salivary progesterone levels in females. Thus, we collect information on physical exercise scheduled. Brown et al. (2009) indicate that "social closeness" may effect progesterone. We asked for dating activities, whether students live alone, with family etc. Hooper et al. (2009) report associations between soy consumption and endocrinological factors. While they did not find an effect of soy consumption on estradiol, they found significantly reduced FSH and LH and increased menstrual cycle length. Our sample contains a large fraction of Asians and soybean protein is relatively common in ethnic asian food. Besides race, we also collect data on dietary preferences. Obesity has been linked to abnormal menstrual cycles and deficient progesterone secretion (Jain et al. 2007). Therefore we collect information on height

and weight. While all those factors may affect hormones, they may not necessarily affect the quality of the assays. Thus, we do not include them in the analysis of quality. Yet, the analysis is available from the author on request and can be produced from the datasets and the Stata do-file available from `https://faculty.econ.ucdavis.edu/faculty/schipper/`.

In Table 19, we present results from OLS regressions of salivary hormone levels normalized by their standard deviation on above-mentioned variables and session dummies by gender. "T", "E", "P", and "C" refer to testosterone, estradiol, progesterone, and cortisol, respectively. "F" and "M" refer to female and male, respectively. We use robust standard errors to adjust for potential heteroscedasticity and non-normality. We observe that whenever a variable is significant, then the coefficient is close to zero with four exceptions. Testosterone and cortisol of males (specifications TM and CM, respectively) where "'When lunch today" on average decreases testosterone by 0.07 of its standard deviation and cortisol by 0.13 of its standard deviation, respectively. Moreover, for estradiol in males we find that the frequency of smoking is positively correlated with salivary estradiol (specification EM). Finally, the frequency of brushing teeth is positively correlated with cortisol in males only (specification CM). This is somewhat surprising given that Kivlighan et al. (2004) found that cortisol is irresponsive to microinjuries in the mouth or teeth brushing. Some of the variables we used in robustness checks of our results.

B.6 Assessing Measurement Errors in Salivary Hormones

Measurement error cannot be avoided in salivary hormones measurements. Our results may by downward biased (i.e., attenuation bias). It is possible that our null findings are due to measurement error. In this section we study to what extent we can use the ex post salivary hormone measures to assess the measurement error of the basal (i.e., first sample) salivary hormone measures.

Let H_i^t denote the hormone measure of subject i at $t = 1, 2$, where $t = 1$ stands for the basal measure and $t = 2$ for the ex post measure. We assume that the basal measure H_i^1 is noisy but related to the true underlying hormone level H_i by $H_i^1 = H_i + \varepsilon_i^1$, where ε_i^1 is the measurement error of the basal measure. Similarly, the ex post measure $H_i^2 = H_i + \Delta_i + \varepsilon_i^2$, where Δ_i is the response of the hormone to events in the auction and ε_i^2 is the measurement error of the ex post measure. We assume $Cov(H_i, \varepsilon_i^1) = Cov(H_i, \varepsilon_i^2) = Cov(\Delta_i, \varepsilon_i^2) = 0$.

When we regress H_i^1 on H_i^2, the coefficient is given by

$$\beta_{12} = \frac{Cov(H_i^2, H_i^1)}{Var(H_i^2)} = \frac{Var(H_i) + Cov(H_i, \Delta_i) + Cov(\varepsilon_i^1, \Delta_i) + Cov(\varepsilon_i^1, \varepsilon_i^2)}{Var(H_i) + Var(\Delta_i) + Var(\varepsilon_i^2) + 2Cov(H_i, \Delta_i)}.$$

Likewise, when we regress H_i^2 on H_i^1, the coefficient is

$$\beta_{21} = \frac{Cov(H_i^2, H_i^1)}{Var(H_i^1)} = \frac{Var(H_i) + Cov(H_i, \Delta_i) + Cov(\varepsilon_i^1, \Delta_i) + Cov(\varepsilon_i^1, \varepsilon_i^2)}{Var(H_i) + Var(\varepsilon_i^1)}.$$

Thus, if $\Delta_i = 0$ for all i, then $\beta_{12} \leq \beta_{21}$ if and only if $Var(\varepsilon_i^1) \leq Var(\varepsilon_i^2)$. That is, in the absence of a hormone response, the coefficients β_{12} and β_{21} allow us to assess the measurement error of the basal hormone measurement relative to the ex post measurement. If there is a hormone response, $\Delta_i \neq 0$, then in order to assess measurement error further information on the variance of the hormone response

Table 19: Quality of Salivary Hormones

	(TF)	(TM)	(EF)	(EM)	(PF)	(PM)	(CF)	(CM)
When lunch today	−0.0099	−0.0703*	−0.0588	0.0150	−0.0901	−0.0220	−0.0529	−0.1341*
	(0.0211)	(0.0295)	(0.0727)	(0.0370)	(0.0660)	(0.0210)	(0.0421)	(0.0567)
Time last eaten	−0.0005	0.0017	−0.0015	−0.0002	−0.0022	−0.0019**	−0.0019*	−0.0029
	(0.0005)	(0.0009)	(0.0017)	(0.0009)	(0.0016)	(0.0007)	(0.0008)	(0.0015)
Time last drank	−0.0024*	−0.0023	−0.0046	0.0013	−0.0044	−0.0003	−0.0035	0.0007
	(0.0012)	(0.0017)	(0.0029)	(0.0017)	(0.0040)	(0.0011)	(0.0022)	(0.0020)
Mouth injuries	−0.0571	0.2321	−0.2568	0.0989	0.1947	−0.0797	0.1736	−0.0773
	(0.0963)	(0.2340)	(0.3165)	(0.2223)	(0.4825)	(0.1793)	(0.2256)	(0.2193)
Freq. toothbrush.	0.1487	0.0585	0.1030	0.1236	−0.5213	0.1655	−0.0380	0.3596*
	(0.1082)	(0.1168)	(0.2610)	(0.1182)	(0.3491)	(0.0974)	(0.1452)	(0.1580)
Time last toothbrush.	−0.0026	−0.0047	0.0020	−0.0088*	−0.0027	−0.0047**	−0.0116	0.0003
	(0.0044)	(0.0024)	(0.0054)	(0.0040)	(0.0040)	(0.0017)	(0.0070)	(0.0029)
Smoking	0.0495	0.1046	0.4001	0.4167*	0.1090	0.0999	−0.0564	0.1444
	(0.1096)	(0.1305)	(0.2703)	(0.1969)	(0.2889)	(0.1124)	(0.2054)	(0.2353)
Daylight Sav. Time	0.2276	−0.1032	0.3933	0.1314	0.3975	0.0109	1.5397	−0.1674
	(0.2136)	(0.2632)	(0.6583)	(0.3033)	(0.8050)	(0.1330)	(0.8122)	(0.2983)
Number of Observations	93	115	93	115	93	114	93	114
R^2	0.0980	0.1630	0.0763	0.1814	0.0738	0.2009	0.2684	0.1605

Robust standard errors in parentheses; Significance levels: * 5%; ** 1%.

and the covariance of the underlying hormone level and the hormone response would be required. This information is typically not available.

Table 20: Regressing Basal on Ex Post Measures and Vice Versa

	(E12)	(E21)	(P12)	(P21)
Ex Post Estradiol	0.6980**			
	(0.0520)			
Basal Estradiol		0.6721**		
		(0.0501)		
Ex Post Progesterone			0.7762**	
			(0.0410)	
Basal Progesterone				0.8229**
				(0.0434)
Number of Observations	206	206	205	205
R^2	0.4691	0.4691	0.6387	0.6387

Standard errors in parentheses. Significance levels: * 5%; ** 1%

As discussed in our companion paper, Schipper (2015), salivary testosterone and cortisol may respond to events in the auction while we do not expect estradiol or progesterone to respond to events in the auction. If we assume this hypothesis to hold, then for these two hormones we can use the ex post salivary measures to assess the measurement error in the salivary measures of those basal hormones. Table 20 shows the results from regressing basal estradiol on ex post estradiol (specification "E12"), ex post estradiol on basal estradiol (specification "E21"), basal progesterone on ex post progesterone (specification "P12"), and ex post progesterone on basal progesterone (specification "P21"). The coefficients for estradiol are of very similar magnitude. The coefficient of ex post progesterone in "P12" is slightly smaller than the coefficient of basal progesterone in "P21" suggesting that basal progesterone has a slightly smaller measurement error. We conclude that our measurements of basal estradiol and progesterone do not have larger measurement errors than our ex post measurements. Nevertheless, we emphasize again that some degree of measurement error is unavoidable in endocrinological economics. This may lead to the underestimation of the size of the true association between behavior and hormones.

C Further Details on Hormonal Contraceptives

Hormonal contraceptives may influence endogenous basal hormone levels. Schultheiss et al. (2003) report that users of oral contraceptives show suppressed levels of salivary testosterone and estradiol.

Roughly 25.6% of women in our sample administer hormonal contraceptives. This number is reasonable given the age of women and their ethnic background.[24] From 21 females using hormonal

[24]The United States Department of Health and Human Services (2010) estimates that in the US roughly over 11% of asian, hispanic, and black women between 15 to 44 years of age use oral contraceptives compared to over 21% of white women. The use of the oral contraceptives varies also with age. In the age group 15 to 19, it is slightly over 15%, while it increases to 26% in the age group 20 to 24. Note that the mean age of women in our sample is 20.1 years old. Another study, Collins and Hershbein (2011), finds higher percentages of users among college women but that study contains only about 10% of Asian women. The use of oral contraceptives is less

contraceptives, 10 females reported the name of the contraceptive. This enabled us to evaluate their typical administration schedules and active ingredients.

Contraceptives can be classified into three categories (see Fritz and Speroff, 2011, Section III, for an extensive overview): First, there are injections like Depo Provera. This is a long-acting reversible contraceptive acting over 12 weeks containing as the active ingredient only a progestin. Second, there are oral birth control pills. While some of the pills available may contain as the active ingredient a progestin only, all the pills reported in the experiments contained both a progestin as well as ethinyl estradiol. Ethinyl estradiol is not converted into estradiol in the body but can bind to estrogen receptors. There are oral contraceptives that contain the active ingredient for three weeks and an inert ingredient (i.e., placebo) for one week during which a withdrawal bleeding usually occurs (e.g. Avian, Desogen, Junel, Microgestin, Ortho-Tri-Cyclen, Sprintec, and Yasmin). Then there are oral contraceptives that contain the active ingredient for 24 days after which an inert ingredient is taken for 4 days during which withdrawal bleeding usually occurs (e.g. Yaz). For some oral contraceptives like Ortho-Tri-Cyclen, the concentration of active ingredient varies across the cycle (i.e., biphasic or triphasic oral contraceptives). Third, there are extended cycle oral contraceptives that contain an active ingredient for 84 days after which an inert ingredient is used for 7 days during which withdrawal bleeding usually occurs (e.g. Seasonale). Fourth, there is the NuvaRing, a flexible vaginal ring that when placed in the vagina releases both a progestin as well as estradiol over a period of three weeks, after which it is removed for a one-week break during which a withdrawal bleeding occurs. Compared to oral contraceptives, whose active ingredients peak about two hours after intake and then decline for the rest of the day, the NuvaRing releases the active ingredients more constantly. Except for Depo Provera, all hormonal contraceptives reported involve a regular break/placebo during which circulating levels of progesterone are expected to be lower than when active ingredients are taken. This break may affect behavior. Not all women may observe the break but skip the placebo or break in order to avoid the inconvenience of withdrawal bleeding.

Different hormonal contraceptives contain different progestins, and different progestins have different effects on the brain. Not all progestins can be converted into the $GABA_A$ receptor–active metabolites (Pluchino et al., 2009). Thus, not all hormonal contraceptives may have the same slight sedating effect that we alluded to in the main text. While we find the progesterone-$GABA_A$-sedation explanation for the correlation with the use of hormonal contraceptives quite attractive, we cannot claim a causal effect since it may be a selection effect. In particular, women who decide to take hormonal contraceptives may also differ systematically in their bidding behavior from women who decide not to take any hormonal contraceptives. It is not clear whether a priori more risk averse women are more likely to use hormonal contraceptives or whether women with more risky sexual behavior are more likely to take hormonal contraceptives. Conclusive evidence could be obtained in an experiment in which oral contraceptives and a placebo are blindly and randomly assigned to women. Obviously, such an experiment would be rather difficult to conduct. Moreover, women who would agree to participate in such a "risky" experiment may systematically differ in their risk preferences from the rest of the population.

---

common in Asian women compared to white.

D Further Details on the 2D:4D Measurements

For each participant, each digit was measured independently by two researchers. To analyze the consistency of agreement of the two finger measurements across the two researchers, we compute intraclass correlations using a two-way mixed model (Shrout and Fleiss, 1979). Each participant's digit is considered as a target that is measured by two researchers. The method requires us to analyze intraclass correlations for each digit separately. For each digit, there are 208 targets and two researchers. The analysis treats targets as random and researchers as fixed.

Table 21: Intraclass Correlations for Measurements

	Second Digit			Fourth Digit		
	ICC	95% Conf.	Interval	ICC	95% Conf.	Interval
Individual	0.987	0.983	0.990	0.992	0.990	0.994
Average	0.994	0.991	0.995	0.996	0.995	0.997

E Correlations of Biological Measures

Table 22: Pairwise Correlations of Biological Measures

Variables	Female	DR	Pre-Men.	Menstr.	Follic.	Peri-Ov.	Luteal	E.a. T	E.a. E	E.a. P	E.a. C	E.p. T	E.p. E	E.p. P	E.p. C
Female	1.000														
Digit Ratio	0.185	1.000													
Pre-Menstrual Phase	0.380	0.067	1.000												
Menstrual Phase	0.356	0.076	-0.119	1.000											
Follicular Phase	0.309	0.060	-0.103	-0.097	1.000										
Peri-Ovulatory Phase	0.232	0.034	-0.077	-0.072	-0.063	1.000									
Luteal Phase	0.417	0.075	-0.139	-0.130	-0.113	-0.085	1.000								
Ex ante Testosterone	-0.752	-0.208	-0.307	-0.278	-0.231	-0.164	-0.313	1.000							
Ex ante Estradiol	0.132	-0.061	0.073	-0.041	0.049	0.096	0.073	0.058	1.000						
Ex ante Progesterone	0.174	0.084	0.244	-0.144	-0.048	0.193	0.092	-0.053	0.434	1.000					
Ex ante Cortisol	-0.106	-0.007	-0.091	0.088	0.002	-0.076	-0.114	0.270	-0.012	0.070	1.000				
Ex post Testosterone	-0.708	-0.232	-0.294	-0.281	-0.198	-0.140	-0.293	0.890	0.012	-0.097	0.187	1.000			
Ex post Estradiol	0.126	-0.054	0.040	-0.071	0.048	0.118	0.108	0.027	0.684	0.359	-0.011	0.061	1.000		
Ex post Progesterone	0.144	0.073	0.204	-0.157	-0.069	0.227	0.091	-0.037	0.296	0.799	-0.001	-0.012	0.475	1.000	
Ex post Cortisol	-0.240	-0.054	-0.099	-0.124	-0.060	-0.068	-0.066	0.393	0.000	-0.058	0.474	0.445	-0.006	0.010	1.000

F Holt-Laury Lottery Task

Instructions for the Lottery Experiment Terminal: __

Along with these instructions, you have received two decision sheets. Each of them shows ten decisions listed on the left. Each decision is a paired choice: either "Option A" or "Option B." On each sheet, you will make ten choices and record these in the final column, but only one of them from each sheet will be used in the end to determine your earnings. Before you start making your ten choices, please let me explain how these choices will affect your earnings for this part of the experiment.

There is a ten-sided die that will be used to determine payoffs in front of your eyes; the faces are numbered from 1 to 10 (the "0" face of the die will serve as 10). After you have made all of your choices, we will throw this die twice for each decision sheet, once to select one of the ten decisions of the sheet to be used, and a second time to determine what your payoff is for the option you chose, A or B, for the particular decision selected. Even though you will make ten decisions on each sheet, only one of these from each sheet will end up affecting your earnings, but you will not know in advance which decisions will be used. Obviously, each decision has an equal chance of being used in the end.

Now, please look at Decision 1 at the top of the first sheet. Option A yields a sure gain of $3.20 (320 cents), and option B yields a sure gain of $0.20 (20 cents). Next look at Decision 2 in the second row. Option A yields $4.00 if the throw of the ten sided die is 1, and it yields $3.20 if the throw is 2-10. Option B yields $7.70 if the throw of the die is 1, and it yields $0.20 if the throw is 2-10. The other decisions on the sheet are similar, except that as you move down the table, the chances of the better payoff for each option increase.

The second decision sheet is identical to the first one except for one difference: all payoffs are negative. For instance look at Decision 1 at the top of the second sheet. Option A yields a sure loss of $3.20 (minus 320 cents), and option B yields a sure loss of $0.20 (minus 20 cents). Payoffs for this choice are negative and will be subtracted from your previous earnings.

To summarize, on each decision sheet you will make ten choices: for each decision row you will have to choose between Option A and Option B. You may choose A for some decision rows and B for other rows, and you may change your decisions and make them in any order. When you are finished, we will come to your desk and collect both decision sheets. Then the market experiment will be run. After the market experiment we will throw the ten-sided die for each decision sheet to select which of the ten Decisions will be used. Then we will throw the die again for each decision sheet to determine your payoff for the Option you chose for that Decision. Payoffs for your choices and will be added/subtracted to/from your previous earnings from the market experiment, and you will be paid the sum of all earnings in cash when we finish.

So now please look at the empty boxes on the right side of the record sheet. You will have to write a decision, A or B in each of these boxes, and then the die throw will determine which one is going to count. We will look at the decision that you made for the choice that counts, and circle it, before throwing the die again to determine your earnings for this part. Then you will write your earnings in the blank at the bottom of the page. Please note that these gains/losses will be added/subtracted to/from your previous earnings up to now.

Are there any questions? Now you may begin making your choices. Please do not talk with anyone while we are doing this; raise your hand if you have a question.

Terminal: ___ Session No.: _____

Decision Sheet (Gains)

	Option A	Option B	Your Choice A or B
Decision 1	$3.20 if throw of die is 1 to 10	$0.20 if throw of die is 1 to 10	
Decision 2	$4.00 if throw of die is 1 $3.20 if throw of die is 2 to 10	$7.70 if throw of die is 1 $0.20 if throw of die is 2 to 10	
Decision 3	$4.00 if throw of die is 1 or 2 $3.20 if throw of die is 3 to 10	$7.70 if throw of die is 1 or 2 $0.20 if throw of die is 3 to 10	
Decision 4	$4.00 if throw of die is 1 to 3 $3.20 if throw of die is 4 to 10	$7.70 if throw of die is 1 to 3 $0.20 if throw of die is 4 to 10	
Decision 5	$4.00 if throw of die is 1 to 4 $3.20 if throw of die is 5 to 10	$7.70 if throw of die is 1 to 4 $0.20 if throw of die is 5 to 10	
Decision 6	$4.00 if throw of die is 1 to 5 $3.20 if throw of die is 6 to 10	$7.70 if throw of die is 1 to 5 $0.20 if throw of die is 6 to 10	
Decision 7	$4.00 if throw of die is 1 to 6 $3.20 if throw of die is 7 to 10	$7.70 if throw of die is 1 to 6 $0.20 if throw of die is 7 to 10	
Decision 8	$4.00 if throw of die is 1 to 7 $3.20 if throw of die is 8 to 10	$7.70 if throw of die is 1 to 7 $0.20 if throw of die is 8 to 10	
Decision 9	$4.00 if throw of die is 1 to 8 $3.20 if throw of die is 9 or 10	$7.70 if throw of die is 1 to 8 $0.20 if throw of die is 9 or 10	
Decision 10	$4.00 if throw of die is 1 to 9 $3.20 if throw of die is 10	$7.70 if throw of die is 1 to 9 $0.20 if throw of die is 10	

Decision used: _____ Die throw: _____ Your earnings on this sheet: _____

Terminal: ___ Session No.: _____

Decision Sheet (Losses)

	Option A	Option B	Your Choice A or B
Decision 1	-$3.20 if throw of die is 1 to 10	-$0.20 if throw of die is 1 to 10	
Decision 2	-$4.00 if throw of die is 1 -$3.20 if throw of die is 2 to 10	-$7.70 if throw of die is 1 -$0.20 if throw of die is 2 to 10	
Decision 3	-$4.00 if throw of die is 1 or 2 -$3.20 if throw of die is 3 to 10	-$7.70 if throw of die is 1 or 2 -$0.20 if throw of die is 3 to 10	
Decision 4	-$4.00 if throw of die is 1 to 3 -$3.20 if throw of die is 4 to 10	-$7.70 if throw of die is 1 to 3 -$0.20 if throw of die is 4 to 10	
Decision 5	-$4.00 if throw of die is 1 to 4 -$3.20 if throw of die is 5 to 10	-$7.70 if throw of die is 1 to 4 -$0.20 if throw of die is 5 to 10	
Decision 6	-$4.00 if throw of die is 1 to 5 -$3.20 if throw of die is 6 to 10	-$7.70 if throw of die is 1 to 5 -$0.20 if throw of die is 6 to 10	
Decision 7	-$4.00 if throw of die is 1 to 6 -$3.20 if throw of die is 7 to 10	-$7.70 if throw of die is 1 to 6 -$0.20 if throw of die is 7 to 10	
Decision 8	-$4.00 if throw of die is 1 to 7 -$3.20 if throw of die is 8 to 10	-$7.70 if throw of die is 1 to 7 -$0.20 if throw of die is 8 to 10	
Decision 9	-$4.00 if throw of die is 1 to 8 -$3.20 if throw of die is 9 or 10	-$7.70 if throw of die is 1 to 8 -$0.20 if throw of die is 9 or 10	
Decision 10	-$4.00 if throw of die is 1 to 9 -$3.20 if throw of die is 10	-$7.70 if throw of die is 1 to 9 -$0.20 if throw of die is 10	

Decision used: _____ Die throw: _____ Your earnings on this sheet: _____

G Questionnaire

SURVEY (collected on the subject's computer terminal)

We are interested in whether there is a correlation between participants' bidding behavior and some socio-psychological and biological factors. It is an extremely important part of our research. This information will be strictly confidential.

1. What is your (biological) sex?
    - o Male
    - o Female

2. What is your sexual orientation?
    - o Heterosexual
    - o Homosexual
    - o Bisexual
    - o Transsexual

3. Are you currently in a relationship?
    - o No
    - o Married
    - o Boyfriend/girlfriend

4. How many people did you date within the last year? (drop down menu)
    - o None
    - o 1 person
    - o 2 persons
    - o 3 persons
    - o 4 persons
    - o 5 persons
    - o 6 persons
    - o 7 persons
    - o 8 persons
    - o 9 persons
    - o 10 persons
    - o More than 10 persons

5. Do you have children? (drop down menu)
    - o No
    - o 1 child
    - o 2 children
    - o 3 children
    - o 4 children
    - o More than 4 children

6. What is your ethnic origin? (You may choose several.)
   - o White
   - o Asian/Asian American
   - o African American
   - o Hispanic/Latino
   - o Native American
   - o Other

7. What is your age (in years)? _____

8. What is your weight (in pounds)? _____

9. What is your height (in inches)? _____ *(Remark: We helped them to calculate if known only in feet or cm)*

10. How many siblings do you have?
    I have ___ younger siblings.
    I have ___ older siblings.

11. How often do you exercise in an average week?
    - o Never
    - o At least once a week
    - o At least twice a week
    - o At least three times a week
    - o Four or more times a week

12. Have you ever broken a finger on your right hand?
    - o No
    - o Yes

13. If yes, was it the pointer or ring finger?
    - o Yes
    - o No

14. Would you describe your personality as (please choose one)
    - o optimistic
    - o pessimistic
    - o neither

15. Which of the following emotions did you experience during the experiment? (You may choose any number of them.)
    - o anger
    - o anxiety

- o confusion
- o contentment
- o fatigue
- o happiness
- o irritation
- o mood swings
- o withdrawal

16. Do you live
    - o alone
    - o with your parents
    - o with your partner/boyfriend/girlfriend/spouse
    - o with a roommate?

For female participants only:

17. Are you pregnant?
    - o No
    - o Yes
    - o May be

18. How many days ago was the first day of your **last** menstrual period? **_____**

19. What is your best guess on how many days until your **next** menstrual cycle? _____

20. On average, how many days are there between your menstrual periods?
    - o less than 25 days
    - o 25 days
    - o 26 days
    - o 27 days
    - o 28 days
    - o 29 days
    - o 30 days
    - o 31 days
    - o 32 days
    - o 33 days
    - o 34 days
    - o 35 days
    - o more than 35 days

21. Do you often experience changes in the length of your menstrual cycle?
    - o No, it is quite regular and almost always takes the same number of days.
    - o The length is irregular.

22. Do you keep a menstrual cycle calendar?
    - o Yes
    - o No

23. Do you usually experience any symptoms of PMS? (please choose one)
    - o None
    - o **M**ild
    - o Severe

24. Are you currently experiencing any symptoms of PMS (please choose one)
    - o None
    - o Mild
    - o Severe

25. Do you currently use a hormone-based contraceptive (birth control pill, IUD, contraceptive patch [OrthoEvra], vaginal ring [Nuvaring], Norplant, IUS, injection [DepoProvera, Lunelle], etc.)?
    - o Yes
    - o No

26. If yes, what type? _____

For all participants:

27. Do you smoke?
    - o Daily
    - o Occasionally
    - o Never

28. Do you regularly take dietary supplements that help you perform better in sports?
    - o No
    - o Yes

29. If yes, what type? _____

30. Are you vegetarian or vegan?
    - o No
    - o Yes

31. Do you regularly eat soybean-based food like tofu, soymilk etc.?
    - o Not at all
    - o Not very often
    - o Yes, daily

- o Yes, several times a week

32. When did you have lunch today?
    - o I skipped lunch
    - o 11.00 am
    - o 12.00 pm
    - o 1.00 pm
    - o 2.00 pm
    - o 3.00 pm

33. Before arriving at the experiment, how long has it been since you last ate?
    - o 30 min
    - o 1 hour
    - o 2 hours
    - o 3 hours
    - o 4 hours
    - o More than 4 hours ago

34. What did you eat last? _____

35. Did you drink coffee/tee/other drinks in the past two hours before arriving at the experiment?
    - o Yes, within 30 min before the experiment
    - o Yes, within 1 hour before the experiment
    - o Yes, within 1.5 hours before the experiment
    - o Yes, within 2 hours before the experiment

36. What did you drink last? _____

37. Do you currently have any small injuries in your mouth or gums (cuts, sores, bleeding)?
    - o Yes
    - o No

38. How many times a day do you brush your teeth?
    - o Never
    - o Once a day
    - o Twice a day
    - o Three times a day
    - o More than three times a day

39. When was the last time you brushed your teeth?
    - o 30 minutes ago
    - o 1 hours ago

- o 2 hours ago
- o 3 hours ago
- o More than 3 hours ago

40. What was your SAT score? ____

41. What is your major field of study?
    - o Economics
    - o Mathematics
    - o Other Social Science
    - o English
    - o Other Arts/Humanities
    - o Chemistry/Biology/Physics
    - o Other Natural Science
    - o Engineering

42. What is your current GPA? ____

43. If you are student, how many quarters have you completed? ____

References

[1] Al-Dujaili, E.A.S., Bryant, M.L. (2005). Effect of meal fat content on salivary testosterone and cortisol levels in healthy female volunteers, Endocrine Abstracts 10, P75.

[2] Arnold, A.P., Breedlove, S.M. (1985). Organizational and activational effects of sex steroids on brain and behavior: A reanalysis, Hormones and Behavior 19, 469–498.

[3] Attia, A.M., el-Dakhly, M.R., Halawa, F.A., Ragab, N.F., Mossa, M.M. (1989). Cigarette smoking and male reproduction, Archives of Andrology 23, 45-49.

[4] Baron-Cohen, S., Lutchmaya, S., Knickmeyer, R. (2006). Prenatal testosterone in mind, The MIT Press.

[5] Brown, S.L., Fredrickson, B.L., Wirth, M.M., Poulin, M.J., Meier, E.A., Heaphy, E.D., Cohen, M.D., Schultheiss, O.C. (2009). Social closeness increases salivary progesterone in humans, Hormones and Behavior 56, 108-111.

[6] Collins, E.G., Hershbein, B. (2011). The impact of subsidized birth control on college women: Evidence from the Deficit Reduction Act, University of Michigan.

[7] Ditzen, B., Hoppmann, C., Klumb, P. (2008). Positive couple interactions and daily cortisol: On the stress-protecting role of intimacy, Psychosomatic Medicine 70, 883–889.

[8] Ditzen, B., Neumann, I.D., Bodenmann, G., von Dawans, B., Turner, R.A., Ehlert, U., Heinrichs, M. (2007). Effects of different kinds of couple interaction on cortisol and heart rate responses to stress in women, Psychoneuroendocrinology 32, 565-574.

[9] Ellison, P.T., Lager, C. (1986). Moderate recreational running is associated with lower salivary progesterone profiles in women, American Journal of Obstetrics and Gynecology 154, 1000–1003.

[10] Fleischman, D.S., Fessler, D.M.T. (2011). Progesterone's effects on the psychology of disease avoidance: Support for the compensatory behavioral prophylaxis hypothesis, Hormones and Behavior 59, 271–275.

[11] Field, T., Hernandez-Reif, M., Diego, M. (2005). Cortisol decreases and serotonin and dopamin increae following massage therapy, International Journal of Neuroscience 115, 1397–1413.

[12] Fritz, M.A., Speroff, L. (2011). Clinical gynecologic endocrinology and infertility, Eight edition, Lippincott Williams & Wilkins.

[13] Granger, D.A., Shirtcliff, E.A., Booth, A., Kivlighan, K.T., Schwartz, E.B. (2004). The "trouble" with salivary testosterone, Psychoneuroendocrinology 29, 1229-1240.

[14] Hamilton, L. D., Meston, C.M. (2011). The role of salivary cortisol and DHEA-S in response to sexual, humorous, and anxiety-inducing stimuli, Hormones and Behavior 59, 765–771.

[15] Hooper, L., Ryder, J.J., Kurzer, M.S., Lampe, J.W., Messina, M.J., Phipps, W.R., Cassidy, A. (2009). Effects of soy protein and isoflavones on circulating hormone concentrations in pre- and post-menopausal women: A systematic review and meta-analysis, Human Reproduction Update 15, 423-440.

[16] Jain, A., Polotsky, A.J., Rochester, D., Berga, S.L., Loucks, T., Zeitlian, G., Gibbs, K., Polotsky, H.N., Feng, S., Isaac, B., Santoro, N. (2007). Pulsatile luteinizing hormone amplitude and progesterone metabolite excretion are reduced in obese women, Journal of Clinical Endocrinology and Metabolism 92, 2468-2473.

[17] Kantermann, T., Juda, M., Merrow, M., Roenneberg, T. (2007). The human circadian clock's seasonal adjustment is disrupted by Daylight Saving Time, Current Biology 17, 1996–2000.

[18] Kapoor, D., Jones, T.H. (2005). Smoking and hormones in health and endocrine disorders, European Journal of Endocrinology 152, 491-499.

[19] Kelly, S.J., Ostrowski, N.L., Wilson, M.A. (1999). Gender differences in brain and behavior: Hormonal and neural bases, Pharmacology Biochemistry and Behavior 64, 655–664.

[20] Kivlighan, K.T., Granger, D.A., Schwartz, E.B. (2005). Blood contamination and the measurement of salivary progesterone and estradiol, Hormones and Behavior 47, 367-370.

[21] Kivlighan, K.T., Granger, D.A., Schwartz, E.B., Nelson, V., Curran, M., Shirtcliff, E.A. (2004). Quantifying blood leakage into the oral mucosa and its effects on the measurement of cortisol, dehydroepiandrosterone, and testosterone in saliva, Hormones and Behavior 46, 39-46.

[22] Leproult, R., Copinschi, G., Buxton, O., van Cauter, E. (1997). Sleep loss results in an elevation of cortisol levels the next evening, Sleep 20, 865–870.

[23] Lovallo, W.R., Farag, N.H., Vincent, A.S., Thomas, T.L., Wilson, M.F. (2006). Cortisol responses to mental stress, exercise, and meals following caffeine intake in men and women, Pharmacology, Biochemistry and Behavior 83, 441–447.

[24] Maner, J.K., Miller, S.L. (2014) Hormones and social monitoring: Menstrual cycle shifts in progesterone underlie women's sensitivity to social information, Evolution and Human Behavior 35, 9–16.

[25] Mazur, A., Booth, A. (1998). Testosterone and dominance in men, Behavioral and Brain Sciences 21, 353–397.

[26] Mehta, P.H., Josephs, R.A. (2010). Testosterone and cortisol jointly regulate dominance: Evidence for a dual-hormone hypothesis, Hormones and Behavior 58, 898–906.

[27] Mehta, P.H., Josephs, R.A. (2006). Testosterone change after losing predicts the decision to compete again, Hormones and Behavior 50, 684-692.

[28] Mechoulam R., Brueggemeier. R.W., Denlinger D.L. (1984). Estrogens in insects, Journal Cellular and Molecular Life Sciences 40, 942–944.

[29] Nelson, R.J. (2011). An introduction to behavioral endocrinology, 4th edition, Sinauer Assoc.

[30] Pluchino, N., Cubeddu, A., Giannini, A., Merlini, S., Cela, V., Angioni, S., Genazzani, A.R. (2009). Progestogens and brain: An update, Maturitas 62, 349–355.

[31] Phoenix, C., Goy, R., Gerall, A., Young, W. (1959). Organizing action of prenatally administered testosterone propionate on the tissues mediating mating behavior in the female guinea pig, Endocrinology 65, 369–382.

[32] Schipper, B.C. (2015). Sex hormones and competitive bidding, Management Science 61, 249–266.

[33] Schultheiss, O.C., Dargel, A., Rohde, W. (2003). Implicit motives and gonadal steroid hormones: Effects of menstrual cycle phase, oral contraceptive use, and relationship status, Hormones and Behavior 43, 293–301.

[34] Schultheiss, O.C., Wirth, M.M., Stanton, S.J. (2004). Effects of affiliation and power motivation arousal on salivary progesterone and testosterone, Hormones and Behavior 46, 592–599.

[35] Schultz, K. M., Molenda–Figueira, H.A., Sisk, C.L. (2009). Back to the future: The organizationalCactivational hypothesis adapted to puberty and adolescence, Hormones and Behavior 55, 597–604.

[36] Shrout, P.E., Fleiss, J.L. (1979). Intraclass correlations: Uses in assessing rater reliability, Psychological Bulletin 86: 420–428.

[37] Thomas, E.J., Edridge, W., Weddell, A., McGU, A., McGarrigle, H.H.G. (1993). The impact of cigarette smoking on the plasma concentrations of gonadotrophins, ovarian steroids and androgens and upon the metabolism of oestrogens in the human female, Human Reproduction 8, 1187–1193.

[38] U.S. Department of Health and Human Services (2010). Use of contraception in the United States: 1982 – 2008, Vital and Health Statistics Series 23, No. 29.

[39] Valdez, P., Ramirez, C., Garcia, A. (2003). Adjustment of the sleep-wake cycle to small (1-2h) changes in schedule, Biological Rhythm Research 34, 145–155.

[40] Van Cauter E., Turek, F.W. (1995). Endocrine and other biological rhythms, in: De Groot L.J. (ed.), Endocrinology, Philadelphia: Saunders, 2487-2548.

[41] Wirth, M.M., Schultheiss, O.C. (2006). Effects of affiliation arousal (hope of closeness) and affiliation stress (fear of rejection) on progesterone and cortisol Hormones and Behavior 50, 786–795.

[42] Wu, M.V., Manoli, D.S., Fraser, E.J., Coats, J.K., Tollkuhn, J., Honda, S.I., Harada, N., Shah, N.M. (2009). Estrogen masculinizes neural pathways and sex-specific behaviors, Cell 139, 61–72.

[43] Wu, C.H., Motohashi. T., Abdel-Rahman, H.A., Flickinger, G.L., Mikhail, G. (1976). Free and protein-bound plasma estradiol-17 beta during the menstrual cycle, Journal of Clinical Endocrinology and Metabolism 43, 436-445.

[44] Zumoff, B., Miller, L., Levi, C.D., Miller, E.H., Heinz, U., Kalin, M., Denman, H., Jandorek, R., Rosenfeld, R.S. (1990). The effect of smoking on serum progesterone, estradiol, and luteinizing hormone levels over a menstrual cycle in normal women, Steroids 55, 507–511.