The Relationship between Social Cognition, Sex Steroids, and Economic Decision-Making

by

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Abstract

Decision-making involves the selection of one from several distinct options with distinct outcomes. Although previous research on economic decision-making under risk probed social, psychological, and biological influences, including endocrine factors such as gonadal and adrenal steroids, the intersection between these factors on economic decision-making is not well understood. As these hormone classes are functionally related to many aspects of social cognition, this dissertation describes three studies designed to explore the reciprocal relationship between steroid hormones (testosterone, estradiol, and cortisol), social cognition, and economic decision making. The first study tests whether baseline levels of steroid hormones have a differential effect on equivalent risky decisions in a simple gambling task when they are framed as either social or non-social, and whether such framing differences likewise cause changes in steroid hormone levels. The results show that female estradiol and male testosterone decrease in the non-social but not social framing of the same task, and that cortisol moderates some of this difference. The second study tests whether baseline levels of steroid hormones have a differential effect on decision-making after social facilitation versus provocation, and whether social facilitation versus provocation have differential effects on steroid hormone levels and decision-making in the Ellsberg Urns task. This task can characterize individual preferences for risk (where outcome probabilities are known) and ambiguity (where outcome probabilities are unknown), called the ambiguity premium. The results show that increases in female estradiol predict increases the ambiguity premium, but only after social facilitation. The third study tests whether baseline levels of steroid hormones have a differential effect on decision-making to fair versus unfair offers in the Ultimatum Game, and whether these differences in offers likewise cause changes in steroid hormone levels. The results show that female testosterone decreased in response to fair offers, female estradiol increased in response to unfair offers, and that distinct hormone level profiles predicted acceptance rates to unfair offers. Overall, these studies demonstrate that, in a sex-specific manner, social context affects how gonadal steroids differentially influence decision-making under risk and ambiguity. While testosterone is typically associated with risk-seeking in males, here female estradiol is more associated with risk-aversion.

Keywords: Hormones; Sex steroids; Decision making; Social cognition; Game theory

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Table of Contents

List of Tables

List of Figures

List of Acronyms

ACTH	Adrenocorticotropic releasing hormone
ANOVA	Analysis of variance
AR	Androgen receptor
ACC	Anterior cingulate cortex
ANS	Autonomic nervous system
CNS	Central nervous system
CRH	Corticotropin-releasing hormone
DMN	Default mode network
dlPFC	Dorsolateral prefrontal cortex
ENS	Enteric nervous system
ELISA	Enzyme-linked immunosorbent assay
ERα	Estrogen receptor alpha
ERβ	Estrogen receptor beta
EPSP	Excitatory postsynaptic potential
FSH	Follicle-stimulating hormone
FP	Frontopolar cortex
GPCR	G protein coupled receptor
GABA	Gamma amino butyric acid
GR	Glucocorticoid receptor
GnRH	Gonadotropin-releasing hormone
HUG	Hierarchical ultimatum game
HPA	Hypothalamic-pituitary-adrenal
HPG	Hypothalamic-pituitary-gonadal
IPSP	Inhibitory postsynaptic potential
LH	Luteinizing hormone
mER	Membrane estrogen receptor
OFC	Orbitofrontal cortex
PNS	Peripheral nervous system
PCC	Posterior cingulate cortex
PD	Prisoner's dilemma
REB	Research ethics board
RPS	Research participation system
SFU	Simon Fraser University
SNS	Somatic nervous system
UG	Ultimatum game
vmPFC	Ventromedial prefrontal cortex

Glossary

Ambiguity	Decisions in which the probabilities of outcomes are unknown
Ambiguity premium	The utility of the perceived difference between risk and ambiguity
Androgen	Class of sex steroids produced primarily, but not exclusively, in testes
Cortisol	The most abundant glucocorticoid produced in humans
Estradiol	The most abundant estrogen produced in primarily in ovaries
Estrogen	Class of sex steroids produced primarily, but not exclusively, in ovaries
Glucocorticoid	Class of steroid hormones produced primarily, but not exclusively, in the adrenal glands
Hormone	Chemical messenger synthesized in and released from glands to distant target cells via the circulatory system
Risk	A factor related to the perceived probability of various outcomes of a decision
Sex steroid	Steroid hormone synthesized in and released by the gonads
Steroid	Small molecule hormone ultimately derived physiologically from cholesterol
Steroid receptor	A protein embedded in the membrane or within a steroid hormone target cell, which causes biochemical changes to the target cell when bound with a matching steroid hormone
Testosterone	The most abundant androgen produced in primarily in testes
Ultimatum game	A two-player game in which a proposer makes an offer to a responder from an endowment of finite value, which can be either accepted or rejected based on the perceived fairness of the offer
Utility	The subjective value of decision outcomes

Chapter 1.

General Introduction

Three maxims were inscribed at the entrance to the ancient temple of Apollo at Delphi. The first, γνῶθι σεαυτόν (“know thyself”), is traditionally interpreted as a call for self-understanding. Since we are instantiations and extensions of the natural world, all modern science can be framed as a quest for self-understanding. However, the biological and social sciences, particularly their intersection in behavioural neuroscience, are the most targeted towards uncovering truths about the human condition. Discoveries in these domains often extend far beyond and run contrary to our culturally endowed intuitions about ourselves, and this forms the basis for the immense value that science has in human life. In this tradition, the following dissertation is an attempt to synthesize and extend some interesting insights from the biological (neuroendocrine) and psychological (social and economic decision making) disciplines.

The two major themes of the research described in this dissertation are: i) the influence of hormonal states, specifically the sex steroids testosterone and estradiol, on risky economic decisions with and without social provocation; and ii) whether that provocation comes from an actual person or pure “random” chance (i.e., a non-social or deterministic algorithm). In a world where we increasingly interact with simulated or artificial cognitive agents, the degree to which our evolved toolkit for social cognition can be circumvented to modify our decision thresholds and economic risk tolerance is a fundamental and pragmatic question.

To explore these issues, I draw from several components of biology, including evolution, physiology, and biochemistry, as well as from social sciences such as cognitive psychology, social psychology, and behavioural economics. The specific confluence of these subdisciplines explored here is expressed in the dynamics of the endocrine system and social cognition. This chapter will present a general and relatively brief overview of these relevant subdisciplines, and it will be followed by a synthesis of their specific insights that helped to frame the set of experiments detailed in subsequent chapters.

1.1. Biological Background

The problems of survival and reproduction are challenging enough for single-cell organisms to navigate. These problems become considerably more difficult for multicellular lifeforms due to several additional requirements. These requirements include the need for cells to subordinate their immediate individual interests to the collective needs of the larger organism when those interests conflict, the need for specialized and differentiated cells to adapt to new environmental contexts that emerge from their own methods of coordination, and the need for efficient communication and coordination across multiple spatiotemporal scales.

The first of these requirements is ubiquitous at all social scales throughout the major evolutionary transitions (Bourke, 2011) and will be discussed in more detail with respect to human social coordination in the general discussion. The details of the second requirement fall within the domain of complexity theory (Strogatz, 2018), and while ultimately relevant to human behaviour, it is beyond the scope of this dissertation. The third requirement, that of efficient intercellular communication over relatively long distances and intervals, has been evolutionarily solved in several ways.

There are three macroscopic communication systems in the human body (Breedlove & Watson, 2023). The fastest and most complex is the nervous system, which conveys signals throughout the body through electrochemical channels. The endocrine system, a less complex network of glands distributed throughout the body, conveys slower chemical signals over longer intervals via the cardiovascular circulatory system. The immune system, another extremely complex communication system, is partly comprised of a network of lymph nodes and lymphatic vessels, as well as other integrated organs for the purpose of defense against infectious agents. The current research is concerned only with the first two systems, emphasizing the neural network architectures that underlie social cognition, and context-specific hormonal modulation of these functional networks.

1.1.1. The Nervous System

The human nervous system is conventionally divided into two primary divisions: the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system (PNS), which includes the somatic nervous system (SNS), the

autonomic nervous system (ANS), and enteric nervous system (ENS). However, when considering the evolutionary origins of the human nervous system, the order in which these systems are presented must be reversed. Such considerations are useful for contextualizing the relationship between physiology and psychosocial dynamics in modern humans.

1.1.1.1. Nervous System Evolution

The nervous system is a characteristic feature of animals. The simplest evolutionary explanation for its emergence is that it offers multicellular organisms a means by which they can: i) process and digest food (the ENS); ii) regulate the internal physiology of the organism (ANS); iii) sense and respond to the environment (SNS); and iv) use high-level perceptual information to actively pursue food sources and other fitness opportunities while avoiding existential threats (CNS). While the function of the ENS may have originally been distinct from the others, for most animal clades these processes have become well integrated, often to the point that they operate as a single system. However, each functional component can also operate independently when their relatively minimal connections are severed, and some recognized psychopathologies (e.g., autism spectrum disorder) are even hypothesized to result from developmental abnormalities in their integration (Porges, 2011).

Neurons and glial cells are the primary cell types of the nervous system and are derived over the course of development from an embryonic structure called the ectoderm. Because other cell lines, such as those in the epidermis, are also derived from pluripotent stem cells found in the ectoderm, it has been suggested that neural tissue was ultimately derived from the deep evolutionary progenitor of both these structures (Arendt et al., 2016). The phylogenetic details of their emergence are not definitively known, but regardless of the specific mechanisms by which the nervous tissues evolved into elaborate structures, their functions are of primary concern for a science of mind. A few of these elaborations will now be discussed to provide some broad context for understanding the systems that are probed in the research described in Chapters 2-4.

1.1.1.2. Nervous System Physiology (Basic Principles of Modularity and Integration)

Neurons and glial cells have unique gene expression profiles, changing throughout their development from pluripotent stem cells to mature, specialized cells, directed by mechanisms such as epigenetic influences, intercellular chemical gradients, and intracellular genetic positive feedback loops (Evans & Kaufman, 1981; Takahashi & Yamanaka, 2006; Kim et al., 2010; Alberts et al., 2018).

Previously deemed as mere "support" cells for neurons, glial cells are now recognized for their vital computational role. Different types of glial cells contribute to the vast computational capacity of neural networks. Microglia perform immunological functions, oligodendrocytes speed up information transfer by sheathing neuronal axons with myelin, and astrocytes serve multiple roles, including neurotransmitter regulation and maintaining the blood-brain-barrier (Pfreiger & Barres, 1997; Auld & Robitaille, 2003; Pfreiger, 2010; Bradl & Lassmann, 2010; Sofroniew & Vinters, 2010; Kettenmann et al., 2011).

Neurons, the most vital cell type in the nervous system, have over a hundred subtypes distinguishable by their genetic expression, structure, or function. Their structural variation may contribute to different subjective states, and they create the hierarchical circuits that underpin virtually all perception, cognition, and behavior. The full extent of this complexity is beyond this dissertation's scope, but it helps illustrate how the mind arises from diverse, multiscale integrated processes (Kandel et al., 2021; Butti et al., 2013).

1.1.1.2.1. Microstructure: Neural Communication

Neurons employ several distinct and complex mechanisms to transmit information, but which can be captured by a simple two-factor model. The factors can be described as receiver identity (self vs. other, or equivalently intracellular vs. extracellular) and medium of signal transmission (electrical vs. chemical). The table below outlines some important examples in each quadrant of this model.

Table 1.1: Two-factor model of neural communication

	Electrical	**Chemical**
Intracellular	Post-synaptic & action potentials	Intracellular signaling cascades
Extracellular	Gap junctions	Neurotransmission

Beginning in the lower right quadrant, the characteristic form of neural communication is neurotransmission where one neuron serving as a signal transmitter, the presynaptic neuron, is osmotically induced to release chemical messengers called neurotransmitters across a synaptic cleft. The synaptic cleft is a small space between the presynaptic neuron and the signal receiver, the postsynaptic neuron. The postsynaptic neuron is endowed with a subset of protein receptor subtypes from a larger pool of potential receptors that may bind with neurotransmitters released by the presynaptic neuron. Once bound, the postsynaptic receptors are activated in a variety of ways depending on their structure.

Some postsynaptic receptors are ion channels whose activation is a conformational change that modifies the selective flow of electrically charged ions to locally depolarize or hyperpolarize the postsynaptic neuron. These local changes in electrical charge are called excitatory- or inhibitory-postsynaptic potentials respectively (EPSPs & IPSPs). They are graded signals that propagate away from the signal source along the postsynaptic cell's membrane via voltage-gated ion channels embedded throughout the cell's surface. They can be intuitively thought of as propagating electrical waves that can undergo destructive and constructive interference with other EPSPs and IPSPs (i.e., spatial and temporal summation). The resting state membrane potential of a typical neuron is approximately -65mV, and when constructive interference between multiple EPSPs is sufficient to bring the membrane potential of the postsynaptic neuron to approximately -40mV at the axon hillock (a structure especially rich in voltage-gated sodium channels between the soma and axon), those integrated signals trigger an action potential. Action potentials are electrical signals comparable to EPSPs, but rather than being graded they are all-or-none and typically move unidirectionally down the length of an axon to ultimately trigger the release of neurotransmitters into another synapse.

Other postsynaptic receptors are metabotropic whose members mostly consist of G protein-coupled receptors (GPCRs). When a particular GPCR subtype is activated by binding to a neurotransmitter with which it has a high affinity, the conformational change in its structure results in the activation (phosphorylation) of an intracellular G protein, and this activation begins one of several possible intracellular signaling cascades depending on the specific GPCR subtype and version of the G protein activated. The intracellular chemical signaling pathway is therefore much more divergent and its consequences are more variable than the simple opening of ion channels and propagation of EPSPs or IPSPs, but some of those consequences can include the opening or closing or many postsynaptic ion channels, the promotion or inhibition of neurotransmitter synthesis, and changes to gene expression. Such changes in gene expression can contribute to the up- or down-regulation of various receptor subtypes, to changes in cell structure, or several other mechanisms for modulating the postsynaptic neuron's signal sensitivity and output dynamics (Alberts et al., 2018). Many of these metabotropic signals serve to change the rate or pattern at which the postsynaptic neuron will fire action potentials.

Neurotransmitters play a crucial role in neural communication, and they come in various forms. Classical small molecule transmitters are derived from proteogenic amino acids, such as glutamate, GABA, and monoamines. Peptides, such as endogenous opioids, and other biochemical sources, like acetylcholine and endocannabinoids, also serve as neurotransmitters (Kandel et al., 2021). In some instances, neurons communicate with one another not through neurotransmission across a synapse, but electrically by directly passing charged ions through protein channels embedded in the membranes of both neurons that are physically aligned with each other. These structures are called gap junctions, and they complete the two-factor model of neural communication. All these forms of communication can aggregate and ultimately integrate into the multiscale set of signaling networks that constitute the brain. This intricate network of interactions allows for the diverse and complex functions that the nervous system performs, from sensation and perception to cognition and motor control.

1.1.1.2.2. Mesostructure: Neural Pathways and Circuits

Collections of neurons give rise to more complex structures such as subcortical nuclei and various cortical structures that span from minicolumns to hypercolumns to cortical subareas, cortical areas, and finally functionally integrated cortical networks

(DeFilippe, 2012; Mountcastle, 1998). There are no intrinsic delineations between the micro-, meso-, and macrostructures of the brain, but subcortical nuclei and cortical structures up to functional areas can be reasonably described as occupying a “mesoscale” of organization. Some characteristic neural structures in the mesoscopic range are neural pathways and neural circuits, which ultimately aggregate at the macroscopic scale into functional neural networks. Neural pathways are linear sequences or chains of information transmission over two or more neurons, while a neural circuit can be thought of as a pathway with some form of feedback from a neuron further along in the pathway back to an earlier one.

These structural classifications offer ample opportunity for the development of a taxonomy for neural architecture and computation. However, while the term “mesostructure” can be appropriately applied to any such structure that occurs between the level of individual cells and the whole nervous system, it will be limited in this context to describing structures smaller than neural systems that serve as the basis for modular cognitive, affective, and behavioural domains. Such systems are relevant objects of psychological study, and if by analogy one of these systems can be likened to a narrative, then its neural mesostructures would constitute the phonemes, words, and sentences.

For example, social interaction is guided by brain networks for social perception, aversion, and affiliation that are centered on the amygdala as an important hub node, with distinct nuclei subserving each respective component of social behaviour (Bickart et al., 2014). Noncentral nodes for each (sub)system (and their corresponding behavioural expression) may consist of neural mesostructures which may also be variously employed by other neural systems, but their functions between systems may be distinct or even contradictory (e.g., a bite that employs the trigeminal nerve can be used in an act of aggression, courtship, or hunger). A unifying feature of neural mesostructures is that they are in some sense modular but psychologically meaningless in the absence of context provided by the larger neural system in which they are functionally embedded. Importantly, the distribution of neurotransmitter and hormone receptor subtypes on neurons within system nodes may offer (occasionally dubious) hints about the role of their signaling molecules on neural systems, in proportion to node centrality.

1.1.1.2.3. Macrostructure: Functional Network Systems

The nervous system is organized both hierarchically and in terms of broadly distributed functional systems (Kandel et al., 2019). Sensory neurons at the bottom of the hierarchy transduce environmental energy forms into electrochemical information, transmitted to higher regions for conversion into adaptive motor responses. Notably, the prefrontal and cingulate cortices, comprising the default mode network (DMN), might be considered to represent the highest association cortices (Raichle et al., 2001; Raichle, 2015), relaying top-down model expectations to lower areas.

Functional network systems are characterized by temporally correlated activity between various brain neuron subpopulations (Sporns & Kotter, 2004; Bullmore & Sporns, 2009). Examples include the DMN, the amygdala-centric social cognition networks (Bickart et al., 2014), and attentional networks (Szczepanski et al., 2013; Mars et al., 2012; Krall et al., 2015). These networks exhibit a scale-free structure, where local structures mirror global ones.

A high-level functional system example is the integration of cognitive neural networks and the autonomic nervous system's dynamics, as per the somatic marker hypothesis (Bechara & Damasio, 2005; Poppa & Bechara, 2018). This theory links decision-making and affect with physiological processes like respiration and heart rate. When considering such integrative theories, it's crucial to acknowledge the endocrine system's role, which collaborates with the nervous system's central and peripheral components to regulate behavior. The next section will delve into the endocrine system's structure and function for a more comprehensive understanding of its interplay with the nervous system.

1.1.2. The Endocrine System

Along with the nervous system described above, and the immune system which has no direct bearing on the studies described below, the other macroscopic communication system in the body is the endocrine system. Endocrine communication works similarly to neurotransmission, but on somewhat different spatiotemporal scales. The characteristic time scale of hormone information transmission can be on the order of seconds, minutes, or years. This is several orders of magnitude greater than the time scale of neurotransmission. The difference between neurotransmitters and hormones may therefore crudely be likened to ocean waves and tides.

Rather than release its chemical signaling molecules into a small synaptic cleft for fast and highly localized between-cell communication as neurons do, endocrine cells release them into the cardiovascular circulatory system where they can be transported to virtually any target destination in the body. Before explaining neuroendocrine integration and its relevance for social cognition, some important physiological aspects and the broad evolutionary strokes of the endocrine system will be outlined.

1.1.2.1. Endocrine Physiology

The endocrine system's glands are diverse and spread throughout the body, with the pituitary gland, ventral to the hypothalamus, coordinating their activity (Nelson, 2023). The interaction between various hypothalamic nuclei and the pituitary gland constitutes the central interface between the nervous and endocrine systems. The hypothalamus secretes a variety of releasing factors (hormones) that trigger the anterior pituitary to release tropic hormones, targeting receptors on glands across the body. Key pathways from the hypothalamus to pituitary and glands are known as axes, including the hypothalamic-pituitary-adrenal (HPA) and hypothalamic-pituitary-gonadal (HPG) axes. Furthermore, the paraventricular and supraoptic hypothalamic nuclei release oxytocin and vasopressin into the posterior pituitary for body-wide distribution.

Hormone release coordination involves various complex feedback loops within and between axes. Hormones display inherent rhythmic variation aligning physiology with environmental rhythms, with much coordination managed by temporal regulation anticipating cyclic behavioral demands. Most hormone signaling systems exhibit emergent rhythms of various durations, providing regularity for ecologically relevant physiological and behavioral adaptation. Conventionally, biological rhythms are categorized as ultradian (less than a day), circadian (around a day), or infradian (greater than a day). In humans, the endocrine system's ultradian rhythms of approximately 90-120 minutes and 3-8 hours overlay a more dominant circadian rhythm (Reppert & Weaver, 2001; Buzsaki, 2006). The circadian rhythm, controlled by the hypothalamic suprachiasmatic nucleus or "master clock" (Mohawk & Takahashi, 2011), can be generated by virtually every cell. Steroid hormones have a diurnal peak in the early morning and a gradual decline throughout the day (Tenover et al., 1988; Bao et al., 2003). The most notable human infradian hormonal rhythm is the menstrual cycle, though some evidence suggests subtle annual seasonal

changes in various hormones, including testosterone (Smals et al., 1976; Dabbs, 1990; however, see Smith et al., 2013).

The HPA and HPG axes are critical for modulating the effects of sex steroids on social cognition and behavior. The HPA axis, involved in stress responses and energy regulation, starts with parvocellular neurons in the hypothalamus's paraventricular nucleus releasing corticotropin-releasing hormone (CRH). CRH stimulates the anterior pituitary to produce adrenocorticotropic hormone (ACTH), which prompts the adrenal cortex to release glucocorticoids like cortisol (Khani & Tayek, 2001). The primary focus here will be on cortisol's role in modulating sex steroids' effects on social cognition and behavior.

The HPG axis, responsible for producing sex steroids such as progesterone, testosterone, and estradiol, begins in the anterior hypothalamus's medial preoptic nucleus, where gonadotropin-releasing hormone (GnRH) is synthesized. GnRH triggers the anterior pituitary to release luteinizing hormone (LH) and follicle-stimulating hormone (FSH), regulating sex steroids production in the gonads.

Testosterone, an androgen significant for both sexes' physiology and behavior, is mainly synthesized in males' testes' Leydig cells and in females' adrenal cortex and ovaries. Testosterone actions are mediated via the androgen receptor (AR), and genetic differences in the AR gene can subtly affect the ligand-receptor complex's function (Ackerman et al., 2012). Each androgen has at least slightly different binding affinity for the AR (Gloyna & Wilson, 1969; Grover, 1975; Norman & Litwack, 1997), and generally, androgens are crucial in sex determination, differentiation, and secondary sexual characteristics development.

Estradiol, an estrogen, can be synthesized from testosterone or through other enzymatic pathways. Estrogens have a complex cellular signaling pathway system, binding to intracellular receptors (ERα and ERβ) and membrane estrogen receptors (mERs), some resembling the GPCRs used by many neurotransmitters (Levin, 1999; 2002; 2009). Estradiol, primarily synthesized in females' ovaries' granulosa cells, plays a role in secondary sexual characteristics development. The influence of sex steroids on social cognition and behavior is mediated through their direct effects on the nervous system, including organizational and activational effects, which I will discuss next.

1.1.2.2. Homeostasis, Sex, Social Status: Some Evolutionary Aspects of Glucocorticoids, Androgens, and Estrogens Relating to Social Cognition

Claude Bernard, a 19th-century French physiologist, introduced the concept of homeostasis (although the term homeostasis is attributed to Walter Cannon [Cannon, W., 1929]):

> "[I]t is the fixity of the milieu intérieur which is the condition of free and independent life… and all the vital mechanisms, however varied they may be, have only one object, that of preserving constant the conditions of life in the internal environment."
>
> Bernard, C., 1878, p. 113.

Homeostatic regulation of bodily processes is crucial for adaptive survival and reproduction. Physiological and behavioural homeostasis involve numerous mechanisms for adjusting fluid, pH, energy balance, temperature, and ion concentrations. It may also be meaningful to extend the concept to social network regulation (a kind of milieu extérieur), as reliable social support strongly influences stable homeostasis for physiological variables. Conversely, antagonistic social interactions or the absence of affiliative others can jeopardize stability. Although there is substantial evidence linking nonapeptide hormones like oxytocin and vasopressin to social affiliation and aggression in humans and nonhuman animals (Goodson & Thompson, 2010; Baran, 2017), this section will focus on the roles sex steroids play in social cognition and decision making.

Hormones can have two broad types of effects on bodily tissues: organizational and activational (Arnold, 2009; Schulz et al., 2009). Both types of effects are mediated by the same receptors on hormone target cells, but organizational effects cause lasting structural and functional changes in the target tissue, while activational effects are more transient without causing permanent changes. Organizational effects predominantly occur during sensitive developmental windows, such as perinatally or during adolescence, while activational effects can occur at any developmental stage, influencing physiology and behaviour in a time-restricted manner. The sex steroids, testosterone, and estradiol exert both types of effects in a sexually dimorphic manner.

Intrasexual competition is a strong selection pressure for individuals of both sexes. Direct effects of intrasexual competition for mates on physiology and behaviour promote

a universal feature of competition: risk-taking (Baker Jr & Maner, 2008; 2009; Fisher, 2013). Behaviours with typically high fitness costs may be worthwhile if the fitness benefits of intrasexual competition victory are also high. This logic can be observed in animals with seasonal mating strategies (Crowley et al., 1991) and humans in contexts where opportunities for fitness-promoting social status gains serve as direct or indirect proxies for mating opportunities in intrasexual competition.

The sex steroids testosterone and estradiol contribute to social status organizationally, through sexual differentiation of the body, and activationally by motivating behaviours associated with social status (Anderson et al., 2001). The activational effects of sex steroids on promoting social status and social status-seeking behaviour are well-documented in nonhuman animals (De Almeida et al., 2015; Hamilton et al., 2015; Sapolsky, 2004) and humans (Sapolsky, 2004; Goyman & Wingfield, 2004; Knight & Mehta, 2014). The most relevant social status-seeking behaviour for the present research is risk-taking (Ashton et al., 2010; Mehta et al., 2015). Cognitive and social appraisal of risk and decision making will be described in section 1.2, and its bidirectional relationship with sex steroids will be integrated in section 1.3.

1.1.3. Intermediate Summary and Intersection: Neuroendocrinology

As we've seen, the nervous system is a characteristic feature of all animals, serving as the functional basis for the input of environmental energy (sensation), synthesis and computational transformation of sensory stimuli (cognition), and output primarily in the form of motor plan executions (behaviour). It is organized hierarchically in terms of pathways and circuits (bottom-up and top-down flow of information) and scale (from neurons to nuclei to networks). Coordination between the ANS, SNS, and CNS underlies our ability to experience and express emotion, and to perceive emotion in others for rapid social adaptation. Social emotions (e.g., pride/shame, anger, gratitude, envy, etc.) are particularly useful to deploy appropriately or strategically, and to accurately perceive in others, which are important aspects of social cognition.

A central hub for social cognition in the brain is the amygdala, whose subnuclei form the core of distinct functional brain networks related to different facets such as social perception, affiliation, and aversion/aggression. The amygdala helps coordinate appropriate social behaviour by integrating memory and cognitive appraisals of social

schemas (discussed below) with real-time social perception to organize those parts of the nervous and endocrine systems involved in autonomic and somatic output. In the brain, this includes volitional and non-volitional motor plan execution via motor cortex and cranial nerve nuclei, including the vagus nerve as the source of most parasympathetic fibers of the ANS, as well as non-volitional recruitment of the endocrine system via various nuclei of the hypothalamus and their signaling of the pituitary gland.

As described above, the endocrine system is structurally a constellation of glands and functionally one of the three macroscopic communication systems in the body, signaling other cells like neurons via hormones, which are chemical messengers transmitted through the cardiovascular system. Hormones operate on longer time scales than the nervous system, and influence behaviour by priming particular neural networks, especially through receptors in brain areas associated with social affect and cognition (e.g., the amygdala, hypothalamus, periaqueductal gray, and associated cortical areas). While the presence of specific hormones rarely determines a particular behaviour, they do stochastically increase the likelihood of certain behaviours depending on the hormone and ecological context. A ubiquitous type of behaviour is decision making under risk, as it appears in both social and non-social contexts. This behaviour and its cognitive bases will be discussed in the next section.

1.2. Psychological Insights

The dynamics of the central nervous system, in concert with environmental input and functionally integrated systems like the endocrine system, are the physiological reflections of an even more interesting phenomenon: the mind. This relationship is reciprocal, as the mind is also a reflection of these physiological processes. In a curious sense, originally expounded by Descartes by the aphorism *cogito ergo sum*, the mind is the only thing that undoubtedly exists. Even the set of assumptions underlying philosophical materialism are necessarily a subjective mental experience. Still, science operates under the premises of materialism, and there is enough consistent structure of the mind to allow coherent discussion. The human mind in particular displays capacities that are especially interesting, at least to itself.

Many of our mental capacities are shared with other animals because of common lineage or convergent evolution, including many aspects of general and social cognition.

However, some novel and perhaps unique mental traits have emerged solely in our species, such as recursive grammar (i.e., language), mythos and narrative, music, mathematics, mental time travel, and others, making us what Terrance Deacon calls "a symbolic species" (Deacon, 1998; Deacon, 2012). These unique capacities may derive from a smaller set or even a single fundamental cerebral novelty, but regardless of their origins, it is fascinating to consider ways in which these traits must have incorporated themselves into already existing mental/neural circuits. A small set of restricted questions pertaining to these issues will be explored in the following chapters, but first it will be necessary to explore what is already known about the relevant aspects of human cognition. Since a unifying theme of my studies is social cooperation and competition under risk, this section will expound on elements of cognitive psychology, social psychology, and behavioural economics.

1.2.1. Cognitive Modularity for Decision-Making Under Risk

Cognitive psychology is the branch most concerned with the computational architectures and mechanisms of the mind. Prior to its emergence, the dominant paradigm in psychology was behaviourism. Behaviourism largely ignored the inner workings of the mind and brain, focusing on input-output relationships only, and proposed that the information transformations between sensory transducers and neuromuscular junctions were either of little significance for explaining behaviour or could not reasonably be established empirically and objectively.

If any watershed moment exists to demarcate the transition between paradigms, it might be the publication of Noam Chomsky's "A Review of B.F. Skinner's Verbal Behavior" (Chomsky, 1959), in which he demonstrates that behaviourist learning principles are insufficient to account for the complexity of language and the rapidity with which it is learned exclusively by human children. He also posits the existence of a "deep grammar", a form of cognitive innatism for language acquisition and production. Since then, cognitive science has attempted to model language and many other mental faculties such as memory, attention, perception, and thinking (Boden, 2008) by attempting to peer into the black box previously ignored by behaviourists. Each of these faculties or *cognitive domains* is interrelated, and they are composed of often overlapping or networked submodules. It is a goal of cognitive psychology to model the processes of these mental modules accurately and parsimoniously.

For the purposes of my studies on the aspects of decision-making under social and non-social risk, there are several aspects of general and social cognition that are relevant and need to be elaborated. Three basic processes will be especially relevant: stimulus valuation, risk propensity or tolerance, and decision-making. Although there is considerable overlap between these processes, they are presented in a logically connected sequence since the kinds of decisions that participants make in the studies described in subsequent chapters rely on risk perception, proneness, or aversion, which in turn relies on the appropriate relative valuation of incentives and aspects of social relationships. How these cognitive processes are contextualized in the framework of social psychology and, for methodological and experimental design purposes, behavioural economics, will be the subject of the rest of this section.

1.2.1.1. Stimulus Valuation

From a broad evolutionary perspective, stimulus valuation can be thought of as the foundation upon which an organism relates to perceived environmental objects. These mental objects or stimuli can be exteroceptive or interoceptive, but the important consideration is ultimately the degree to which the organism can accurately identify it (i.e., categorization), and contextually assign to it a metric that will serve as an estimate for how various forms of interaction with the object will cash out as increases or decreases in fitness. Conscious awareness of stimulus valuation estimates is not necessary for their existence or application, but neither is it necessarily excluded. In humans, the anterior cingulate cortex (ACC) in the medial surface of the forebrain has been specifically identified as a brain area embedded within a larger neural network that is crucial for the "higher" level cognitive component of stimulus valuation (Camille et al., 2011; Kaping et al., 2011; Kolling et al., 2016; Shenhav et al., 2016). Another important cortical area in this network is the ventromedial prefrontal cortex (vmPFC) (Glascher et al., 2009; McNamee et al., 2013; Rudorf & Hare, 2014).

Stimulus valuation serves as a crucial component of memory that keeps track of circumstances and behaviours that lead to reinforcing or punishing outcomes for the organism. It also serves as a means by which various types of stimuli might be contextually compared and heuristically matched with ways in which the organism might interact with an object (Mahon, B.Z., & Caramazza, A., 2010). Additionally, it allows cost-to-benefit ratios to be calibrated so that higher-order estimates of risk might be generated (Croxson

et al., 2009; Rangel & Clithero, 2014; Clithero & Rangel, 2014; Klein-Flügge et al., 2016; Chong et al., 2017), and the likelihood of making optimal decisions about how to relate to the object. While stimulus valuation is an important factor in decision-making, there are several other factors that contribute to the timing and direction of our decisions.

1.2.1.2. Effort, Cost, and Risk

Every decision in the absence of perfect knowledge about future outcomes involves some element of risk. Risk implies the prospect of potentially losing something of subjective value, including simple opportunity costs. Even in the most controlled environments, there is a plethora of unknowns that introduce enough chaos and unpredictability for risk to exist. It must be asked, what do cognitive agents value? From an evolutionary perspective, there is certainly a primacy in the value of continued existence, including survival and reproduction. These are things of ultimate value (Laland et al., 2011) but there is also a vast array of things that have proximate value. To complicate matters, individuals have different subjective weights for things of proximate value, either because of the demands of our individual lives or because of biases in our valuation of fitness opportunities and threats. These subjective weights are asymmetric, dynamic, and can differ considerably between individuals.

Appraisals of risk include an emotional component (Slovic & Peters, 2006; Tompkins et al., 2018) that captures the intuitive and visceral sense of danger or excitement that comes from perceived prospective loss or gain, respectively. They also include a cognitive component that can involve stimulus valuation, fast heuristics (Pachur et al., 2012), numeracy (Peters et al., 2012), and/or the perceived cost of effort for behaviours associated with risky decisions (Bailey et al., 2016). Again, individuals differ in their tolerance and appraisals of risk, which are asymmetrically updated based on the consequences of our decisions. Therefore, trait and state risk tolerance or propensities inform decision-making, and the outcome of our decisions informs our inclination to domain-specific risk-taking.

1.2.1.3. Decision-Making

Etymologically the term decision is derived from the Latin *de-* (“off”) and *caedere* (“to cut). Decision-making is the process of cutting off prospective paths forward from a

range of options. Even vacillating about which of many options to choose is itself almost always one of the available options one has, at least for humans endowed with rudimentary metacognition (e.g., thinking about thinking), and can therefore be considered a kind of decision. Decision-making has been one of the most fruitfully researched aspects of cognitive psychology, with a rich history of often complementary models (Gigerenzer & Gaissmaier, 2011; Gigerenzer & Gaissmaier, 2015; Rilling & Sanfrey, 2011; Slovic et al., 2005). The overarching paradigm in this area is simply called decision theory (Peterson, 2017), which breaks the field into 'normative' decision making, which is virtually equivalent to optimization problems, and 'descriptive' decision making, or the analysis of how cognitive agents actually decide. The latter is of primary concern here.

Some key facets of decision theory involve the analysis of how decisions are made under uncertainty (ignorance, risk, ambiguity, etc.), how outcomes and the information relevant to arriving at one are dynamically weighted (e.g., intertemporal choice), and how our social world influences all the above. This third facet is part of the motivation for the integration of social psychology and game theory from behavioural economics, which will be discussed in the next subsection. However, we must have a sense of the more general facets of decision making before delving into these elaborations.

In learning theory, there is a fundamental behavioural trade-off known as the explore-exploit dilemma (Wilson et al., 2014) in which a choice to search for new and potentially greater-than-baseline payoff opportunities necessitates a cost paid in the reduced exploitation of a resource with known value, and vice versa. This dilemma can be found in disciplines that span cognitive science, as well as at multiple scales of social organization (e.g., explore-oriented progressive versus exploit-oriented conservative political orientations). A prototypical technique for researching the explore-exploit dilemma in psychology is known as the multi- or n-armed bandit paradigm (Racey et al., 2011) in which a participant is confronted with multiple “bandits” (e.g., slot machines) with unique payoff distributions. The participant must decide on how to allocate their limited time in either exploiting a “good enough” bandit or exploring the payoff distributions of other bandits. There is risk and opportunity in each option. There are unique and learnable solution sets to the n-armed bandit problem when the number of bandits is finite, and their payoff distributions are stable. However, if neither of those conditions are met, as is the case for most important features of complex cognition, then there is no necessarily optimal algorithmic approach to the n-armed bandit paradigm or the explore-exploit dilemma.

In addition to understanding decision making under uncertainty, such as the explore-exploit dilemma, it is crucial to examine the impact of time on valuing particular outcomes. This idea can be observed in the well-known Stanford marshmallow experiments, where children were given a choice between an immediate reward and a delayed, larger reward (Mischel et al., 1989). Studies on adult participants by Daniel Kahneman and Amos Tversky further explored value preferences over time, leading to the development of prospect theory (Kahneman & Tversky, 2013; Tversky & Kahneman, 1981; Tversky & Kahneman, 1992). While there is considerable individual and group variability on the expression of reinforcement time preferences (Gurven, 2018), this theory highlights humans' general loss aversion and the asymmetric temporal discounting of value for gains and losses, which can be modeled using a hyperbolic discounting curve for future prospects.

While there is a general consistency in how humans approach problems like the explore-exploit dilemma, gains versus losses, and temporal discounting of future rewards, it is important to recognize the significant degree of subjectivity involved in valuing different types of stimuli. This means that even though proximal rewards ultimately relate to evolutionary fitness, individuals can assign different weights to these rewards and their relative importance. Moreover, these weights can change over a person's lifespan. Further exploration of these aspects will be carried out in section 1.2.3. Before delving into that, it is essential to examine the influence of social factors on decision making, as human decisions are often made in the context of our relationships with others.

1.2.2. Social Cognition: A Distinct Mental Module for Decision-Making

One effective way to functionally differentiate human cognition at a high level is to distinguish between physical and social cognition, or mechanistic and mentalizing processes (Crespi & Badcock, 2008). Although there is considerable overlap and shared submodules between these two cognitive modes, social species face unique and complex challenges that do not directly stem from mastering the physical world. For humans, it can be especially computationally demanding to track dynamic social networks, shifting social ranks and roles, and the motivations and intentions of others in scenarios that intertwine deception with recursive theory of mind.

In many ways, social cognition can be viewed as an application of general cognition (e.g., attention, memory, categorical schemas, action planning, decision-making, etc.) to the social world. However, there is ample evidence suggesting that social cognition utilizes computational capacities uniquely tailored for agency inference and adaptation (Leslie et al., 2004; Frith & Frith, 2005). Examples include joint attention and triadic relations (Mundy & Newell, 2007; Moore et al., 2014), theory of mind (Wellman, 2018), observational learning and cultural transmission (Reed et al., 2010; Henrich, 2016), empathy and moral reasoning (Cuff et al., 2016; Paxton & Greene, 2010), and a set of processes often referred to as "social skills" or "social intelligence" (Little et al., 2017). For the studies discussed below, the two most relevant and related components of this social cognitive toolkit are representational theory of mind and intention inference, or mentalizing (Freeman, 2016).

Mentalizing occurs when we infer the mental state of another person (Frith & Frith, 2006). One aspect of mentalizing that may be uniquely human is recursion, in which inferring another's mental state can include their inference of our own mental state. In principle, this process of iterative mental modeling could continue indefinitely if not for constraints such as time, energy, and motivation. However, in practice, most mentalizing concludes after a few or often a single iteration and is useful if it informs us about the emotional and intentional states of others. The latter has been referred to as "the intentional stance" by philosopher Daniel Dennett (Dennett, 1989) and forms the basis for several research questions explored in this dissertation. Specifically, does a socially provocative game player influence one's decisions under risk and ambiguity equally when perceived as an agential algorithm versus a person with intentionality? One important consideration for this question is the role that social emotions play in decision-making.

1.2.2.1. Evolution of Emotions for Socialization

Emotions are a pervasive feature of the human psyche, with every subjective experience being influenced by a combination of autonomic arousal and various valences. Despite being a longstanding area of psychological research (James, 1890) and significant progress made in understanding their universality (Ekman, 1992; 1999) or lack thereof (Barrett, 2006; Gross & Barrett, 2011), emotions remain challenging to model effectively. However, comparative neurophysiology, endocrinology, and ethology can

provide valuable insights into the taxonomy of emotions, especially in relation to social emotions.

Understanding emotions necessitates recognizing that valence, or the positive or negative nature of an emotion, is an extension of the basic approach-avoid behavioural drives. Valence reflects the fundamental motivation of emotional responses, while specific emotional characteristics guide the form of these responses. Primary emotions, if they exist (Panksepp & Biven, 2012; Feldman-Barrett, 2017), have a relatively straightforward basis in psychophysiological homeostasis and fitness. Higher-order compound emotions, particularly species-specific social emotions, must be considered within the context of a species' ecological and social niches.

Social emotions are emotions elicited by social circumstances, which reorient cognition and behaviour to resolve social challenges or capitalize on social opportunities (Hareli & Parkinson, 2008; Klimecki, 2015). Examples include pride, shame, anger, gratitude, and envy (Sznycer et al., 2021). These emotions carry no fitness benefits for asocial animals, so comparative insights about their forms and functions in humans should begin with our closest evolutionary relatives: other primates. Emotions like pride-shame, anger, and dejection are particularly relevant for the studies discussed below.

The pride-shame emotional spectrum reflects an individual's perceived valuation by others (Robertson et al., 2018; Sznycer et al., 2016; Sznycer & Cohen, 2021). Shame signifies perceived personal devaluation, while pride signifies heightened personal valuation. These emotions result in various forms of social retreat (avoidance) or engagement (approach) and serve to align individuals with the expectations of their social group. For social species like humans, there are fitness benefits of being emotionally well-calibrated to one's social group as it increases the ease in which an individual navigates complex social dynamics.

Additionally, anger and dejection are social emotions that facilitate active and passive responses to social provocation or rejection. These emotions help individuals tune in to context-specific social risk by encouraging or discouraging specific behaviours. Emotions color all experience and guide all behaviour, facilitating perceptual attunement for context-relevant behaviour and orienting us towards the deployment of specialized cognitive tools. To fully appreciate this, it is necessary to explore social cognitive

capacities, including the evolutionary origins of their structure and function within the nervous system.

1.2.3. Behavioural Economics

The second maxim above the Delphic temple, μηδὲν ἄγαν (“nothing in excess”), and its complement "everything in moderation," is an appeal to wise temperance. Comparable aphorisms can be found in the wisdom traditions of many cultures, as their necessity springs from universal, deep-seated impulses for immediate reinforcement that have presumably been selected for over the course of vertebrate evolution. Among others, Kahneman (2011) has proposed and popularized the idea that humans have at least two distinct cognitive modes or systems that can be distinguished based on the timescale over which they operate. System 1 in his framework is the more immediately impulsive mode, using fast cognitive and affective heuristics to make expedient decisions, while System 2 is the slower, more deliberate, and rational cognitive mode that can carefully weigh options and potentially overcome the intrinsic biases of System 1.

Both systems proposed by Kahneman are employed in the service of decision-making, and they serve as an important starting point for understanding economic and social decision-making under risk. Decision-making in the economic and social domains is arguably best captured respectively by prospect theory and game theory, two complementary branches of behavioural economics. This section will briefly outline the relevant aspects of prospect theory, which was proposed by Kahneman to account for some seemingly non-rational peculiarities in human perception and decision-making in economic contexts (Tversky & Kahneman, 1992). Once grounded in the basics of prospect theory, this section will conclude with a brief introduction to game theory, as it has been one of the most successful methods employed in the analysis of social interactions, particularly the fascinating phenomenon of social dilemmas, which are used in several of the studies in this dissertation.

1.2.3.1. Prospect Theory

All else being equal, would you prefer to receive $100 today or $110 tomorrow? What about $1000 a year from today or $1010 in a year and one day? Perhaps your answers to these questions followed a similar logical sequence and were rationally

consistent, but if you intuitively felt the same degree of preference for either the smaller or larger amounts between these choice pairs then you can count yourself as an outlier. Consider another comparison: would you prefer a 5% chance to win $100 or a 10% chance to win $51? Would your choice preference be the same if instead of a chance to win money it was the probability of having to pay the equivalent cost or penalty? These kinds of questions generate raw data upon which prospect theory is based. They probe our perception of comparative value over time, and several interesting conclusions have been drawn about how, on average, our intuitions and decisions deviate from what a perfectly rational agent would generate.

The systematic deviations from rationality that people make are: i) that small payoffs which are guaranteed are typically preferred over larger ones that are uncertain; ii) that we are risk-seeking for losses, but risk-averse for gains (termed the “reflection effect”); iii) that the subjective value of some economic prospect can be influenced by how it is framed by the individual (e.g. narrow or broad, gain or loss, or place in a sequence of choices – termed the “isolation effect”); iv) small probabilities are overweighted (e.g. the difference between 1% and 2% is seen as much greater than that between 51% and 52%; and v) the magnitude of perceived value is nonlinear (e.g. the difference between $10 and $20 is seen as greater than the difference between $110 and $120). Most economic intuitions and decisions can be captured by these principles of prospect theory, as confirmed by a large cross-cultural set of replication studies (Ruggeri et al., 2020). It is often emphasized that these deviations from rationality are systematic, or as Dan Ariely puts it, we are “predictably irrational” (Ariely & Jones, 2008).

It should be remarked that these findings are generalizations from many specific decision sets, and that like most psychological factors there is both quantitative and qualitative individual variance in how value is perceived and weighed. In fact, it is the sources of variance that are of primary interest for the studies described below. The degree to which biological (i.e., hormonal) and related psychological factors explain within- and between-subject variance in these types of economic decisions is one of the core research questions being explored. Another core research question concerns how the same hormones influence economic decision making when equivalent decisions are framed as being purely economic versus social in nature. To explore the latter, it is necessary to understand the basics of game theory.

1.2.3.2. Game Theory

Our ability to predict the thoughts, feelings, behaviors, and reactions of others across various situations, which underscores the intricate complexity of human social interaction, is often taken for granted. While the computational mechanisms of our social cognition largely operate outside our conscious awareness, we have developed tools to improve social self-awareness. Cognitive and social psychology have identified numerous heuristics and biases that shape our social intuitions, but game theory potentially provides a more holistic theoretical framework.

Modern game theory, dating back to the publication of Theory of Games and Economic Behavior (von Neumann & Morgenstern, 1944/2007), aims to formalize social interactions by defining quantifiable parameters within explicit rules. Particularly illustrative of this theory are simultaneous non-cooperative games, where self-interested social agents or "players" choose their strategies without knowledge of others' strategies. These games are defined by four parameters: i) the number of players; ii) the available strategies for each player; iii) the payoffs for each combination of strategies, depicted as a 'payoff matrix'; and iv) the information each player has access to. The richness of social perception and cognition is largely encompassed in the fourth parameter.

In this context, social agents represent individuals, strategies signify available options for any social interaction or decisions, payoffs are the outcomes of social interactions (measured in any proximate "currency" that translates to evolutionary fitness), and information includes any factors that might sway a social agent to pick a particular strategy to boost their payoffs. Payoffs can be in various forms, such as money, information, affiliation, or any other subjectively valuable element. With these parameters, a wide range of game structures can be delineated, with social dilemmas being some of the most intriguing. These games have payoff matrices that compel a conflict between a player's personal interests and those of a group they belong to (Komorita, 2019). Classic instances include the prisoner's dilemma (PD) (Rapoport & Chammah, 1965) and the ultimatum game (UG).

In the PD, the highest individual payoff is obtained by the player who chooses to defect when the other cooperates, whereas both players receive the next highest payoff for mutual cooperation. Mutual defection leads to the third highest payoff, and the lowest

is given to the player who cooperates while the other defects. The UG, on the other hand, involves one player proposing a split of an endowment, which the other player can either accept or reject. If rejected, both players get nothing.

Two other key concepts from game theory—Nash equilibrium and Pareto efficiency—further highlight its utility in social analysis. The Nash equilibrium refers to a set of strategies where no player can better their payoff by unilaterally changing strategies. In contrast, Pareto efficiency refers to a set of strategies where no player can improve their payoff without causing another player to lose out.

While these concepts can predict the actions of simple artificial agents programmed to maximize payoffs, humans are much more complex. Our minds are subject to cognitive biases, situational factors, and other forms of noise, and we are often motivated by more than just maximizing payoffs. As a result, human behavior often deviates from predictions based on these assumptions.

1.3. Biopsychosocial Framework: Rationale, Design, and Hypotheses of Present Studies

To understand how social factors influence the biology of decision-making, we must integrate knowledge about the functions of the nervous and endocrine systems with social and cognitive appraisals of economic risk. Specifically, social challenges or provocations (e.g., threats, social exclusion, etc.) are known to induce a rise in testosterone, and both testosterone and estradiol have been implicated as biological factors that activationally influence the nervous system to promote increased risk-taking in both social and non-social contexts.

1.3.1. Summary and Research Objectives

A summary of the general introduction might be helpful before outlining the studies described in the following chapters. Section 1.1 provided biological background on the nervous system, the endocrine system (emphasizing testosterone and estradiol), and some evolutionary considerations relevant to understanding the structural and functional organization of these systems, including their integration. Section 1.2 offered psychological background on three aspects of cognitive psychology: stimulus valuation,

risk/reward/reinforcement, and decision-making. It also reviewed aspects of social psychology related to social status and revisited pertinent evolutionary considerations. Lastly, it delved into two branches of behavioural economics—prospect theory and game theory—which can be respectively understood as extensions of cognitive and social psychology. Considering these conceptual components, we can now frame the rationale and basic design for the research presented in the subsequent chapters.

With this integrated framework in place, the motivation, basic design, and general hypotheses of the empirical studies can be outlined. Three experimental studies were designed to explore the central theme of the influence of social provocation on the biology of decision-making under risk. To elucidate these relationships, three distinct sets of research questions emerged:

I) What is the relationship between social versus non-social provocation or facilitation and framing of otherwise equivalent economic decision-making?

   a. Does provocation affect circulating levels of the sex steroids testosterone (T) and estradiol (E)?
   b. Do equivalent risky decisions differentially influence testosterone and estradiol when the outcomes are framed as coming from people versus algorithms?
   c. Do testosterone and estradiol influence economic decisions differently when provocation payoffs to simple gambling decisions come from a social versus non-social agent?

II) What is the relationship between social versus non-social provocation or facilitation specifically to decisions under risk or ambiguity?

   a. Does provocation affect circulating levels of the sex steroids testosterone and estradiol?
   b. Do testosterone and estradiol influence economic decisions under risk differently when provocation comes from a social versus non-social agent in the form of unfair treatment than when socially facilitated by fair treatment?

c. Do testosterone and estradiol influence economic decisions under conditions of ambiguity differently than decisions under simple risk when provocation comes from a social versus non-social agent?

III) Is the effect of a competitive social provocation on economic decision-making influenced by a broader contextual framing of cooperation?

a. Does social facilitation and provocation affect circulating levels of the sex steroids testosterone and estradiol differently?
b. Do testosterone and estradiol influence economic decisions differently under conditions of social facilitation versus provocation?

The first set of questions will be addressed by a simple 2 x 2 between-subjects experiment in which participants play several rounds of the ultimatum game. In each condition, participants are the responder, with the experimental factors being distinguished by whether the proposer is conveyed to be a person or an algorithm, and whether the proposer makes primarily fair or unfair offers. The first factor is meant to address the social versus non-social aspect of the research questions, whereas the fair and unfair offers are meant to capture the facilitation versus provocation aspect.

The second set of questions will be addressed by another simple 2 x 2 between-subjects experiment in which participants play several rounds of the ultimatum game. The factors and conditions are identical to the first experiment, but with an additional dependent variable of an Ellsberg Urns task to probe the effects of social and non-social offers of fair or unfair amounts in the UG on risk and ambiguity, as measured by the ambiguity premium. In the Ellsberg Urns task, participants are told that there are some ratio of black and white marbles in an urn, and that they can either choose a colour and gamble on winning or losing some payoff by reaching in and pulling out a marble to match their colour choice, or they can sell their decision for a more certain payoff that is less than the expected value of the gamble. Because the participant knows the ratio of marbles it is considered a risky decision. The same choice is given for another urn whose marble colour ratio is not known, which is what makes it an ambiguous decision. The difference between the participant's sale price for the ambiguous and risky urns is their ambiguity premium.

For the third set of questions, I developed a novel, tournament-style form of the UG called the hierarchical ultimatum game (HUG). This is framed as a second-order social dilemma, in that the proposer of the UG goes on to play another gambling game with their winnings from the first game with the participant, and their subsequent gambling winnings are shared equally with the participant. It is a 2 x 2 between-subjects design with fair versus unfair offers and prosocial versus antisocial proposers serving as the levels of the two factors. The direction of pre- and post-task hormone change is unspecified *a priori* for all three sets of hypotheses because for estradiol there is insufficient previous research upon which to base directional hypotheses, and because for all three hormones these are sufficiently novel research protocols that expectations of directional change are unwarranted.

These three studies and sets of hypotheses explore distinct aspects of the intersection between behavioural endocrinology of decision-making and social cognition. Each of them is designed to both elaborate or combine previous research in each respective subfield, as well as to include novel contributions. The most salient example of the latter is the inclusion of baseline estradiol and estradiol change as an outcome variable, which is an important and understudied hormone for human behavioural endocrinology, decision-making, and social cognition.

Chapter 2.

Sex-Specific Sex Steroid Responses to Social Versus Non-Social Decisions Under Risk

2.1. Introduction

In nearly all behavioural domains decisions are often made without certainty about their outcomes or consequences, meaning that there is some element of risk associated with the decision. How humans make decisions under risk has been a topic of considerable debate and research, and many theories have been proposed to account for this broad range of decisions (Mishra, 2014). Some of the most influential theories have been expected utility theory (von Neumann & Morgenstern, 1944/2007), prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992; Barberis, 2013), risk-sensitivity theory (Kacelnik & Bateson, 1997; Hintze et al., 2015), and cognitive heuristics (Tversky & Kahneman, 1974; Gigerenzer, 2008).

2.1.1. Decision-making under risk

Expected utility theory was first formalized in the 1944 “Theory of Games and Economic Behavior” (von Neuman & Morgenstern, 1944/2007), and the core of the theory is a set of utility functions that were intended to describe relatively consistent profiles in decision-making behaviour. The utility functions graph changes in subjective utility (i.e., individual preferences for some quantifiable reward) as a function of absolute reward value, where convex curves reflect increasing risk-preference, linear relationships reflect risk-indifference, and concave curves reflect increasing risk-aversion (Mishra, 2014). For example, if someone subjectively values the relative difference of $30 compared to $20 more than they value the difference of $20 compared to $10, they are risk-preferring, whereas someone who subjectively values those differences equally is risk-indifferent, and someone who values the latter difference more than the former is risk-averse.

While there is an appealing elegance to the way that expected utility theory categorizes decision makers, the actual behaviour of humans is more complex than what can be captured by simple utility functions. This was prominently demonstrated by

the experiments that led to prospect theory (Kahneman & Tversky, 1979). Prospect theory empirically extended the framework of expected utility theory by demonstrating that even when considering a single reward currency (e.g., dollars), there are consistent asymmetries in how humans think about and decide on gains versus losses. That is, while most people tend to be, on average, risk-averse when it comes to gains (i.e., value an increase of $1 more when it is framed as the difference between $1 and $2 than when it is framed as a difference between $99 and $100), there is also a general tendency to be *even more* risk-averse to losses compared to gains.

One of the reasons for these asymmetries that is proposed by prospect theory is *framing effects*, where people tend to make different decisions on equivalent risky scenarios based on positive (gain) or negative (loss) framing, and that the framing is normalized around a zero-point anchor. The anchor concept is used to explain reliable decision tendencies in the population, such as large differences in preferences for financial gains or losses between individuals who are relatively poor compared to those who are relatively rich. While there are several innovations and advantages to prospect theory compared to expected utility theory, one of the primary disadvantages is that it underspecifies the currency of utility, and it does not generate reliable exchange rates between different utility currencies (e.g., dollars and calories).

Another prominent theory of decision-making under risk is risk-sensitivity theory (Hintze et al., 2015; Kacelnik & Bateson, 1997), which was inspired by foraging behaviour of animals in natural ecologies. This theory incorporates the explore-exploit trade-off in reinforcement learning, which is a behavioural dilemma between time spent searching for better fitness-enhancing opportunities, and time spent taking advantage of known resources. The primary insight from risk-sensitivity theory is that scarcity and need tend to induce risk-seeking behaviours on animals who are otherwise risk-averse in situations and times of plenty. That is, the state of the organism and its current environmental context are directly relevant to the degree to which risky decisions are made. While this theory does not fully address the currency issue, it does incorporate the context in which decisions are made, which is an important feature of decision-making under risk.

The last of the most influential approaches to decision-making is cognitive heuristics (Mishra, 2014). Cognitive heuristics are simple strategies for making decisions

that circumvent the need to spend excessive time collecting the complete information set that could usefully inform a decision. Herbert Simon's satisficing heuristic (Simon, 1956; Parker et al., 2007) is one example, where the decision space is searched only until an option is found that sufficiently satisfies some pre-determined decision preference. For example, when searching through a lengthy menu of a new restaurant, a satisficing rule might be to choose the first unfamiliar dish that is in one's price range. Another example of a decision heuristic is maximization, in which the maximum value along one or more dimensions must be satisfied for a choice option to be selected.

2.1.2. *Domain-specificity of decision-making*

It is widely accepted that evolution has endowed us with a toolkit of such cognitive heuristics to navigate complex decision making under risk and uncertainty (Johnson & Busemeyer, 2010; Mousavi & Gigerenzer, 2014), and that decisions under risk are generally subject to the cognitive biases outlined in prospect theory and risk-sensitivity theory. It is also generally accepted that humans employ complementary but different sets of heuristics for dealing with distinct cognitive domains. One of the most prominent distinctions in cognitive domains is between the non-social and social worlds, which is reflected in both behaviour and activity within neural networks (Spunt & Adolphs, 2017). In addition, there is a growing literature on the relationship between hormones and economic decision-making under conditions of simple risk (Stenstrom & Saad, 2011; Barel et al., 2017; Kusev et al., 2017), as well as in cooperative and competitive social contexts (Reimers & Diekhof, 2015; Zilioli & Bird, 2017).

Previous research has indicated that economic decision-making is influenced by whether the decision is framed as a non-social or social event (FeldmanHall et al., 2015). This may be due at least partly to the involvement of trust in social but not non-social events, where trust is "a psychological state comprising the intention to accept vulnerability based upon the positive expectations of the intentions or behaviour of another" (Rousseau et al., 1998; Evans & Krueger (2009); Krueger & Meyer-Lindenberg (2019). From an evolutionary perspective trust is a crucial component of reliable and mutual cooperation, with implications for individual and group level fitness (Nowak, 2006). There is even a strong asymmetry in what and how many indicators are required for someone to be categorized as trustworthy versus untrustworthy (Fiske et al., 2007).

While there are several neural models for both economic decision-making (Basten et al., 2010; Ratcliff et al., 2016), and social cognition involving trust (Lee, 2008; Ruff & Fehr, 2014), physiological mechanisms for the distinct heuristic sets underlying non-social and social risk-taking have not described. The current study sought to explore the relationship between hormones that are known to be associated with risk decisions when the risks are framed as either algorithmic (non-social) or determined by another person (social).

2.1.3. Endocrine effects on economic decision-making under risk

In the non-social realm, the sex steroids (e.g., testosterone and estradiol) have each been individually and jointly implicated in risk-taking (Schipper, 2014; Apicella et al., 2015; Kurath & Mata, 2018). For example, both baseline and increasing levels of testosterone have been associated with variation in financial risk-taking (Stanton et al., 2011; Apicella et al., 2014; Stanton et al., 2021). Cortisol, an important hormone for the stress response, has also been implicated in moderating the effect of testosterone on financial risk-taking (Cueva et al., 2015; Mehta et al., 2015; Herbert, 2018). In addition, recent evidence suggests endogenous estradiol affects decision-making under risk (Diekhof, 2015; Diekhof et al., 2020). This body of research generally implicates higher sex steroid levels with riskier economic decision-making, except when cortisol levels are high.

Steroid hormones have also been investigated for their role in social decision making (Eisenegger et al., 2011; Zilioli & Bird, 2017; Geniole & Carre, 2018). For example, testosterone has been implicated in sensitivity to trust and perceptions of fairness (Burnham, 2007; Bos et al., 2010; Maarten et al., 2013) as well as generosity (Zak et al., 2009). Cortisol has similarly been studied for its moderating effects on testosterone in game scenarios (Mehta & Prasad, 2015b). While estradiol has been implicated in threshold changes for framing and acceptance of unfair offers in the ultimatum game (Coenjaerts et al., 2011), other reports call the relationship between steroid hormones and trust into question (e.g., Zethraeus et al., 2009). Together these studies point to a robust and complex relationship between steroid hormones and decision making under risk in both non-social and social contexts.

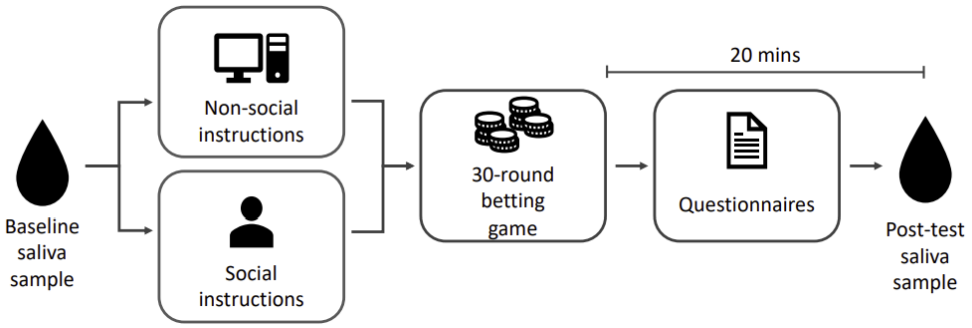
While there is an abundance of research regarding risk taking for economic decisions in both non-social and social contexts separately, few studies have directly compared these two cognitive and behavioural domains (Stenstrom et al., 2011; FeldmanHall et al., 2015). Additionally, while the effects of steroid hormones have been investigated for their role in risky economic decisions, and in a variety of social scenarios drawn from game theory, they do not appear to have been studied in a paradigm that directly compares these two decision types. Two outstanding questions exist regarding their possible relationship. The first outstanding question is, do baseline levels of sex steroid and glucocorticoid hormones differentially influence economically equivalent risky decisions when they are framed as non-social versus social? That is, do our steroid hormones influence quantitative decision-making when those decisions are contingent on our perception of another person's intentions compared to when no such intentions are perceived, all other things being equal? A real-world example of this difference would be playing a slot machine or any other *n-armed bandit* (non-social) or investing in a company whose CEO has a large stock option grant (social), where the amount of invested money and the expected payoffs for both are the same. Another example is simply playing poker against a poker-playing algorithm compared to playing against another person. The second outstanding question is, does the social versus non-social framing of equivalent economic outcomes to risky decisions influence sex steroid and glucocorticoid levels?

2.2. Methods

The primary dependent variable was the risk propensity of the participant, measured by the degree to which they were willing to make risky decisions in a gambling task. The task was incentivized by the prospect of winning a prize whose value was $100, as participants gambled with virtual tokens which at the end of the experiment served as ballots for a lottery to win the gift certificate prize.

2.2.1. **Subjects**: Undergraduate participants (N = 100; 49 male) were recruited from the SFU Psychology Department's research participation system (RPS) to participate in a single-factor between-subjects experiment. A power analysis for within-subjects' effects was performed using G*Power (Faul et al., 2009). With an alpha level of 0.05, an effect size of d = 0.4 and d = 0.41 is expected to be detected with a beta level of 0.2 for females and males, respectively. For between-subjects' effects with the same alpha and

beta levels, an effect size of d = 0.57 is expected to be detected. Both power analyses assumed two-tailed critical t-values and are each conservative estimates. The factor of the experiment was the identity of the proposer in the ultimatum game and gambling task, with two levels: algorithm and human. That is, in the control condition participants were led to believe that the payoffs of their bets were being determined by a stochastic process, while in the experimental condition they were led to believe that the payoffs of their bets were being determined by another person. In both cases the frequency of wins and losses were actually equivalent and stochastic. Participants were randomly assigned to either the non-social framing condition or the social framing condition.



2.2.2. **Procedure**: Participants provided their informed consent as outlined in the SFU REB approved experimental protocol (#30000494) and were then instructed to provide saliva which was then immediately placed in a freezer by the participant. Once the initial saliva sample for baseline hormone measurement was collected, participants were brought to the testing room where they were instructed to follow computer prompts that explained the gambling task and provided the initial framing for the non-social and social framing of feedback payoffs in the gambling task.

2.2.3. Hormones: two saliva samples were collected from each participant, one immediately after participants gave informed consent to participate in the experiment, and one twenty minutes after they completed the last trial of the gambling task. All experimental sessions, including the collection of saliva samples, were done between 1pm-5pm to control for the effects of circadian rhythms on steroid hormone concentrations (Czeisler, & Klerman, 1999). For each sample the participants were instructed to provide at least 2mL of saliva via a passive drool into a 15mL plastic centrifuge test tube. Once they reached the 2mL mark the participant immediately placed the tube into a freezer for storage until the samples could be analyzed for steroid

hormone concentrations. Hormone analysis was performed by Salimetrics Inc. (Carlsbad CA) using enzyme-linked immunosorbent assays (ELISA) (Lequin, 2005). Each sample was analyzed for testosterone and cortisol, and the female participants' samples were analyzed for estradiol.

2.2.4. Gambling task: a simple decision-making task was designed and employed to capture individual risk preferences. The task instructions were for participants to bet a relatively small (1), medium (2), or large (3) number of virtual tokens in each trial. There were thirty trials in total, with the participants not being aware of the number of trials in advance. Participants were not under a time constraint to decide on the value of their bet, and the result of each gamble was either a gain or loss of the number of tokens that were bet in that trial. That is, if a player made a small bet of 1 token and won, they would receive a feedback payoff of 2 tokens, whereas winning bets of 2 or 3 tokens would result in payoffs of 4 and 6 respectively, and loss trials simply resulted in losing the amount that the participant bet. The total number of wins and losses out of the thirty trials was predetermined (18 +/- 1 gains) but the ordering of the feedback (wins or losses) was stochastic for each participant to randomize and control for ordering effects. Participants always doubled their bet in "win" trials and lost their bet in "loss" trials. This structure ensured that there were no specific ordering effects to the wins and losses for each participant's bets, while holding the frequency distribution of wins and losses constant for all participants.

2.2.5. Psychometric measures: several psychometric questionnaires were filled out by participants to collect data described in previous literature as mediating or moderating the relationship between steroid hormones and risk-taking propensity. The questionnaires collected data about: i) hubristic and authentic pride (Tracy et al., 2009), which has been associated with positive emotions, social status, and testosterone (Tracy et al., 2010; Martens et al., 2011; Verbeke et al., 2018) ii) self-construal (Hardin et al., 2004), which mediates testosterone's effects on aggression and risk (Welker et al., 2017; Welker et al., 2019); iii) social dominance orientation (Pratto et al., 1994), which has been shown to be a relevant factor in mediating the effect of testosterone to competitive defeats (Maner et al., 2008); and iv) big-five personality dimensions (Gosling et al., 2003), as extroversion is associated with higher testosterone during male mating efforts (Alvergne et al., 2010), and conscientiousness has a weak negative correlation with testosterone (Sundin et al., 2021). A final questionnaire was administered which

collected demographic and other miscellaneous information that was considered relevant to the research questions (Appendix A).

2.3. Results

All analyses were performed using SPSS 27 for Windows.

2.3.1. **Descriptive Statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
T0	96	27.75	424.85	164.4582	109.66049
T1	95	28.22	346.23	156.3855	92.71475
E0	47	.66	2.07	1.2930	.35442
E1	46	.67	2.07	1.2628	.31815
C0	91	.06	.59	.2568	.12020
C1	90	.06	.61	.2090	.09320
FirstBet	100	1.00	3.00	1.8300	.68246
Avg_Bet	100	1.27	2.83	2.0445	.26644
Avg_Outcome	100	-.07	.73	.4025	.15948
Total_Bet1s	100	.00	23.00	9.6800	4.32255
Total_Bet2s	100	.00	19.00	9.5800	3.39988
Total_Bet3s	100	1.00	26.00	10.7400	4.32661

Table 2.1. Descriptive statistics for endocrine and behavioural data. Pre-task testosterone (T0); post-task testosterone (T1); pre-task estradiol (E0); post-task estradiol (E1); pre-task cortisol (C0); post-task cortisol (C1); participant's first trial bet (FirstBet); participant's mean bet value (Avg_Bet); Participant's mean trial payoff (Avg_Outcome); The respective number of small, medium, and large bets (Total_Bet1s/2s/3s).

2.3.2. **Behavioural measures:** The first set of analyses sought to identify differences in betting behaviour between experimental conditions, which was first done using between-subjects t-tests. While there were no differences between conditions for the average bets, or the frequency of low, medium, or high bets, the average outcome was different between conditions ($t_{(98)} = 2.022$; $p = 0.046$). This average difference in payoff outcome

can be explained by an interaction between sex and condition, which emerged from a two-factor ANOVA, where female participants in the social condition made significantly fewer high bets than males and all participants in the non-social condition ($F_{(1,96)} = 4.59$; $p < 0.05$).

2.3.3. **Endocrine measures**: Between-subjects t-tests revealed no significant differences in baseline hormone levels between conditions, and no relationship between baseline hormone levels and betting behaviour was found within each condition when all participants were included. However, repeated measures ANOVAs revealed that there was a significant effect of condition on hormone changes. Sex steroid levels were maintained for both sexes in the social condition but dropped in a sex-specific way for both females and males in the non-social condition. That is, estradiol was lower post-task in females in the non-social condition ($F_{(1,44)} = 6.556$, $p = 0.014$; figure 2.1), and testosterone was lower post-task in males in the non-social condition ($F_{(1,46)} = 9.59$; $p = 0.003$; figure 2.1). A cortisol and condition interaction was also found, where cortisol decreased in the non-social condition, but not in the social condition ($F_{(1,86)} = 4.203$, $p = 0.043$; figure 2.2). However, there was no significant 3-way interaction between cortisol, condition, and sex. ANOVA summary tables can be found in Appendix B.

When the sample was split into low and high levels of baseline hormones by sorting participants into the lowest and highest thirds of their respective distributions, only one significant behavioural difference was observed. Females with low baseline estradiol made larger first bets than females with high estradiol ($t_{(30)} = 3.397$; $p = 0.002$). There was no difference in first bet for either sex between conditions, and there was no comparable effect of baseline testosterone for males on their first bet. No other relevant behavioural measure that was investigated (i.e., average bet, frequency of low/medium/high bets) showed a significant difference between individuals who were respectively low and high in sex steroid concentrations. There were also no significant differences in relevant behavioural measures between individuals who were comparably split into low and high in cortisol.

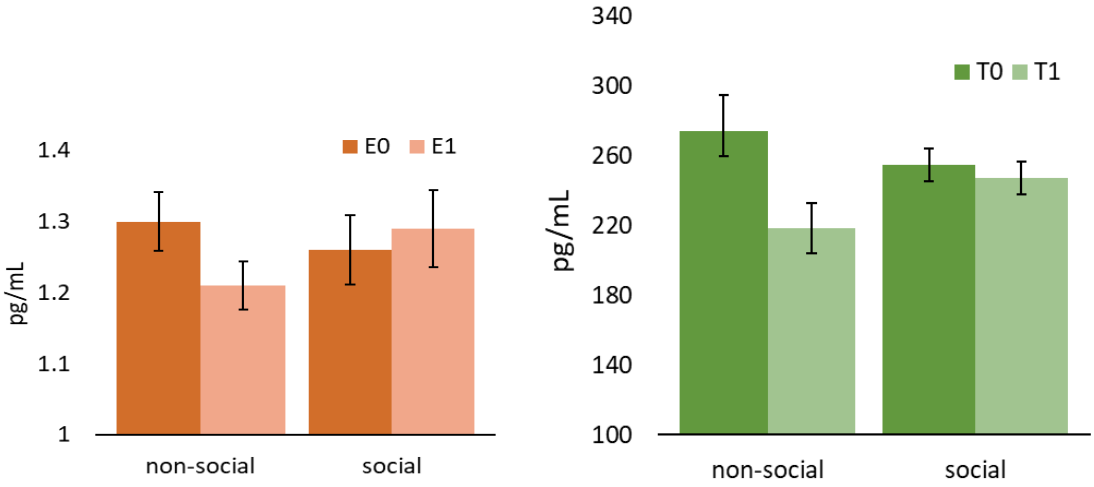


Figure 2.1 – Sex-specific changes in sex steroid hormones in the non-social condition but not in the social condition. In the non-social condition, decreases were observed for both estradiol for females (left) and testosterone for males (right). Neither sex steroid hormone showed a significant change in the social condition.

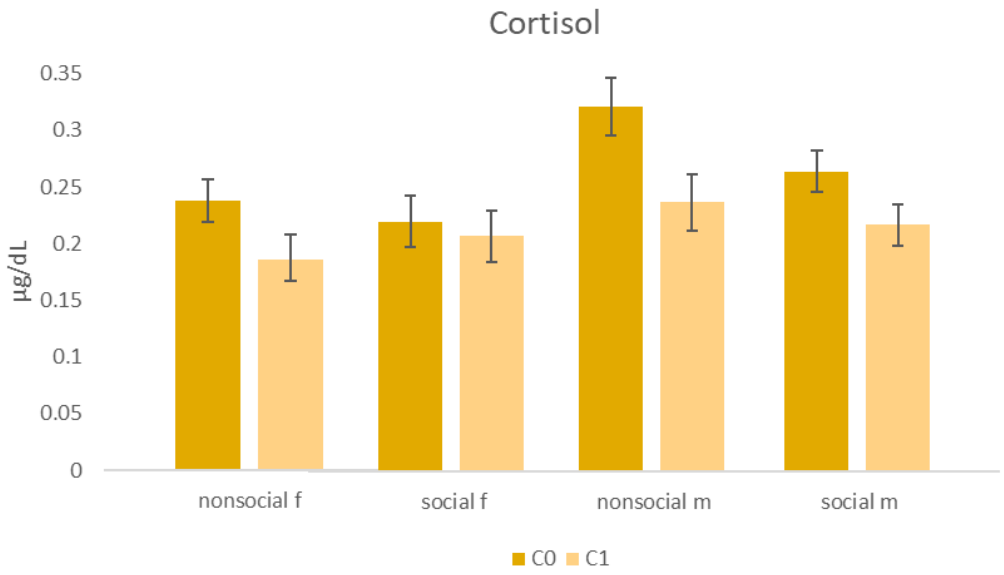


Figure 2.2 – Sex-specific cortisol changes in both non-social and social conditions. A significant decrease occurred for all conditions except for the social condition for females.

There are glucocorticoid receptors on cells in the HPG-axis (Chandran et al., 1994; Bourke et al., 2012) which contribute to the differential regulation of sex steroids in both males and females. It is therefore appropriate to treat cortisol as a moderating variable for the sex steroids, as high levels of cortisol tend to inhibit the release of the sex steroids (Toufexis et al., 2014). To that end, a set of moderation analyses were performed using the Process Macro in SPSS (Hayes et al., 2017) to test whether the condition-specific changes in female estradiol and male testosterone were respectively influenced by cortisol reactivity (i.e., change in cortisol between pre- and post-task samples). A moderating variable is one whose values influence the effect that the independent variable has on the dependent variable. In this case the moderation analyses tested whether variance in cortisol reactivity influenced the degree to which experimental condition affected changes in female estradiol and male testosterone. Both models indicated significant interactions but in opposite directions for females and males. For females there was a cortisol by condition interaction ($F_{(3,39)} = 11.36$, $p < 0.001$; figure 2.3), such that increases in cortisol were associated with increases in estradiol in the social condition but not the non-social condition. For males there was also a cortisol by condition interaction ($F_{(3,41)} = 6.47$, $p < 0.001$; figure 2.3), such that

decreases in cortisol were associated with decreases in testosterone in the non-social but not the social condition. It is noteworthy that the moderating effect of cortisol on female estradiol and male testosterone involved opposite direction of cortisol changes, opposite direction of sex steroid changes, and the effects were on different conditions for females and males.

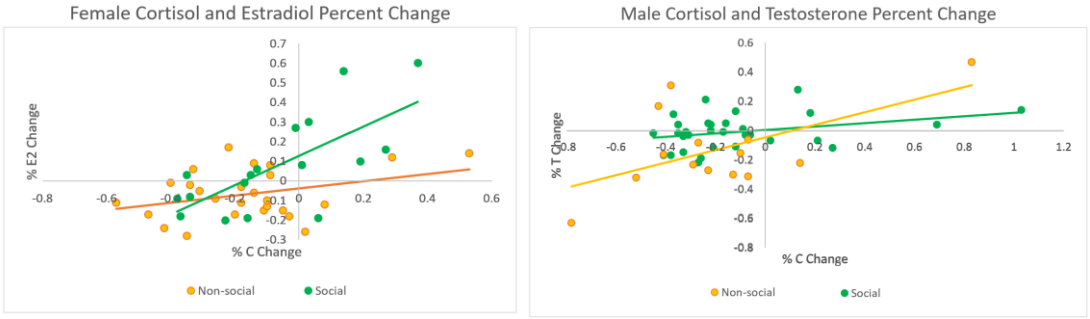


Figure 2.3 – Cortisol moderates the effect of condition on sex steroid reactivity for females (A) and males (B), in opposite directions and in different conditions.

2.4. **Interim Discussion**

To summarize the findings, there were no significant differences in average betting behaviour between the conditions, but a narrowly significant difference in the average outcome (i.e., payoffs) from the bets was observed and accounted for by a low rate of high bets for females in the non-social condition. As expected, baseline hormones did not differ between conditions, but significant decreases in female estradiol, male testosterone, and cortisol for both sexes were observed in the non-social condition. In the social condition sex steroids remained stable and cortisol decreased for males only. A moderation analysis revealed that increases in female cortisol were associated with increases in estradiol in the social condition, while decreases in male cortisol were associated with decreases in testosterone in the non-social condition.

There are distinct lines of research that describe the dynamics of economic decision making under risk (Wakker, 2010; Johnson & Busemeyer, 2010) and how hormones influence decision making in non-social and social contexts (Zilioli & Bird, 2017; Orsini et al., 2021), but no prior research exists that has directly compared the endocrinology of non-social and social economic decision making. The present study explored this relationship with a simple incentivized gambling task in which hormones

were measured immediately prior and twenty minutes after the task for a baseline and post-task effect measure, respectively. Consistent with established findings on sex differences in economic decision making (Sapienza et al., 2009; Derntl et al., 2014), it was found that female participants made fewer high-risk bets than males. However, in the current study, this effect was restricted to the social condition. In addition to the sex difference in betting behaviour, there was an observed decrease in female estradiol and male testosterone in the non-social condition, but a post-task maintenance of baseline sex steroid levels in the social condition. A decrease in cortisol was observed in both sexes and in both conditions.

The current experiment offers the first evidence that there is a domain specificity in the engagement of sex steroids for decision making under risk, and there are at least two possible explanations for this effect. The first explanation is that merely interacting with another individual requires the maintenance of sex steroids, which are known to be engaged in many aspects of social cognition (McCall & Singer, 2012). In the non-social condition the lower post-task sex steroid levels may reflect a typical circadian and/or pulsatile decrease in sex-steroids, and in the social condition the stable post-task sex steroid levels may actually be a relative compensatory increase away from an otherwise expected decrease. A second and possibly complementary explanation relates to another result from the study, that there was a statistically significant difference in the overall betting outcomes between conditions, with participants receiving greater overall returns on their bets in the non-social condition than in the social condition. This is a possible explanation for the difference in female estradiol and male testosterone between the non-social and social conditions.

One reason why there may exist a domain specificity in the hormone response to non-social and social economic risk is that different cognitive heuristics are used when making decisions in these distinct behavioural domains (Adolphs, 2009). While the non-social domain involves decision processes that have been well characterized in behavioural economics, decisions that are matched in economic risk but framed as a kind of social decision making is thought to rely on distinct cognitive processes. For example, social decision making can be biased by one’s impression of the individual making an offer, considerations of their motivation and incentives, and how these change from one offer to the next depending on outcomes (FeldmanHall & Shenhav,

2019). In contrast, algorithmically generated (non-social) outcomes are typically assumed to be independent from one trial to the next.

Since social cognition relies on a distinct set of mental processes, and since at least part of the role of sex steroids is to influence behaviours that are unique to reproductive and social challenges (O'Connell & Hofmann, 2012; McCall & Singer, 2012), it is natural to conclude that the difference in sex steroid dynamics between equivalent economic risks when framed as either non-social or social is a result of the roles that sex steroids generally play in social cognition. The data in the current study implicate sex steroids in meeting the unique challenges of the social world, even when these challenges are equivalent to non-social ones. The mere framing of economic decisions under risk as being non-social or social affects sex steroid release in a sex-specific manner, and the maintenance of female estradiol and male testosterone may serve to promote cognitive processes that are unique to social interaction.

Chapter 3.

Social Context Influences Sex-Specific Sex-Steroid Effects on Risk and Ambiguity Judgments

3.1. **Introduction**

Economic decisions are rarely made with certainty about what outcomes will follow, and therefore there is often an element of risk involved in such decision-making. Additionally, in behavioural economics there is a distinction made regarding the degree of uncertainty about decision outcomes. The term *risk* is used to describe decisions in which the probabilities of various possible outcomes are known (e.g., a fair coin flip has two possible outcomes and there is a 50% likelihood that either outcome will occur), and an individual's approach to such decisions can be described in terms of their risk thresholds or risk tolerance or risk aversion (Klibanoff et al., 2005; Krain et al., 2006; Jackson et al., 2016). There is also a kind of second-order risk called *ambiguity* which is used to describe decisions in which the possible outcomes are either known or unknown, but their respective probabilities are not known (e.g., blind auction bidding). While there has been a considerable amount of research done at the intersection of behavioural endocrinology and economic decision-making under risk (Stenstrom & Saad, 2011; Barel et al., 2017; Kusev et al., 2017), very little comparable work has been done investigating risk propensities with decisions-making under ambiguity (Danese et al., 2017).

3.1.1. *Economic decision-making under risk*

There are several models of decision-making that have been variably applied to help explain some asymmetries within and between individuals regarding risk (Mishra, 2014). These are outlined in the previous chapter and include expected utility theory (von Neumann& Morgenstern, 1944/2007), prospect theory (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992; Barberis, 2013), risk-sensitivity theory (Kacelnik & Bateson, 1997; Hintze et al., 2015), and cognitive heuristics (Tversky & Kahneman, 1974; Gigerenzer, 2008). Examples of such asymmetries are i) stronger risk- and loss-aversion than a comparable propensity for reward; and ii) subjective over-weighting of small probability differences for gains and losses when the base rate probabilities are

relatively low, and underweighting of comparable probability differences for gains and losses when the base rate probabilities are relatively high. For example, an increase from a 1% to 2% chance of winning a lottery is typically more actionable for people than an increase of 98% to 99%, all else being equal. While the improvement in the likelihood of winning is equivalent in both cases, a simple cognitive heuristic may be responsible for the difference in asymmetric subjective weighting (i.e., the likelihood “doubles” in the first case).

Prospect theory captures these asymmetries graphically, as illustrated below (figure 3.1). However, while there is a general tendency toward risk- and ambiguity-aversion, there are situational and dispositional factors that can incline individuals toward risk-seeking and ambiguity-imprudence, or even insensitivity to risk or ambiguity (Trautman & van de Kuilen, 2018; Kocher et al., 2018). Some situational factors include the information source (e.g., preference for familiar ambiguity over equal unfamiliar ambiguity), provisional subjective competence about other unrelated decisions (e.g., confidence in successful prior risky decisions can decrease risk- and ambiguity aversion), and whether the decision maker is being observed, while some common dispositional factors include age, sex, and personality traits (Aumeboonsuke & Caplanova, 2021; Trautman & van de Kuilen, 2015).

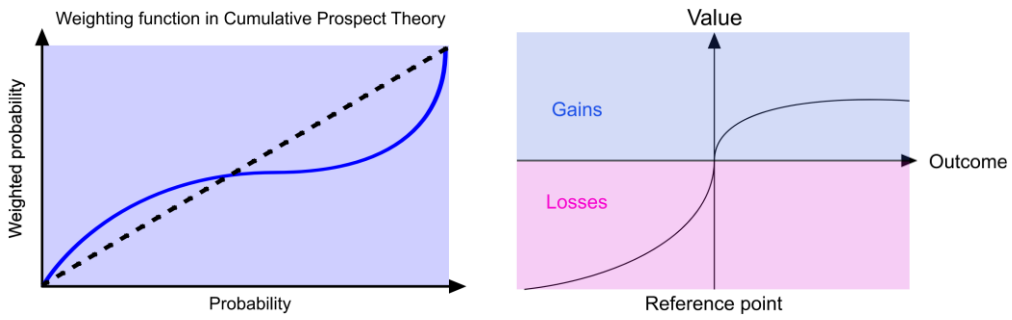


Figure 3.1 – Graphical representation of asymmetric subjective weighting of probabilities for gains (left), and for the subjective utility of gains and losses (right).

3.1.2. Endocrine influence on the nervous system and decision-making

Hormones can also serve as both situational or dispositional factors that can influence risk or ambiguity attitudes and behaviours, depending on context. The kinds of effects can be broadly characterized as “activational” and “organizational”, where activational effects of hormones are transient changes to physiology and/or behaviour resulting from dynamical hormone fluctuations, and organizational effects of hormones are more permanent changes that occur during sensitive/critical developmental windows (e.g., perinatal or adolescence) (Romeo, 2003; Schultz et al., 2009; Berenbaum & Beltz, 2011). There are evolutionary considerations for both types of effects, with sex differences in physiology and behaviour being especially prominent. For example, male and female gonads typically produce distinct ratios of androgenic and estrogenic sex-steroids (Ruiz-Cortes, 2012; Svechnikov & Soder, 2008). The androgen testosterone is known to masculinize several organs, including the brain via its conversion to estradiol by the enzyme aromatase (Simpson et al., 2002). That is, exposure to differential levels of testosterone in utero and during adolescence developmentally organizes the brain in a sexually dimorphic (or polymorphic, as there may be more than two human “morphs” [Mank, 2023]) manner. Some sexually dimorphic brain areas include various nuclei in the hypothalamus (Fernandez-Guasi et al., 2000; Swaab et al., 2001), and several other cortical and subcortical regions that can influence decision-making under risk (Broche-Perez et al., 2016; Daw et al., 2006; Neubert et al., 2015). Sexually dimorphic brain regions that express protein receptors for androgenic or estrogenic sex steroids are expected to respond to comparable concentrations of these hormones in different ways. However, males and females typically produce quite distinct concentrations of sex steroids, further exaggerating their pronounced effects on each sex.

Two prominent evolutionarily relevant considerations for the effects of sex steroids on behaviour are: first, that there is a sex difference in social cognition that is influenced by sex steroids, and second, that decision-making for economic utility is influenced by sex steroids. The first consideration is relevant for the present study because the experimental manipulation involves the social context of fair versus unfair offers in a social game interaction (discussed below; Dreber & Johannesson, 2018), and the second consideration is relevant because economic decision-making under risk and ambiguity has also been observed to show reliable sex differences, which presumably

reflect distinct ancestral socio-ecological niches between the sexes (Migliano & Vinicius, 2022). Empirical support for these considerations is described below.

3.1.3. Sex steroids in (social) decision-making under risk and ambiguity

It is known that there is typically an endocrine response to social provocation (Archer, 2006; Carre et al., 2014; Wagels et al., 2018; Carre & Archer, 2018) and that one's hormonal state can directly influence social decision-making and subjective risk propensity. For example, Eisenegger et al., (2010) showed that testosterone administration in women increased fairness in their offers in the ultimatum game (UG), and Bos et al, (2010) showed that testosterone administration in women decreased perception of trustworthiness when evaluating pictures of faces. These findings about fairness and trust were partially synthesized in later research that demonstrated that testosterone administration decreased trust and increased reciprocity when subjects played the trust game as investor and trustee, respectively (Boksem et al., 2013). Together these studies point to a general social function for testosterone where is increases social vigilance but enhances mutualistic and cooperative behaviours (Van Honk et al., 2012).

In the current experiment I sought to replicate and extend a previously published experiment on hormones, risk, and ambiguity (Danese et al., 2017). In that experiment male participants gave their subjective sale prices for the "risk" and "ambiguity" decisions in the Ellsberg Urns paradigm. In addition, pre- and post-task saliva samples were collected for hormone analysis and revealed that testosterone and cortisol jointly influence the difference between the sale prices of these two urns (their "ambiguity premium"). In this Ellsberg Urns paradigm, there are two urns that are referred to as either the "risk" urn or the "ambiguity" urn. Participants are asked to make a bet on drawing a marble from each urn. In the In the "risk" urn there are a known number of black and white marbles, and therefore a known probability of drawing a chosen colour. In the "ambiguity" urn the ratio of black and white marbles, and therefore the probability of selecting a particular colour, is unknown. When participants are told that the "risk" urn contains an equal number of black and white marbles, then the estimate on the number of each type of marble for both urns is the same. However, despite this equivalence, it is typical to find that participants prefer to make bets on the "risk" urn, with a known probability for their bet. This finding has been dubbed the Ellsberg Paradox, which is a

specific type of ambiguity aversion, and is an example of a deviation from the classical behavioural economics assumption of rational human behaviour.

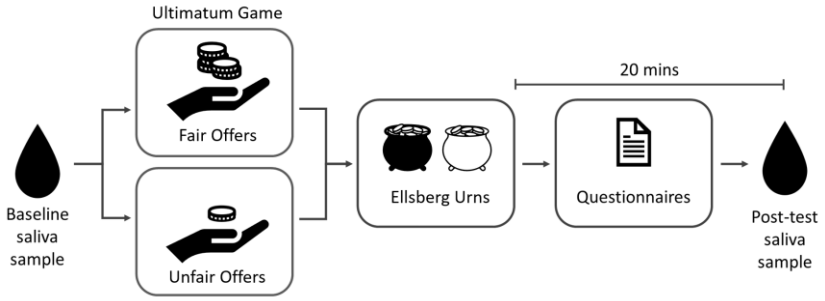
The present experiment modifies this by introducing an initial social provocation (unfair UG offers) or facilitation (fair UG offers) and exploring a possible role for estradiol in female participants. Along with testosterone, estradiol has also been studied in the context of economic risk and social cognition, however the literature is relatively limited compared to testosterone. In postmenopausal women estradiol did not influence trust or reciprocal fairness (Zethraeus et al., 2009). However, testosterone also did not influence these aspects of social cognition in the same study, perhaps suggesting that there is an important difference in age and/or endocrine status. Indeed, a recent review does corroborate a role for both testosterone and estradiol in risk-taking behaviour (Kurath & Mata, 2018).

The research questions for the current experiment are fourfold. Firstly, do the findings from Danese et al., (2017) replicate with males and extend to female participants? Secondly, is there an influence of social provocation or facilitation on risk and ambiguity thresholds in an Ellsberg Urns task as reflected by unfair and fair UG offers, respectively? Thirdly, does fairness in UG offer differentially influence hormonal states (testosterone, estradiol, and/or cortisol)? Finally, if there is an observed effect on risk and ambiguity decision-making, can they be attributed to changes in hormone state for males and females?

3.2. **Methods**

3.2.1. **Subjects**: Undergraduate participants (N = 76; 26 male) were recruited from the SFU Psychology Department's research participation system (RPS) to participate in a single-factor between-subjects experiment. A power analysis for within-subjects' effects was performed using G*Power (Faul et al., 2009). With an alpha level of 0.05, an effect size of d = 0.41 and d = 0.6 is expected to be detected with a beta level of 0.2 for females and males, respectively. For between-subjects' effects with the same alpha and beta levels, an effect size of d = 0.66 is expected to be detected. Both power analyses assumed two-tailed critical t-values and are each conservative estimates. The between-groups factor of the experiment was social equity or fairness as reflected by the average

payoff in five rounds of the Ultimatum Game (UG), where participants in the “control” condition were given offers that were nearly fair on average, and participants in the “experimental” condition were given offers that were unfair on average. The UG is a standard scenario from game theory in which one player, the proposer, is endowed with some endowment of value and makes an offer from that endowment to the other player, the responder, about how to split the endowment. If the responder accepts the offer, then the endowment is split according to the offer, but if the offer is rejected then both players receive nothing. While it has been traditionally argued by game theorists that it is rational for the responder to accept any non-zero offer (Von Neumann & Morgenstern, 1944/2007), offers that are perceived to be unfair are often rejected (Rand et al., 2013). This is often interpreted as a form of punishment for the proposer’s selfishness.



3.2.2. **Procedure**: Participants provided their written informed consent as outlined in the SFU REB approved experimental protocol (#20180452) and were then instructed to provide saliva which was immediately placed in a freezer by the participant. Once the initial saliva sample for baseline hormone measurement was collected, participants were brought to the testing room where they were instructed to follow computer prompts that explained the UG. When the participant indicated that they understood the UG they played five rounds on the computer in either the high (near fair) or low (unfair) payoff conditions. The endowment of the proposer, a research assistant in another room, in each round was 10 points, where an offer of four was considered high or nearly fair and an offer of one was considered low or unfair. Participants were incentivized to maximize their payoffs with the expectation that each point they acquired would translate into a ballot that would be entered into a lottery with other participants and the researchers for a chance to win a gift certificate valued at $100. Once the UG rounds were done the participants completed the biometric and psychometric measurements, followed by the Ellsberg Urns task.

3.2.1. Ellsberg Urns: A computer version of the Ellsberg Urns task (Ellsberg, 1961; Danese et al., 2017) was given to participants in which they were asked to provide a "sell price" for an opportunity to draw a marble first from a "risk" urn, and then another sell price to draw a marble from an "ambiguity" urn, where the urns appeared to participants on the computer screen as images of black bags. Participants were instructed that the risk urn contained 10 marbles composed of exactly five white and five black marbles and that if they drew a marble of the colour they had been assigned then they would win 15 ballots for the same lottery described for the UG. Participants were also instructed that the sell price they indicated for each urn could be accepted by the researcher (research assistant), in which case the sell price would be the number of ballots they received. The expectation of drawing an assigned marble colour is 0.5, which corresponds to a sell price of 7.5 in the risk bag and is completely unknown in the ambiguity bag, Therefore, sell prices of 7 or lower are indicative of risk aversion, and sell prices of 8 or higher are indicative of risk seeking. The difference between the sell price for the ambiguity urn and the risk urn reflects the participant's *ambiguity premium* (Danese et al., 2017), or the direction and magnitude of change in their second-order- compared to first-order risk preferences.

3.2.2. Hormones: two saliva samples were collected from each participant, one immediately after participants gave informed consent to participate in the experiment, and one twenty minutes after they completed the last round of the UG. All experimental sessions, including the collection of saliva samples, were done between 9am-1pm to control for the effects of circadian rhythms on steroid hormone concentrations (Czeisler, & Klerman, 1999). For each sample the participants were instructed to provide at least 2mL of saliva via a passive drool into a 15mL plastic centrifuge test tube. Once they reached the 2mL mark the participant immediately sealed and placed the tube into a freezer for storage until the samples could be analyzed for steroid hormone concentrations. Hormone analysis was conducted by Salimetrics Inc. (Carlsbad CA) using enzyme-linked immunosorbent assays (ELISA) (Lequin, 2005). Each sample was analyzed for testosterone, cortisol, and the female participants' samples were analyzed for estradiol.

3.2.3. Psychometric measures: several psychometric questionnaires were filled out by participants to collect data described in previous literature as mediating or moderating the relationship between steroid hormones and risk-taking propensity. The

questionnaires collected data about: i) hubristic and authentic pride (Tracy et al., 2009), which has been associated with positive emotions, social status, and testosterone (Tracy et al., 2010; Martens et al., 2011; Verbeke et al., 2018) ii) self-construal (Hardin et al., 2004), which mediates testosterone's effects on aggression and risk (Welker et al., 2017; Welker et al., 2019); iii) social dominance orientation (Pratto et al., 1994), which has been shown to be a relevant factor in mediating the effect of testosterone to competitive defeats (Maner et al., 2008); and iv) big-five personality dimensions (Gosling et al., 2003), as extroversion is associated with higher testosterone during male mating efforts (Alvergne et al., 2010), and conscientiousness has a weak negative correlation with testosterone (Sundin et al., 2021). A final questionnaire was administered which collected demographic and other miscellaneous information that was considered relevant to the research questions (Appendix A).

3.3. **Results**

All analyses were performed using SPSS 27 for Windows.

3.3.1. **Descriptive Statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
T0	61	33.96	398.90	152.2408	104.01485
T1	61	30.60	380.41	155.0059	92.53091
E0	38	.48	1.95	1.2255	.36610
E1	38	.27	2.19	1.3318	.38143
C0	61	.08	.91	.3227	.19089
C1	61	.05	.70	.2762	.15387
sellprice1	58	1.00	15.00	8.1207	4.04396
sellprice2	58	1.00	15.00	8.5000	3.97470
AmbiguityPremium	58	-5.00	9.00	.3793	1.89021

Table 3.1. Descriptive statistics for endocrine and behavioural data. Pre-task testosterone (T0); post-task testosterone (T1); pre-task estradiol (E0); post-task estradiol (E1); pre-task cortisol (C0); post-task cortisol (C1); participants' sell price for risk decisions (sellprice1); participants' sell price for ambiguity decisions (sellprice2)

3.3.2. **Behavioural measures**: A between-subjects t-test was first performed to confirm the effectiveness of the experimental manipulation of social facilitation (high initial UG offers) or provocation (low initial UG offers). The t-test included the entire pooled sample (males and females) and showed a significant difference between these groups, with participants in the “high offers” or social facilitation group accepting more offers and participants in the “low offers” or social rejection/provocation group rejecting more offers ($t_{(71)}$ = -2.585; p = 0.012). The experimental manipulation, therefore, can be regarded as successful and the experimental condition can be reliably used for further analyses.

The behavioural measures of risk, ambiguity, and the ambiguity premium as reflected in the sell prices for the risk and ambiguity bags and the ratio between them, were then analyzed by between-subjects t-tests. No differences were found between experimental conditions in any of these behavioural measures alone ($t_{(71)}$ = 0.137, p = 0.89; $t_{(71)}$ = 0.931, p = 0.36; $t_{(71)}$ = 1.624, p = 0.16). However, the main experimental hypotheses involved the inclusion of endocrine measures. Therefore, the next set of analyses included the prospective influence of baseline testosterone, estradiol, cortisol, and their condition-specific percentile change on the behavioural measures.

3.3.3. **Endocrine measures**: After splitting the sample by sex, one-way ANOVAs were used to establish that the baseline levels of testosterone, estradiol, and cortisol did not differ between experimental conditions, and this was confirmed with no baseline hormone measure being significantly different between conditions. Next, the hypothesis that the experimental manipulation would lead to a differential change in hormones was tested, where the sample was again split by sex. There are two reasons for splitting the sample by sex rather than using sex as a between-subjects factor. The first is that the means and variance for testosterone are known to be quite different in males and females, and the second is that the different hormone concentrations and sex steroid receptor distributions underlie distinct psychophysiological functions between the sexes. For the analyses each hormone served as the within-subjects factor and condition served as the between-subjects factor in mixed-model ANOVAs. For testosterone, females had an increase for both conditions ($F_{(1, 36)}$ = 20.34, p < 0.001; figure 3.2) while males did not show a difference in either condition ($F_{(1,20)}$ = 3.58, p = 0.073); figure 3.2). For estradiol, females also showed an increase in both conditions ($F_{(1,36)}$ = 10.37, p = 0.003; figure 3.3), and estradiol was not measured in males. For cortisol, females showed an overall decrease in both conditions ($F_{(1,36)}$ = 9.70, p = 0.004; figure 4) but did

not show an interaction by condition, while males did not show a difference in either condition ($F_{(1,20)}$ = 1.77, p = 0.198; figure 3.4). ANOVA summary tables can be found in Appendix C.

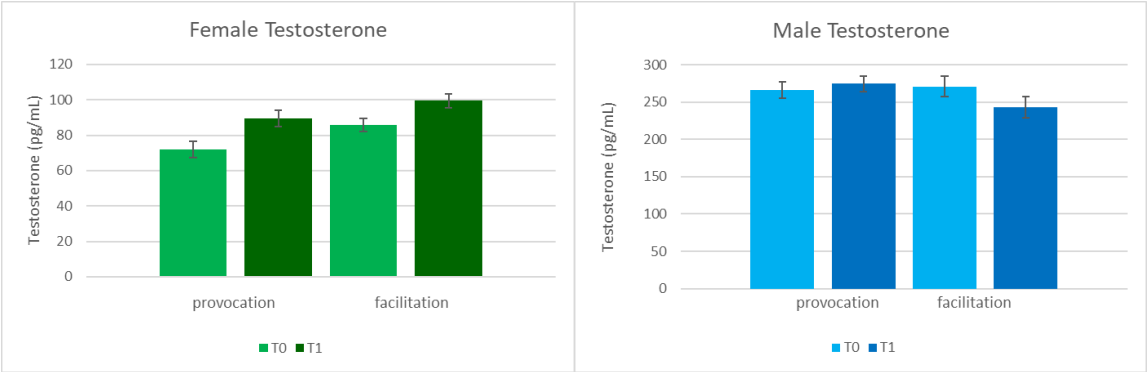


Figure 3.2 – Testosterone measures for females (left) and males (right) in both social provocation and social facilitation conditions. Female testosterone increased in both conditions, while no changes in testosterone were observed in either condition for males.

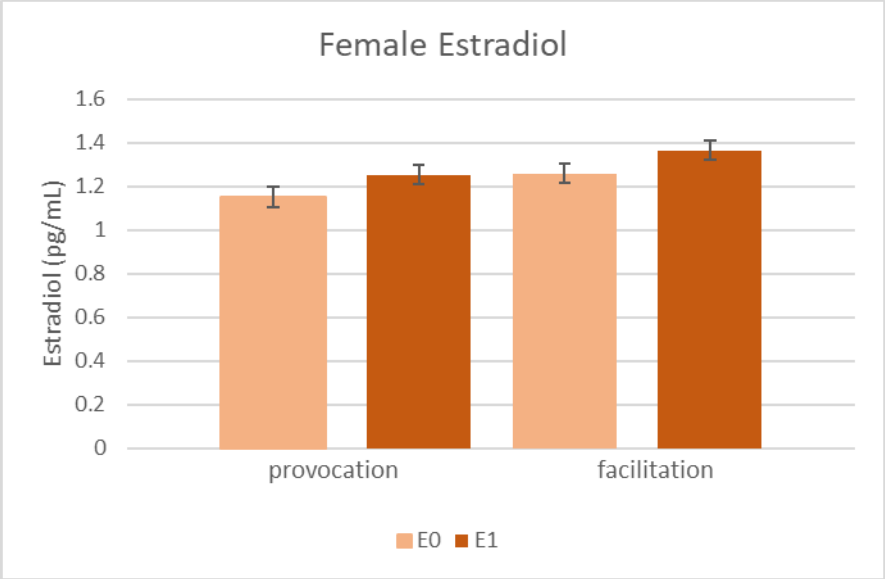


Figure 3.3 – Female estradiol increased in both social provocation and social facilitation conditions.

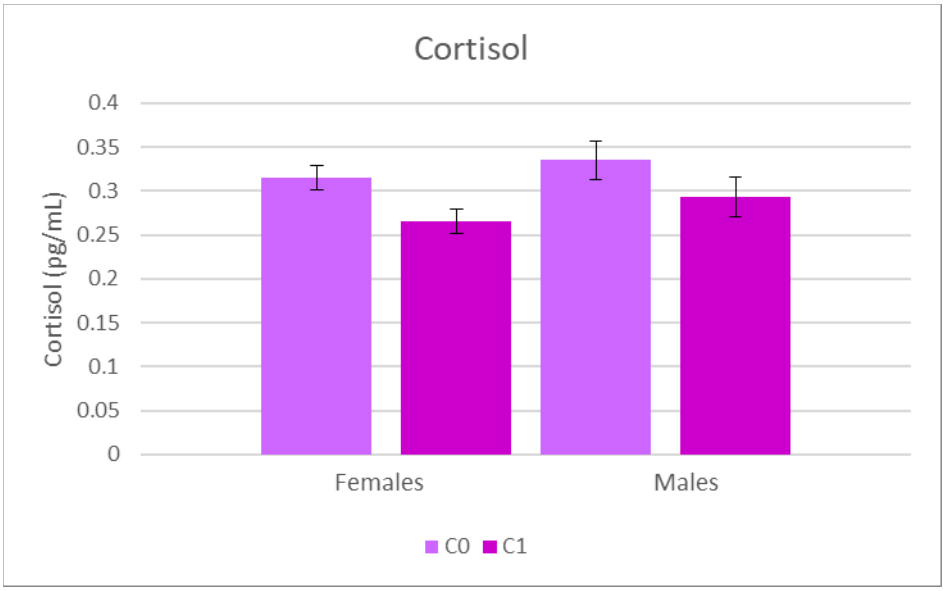


Figure 3.4 – Cortisol measures for females and males for combined social provocation and facilitation conditions. A statistically significant decrease in cortisol was seen in females in both conditions, but males did not show a change in cortisol in either condition.

The behavioural data were re-examined in light of these observed hormonal differences using linear regression analyses with ambiguity premium as the outcome

variable and baseline estradiol and estradiol reactivity as the predictor variables. The model showed an effect of baseline female estradiol on decisions for ambiguity (B = -5.475, t = -3.017, p = 0.007) and the ambiguity premium (B = -1.633, t = -3.096, p = 0.006) in the social facilitation condition, but no effect in the social provocation condition. The model also showed an effect of estradiol percent change on ambiguity premium (B = 3.132, t = 3.710, p = 0.002; figure 3.5) in the social facilitation condition but not for the social provocation condition. No effects were found on regressions on behavioural measures for baseline cortisol or percent cortisol change for either sex, in either condition.

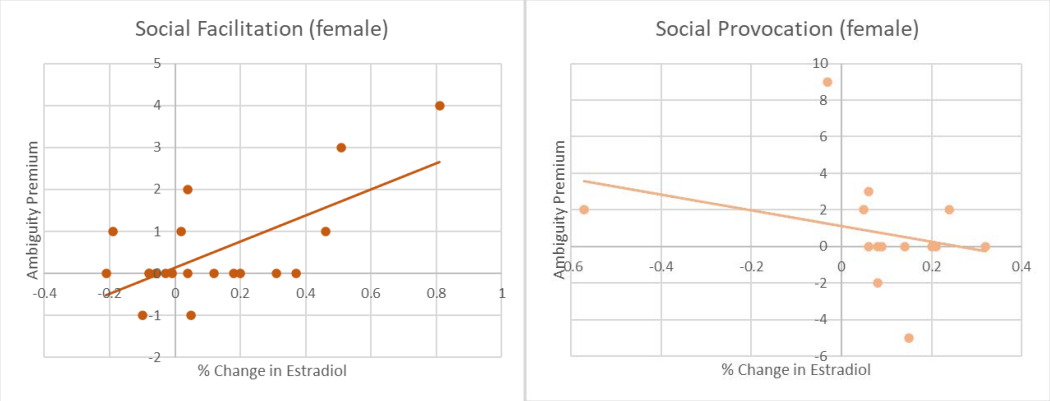


Figure 3.5 – Percent change in female estradiol predicts increases in the ambiguity premium in the social facilitation condition but not the social provocation condition.

3.4. **Interim Discussion**

To summarize the findings, the experimental condition of social facilitation versus social provocation led to a difference in the acceptance rates of UG offers, as expected. The experimental factor also led to condition-specific changes in sex steroids. Female testosterone and estradiol increased, and cortisol decreased, in both conditions, whereas male testosterone decreased but only in the social facilitation condition and no change in male cortisol was observed. Changes in female estradiol were significant in predicting participants' ambiguity premium, but in opposite directions between experimental conditions. Increases in estradiol predicted increases in the ambiguity premium after social facilitation, the opposite pattern emerged in the social provocation condition.

Sex differences in risk taking scenarios are often studied through the lens of behavioural economics and have been characterized in various ways over several decades of research (Arch, 1993; Cross et al., 2011; Pawlowski et al., 2008; Charness & Gneezy, 2012; Friedl et al., 2020). While there are many nuanced facets to decision making under risk and ambiguity, the weight of evidence in the literature demonstrates that sex differences exist. For example, males reliably take more health and financial risks than females on average, including physical aggression and gambling. Males also tend to have a higher propensity for sensation-seeking, which can be related to these and other behavioural domains of risk. What has not previously been characterized are sex-specific biological mediators or modulators of subjective thresholds for decisions under risk and ambiguity. The present study explored this relationship using a common experimental paradigm from behavioural economics, the Ellsburg Urns (Ellsberg, 1961, Danese et al., 2017), but with an important prior manipulation: participants were either socially “provoked” or “facilitated” by being offered relatively fair or unfair offers in the UG prior to the Ellsburg Urns task. Results indicated that there were no significant differences in risk, ambiguity, or ambiguity premium measures between conditions or sexes when analyzed in isolation, but that several significant results were apparent when baseline and hormonal change measures were included.

While there were no apparent differences between experimental conditions in hormone concentration changes, there were several distinct changes that were seen for females but not for males when the conditions were collapsed. For females, both testosterone and estradiol increased between baseline and post-task measures, while cortisol decreased. For males there were trends toward significance in both testosterone and cortisol change, but only in the social facilitation condition. The lack of significance may have been a result of relatively low statistical power for males compared to females. If this is the case, then it would likely reflect a sex difference in the way that hormones respond to simple social provocation versus facilitation. However, the primary hormonal finding here remains the condition-independent increase in sex-steroids and decrease in cortisol for females. Since the experimental condition had no effect on these changes, it is difficult to interpret their cause or function. The possibility that these significant changes reflect typical circadian or pulsatile cycling of hormones (Czeisler & Klerman, 1999; Herbison, 2018) is immediately challenged by the fact that male participants did not show comparable changes. Another possibility is that these changes reflect a

generalized response to one or more aspects of the experimental protocol, but this possibility begs the question of which aspects were necessary or sufficient to generate the significant hormonal changes. Two aspects of the experimental protocol correspond to behavioural domains that have previously been shown to show sex differences relating to hormone functions.

The first is the mere requirement that participants engage in tasks that involve economic risk and ambiguity (both the UG and Ellsberg Urns). Stanton et al. (2011) reported that both males and females with especially low and high levels of testosterone tended to be risk, ambiguity, and loss neutral compared to the more typical risk, ambiguity, and loss aversion seen in individuals within +/- 1.5 SD of the mean for their sex. It is possible that the increase in testosterone and estradiol in female participants in the present study reflects a kind of psychophysiological risk homeostasis, but if this were the case then the direction of the effect (i.e., whether those low in sex-steroids show an especially strong increase to prime a conservative approach to risk, or whether those in the mid-range show an increase to prime risk-seeking behaviours) would have to be explored in a follow-up study.

The second aspect is social interaction or social cognition (Proverbio, 2021; Paletta et al., 2022). While sex differences have been reported for a variety of perceptual, affective, cognitive, and behavioural outcomes, one that is especially pertinent for interpreting the present results involve bargaining behaviour, where administration of exogenous testosterone has been shown to increase fair bargaining behaviour in women (Eisenegger et al., 2010). This suggests a possible functional role for the increase in testosterone, and perhaps a similar role for estradiol, seen in the present study. Under this scenario, the increase in sex steroids may reflect an attempt to promote a behavioural tendency toward social fairness in the UG regardless of condition (provocation or facilitation). It is not possible to resolve whether such a mechanism is influenced more by more distant evolutionary or proximal socialization considerations, and this interpretation is also compatible with the risk-management interpretation proposed above. Future studies are necessary to distinguish between these various possibilities.

Finally, neither baseline nor change in testosterone significantly predicted any behavioural measures, including the ambiguity premium for males. Conversely, both

baseline and the change in female estradiol in the social facilitation condition were predictive of the ambiguity premium. One reason for this difference in the effects (or lack thereof) of sex-specific sex steroids on the ambiguity premium may simply be a difference in sample size between males and females, which complicates any hormonal and behavioural comparisons between the sexes. This possibility is supported by previous literature of the effects of male testosterone on risk taking (Stanton et al., 2011; Apicella et al., 2015; Stanton et al., 2021), even in the Ellsberg Urns task (Danese et al., 2017). Another reason for the difference may be a genuinely distinct sex-specific use for testosterone and estradiol in social cognition and subjective thresholds for economic risk and ambiguity. This possibility is supported by the present data and would imply that in instances of relative social facilitation, estradiol serves to decrease women's preference for known odds, or rather increases their preferences for higher-order risk (i.e., ambiguity). This would mean that when it comes to decision making under ambiguity, women respond to estradiol similarly to the way men have been widely shown in the literature to respond to testosterone.

Chapter 4.

Differential Sex Hormone Responses to Fair versus Unfair Offers in the Ultimatum Game, in Women

4.1. **Introduction**

There is often an element of risk involved in decision making, and that can be especially true for decisions in social contests (Frith & Singer, 2008). A considerable amount of research in psychology and behavioural economics has explored decision making in a variety of game theoretical paradigms showing that humans are not strictly rational self-interested agents, but often behave in ways that can undermine self-interest in either anti- or prosocial ways (Henrich et al., 2001; Urbina et al., 2019). One simple example is the Ultimatum Game (Sanfey et al., 2003), which involves two game players. The first player is a proposer with some initial endowment who makes an offer to split the endowment with the second player, the responder. The responder can decide to either accept or reject the offer, where an acceptance leads to the proposed split of the endowment and a rejection leads to both players losing the entire endowment. While it is in the narrow self-interest of the responder to accept all positive offers, however unfair they may be, there are other considerations and factors that contribute to the more frequently observed decision of typical responders to reject most unfair offers (Van't Wout et al., 2006; Emanuele et al., 2008; Burnham, 2007; Van Honk et al, 2012; Peterburs et al., 2017).

One important factor that influences decisions in UGs is sex (Solnick, 2001; Croson & Gneezy, 2009, Charness & Gneezy, 2012; but see Nelson, 2016), where male responders are typically offered more than female responders, and responders of both genders accept lower offers from male compared to female proposers. There is also evidence for an effect of offer frame (responder "gives" vs. "takes") that interacts with gender to differentially affect men's but not women's autonomic responses (Sarlo et al., 2011). Additionally, endocrine status has been shown to influence economic decision making, including in the UG (Burnham, 2007; Zethraeus et al., 2009; Van Honk et al., 2012; Kopsida et al., 2016; Inoue et al., 2017). However, while there is substantial evidence for an effect of testosterone on behaviour of men in the UG in both the

proposer and responder roles, there is little comparable research on endogenous estradiol in women. Since there is an empirical basis to suppose that endogenous estradiol influences female decision making in the UG (Eisenbruch & Roney, 2016; Coenjaerts et al., 2021), the present study sought to explore and compare the role of male testosterone and female estradiol and testosterone in UG decisions.

4.1.1. *Estradiol and decision-making*

While there is substantial evidence for an effect of testosterone on behaviour of men in the UG in both the proposer and responder roles, there is little comparable research on endogenous estradiol in women. However, there is an empirical basis to suppose that endogenous estradiol influences female decision making in the UG (Stanton, 2017). For example, when placed in same-sex pairs, females who were in the luteal phase (especially late luteal phase) of their menstrual cycles were more likely to reject low offers (Eisenbruch & Roney, 2016), and estradiol typically increases over the luteal phase and peaks just prior to ovulation. Additionally, while exogenous estradiol increases men's acceptance of fairly framed UG offers, it decreases acceptance offers in women (Coenjaerts et al., 2021). Finally, the gene length for different alleles of the β estrogen receptor was found to be related to the threshold for offer acceptance in both Chinese and Israeli participants, where longer alleles were associated with a higher minimum offers to be accepted (Chew et al., 2013). Therefore, because there is sufficient theoretical and empirical rationale, the present study sought to explore and compare the role of male testosterone and female estradiol and testosterone in UG decisions.

4.1.2. *Research questions*

There are primary and secondary research questions in this study for both endocrine and behavioural outcomes. The main between-subjects experimental factor was average fairness in UG offers, with offers being made by research assistants and study participants playing the role of the responder. In the first condition participants were made low or unfair offers. Since the proposer started each game round with an endowment of 10 tokens, the unfair offers were nearly all 1 or 2 tokens, with some fewer offers of 3 to keep the participants cognitively engaged. In the second condition participants were made relatively fair offers, which were nearly all 4 or 3 tokens, with

some fewer offers of 2 for symmetry with the first condition. This cutoff was chosen because it has been previously demonstrated that approximately half of UG participants will reject offers below 30% of the proposer's endowment (Nowak et al., 2000). No perfectly fair offers of 5 were made as they are expected to be accepted at very high rates and offer little variability in behavioural data. The primary endocrine research questions were, i) do unfair offers induce a distinct steroid hormone profile change compared to fair offers?; and ii) is there a sex difference in hormone change as a result of unfair versus fair UG offers?

There were also some distinct behavioural hypotheses that were explored. The primary behavioural research questions were, i) are acceptance rates of unfair and fair UG offers differentially influenced (predicted) by baseline sex steroid levels in males and females?; and ii) would any experimentally induced changes in sex steroid levels predict acceptance rates for unfair and fair offers? Finally, the framing of the UG was novel in that participants were instructed that the sum of all tokens received in all rounds by the proposer (research assistant) would subsequently be used in a gambling game whose payoff (additional ballots in a lottery) would be split equally with the participant. This gave the game a minimal tournament structure which I called the "hierarchical ultimatum game" (HUG). It was hypothesized that responses to HUG offers would have a higher average acceptance rate than the classic UG because the HUG offers an additional cooperative element to the game between the proposer and responder by adding a competitive element with other game player pairs. This kind of intergroup competition is known to increase intragroup cooperation (Nowak, 2006), and cooperation in this context would mean an increase in acceptance rates for both fair and unfair offers.

4.2. **Methods**

4.2.1. **Subjects**: Undergraduate participants (N = 71; 50 female) were recruited from the SFU Psychology Department's research participation system (RPS) to participate in a single-factor between-subjects experiment. A power analysis for within-subjects' effects was performed using G*Power (Faul et al., 2009). With an alpha level of 0.05, an effect size of d = 0.41 and d = 0.65 is expected to be detected with a beta level of 0.2 for females and males, respectively. Both power analyses assumed two-tailed critical t-

values and are each conservative estimates. The between-subjects factor of the experiment was social equity of fairness as reflected by the average payoff in thirty rounds of the HUG, where participants were assigned to one of two groups. Participants in the first group received almost exclusively unfair offers, and participants in the second group received almost exclusively near-fair offers.

4.2.2. **Procedure**: Participants provided their informed consent as outlined in the SFU REB approved experimental protocol (#20180451) and were then instructed to provide ~2mL of saliva, which was immediately placed in a freezer by the participant. Once the initial saliva sample for baseline hormone measurement was collected, participants were brought to the testing room where they were instructed to follow computer prompts that explained the HUG. When the participant indicated that they understood the HUG they played thirty rounds in either the low (unfair) or high (near fair) payoff conditions. The endowment of the proposer (a research assistant) in each round was 10 points, where an offer of four was considered high or nearly fair and an offer of one was considered low or unfair. Participants were incentivized to maximize their payoffs with the expectation that each point they acquired would translate into a ballot that would be entered into a lottery with other participants and the researchers for a chance to win a gift certificate valued at $100. Once the HUG rounds were done the participants completed the biometric and psychometric measurements, and a second saliva sample was collected approximately 20 minutes after the end of the last HUG round.

4.2.2.1 Hormones: two saliva samples were collected from each participant, one immediately after participants gave informed consent to participate in the experiment, and one twenty minutes after they completed the last round of the UG. All experimental sessions, including the collection of saliva samples, were done between 1pm-5pm to control for the effects of circadian rhythms on steroid hormone concentrations (Czeisler, & Klerman, 1999). The twenty-minute delay is required to allow time for circulating hormone changes to be detected in saliva (Kirschbaum & Hellhammer, 1994). For each sample the participants were instructed to provide at least 2mL of saliva via a passive drool into a 15mL plastic centrifuge test tube. Once they reached the 2mL mark the participant immediately placed the tube into a freezer for storage until the samples could be analyzed for steroid hormone concentrations. Hormone analysis was performed commercially by Salimetrics Inc. (Carlsbad, CA) using enzyme-linked immunosorbent

assays (ELISA) (Lequin, 2005). Each sample was analyzed for testosterone, cortisol, and the female participants' samples were analyzed for estradiol.

4.3. **Results**

All analyses were performed using SPSS 27 for Windows.

4.3.1. **Descriptive Statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
T0	69	33.18	463.88	140.0239	100.02933
T1	69	25.06	407.17	131.3470	89.76286
E0	47	.75	2.43	1.3468	.35120
E1	47	.69	2.29	1.4411	.40028
C0	68	.05	.77	.2411	.14557
C1	68	.04	.69	.2086	.11807
Avg_Decision	67	.07	1.00	.5796	.27646
A_given_2	67	.00	10.00	3.9254	3.64448
A_given_3	67	.00	11.00	4.1642	2.57374

Table 4.1. Descriptive statistics for endocrine and behavioural data. Pre-task testosterone (T0); post-task testosterone (T1); pre-task estradiol (E0); post-task estradiol (E1); pre-task cortisol (C0); post-task cortisol (C1); participant mean decision where reject=0 & accept=1 (Avg_Decision); number of accepts for offers of 2 (A_given_2); number of accepts for offers of 3 (A_given_3).

Behavioural measures: A pair of two-factor ANOVAs were done to establish behavioural differences between the groups. As the only meaningful overlap between the conditions were the offers of two or three tokens, two tests were performed to see if their acceptance rates differed by condition. The first ANOVA tested the factors of sex and condition for their effect on acceptance rate for offers of two. Results indicated that there was no interaction between sex and condition on the rate of acceptance for offers of two tokens ($F_{(1,62)} = 0.004$, $p = 0.95$). However, there was a significant effect of condition ($F_{(1,65)} = 3882.54$, $p = 0.01$) with both males and females accepting more offers of two tokens when they were in the "unfair offers" condition. This suggests that the

manipulation was effective in that participants differentially renormalized their acceptance thresholds for what kinds of offers were considered fair between the conditions. Additionally, there was a significant effect of sex ($F_{(1,65)} = 331.75$, $p = 0.035$), where females were more likely to reject offers of two than males. The second ANOVA did the same thing for offers of three. There was no interaction between condition and sex on acceptance rates of offers of three, nor were there significant main effects in the acceptance rate for offers of three between conditions or between sexes.

A two-factor ANOVA revealed that there was also no significant difference in the overall average acceptance rate between conditions or between sexes, indicating that the increased acceptance rates for lower offers of two tokens in the unfair condition compensates for the overall lower offers made in that condition. Lastly, the hypothesis that HUG offers would be rejected less frequently than UG offers was tested with an independent samples t-test, and no significant differences were found. Therefore, the HUG can be interpreted here comparably to the classic UG.

Endocrine measures: The first hypothesis that the overall fairness in offers would affect hormone levels was tested using a two-factor ANOVA was done to test the between-subjects effect of the condition (unfair vs. fair) and sex on the percent change in testosterone, estradiol, and cortisol. For testosterone the analysis revealed a significant interaction ($F_{(1,61)} = 10.373$, $p = 0.002$), where males showed no change in testosterone in either condition, but females showed a decrease in testosterone in the unfair condition (figure 4.1). For estradiol there was a significant effect of condition ($F_{(1,42)} = 5.99$, $p = 0.019$), where females showed an increase in estradiol only in the fair offers condition (figure 4.1). For cortisol there was no significant interaction between sex and condition ($F_{(1,60)} = 1.59$, $p = 0.212$), and no main effects of either sex or condition either. ANOVA summary tables can be found in Appendix D.

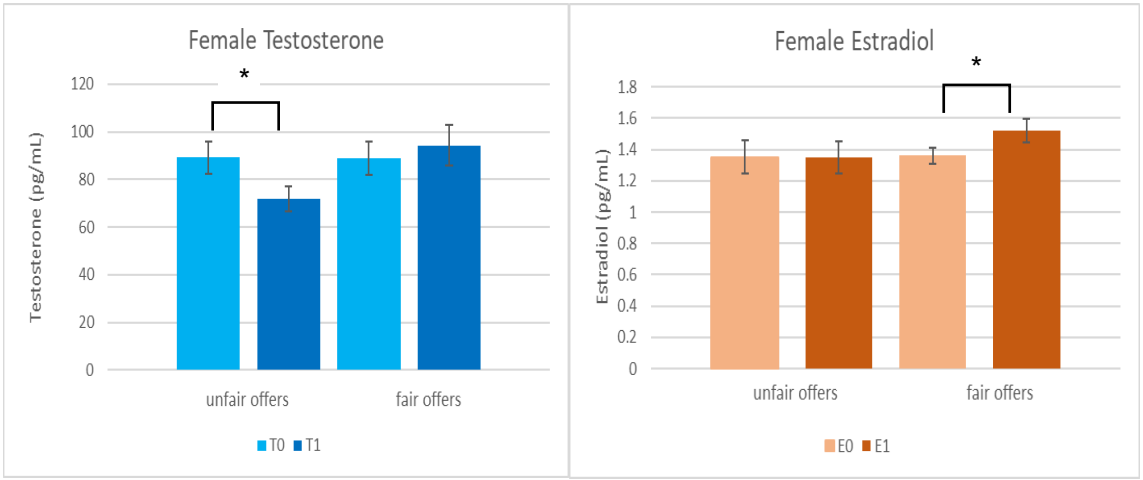


Figure 4.1 – Sex steroid changes in females are contingent on experimental condition. Female estradiol (left) increased in response to fair offers, while female testosterone (right) and cortisol (not shown) decreased in response to unfair offers. (T0 = baseline testosterone; T1 = post-task testosterone; E0 = baseline estradiol; E1 = post-task estradiol). Asterisks show significant differences at $p < 0.05$.

Having established that there was an effect of overall unfair verses fair offers on female testosterone and estradiol, the behavioural data were analyzed with linear regression analyses to test the hypothesis that hormone levels influence participants' response to accept or reject UG offers. Therefore, in the first analysis, baseline hormones were input as predictor variables and average acceptance rate was input as the outcome variable. There were no significant effects of baseline hormones on male or female average acceptance rates in either unfair or fair conditions. A similar subsequent analysis was done with response ratios for offers of two and three as outcome variables (ratios being used to control for differences in the number of offers of twos and threes in each condition). The baseline cortisol was a significant predictor of the acceptance rate for offers of three (but not for offers of two) for females when in the "unfair offers" condition ($F_{(1,17)} = 5.303$, $p = 0.012$), but not in the "fair offers" condition ($F_{(1,25)} = 1.266$, $p = 0.312$). That is, higher baseline cortisol was associated with lower rates of acceptance

for offers of three tokens (figure 4.2). No other baseline hormone predicted response offers for either sex in either condition. Another analysis was done using the percent changes in each of the three hormones, and it was revealed that testosterone percent change was a significant predictor of the acceptance rate for offers of three tokens (but not for offers of two tokens) for females in the “unfair offers” condition ($F_{(1,18)} = 5.545$, $p = 0.03$), but not in the “fair offers” condition (figure 4.2). No other change in hormones predicted acceptance rates.

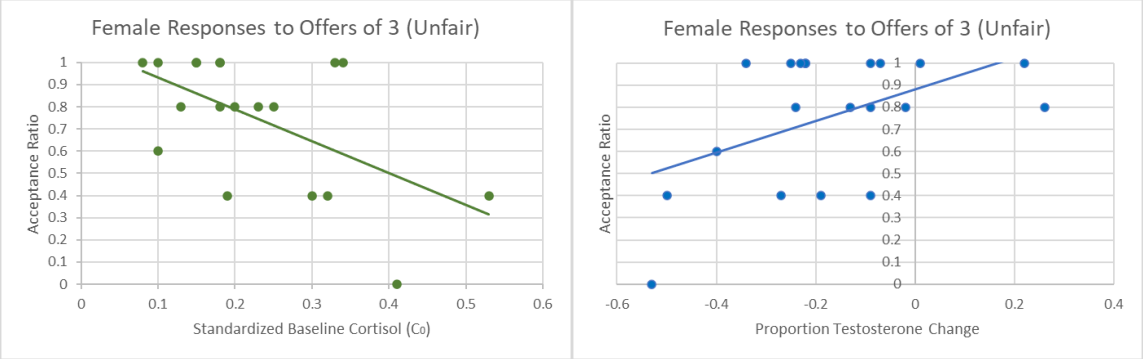


Figure 4.2 – Hormone state predicts the rate of offer acceptance for females. Female participants in the “unfair offers” condition, baseline higher baseline cortisol was associated with a reduced acceptance ratio, while changes in testosterone positively predicted the acceptance rate for offers of “3”.

4.4. Interim Discussion

To summarize the findings, males generally rejected more unfair offers compared to females regardless of condition, but males did not show a change in testosterone for either condition. Conversely, females showed changes in testosterone and estradiol in opposite directions in the unfair offers condition, where testosterone decreased but estradiol increased. The change in estradiol did not seem to affect acceptance rates, but female participants who were higher in baseline cortisol tended to show lower acceptance rates in the unfair offers condition, and the degree of female testosterone increases tended to reflect higher acceptance rates in the unfair offers condition.

There is a large literature on cognitive and behavioural sex differences, and previous research strongly indicates that overall, females are more risk- and loss-averse than males (Charness & Gneezy, 2012; Bouchouicha et al., 2019). There is also a relatively large body of work that implicates testosterone in both male and female decision-making under risk (Carre & Archer, 2018; Eisenegger et al., 2010), but comparatively little research published about the effects of endogenous estradiol on female decision-making (Kurath & Mata, 2018). The present study sought to replicate previous work on male testosterone and bargaining behaviour in the UG and extend the scope of the published research to include how endogenous estradiol might differentially affect female UG bargaining toward relatively fair and unfair proposers.

Results indicated that for males there was no change in testosterone for participants in either the condition. This result is unexpected since previous work has reported an effect of male testosterone on exactly this type of decision-making. However, the present study perhaps simply did not have the statistical power to detect a genuine difference. As is the case for most psychology experiments that rely on undergraduate participation, the present study much more readily recruited female participants, while male participation was much lower by comparison. Therefore, considering relatively consistent previous work mentioned above, it would seem prudent not to make any definitive claims about the relationship between testosterone and male decision-making in the present experimental setup.

While it is tempting to suspect that testosterone levels decreased in females in the “unfair offers” condition in conjunction with an increase of cortisol since female testosterone is primarily produced in the adrenals, there was also a significant decrease in female cortisol in the “unfair offers” condition. This rules out a hormonal trade-off interpretation of this decrease in testosterone, and suggests a distinct sex-specific function for testosterone in females compared to males when engaged in bargaining behaviour. While previous research (Derntl et al., 2014) indicates that testosterone may not play a significant role in female decision-making under risk, changes in testosterone in the present study did predict acceptance rates of 3 tokens in the “unfair offers” condition, suggesting that the female drop in testosterone does serve to influence adaptive responses to social slights, but in the opposite direction from males. Conversely, a novel finding of the present study was a significant increase in female estradiol in the “fair offers” condition, while estradiol remained constant on average in

the “unfair offers” condition. This condition-specific change in estradiol did not, however, predict any differences in the acceptance rates for UG offers. Therefore, while this finding is novel, its functional significance is uncertain, and may involve physiological or other cognitive domain-specific changes in behaviour. This possibility should be explored in future research, and a broader contextualization of these results will follow in the general discussion.

Chapter 5.

General Discussion

5.1. Summary and General Interpretation

5.1.1. Summary of Results

Taken together there are both converging and diverging themes that emerge in the present studies relating to the relationship between steroid hormones and decision making under risk. These themes can be viewed through the behavioural and physiological lenses, and it will be instructive to take both perspectives to sufficiently unpack the results. In that regard, the structure of this initial summary will be to first outline the influence that the various experimental manipulations had on steroid hormone concentrations for participants in each study, and then to outline the variable relationships that were observed between baseline and dynamic hormone levels and behaviour, especially where hormone levels can be said to influence economic decision making directly and differentially.

The primary experimental manipulations and behavioural outcome variables for the three studies were: i) whether outcome feedback for simple gambles were framed as being decided by a person (social condition) or an algorithm (non-social condition) differentially affected betting behaviour; ii) whether “provocative” ultimatum game offers (unfair condition) versus “facilitative” ultimatum game offers (fair condition) affected economic decision making under risk and ambiguity; and iii) whether low (unfair) versus high (fair) ultimatum game offers affected decisions on comparable offers. The influence of each experimental condition on the steroid hormones of testosterone, estradiol, and cortisol, and the reciprocal effect that these hormones had on behavioural outcomes, will be outlined below. However, even aside from hormone dynamics there were some notable results in two of the three studies. In study 1, females in the “social” condition made fewer “high” bets, and in study 3 there was a general tendency for participants to accept more offers of “2” (from a proposer’s endowment of 10) when they were in the “unfair offers” condition compared to the “fair offers” condition. However, while statistically significant, these hormone-independent results are relatively minimal, and it

is difficult to extrapolate a unifying general interpretation for them. Therefore, the hormone results will be summarized next.

The main hormonal findings will be summarized sequentially like the behavioural results above. In the first study there were sex- and condition-specific changes relating to all three measured steroid hormones. While sex steroids displayed no change for both sexes in the “social” framing condition of the simple gambling task, female estradiol and male testosterone decreased in the non-social condition. Female testosterone showed no difference in the non-social condition and male estradiol was not measured. Additionally, cortisol decreased for males in both conditions, but only in the non-social condition for females. Therefore, given the fact that the weight of findings leans more toward a general decrease in steroid hormone levels, it might be more sensible and appropriate to emphasize the instances in which hormone levels were maintained. This interpretation will be upacked in the next section. In addition, low baseline female estradiol was associated with larger initial bets for both conditions, and the lack of a difference between conditions is not surprising given that there is no feedback prior to the first bet.

In the second study there were no significant hormone-independent results, meaning that participants in the “provocation” (unfair UG offers) and “facilitation (fair UG offers) showed no differences in risk, ambiguity, or the ambiguity premium measures of the Ellsberg Urns task when behavioural measures were considered alone. In contrast, the females in both conditions showed an increase in testosterone and estradiol, and a decrease in cortisol. Males showed no change in testosterone or cortisol between pre- and post-task measurements, although there was less statistical power to observe potential effects in males. The most notable finding from study 2 was that, while no differences were observed between experimental conditions on either hormone levels or behavioural measures, both baseline estradiol and percent changes in estradiol in females, but not of testosterone, were predictors of the ambiguity premium such that higher baseline and increases in estradiol were associated with a higher ambiguity premium. This suggests that higher levels of estradiol increase ambiguity-aversion in the Ellsberg Urns task.

Finally, in the third study there were some notable effects of experimental condition on hormone levels for females, but not males. In the “low offers” (i.e., unfair

UG offers) condition there was an apparent decrease in both testosterone and cortisol for females, while in the “high offers” (i.e., fair UG offers) condition there was an apparent increase in estradiol. Unlike the previous two studies, female estradiol (both baseline and percent change) was not associated with UG acceptance rates in either condition. However, in both conditions there was a positive correlation between changes in female testosterone and acceptance of offers of “3”, while baseline female cortisol was negatively correlated with acceptance of offers of “3” in the UG. Therefore, with these results sufficiently summarized, they can now be discussed in combination and with an emphasis on where and how these results variously converge toward and diverge from the simplest unifying interpretations.

5.1.2. Integrative Synthesis

In the spirit of parsimony, it is important to ask what simple narrative(s) might account for the results of all the above studies when considered together, and how they might be comparably integrated with existing literature and theory. The latter will be expanded in 5.2., and in both cases there are multiple perspectives and approaches that can help in achieving simple and accurate interpretations of the results. This section will begin with a reminder of the most foundational principles of behavioural endocrinology, which will then increase in specificity until a natural match with the results of the present studies is achieved.

Let us first recall that there is a bidirectional or reciprocal relationship between hormones and behaviour such that differential hormonal baselines and dynamics, in a population and in individuals, can directly and indirectly affect nervous system functioning and therefore behaviour. Complementary to that, specific states and dynamics of individual perception, affect, cognition, and behaviour are able to influence endocrine function, including the concentration or density of hormones and their receptors throughout the body (Shulkin et al., 1994; Porges, 2021; Trofimova & Gaykalova, 2021). There are important evolutionary considerations for the specific patterns of interactions that exist between the nervous and endocrine systems which will be expanded on below, but it is sufficient for now to emphasize that the neural circuitry for social cognition and decision-making are most relevant. A highly influential model for the relationship between these mental processes was dubbed the “somatic marker hypothesis” of economic decision-making (Bechara & Damasio, 2005).

The somatic marker hypothesis was a relatively early attempt to integrate disparate research programs from behavioural economics, emotion, and cognitive neuroscience. The foundation of the model rests on the recognition that human decision-making is not done algorithmically by entirely rational self-interested individuals, as had been assumed in classical economic theory (Von Neumann & Morgenstern, 2007). Rather, it is more natural to assume that much of the bias and noise observed between simple economic decision models and empirical data of actual human choices is a result of systematic situational and dispositional factors, and that there is an inextricable link between "irrational" emotion and cognition. Some important components of the somatic marker hypothesis are that: i) decisions are often guided or influenced by emotions (somatic/physiological responses) regardless of whether the decision is related to emotion; and ii) emotions are induced by primary or secondary means, where the amygdala serves as a network hub node for primary induction and the ventromedial prefrontal cortex (vmPFC) serves as a network hub node for secondary induction. Secondary induction is considered as an initially cognitive process (e.g., thinking about an emotional state, or remembering an emotional event) that can trigger the activation of some emotional circuitry, which then serves as an influencing factor on cognition and decision-making (Bechara & Damasio, 2005). The basic premise of this model is widely accepted, and when considered in tandem with the bidirectional relationship between the endocrine and nervous systems, and that hormones are certainly a "somatic marker" of many emotional states (Van Wingen et al., 2011; Ter Horst et al., 2012) there is good a priori reason to assume that social perceptions, endocrine dynamics, and economic decision-making should be functionally intertwined as was investigated in the present studies.

To further ground this point in existing theory and existing literature, it should also be noted that the amygdala is central to not only emotional processes in the brain, but also social cognition (Adolphs, 2010; Bickart et al., 2014) where distinct nuclei within the amygdala are central in functional neural networks that are associated with social perception, social aversion, and social affiliation. Receptors for testosterone, estradiol, and cortisol have also been found throughout the diencephalon and telencephalon (Sarkey et al., 2008), which is sufficient to implicate these hormones in both emotion, social cognition, and decision-making. For example, androgen receptors are expressed in the human hypothalamus (Fernández-Guasti et al., 2000; Swaab et al., 2003),

amygdala (Manuck et al., 2010), hippocampus (Beyenburg et al., 2000), and cortex (Puy et al., 1995). The same can be said for estrogen receptors (Österlund et al., 2000; González et al., 2007; Azcoitia et al., 2011) and cortisol receptors (Perlman et al., 2007, Cao-Lei et al., 2013). Taken together, these findings strongly implicate these steroid hormones in neural circuitry for emotion, social cognition, and decision-making.

Therefore, with the contextual rationale for the studies being sound, it is pertinent to evaluate how the specific research hypotheses stood up to testing, and how the results might be integrated. The hypotheses of the first study were that i) social versus non-social framing of otherwise equivalent economic gambles would lead to differential hormone changes; and ii) that differences in hormone changes would predict the average magnitude of risks that participants were willing to make in simple betting. These hypotheses were based on previous literature that robustly implicates sex-steroids and cortisol in both social cognition (Paletta et al., 2022) and simple economic decision-making under risk (Schipper, 2015; Stanton, 2017). The second study replicated and extended previous work that showed a relationship between testosterone, cortisol, and both risk and ambiguity (Danese et al., 2017). There were two extensions of this work, with the first being the addition of female estradiol as an outcome and predictor variable, and the second being an initial behavioural manipulation of being socially provoked or facilitated through unfair or fair Ultimatum Game offers prior to the Ellsberg Urns task. Finally, the third study turned the second study's initial manipulation of unfair and fair Ultimatum Game offers into the primary task, and explored the potential differences in the acceptance rates of identical offers when those offers were relatively above or below average in a longer sequence of stochastically determined offers.

There are some results across all three studies that are complementary and some that are dissimilar involving both endocrine and behavioural measures. One prominent set of complementary endocrine results is that in both studies 1 and 2 lower estradiol was associated with more risk-preference and higher estradiol was associated with more risk-aversion. While there are at least two important differences in the overall context in these studies, this commonality in the functional role of estradiol between the studies is noteworthy. In the first study, estradiol was maintained at baseline levels in the social frame condition and female participants made fewer high bets (i.e., were more risk-averse), while female estradiol decreased in the non-social condition where more high bets were also observed. In the second study, high baseline estradiol and estradiol

increases in the social facilitation condition were associated with increases in the ambiguity premium in the Ellsberg Urns task, which is the difference between how much a participant values a risky compared to an ambiguous decision. While it can be argued that there are contexts in which these are qualitatively different types of decisions, ambiguity is generally considered to be a kind of “second-order” risk (Ghosh et al., 1997; Levy et al., 2010; Huettel et al., 2006), and therefore an increased ambiguity premium is a kind of higher-order risk-aversion. Therefore, where significant effects were seen for estradiol, they were generally consistent in reflecting a positive association with risk-aversion. Female estradiol increased in the “fair offer” condition in the third study, but this was statistically unrelated to acceptance rates for Ultimatum Game offers and therefore neither supports nor refutes the complementarity of estradiol findings from the first two studies.

In contrast to the converging estradiol results between the studies, there were primarily diverging results for testosterone both within- and between sexes. While male testosterone decreased in the non-social condition of study 1, making it a complementary finding with female estradiol within that study, and while there was a consistent lack of change for male testosterone in studies 2 and 3, the significant female testosterone changes notably diverged between studies 2 and 3. In study 2 female testosterone increased in both conditions (social provocation (unfair UG offers) and facilitation (fair UG offers)), while it significantly decreased in the “unfair offers” condition in study 3. The most plausible explanation for why this divergence occurs for female testosterone between studies 2 and 3 is that in study 2 the increase was not a result of the experimental manipulation (unfair versus fair UG offers), but rather in response to the main experimental task (Ellsberg Urns), which was the more consistent experience for all participants in the study that differs from study 3. Additionally, while testosterone was unrelated to female decision-making in the Ellsberg Urns task, in study 3 female testosterone both decreased in the “unfair offers” condition and its percent changes positively correlated with acceptance rates for offers of “3” in that same condition. Therefore, the most parsimonious explanation for the diverging female testosterone results between studies is that the testosterone results in study 3 are simply more reliable, being differentiable between conditions and also related to the behavioural measure of UG offer acceptance rate in a condition-specific way.

Finally, results for cortisol were also distinct between studies, and are worth characterizing for studies 1 and 3. In study 2, cortisol decreased in both conditions for females and showed no change for males but were not statistically related to any of the behavioural measures of risk, ambiguity, or ambiguity premium. However, in study 1 cortisol decreased in both conditions for males and in the non-social condition for females, while it remained constant in the social condition. Perhaps consistent with the dual-hormone hypothesis (Mehta & Prasad, 2015), decreases in cortisol were associated with decreases in testosterone for males in the non-social condition, but not in the social condition. Invoking the dual-hormone hypothesis may be dubious, however, as neither hormone was associated with betting behaviour for males in that study. Conversely, cortisol was also a moderating variable for the increase of female estradiol in the social condition but not the non-social condition, and as mentioned earlier both female estradiol and the primary independent factor (social versus non-social feedback) were significant predictors of female betting behaviour.

In keeping with a dual-hormone hypothesis but tailored to estradiol rather than cortisol, it might be proposed here that cortisol and estradiol are co-regulators of the neural circuitry required for social cognition, and that this neuroendocrine profile corresponds with a more conservative approach to risky decision-making in social contexts. However, while this interpretation is tempting, it does not necessarily fit the results from study 3 in which female cortisol was maintained at baseline levels in the “fair offers” condition but decreased in the “unfair offers” condition and was not a moderator of changes in estradiol. The experimental paradigms between studies 1 and 3 are sufficiently distinct to perhaps dismiss this incongruity, but in that case the same logic can be applied to the convergent estradiol findings between studies 1 and 2. However, although baseline cortisol was also negatively correlated with acceptance rates for offers of “3” (i.e., relatively high offers) in the “unfair offers” condition of study 3, and estradiol was not significantly related to any behavioural measures, the interpretation that estradiol and cortisol might serve as co-regulators of social cognition may survive by virtue of the fact that study 1 was the only experiment that directly tested social versus non-social framing of decision-making under risk. Ultimately, each of these findings and speculations must be replicated and independently tested to adequately make a case for each interpretation, but before some concrete proposals are made in that regard it will be instructive to clarify and contextualize what findings in this dissertation are truly novel,

and what implications they have for theories that integrate endocrine function with decision-making under risk.

5.2. Theoretical Implications of Novel Findings

5.2.1. Implications for Theory

While the above considerations are interesting syntheses, it is not enough to outline and interpret convergences merely between results of the present studies. These results must have broader theoretical grounding, and the purpose of the present section will be to outline what I believe to be the most reasonable integration of my studies' results with current theory on the intersection of biology and decision-making under risk. However, before discussing the implications of the present studies for decision-making it is worth noting that while some participants spent the bulk of their psychologically formative time in non-Western contexts, most participants fall under the WEIRD category, or from Western, educated, industrialized, rich, and democratic nations (Henrich et al., 2010; Cheon et al., 2020). It has been well established that WEIRD individuals constitute a global minority, and that the cognitive diversity of humanity has been drastically underestimated (Brady et al., 2018; Rad et al., 2018). It is important to interpret the findings of the present studies in this context because the relationship between hormones, social cognition, and decision-making in humans may involve much more systematic diversity than was captured by these limited samples. With that caveat understood, there are some noteworthy implications of the present studies for theory.

5.2.1.1. Decision-Making

As noted above, the most natural theoretical framework for understanding and embedding the results of the present studies is the somatic marker hypothesis (Bechara & Damasio, 2005), although it is not the only theory that offers useful insight. Beyond the decision-making theories outlined especially in chapter 2, another complementary framework for interpreting the present studies along with the somatic marker hypothesis is the dual-process theory (Brocas & Carillo, 2014; Evans & Stanovich, 2013). The dual-process theories propose that there are (at least) two distinct brain system for evaluating decision options: a relatively fast system that uses simple heuristics for making expedient choices, and a relatively slow system that more rationally deliberates (e.g., the weight of evidence for the probabilities of various choice outcomes). While the fast

system is generally considered to be more driven by emotion, and emotion is more generally associated somatic markers than the higher cognitive capacities employed by the slow system, each of the studies in this dissertation at least allowed for slow deliberation on choices (regardless of whether they were actually used by study participants), and it can be argued that both systems in dual-process theories are heavily reliant on somatic markers as intrinsic factors used in the decision-making process. This section will briefly review how hormones can be considered somatic markers in slow, deliberative decision-making under risk, and why the hormone changes and cognitive-behavioural correlates described in the present studies constitute an important addition to the literature.

Given the well-documented bidirectional relationship between steroid hormones and behaviour outlined in the previous section and chapters, it is uncontroversial to assert that all three steroid hormones measured in the studies of this dissertation can serve as the kind of somatic marker imagined in the somatic marker hypothesis of decision-making. Additionally, while the role that these hormones play both in the peripheral nervous system (Dart et al., 2002; Melcangi et al., 2009) and subcortical regions of the central nervous system that subserve fast heuristic processes (Guerriero, 2009), there is considerable evidence that cortical areas involved in slow deliberative cognition, including decision-making under risk, are directly and indirectly influenced by these somatic markers (Kusev et al., 2017).

For example, while different types of decisions engage or are underpinned by distinct functional brain networks (Si et al., 2019), economic decision-making has been consistently reported to correlate with activity in the prefrontal cortex (Glasher et al., 2012; Neubert et al., 2015), and more specifically in ventromedial prefrontal cortex (vmPFC), dorsolateral prefrontal cortex (dlPFC), anterior cingulate cortex (dACC), orbitofrontal cortex (OFC), and often with regions that are typically coupled with these such as the posterior cingulate cortex (PCC), frontopolar cortex (FP), and others. Importantly, these regions are known to express androgen, estrogen, and glucocorticoid receptors (albeit in heterogenous densities (Aguilera, 1998; Gegenhuber et al., 2022; Zuloaga et al., 2008), such that the neural networks for decision-making that they comprise are directly influenced by these somatic markers. Additionally, indirect bottom-up influence on these cortical decision networks arises from subcortical regions that more abundantly express nuclear steroid receptors, such as the amygdala,

hippocampus, and hypothalamus. Furthermore, cells of the peripheral nervous system also express these steroid receptors, ultimately making them an extremely robust somatic markers for both fast and slow cognitive processes like decision-making under risk.

5.2.1.2. Evolutionary Considerations

The other major theoretical grounding that is appropriate for the present studies is evolutionary (Darwin, 1859). It should be noted before discussing evolutionary considerations that no evolutionary theory was directly tested here, and therefore the integration of the empirical results must be qualified as being somewhat speculative. However, as there was no testing of any evolutionary theory there is also no challenge to them, and therefore this section will attempt to embed the present research within the most relevant and congruent branch of evolutionary theory. While there are several contenders, I argue that evo-devo offers the best lens through which to view these studies (Toth & Robinson, 2007; Muller, 2007). Evo-devo is the branch of evolutionary theory that incorporates developmental aspects, including an organism's life history. In that light, I will argue that the organizational and activational aspects of endocrine function with respect to cognitive processes like decision-making offer both variability and constraints on developmental trajectories through which an individual's propensities for risk taking are crystalized and then modulated throughout adulthood.

Evo-devo is considered by some to be part of an extended synthesis of evolutionary theory whose primary emphasis is on the relationship between developmental and processes and natural selection (Muller, 2007). Some of the main insights of the evo-devo perspective are that variation in individual phenotypes, and therefore phenotype frequencies in larger populations of organisms, are often influenced as much or more by developmental trajectories during juvenile maturation than on genotype or genetic variation within a population. For example, even identical twins display distinct physiological and/or behavioural phenotypes as adults, and subtle differences in gene expression patterns must be responsible for such differences rather than genetic differences, as identical twins have virtually none of the latter. Additionally, it is also more commonly the case that the earlier that a genetic developmental program is influenced by environmental factors, the larger the impact of that influence will be (Berardi et al., 2015; Briley & Tucker-Drop, 2017). While the connection of evo-devo

principles with the present studies is remote, it can be argued that the bidirectional relationship between hormones and behaviour, as well as the existence of distinct organizational and activational effects of hormones (especially sex-steroids), makes them prime candidates for inclusion as developmental factors that can influence physiological and behavioural phyenotypes in adulthood. I suggest that an individual's trait and state propensity for risk might be one such outcome of variable developmental trajectories influenced by the organizational and activational effects of hormones respectively, and this can be argued in a straightforward linear way.

First, while organizational effects of sex steroid hormones may not necessarily be limited to sexual differentiation, they certainly have at least that effect during sensitive periods of development (Romeo, 2003; Schultz et al., 2009; Berenbaum & Beltz, 2011). Second, sex differences in decision-making under risk can be at least partially attributed to organizational effects of sex steroids regardless of activational effects, as such differences are detectable in juveniles who do not produce quantities of sex steroids required for activational effects in adults (Cohen-Bendahan et al., 2005; Auyeung et al., 2013). A potential objection might be made here based on differential "socialization" effects on gendered juveniles. However, this organizational effect is robust across human cultures and even across mammalian species (Kimura, 2002; Sisk, 2016), lending credence to the differential organizational effect of sex steroids on developmental trajectories for risk propensity in adulthood. Third, sensitive developmental periods for the organizational effects of sex steroids also include puberty, which in humans might be said to extend to the middle of the third decade of life (Johnson et al., 2009), as this is when physiological maturation of the brain is generally thought to be complete (e.g., the prefrontal cortex is fully myelinated, and individuals display characteristically adult electroencephalographic waveforms (Epstein, 1980; Fuster, 2002; Miller et al., 2012)). Finally, the structural and functional brain changes brought about by differential exposure to sex steroids during these sensitive developmental periods, which are responsible for sexual differentiation and whose activational effects include changes in risk propensity, are prime candidates for at least partially explaining trait-level individual differences in attitudes and behaviours related to decision-making under risk. While direct tests of this argument would necessarily require longitudinal experiments that are beyond any sensible ethical framework for research in humans, the present studies contribute to a growing literature that can guide future

research aimed at elucidating these complex biophysiological relationships and clarify the degree to which this argument is valid within the evo-devo framework.

5.3. Significance of Aggregate Results & Outstanding Questions

The results of the present studies do not directly test or adjudicate between different decision-making or evolutionary theories, but as discussed, they do provide a novel contribution about the way that sex steroids affect decision-making, and this can be informative to theorists attempting to validate or falsify one or more specific unifying frameworks. While there are some interesting findings on the role that testosterone plays in both males and females, especially in the first study that distinguishes between social and non-social contexts for equivalent decisions under risk, the most robust novel findings involve the role that baseline and context-specific changes in estradiol plays in decision-making. These results point to a distinguishing functional role for the testosterone and estradiol between males and females. There is currently a relatively large literature that suggests one of the primary functional psychological roles for testosterone is the seeking, acquisition, and maintenance of social status, especially in response to status challenges (Ronay et al., 2010; Eisenegger et al., 2011; Mehta et al., 2017; Vermeer et al., 2020). In many of the empirical contexts in which testosterone has been studied, this is seen as increases in risk-taking, especially economic risk taking (Apicella et al., 2015). There is a much smaller body of literature that explores comparable psychological functional roles for estradiol (Standon et al., 2017; Kurath & Mata, 2018; Orsini et al., 2021).

In females, estradiol has been linked to power motivation (Stanton & Schultheiss, 2007; Stanton & Edelstein, 2009), which can be likened to the social status seeking effects often observed for testosterone in males. However, the role that estradiol plays with respect to risk-taking in females is more ambiguous. While a meta-analysis on this relationship found a small effect size (Kurath & Mata, 2018), the literature is smaller and results more heterogenous than for testosterone in males. The present studies add to this growing literature in two ways. First, it lowers overall credence in the notion that higher baseline or increases in female estradiol is associated with increases in risk-taking. In fact, the first two studies observed an opposite direction of effect, where higher estradiol was associated with more conservative approaches to economic risk. Second, a distinguishing functional role for estradiol is suggested by the results of the first and

third studies. In the first study, estradiol decreased in response to a non-social framing of risky economic choices compared to a social framing, and females made fewer high gambles in the social condition in which estradiol remained stable on average. In the third study, female estradiol increased in response to average Ultimatum Game offers that were relatively fair, whereas when average offers were relatively unfair female estradiol levels remained stable.

Taken together, these results might suggest that estradiol has a nuanced role in decision-making, in which social context is crucial for understanding its effects. To fully elucidate what function estradiol serves, several future studies can be suggested that would serve to parse its influence on decision-making under risk in non-social, and distinct social contexts. First, there is a gap in the literature for estradiol administration studies that test general risk-aversion, ambiguity-aversion, prospect framing, and other aspects of decision-making like temporal discounting, but not for testosterone (Takahashi et al., 2006; Doi et al., 2015; Wu et al., 2020). This line of research would be the easiest path to clarifying how increases in exogenous estradiol and decreases in endogenous effect using receptor antagonists, influence this these forms of cognition. An important advantage to administration studies is that changes in a single hormone can be isolated, and the subsequent changes to other hormones (e.g., progesterone) that can also influence decision-making (Derntl et al, 2014) are somewhat easier to interpret. An important caveat is that this line of research also needs to be done cross-culturally, as attitudes and intuitions that affect economic decision-making can vary significantly between cultures (Gurven, 2018; Henrich et al., 2005).

Second, the social context or framing in which risk-taking is measured for prospective studies like those outlined above should be characterized and controlled. For example, whether the potential consequences of risks are incurred by an individual alone or also by social confederates could be an important factor in how choice actions are calculated (Fleishman, 1988). Additionally, the group membership and social identities of the potential beneficiaries of risky decisions is another important consideration. Whether one's decisions primarily affect in-group versus out-group members, and the degree of personal closeness to such conspecifics, are two of several potential social factors that can influence risk-aversion or risk-seeking behaviours (Brewer & Kramer, 1986; Bicchieri, 2002; Van Lange et al., 2013; Montinari & Rancan, 2018). Ultimately, there are a wide range of biological, psychological, and social factors

whose confluence underlies human decision-making under risk, and teasing apart the relative contribution of factors at any of these levels is a long, meticulous process.

5.4. Concluding Remarks

Human behavioural endocrinology is still a nascent field of study, and with some minor exceptions for *some* functions of individual hormones, I believe a case can be made that it is still "pre-paradigmatic" (Kuhn, 1962), and generally lacking in strong theoretical grounding. There are many reasons for this, some of which are unavoidable. First, there are technical and methodological difficulties in both measuring and manipulating hormones, especially in socially dynamic contexts. Second, hyperspecialization has become a dominant trend in academic science, including biology and psychology (Casadevall & Fang, 2014; Fini et al., 2022). This relative myopia among researchers often prevents the kind of wide-boundary perspective that is necessary for foundational theory building. It is also partially unavoidable, given that scientific publications have increased by orders of magnitude in recent decades (Bornmann & Mutz, 2015; Lariviere et al., 2015; Bornmann et al., 2020). Of course, some of that increase does reflect fruitful interdisciplinary collaboration and often the genesis of new research fields. However, even in those cases it is unavoidable that such a wide-boundary perspective which could unite multiple scientific (sub)disciplines would require considerably more time, effort, and collaboration than is built into the rituals of investigation and information dissemination in science today.

Finally, and perhaps most importantly, behavioural endocrinology is a field attempting to unite physiological and psychological phenomena, which are both complex systems in their own right (Ladyman et al., 2012). Even though the specific intersection between hormones and cognition is a relatively narrow subset of human physiology and psychology, they span multiple levels of analysis and are integral with other biopsychosocial systems with which they are interdependent for their functioning. Although the somatic marker hypothesis and various decision theories (e.g., prospect theory, cognitive heuristics, and biases, etc.) can offer some insight to empirical findings like those discussed in this dissertation, in the absence of a well-grounded overarching theory, behavioural endocrinology is somewhat vulnerable to implicit bias and even folk narratives about the psychological function of hormones, or the physiological functions of mind. I believe that at this time theory building should be a priority for the field and given

the complex adaptive systems approach to the discipline I also believe that dynamic field theory (Coombes, 2005; Zavala et al., 2019) is an appropriate place for theoreticians to begin integrating these phenomena in common language, formulas, and terminology. While it is my intention to personally pursue this line of theoretical inquiry in the aftermath of this dissertation, I am also reminded of the third maxim carved above the entrance of the temple of Apollo at Delphi: “Ἐγγύα πάρα δ' Ἄτα” (“Give a pledge and trouble is at hand”).

References

Ackerman, C. M., Lowe, L. P., Lee, H., Hayes, M. G., Dyer, A. R., Metzger, B. E., ... & Hapo Study Cooperative Research Group. (2012). Ethnic variation in allele distribution of the androgen receptor (AR)(CAG) n repeat. *Journal of Andrology*, *33*(2), 210-215.

Adolphs, R. (2009). The social brain: neural basis of social knowledge. *Annual review of psychology*, *60*, 693.

Adolphs, R. (2010). What does the amygdala contribute to social cognition?. *Annals of the New York Academy of Sciences*, *1191*(1), 42-61.

Alberts, B., Hopkin, K., Johnson, A. D., Morgan, D., Raff, M., Roberts, K., & Walter, P. (2018). *Essential cell biology: Fifth international student edition*. WW Norton & Company.

Aguilera, G. (1998). Corticotropin releasing hormone, receptor regulation and the stress response. *Trends in Endocrinology & Metabolism*, *9*(8), 329-336

Alvergne, A., Jokela, M., Faurie, C., & Lummaa, V. (2010). Personality and testosterone in men from a high-fertility population. *Personality and Individual Differences*, *49*(8), 840-844.

Anderson, C., John, O. P., Keltner, D., & Kring, A. M. (2001). Who attains social status? Effects of personality and physical attractiveness in social groups. *Journal of personality and social psychology*, *81*(1), 116.

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

Apicella, C. L., Dreber, A., & Mollerstrom, J. (2014). Salivary testosterone change following monetary wins and losses predicts future financial risk-taking. *Psychoneuroendocrinology*, *39*, 58-64.

Arch, E. C. (1993). Risk-taking: A motivational basis for sex differences. *Psychological reports*, *73*(1), 3-11.

Archer, J. (2006). Testosterone and human aggression: an evaluation of the challenge hypothesis. *Neuroscience & Biobehavioral Reviews*, *30*(3), 319-345.

Arendt, D., Musser, J. M., Baker, C. V., Bergman, A., Cepko, C., Erwin, D. H., ... & Wagner, G. P. (2016). The origin and evolution of cell types. *Nature Reviews Genetics*, *17*(12), 744-757.

Ariely, D., & Jones, S. (2008). *Predictably irrational* (pp. 278-9). New York: HarperCollins.

Arnold, A. P. (2009). The organizational–activational hypothesis as the foundation for a unified theory of sexual differentiation of all mammalian tissues. *Hormones and behavior*, *55*(5), 570-578.

Ashton, M. C., Lee, K., Pozzebon, J. A., Visser, B. A., & Worth, N. C. (2010). Status-driven risk taking and the major dimensions of personality. *Journal of Research in Personality*, *44*(6), 734-737.

Auld, D. S., & Robitaille, R. (2003). Glial cells and neurotransmission: an inclusive view of synaptic function. *Neuron*, *40*(2), 389-400.

Aumeboonsuke, V., & Caplanova, A. (2021). An analysis of impact of personality traits and mindfulness on risk aversion of individual investors. *Current Psychology*, 1-18.

Auyeung, B., Lombardo, M. V., & Baron-Cohen, S. (2013). Prenatal and postnatal hormone effects on the human brain and cognition. *Pflügers Archiv-European Journal of Physiology*, *465*, 557-571.

Azcoitia, I., Yague, J. G., & Garcia-Segura, L. M. (2011). Estradiol synthesis within the human brain. *Neuroscience*, *191*, 139-147.

Bailey, M. R., Simpson, E. H., & Balsam, P. D. (2016). Neural substrates underlying effort, time, and risk-based decision making in motivated behavior. *Neurobiology of Learning and Memory*, *133*, 233-256.

Baker Jr, M. D., & Maner, J. K. (2008). Risk-taking as a situationally sensitive male mating strategy. *Evolution and Human Behavior*, *29*(6), 391-395.

Baker Jr, M. D., & Maner, J. K. (2009). Male risk-taking as a context-sensitive signaling device. *Journal of Experimental Social Psychology*, *45*(5), 1136-1139.

Bao, A. M., Liu, R. Y., Van Someren, E. J., Hofman, M. A., Cao, Y. X., & Zhou, J. N. (2003). Diurnal rhythm of free estradiol during the menstrual cycle. *European Journal of Endocrinology*, *148*(2), 227-232.

Baran, N. M. (2017). Sensitive periods, vasotocin-family peptides, and the evolution and development of social behavior. *Frontiers in Endocrinology*, *8*, 189.

Barel, E., Shahrabani, S., & Tzischinsky, O. (2017). Sex hormone/cortisol ratios differentially modulate risk-taking in men and women. *Evolutionary psychology*, *15*(1), 1474704917697333.

Barrett, L. F. (2006). Are emotions natural kinds?. *Perspectives on psychological science*, *1*(1), 28-58.

Barrett, L. F. (2017). The theory of constructed emotion: an active inference account of interoception and categorization. *Social cognitive and affective neuroscience*, *12*(1), 1-23.

Basten, U., Biele, G., Heekeren, H. R., & Fiebach, C. J. (2010). How the brain integrates costs and benefits during decision making. *Proceedings of the National Academy of Sciences*, *107*(50), 21767-21772.

Bechara, A., & Damasio, A. R. (2005). The somatic marker hypothesis: A neural theory of economic decision. *Games and economic behavior*, *52*(2), 336-372.

Berardi, N., Sale, A., & Maffei, L. (2015). Brain structural and functional development: genetics and experience. *Developmental Medicine & Child Neurology*, *57*, 4-9.

Berenbaum, S. A., & Beltz, A. M. (2011). Sexual differentiation of human behavior: effects of prenatal and pubertal organizational hormones. *Frontiers in neuroendocrinology*, *32*(2), 183-200.

Bernard, Claude (1878). *Leçons Sur Les Phénomènes de la Vie Communs Aux Animaux Et Aux Végétaux*. Oxford University. Bailliere.

Beyenburg, S., Watzka, M., Clusmann, H., Blümcke, I., Bidlingmaier, F., Elger, C. E., & Stoffel-Wagner, B. (2000). Androgen receptor mRNA expression in the human hippocampus. *Neuroscience letters*, *294*(1), 25-28

Bicchieri, C. (2002). Covenants without swords: Group identity, norms, and communication in social dilemmas. *Rationality and Society*, *14*(2), 192-228.

Bickart, K. C., Dickerson, B. C., & Barrett, L. F. (2014). The amygdala as a hub in brain networks that support social life. *Neuropsychologia*, *63*, 235-248.

Boden, M. A. (2008). *Mind as machine: A history of cognitive science*. Oxford University Press.

Boksem, M. A., Mehta, P. H., Van den Bergh, B., Van Son, V., Trautmann, S. T., Roelofs, K., ... & Sanfey, A. G. (2013). Testosterone inhibits trust but promotes reciprocity. *Psychological science*, *24*(11), 2306-2314.

Bornmann, L., Haunschild, R., & Mutz, R. (2021). Growth rates of modern science: a latent piecewise growth curve approach to model publication numbers from established and new literature databases. *Humanities and Social Sciences Communications*, *8*(1), 1-15.

Bornmann, L., & Mutz, R. (2015). Growth rates of modern science: A bibliometric analysis based on the number of publications and cited references. *Journal of the Association for Information Science and Technology*, *66*(11), 2215-2222.

Bos, P. A., Terburg, D., & Van Honk, J. (2010). Testosterone decreases trust in socially naive humans. *Proceedings of the National Academy of Sciences*, *107*(22), 9991-9995

Bouchouicha, R., Deer, L., Eid, A. G., McGee, P., Schoch, D., Stojic, H., ... & Vieider, F. M. (2019). Gender effects for loss aversion: Yes, no, maybe?. *Journal of Risk and Uncertainty*, *59*, 171-184.

Bourke, A. F. (2011). *Principles of social evolution*. New York, NY: Oxford University Press. doi:10.1093/acprof:oso/9780199231157.001.0001

Bourke, C. H., Harrell, C. S., & Neigh, G. N. (2012). Stress-induced sex differences: adaptations mediated by the glucocorticoid receptor. *Hormones and behavior*, *62*(3), 210-218.

Bradl, M., & Lassmann, H. (2010). Oligodendrocytes: biology and pathology. *Acta neuropathologica*, *119*, 37-53.

Brady, L. M., Fryberg, S. A., & Shoda, Y. (2018). Expanding the interpretive power of psychological science by attending to culture. *Proceedings of the National Academy of Sciences*, *115*(45), 11406-11413.

Breedlove, S.M., Watson, N.V. (2023). *Behavioral Neuroscience 10e.* Sunderland, MA. Oxford University Press.

Bressler, S. L., & Menon, V. (2010). Large-scale brain networks in cognition: emerging methods and principles. *Trends in cognitive sciences*, *14*(6), 277-290.

Brewer, M. B., & Kramer, R. M. (1986). Choice behavior in social dilemmas: Effects of social identity, group size, and decision framing. *Journal of personality and social psychology*, *50*(3), 543.

Briley, D. A., & Tucker-Drob, E. M. (2017). Comparing the developmental genetics of cognition and personality over the life span. *Journal of Personality*, *85*(1), 51-64

Brocas, I., & Carrillo, J. D. (2014). Dual-process theories of decision-making: A selective survey. *Journal of economic psychology*, *41*, 45-54.

Bullmore, E., & Sporns, O. (2009). Complex brain networks: graph theoretical analysis of structural and functional systems. *Nature reviews neuroscience*, *10*(3), 186-198.

Butti, C., Santos, M., Uppal, N., & Hof, P. R. (2013). Von Economo neurons: clinical and evolutionary perspectives. *Cortex*, *49*(1), 312-326.

Burnham, T. C. (2007). High-testosterone men reject low ultimatum game offers. *Proceedings of the Royal Society B: Biological Sciences*, *274*(1623), 2327-2330.

Buzsaki, G. (2006). *Rhythms of the Brain*. Oxford university press.

Cao-Lei, L., Suwansirikul, S., Jutavijittum, P., Mériaux, S. B., Turner, J. D., & Muller, C. P. (2013). Glucocorticoid receptor gene expression and promoter CpG modifications throughout the human brain. *Journal of psychiatric research*, *47*(11), 1597-1607.

Carré, J. M., & Archer, J. (2018). Testosterone and human behavior: the role of individual and contextual variables. *Current opinion in psychology*, *19*, 149-153.

Carré, J. M., Iselin, A. M. R., Welker, K. M., Hariri, A. R., & Dodge, K. A. (2014). Testosterone reactivity to provocation mediates the effect of early intervention on aggressive behavior. *Psychological science*, *25*(5), 1140-1146.

Camille, N., Griffiths, C. A., Vo, K., Fellows, L. K., & Kable, J. W. (2011). Ventromedial frontal lobe damage disrupts value maximization in humans. *Journal of Neuroscience*, *31*(20), 7527-7532.

Cannon, W. B. (1929). Organization for physiological homeostasis. *Physiological reviews*, *9*(3), 399-431.

Casadevall, A., & Fang, F. C. (2014). Specialized science. *Infection and immunity*, *82*(4), 1355-1360.

Chandran, U. R., Attardi, B., Friedman, R., Dong, K. W., Roberts, J. L., & DeFranco, D. B. (1994). Glucocorticoid receptor-mediated repression of gonadotropin-releasing hormone promoter activity in GT1 hypothalamic cell lines. *Endocrinology*, *134*(3), 1467-1474.

Charness, G., & Gneezy, U. (2012). Strong evidence for gender differences in risk taking. *Journal of Economic Behavior & Organization*, *83*(1), 50-58.

Cheon, B. K., Melani, I., & Hong, Y. Y. (2020). How USA-centric is psychology? An archival study of implicit assumptions of generalizability of findings to human nature based on origins of study samples. *Social Psychological and Personality Science*, *11*(7), 928-937.

Chomsky, N. (1959). Chomsky, N. 1959. A review of BF Skinner's Verbal behavior. Language, 35 (1), 26–58.

Chong, T. T. J., Apps, M., Giehl, K., Sillence, A., Grima, L. L., & Husain, M. (2017). Neurocomputational mechanisms underlying subjective valuation of effort costs. *PLoS biology*, *15*(2), e1002598.

Clithero, J. A., & Rangel, A. (2014). Informatic parcellation of the network involved in the computation of subjective value. *Social cognitive and affective neuroscience*, *9*(9), 1289-1302.

Coenjaerts, M., Pape, F., Santoso, V., Grau, F., Stoffel-Wagner, B., Philipsen, A., ... & Scheele, D. (2021). Sex differences in economic decision-making: Exogenous estradiol has opposing effects on fairness framing in women and men. *European Neuropsychopharmacology*, *50*, 46-54.

Cohen-Bendahan, C. C., Van De Beek, C., & Berenbaum, S. A. (2005). Prenatal sex hormone effects on child and adult sex-typed behavior: methods and findings. *Neuroscience & Biobehavioral Reviews*, *29*(2), 353-384.

Coombes, S. (2005). Waves, bumps, and patterns in neural field theories. *Biological cybernetics*, *93*, 91-108.

Crespi, B. J. (2016). Oxytocin, testosterone, and human social cognition. *Biological reviews*, *91*(2), 390-408.

Crespi, B., & Badcock, C. (2008). The evolutionary social brain: From genes to psychiatric conditions. *Behavioral and Brain Sciences*, *31*(3), 284-320.

Croson, R., & Gneezy, U. (2009). Gender differences in preferences. *Journal of Economic literature*, *47*(2), 448-474.

Cross, C. P., Copping, L. T., & Campbell, A. (2011). Sex differences in impulsivity: a meta-analysis. *Psychological bulletin*, *137*(1), 97.

Crowley, P. H., Travers, S. E., Linton, M. C., Cohn, S. L., Sih, A., & Sargent, R. C. (1991). Mate density, predation risk, and the seasonal sequence of mate choices: a dynamic game. *The American Naturalist*, *137*(4), 567-596.

Croxson, P. L., Walton, M. E., O'Reilly, J. X., Behrens, T. E., & Rushworth, M. F. (2009). Effort-based cost–benefit valuation and the human brain. *Journal of Neuroscience*, *29*(14), 4531-4541.

Cuff, B. M., Brown, S. J., Taylor, L., & Howat, D. J. (2016). Empathy: A review of the concept. *Emotion review*, *8*(2), 144-153.

Cueva, C., Roberts, R. E., Spencer, T., Rani, N., Tempest, M., Tobler, P. N., ... & Rustichini, A. (2015). Cortisol and testosterone increase financial risk taking and may destabilize markets. *Scientific reports*, *5*(1), 1-16.

Czeisler, C. A., & Klerman, E. B. (1999). Circadian and sleep-dependent regulation of hormone release in humans. *Recent progress in hormone research*, *54*, 97-130

Dabbs Jr, J. M. (1990). Salivary testosterone measurements: reliability across hours, days, and weeks. *Physiology & behavior*, *48*(1), 83-86.

Danese, G., Fernandes, E., Watson, N. V., & Zilioli, S. (2017). Testosterone and cortisol jointly predict the ambiguity premium in an ellsberg-urns experiment. *Frontiers in behavioral neuroscience*, *11*, 68.

Dart, A. M., Du, X. J., & Kingwell, B. A. (2002). Gender, sex hormones and autonomic nervous control of the cardiovascular system. *Cardiovascular research*, *53*(3), 678-687.

Darwin, C. (2004). *On the origin of species, 1859*. Routledge.

Deacon Terrence, W. (1998). The symbolic species. *The coevolution of language and the brain*.

De Almeida, R. M. M., Cabral, J. C. C., & Narvaes, R. (2015). Behavioural, hormonal and neurobiological mechanisms of aggressive behaviour in human and nonhuman primates. *Physiology & behavior*, *143*, 121-135.

DeFelipe, J., Markram, H., & Rockland, K. S. (2012). The neocortical column. *Frontiers in Neuroanatomy*, *6*, 22.

Dennett, D. C. (1989). *The intentional stance*. MIT press.

Derntl, B., Pintzinger, N., Kryspin-Exner, I., & Schöpf, V. (2014). The impact of sex hormone concentrations on decision-making in females and males. *Frontiers in neuroscience*, *8*, 352.

Diekhof, E. K. (2015). Be quick about it. Endogenous estradiol level, menstrual cycle phase and trait impulsiveness predict impulsive choice in the context of reward acquisition. *Hormones and Behavior*, *74*, 186-193.

Diekhof, E. K., Korf, S., Ott, F., Schädlich, C., & Holtfrerich, S. K. (2020). Avoidance learning across the menstrual cycle: A conceptual replication. *Frontiers in endocrinology*, *11*, 231.

Doi, H., Nishitani, S., & Shinohara, K. (2015). Sex difference in the relationship between salivary testosterone and inter-temporal choice. *Hormones and behavior*, *69*, 50-58.

Dosenbach, N. U., Fair, D. A., Miezin, F. M., Cohen, A. L., Wenger, K. K., Dosenbach, R. A., ... & Petersen, S. E. (2007). Distinct brain networks for adaptive and stable task control in humans. *Proceedings of the National Academy of Sciences*, *104*(26), 11073-11078.

Eisenegger, C., Naef, M., Snozzi, R., Heinrichs, M., & Fehr, E. (2010). Prejudice and truth about the effect of testosterone on human bargaining behaviour. *Nature*, *463*(7279), 356-359.

Eisenegger, C., Haushofer, J., & Fehr, E. (2011). The role of testosterone in social interaction. *Trends in cognitive sciences*, *15*(6), 263-271.

Ekman, P. (1992). An argument for basic emotions. *Cognition & emotion*, *6*(3-4), 169-200.

Ekman, P. (1999). Basic emotions. *Handbook of cognition and emotion*, *98*(45-60), 16.

Ellsberg, D. (1961). Risk, ambiguity, and the Savage axioms. *The quarterly journal of economics*, 643-669.

Emanuele, E., Brondino, N., Bertona, M., Re, S., & Geroldi, D. (2008). Relationship between platelet serotonin content and rejections of unfair offers in the ultimatum game. *Neuroscience letters*, *437*(2), 158-161.

Epstein, H. T. (1980). EEG developmental stages. *Developmental Psychobiology: The Journal of the International Society for Developmental Psychobiology*, *13*(6), 629-631.

Evans, M. J., & Kaufman, M. H. (1981). Establishment in culture of pluripotential cells from mouse embryos. *nature*, *292*(5819), 154-156.

Evans, A. M., & Krueger, J. I. (2009). The psychology (and economics) of trust. *Social and Personality Psychology Compass*, *3*(6), 1003-1017.

Evans, J. S. B., & Stanovich, K. E. (2013). Dual-process theories of higher cognition: Advancing the debate. *Perspectives on psychological science*, *8*(3), 223-241.

Faul, F., Erdfelder, E., Buchner, A., & Lang, A. G. (2009). Statistical power analyses using G* Power 3.1: Tests for correlation and regression analyses. *Behavior research methods*, *41*(4), 1149-1160.

FeldmanHall, O., Raio, C. M., Kubota, J. T., Seiler, M. G., & Phelps, E. A. (2015). The effects of social context and acute stress on decision making under uncertainty. *Psychological science*, *26*(12), 1918-1926.

FeldmanHall, O., & Shenhav, A. (2019). Resolving uncertainty in a social world. *Nature human behaviour*, *3*(5), 426-435.

Fernández-Guasti, A., Kruijver, F. P., Fodor, M., & Swaab, D. F. (2000). Sex differences in the distribution of androgen receptors in the human hypothalamus. *Journal of Comparative Neurology*, *425*(3), 422-435.

Fisher, M. L. (2013). Women's intrasexual competition for mates. *Evolution's empress: Darwinian perspectives on the nature of women*, 19-42.

Fiske, S. T., Cuddy, A. J., & Glick, P. (2007). Universal dimensions of social cognition: Warmth and competence. *Trends in cognitive sciences*, *11*(2), 77-83.

Fini, R., Jourdan, J., Perkmann, M., & Toschi, L. (2022). A new take on the categorical imperative: Gatekeeping, boundary maintenance, and evaluation penalties in science. *Organization Science*.

Freeman, C. (2016). What is mentalizing? An overview. *British Journal of Psychotherapy*, *32*(2), 189-201.

Fleishman, J. A. (1988). The effects of decision framing and others' behavior on cooperation in a social dilemma. *Journal of Conflict Resolution*, *32*(1), 162-180.

Friedl, A., Pondorfer, A., & Schmidt, U. (2020). Gender differences in social risk taking. *Journal of Economic Psychology*, *77*, 102182.

Frith, C. D., & Frith, U. (2006). The neural basis of mentalizing. *Neuron*, *50*(4), 531-534.

Frith, C. D., & Singer, T. (2008). The role of social cognition in decision making. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *363*(1511), 3875-3886.

Fuster, J. M. (2002). Frontal lobe and cognitive development. *Journal of neurocytology*, *31*(3-5), 373-385.

Gegenhuber, B., Wu, M. V., Bronstein, R., & Tollkuhn, J. (2022). Gene regulation by gonadal hormone receptors underlies brain sex differences. *Nature*, *606*(7912), 153-159.

Geniole, S. N., & Carré, J. M. (2018). Human social neuroendocrinology: Review of the rapid effects of testosterone. *Hormones and behavior*, *104*, 192-205.

Ghosh, D., & Ray, M. R. (1997). Risk, ambiguity, and decision choice: Some additional evidence. *Decision Sciences*, *28*(1), 81-104.

Gigerenzer, G. (2008). Why heuristics work. *Perspectives on psychological science*, *3*(1), 20-29.

Gigerenzer, G., & Gaissmaier, W. (2011). Heuristic decision making. *Annual review of psychology*, *62*, 451-482.

Gigerenzer, G., & Gaissmaier, W. (2015). Decision making: Nonrational theories. In *International encyclopedia of the social & behavioral sciences* (pp. 911-916). Elsevier.

Gläscher, J., Adolphs, R., Damasio, H., Bechara, A., Rudrauf, D., Calamia, M., ... & Tranel, D. (2012). Lesion mapping of cognitive control and value-based decision making in the prefrontal cortex. *Proceedings of the National Academy of Sciences*, *109*(36), 14681-14686.

Gläscher, J., Hampton, A. N., & O'Doherty, J. P. (2009). Determining a role for ventromedial prefrontal cortex in encoding action-based value signals during reward-related decision making. *Cerebral cortex*, *19*(2), 483-495.

Gloyna, R. E., & Wilson, J. D. (1969). A comparative study of the conversion of testosterone to 17β-hydroxy-5α-androstan-3-one (dihydrotestosterone) by prostate and epididymis. *The Journal of Clinical Endocrinology & Metabolism*, *29*(7), 970-977.

González, M., Cabrera-Socorro, A., Pérez-García, C. G., Fraser, J. D., López, F. J., Alonso, R., & Meyer, G. (2007). Distribution patterns of estrogen receptor α and β in the human cortex and hippocampus during development and adulthood. *Journal of Comparative Neurology*, *503*(6), 790-802.

Goodson, J. L., & Thompson, R. R. (2010). Nonapeptide mechanisms of social cognition, behavior and species-specific social systems. *Current opinion in neurobiology*, *20*(6), 784-794.

Gosling SD, Rentfrow PJ, Swann WB. (2003) A very brief measure of the Big-Five personality domains. *J Res Pers*; 37(6):504–528.

Gross, J. J., & Feldman Barrett, L. (2011). Emotion generation and emotion regulation: One or two depends on your point of view. *Emotion review*, *3*(1), 8-16.

Grossman, R., & Taylor, E. W. (2007). Respiratory sinus arrhythmia, cardiac vagal tone, and a critique of the polyvagal theory: toward a theory of biobehavioral allostasis of energy exchange. *Biol Psychol*.

Guerriero, G. (2009). Vertebrate sex steroid receptors: evolution, ligands, and neurodistribution. *Annals of the New York Academy of Sciences*, *1163*(1), 154-168.

Gurven, M. D. (2018). Broadening horizons: Sample diversity and socioecological theory are essential to the future of psychological science. *Proceedings of the National Academy of Sciences*, *115*(45), 11420-11427.

Hamilton, L. D., Carré, J. M., Mehta, P. H., Olmstead, N., & Whitaker, J. D. (2015). Social neuroendocrinology of status: A review and future directions. *Adaptive Human Behavior and Physiology*, *1*, 202-230.

Hardin, E. E., Leong, F. T., & Bhagwat, A. A. (2004). Factor structure of the self-construal scale revisited: Implications for the multidimensionality of self-construal. *Journal of Cross-Cultural Psychology*, *35*(3), 327-345.

Hareli, S., & Parkinson, B. (2008). What's social about social emotions?. *Journal for the theory of social behaviour*, *38*(2), 131-156.

Hayes, A. F., Montoya, A. K., & Rockwood, N. J. (2017). The analysis of mechanisms and their contingencies: PROCESS versus structural equation modeling. *Australasian Marketing Journal*, *25*(1), 76-81.

Henrich, J. (2016). *The secret of our success: How culture is driving human evolution, domesticating our species, and making us smarter*. princeton University press.

Henrich, J., Boyd, R., Bowles, S., Camerer, C., Fehr, E., Gintis, H., & McElreath, R. (2001). In search of homo economicus: behavioral experiments in 15 small-scale societies. *American Economic Review*, *91*(2), 73-78.

Henrich, J., Boyd, R., Bowles, S., Camerer, C., Fehr, E., Gintis, H., ... & Tracer, D. (2005). "Economic man" in cross-cultural perspective: Behavioral experiments in 15 small-scale societies. *Behavioral and brain sciences*, *28*(6), 795-815.

Henrich, J., Heine, S. J., & Norenzayan, A. (2010). The weirdest people in the world?. *Behavioral and brain sciences*, *33*(2-3), 61-83.

Herbert, J. (2018). Testosterone, cortisol and financial risk-taking. *Frontiers in Behavioral Neuroscience*, *12*, 101.

Herbison, A. E. (2018). The gonadotropin-releasing hormone pulse generator. *Endocrinology*, *159*(11), 3723-3736.

Hintze, A., Olson, R. S., Adami, C., & Hertwig, R. (2015). Risk sensitivity as an evolutionary adaptation. *Scientific reports*, *5*(1), 1-7.

Hönekopp, J., & Watson, S. (2010). Meta-analysis of digit ratio 2D: 4D shows greater sex difference in the right hand. *American Journal of Human Biology*, *22*(5), 619-630.

Huettel, S. A., Stowe, C. J., Gordon, E. M., Warner, B. T., & Platt, M. L. (2006). Neural signatures of economic preferences for risk and ambiguity. *Neuron*, *49*(5), 765-775.

Jackson, S. A., Kleitman, S., Howie, P., & Stankov, L. (2016). Cognitive abilities, monitoring confidence, and control thresholds explain individual differences in heuristics and biases. *Frontiers in Psychology*, *7*, 1559.

James, W. (1890/2007). *The principles of psychology* (Vol. 1). Cosimo, Inc..

Johnson, J. G., & Busemeyer, J. R. (2010). Decision making under risk and uncertainty. *Wiley Interdisciplinary Reviews: Cognitive Science*, *1*(5), 736-749.

Johnson, S. B., Blum, R. W., & Giedd, J. N. (2009). Adolescent maturity and the brain: the promise and pitfalls of neuroscience research in adolescent health policy. *Journal of adolescent health*, *45*(3), 216-221.

Kacelnik, A., & Bateson, M. (1997). Risk-sensitivity: crossroads for theories of decision-making. *Trends in cognitive sciences*, *1*(8), 304-309.

Kahneman, D. (2011). *Thinking, Fast and Slow*. Macmillan.

Kahneman, D., & Tversky, A. (1979). Prospect theory: An analysis of decision under risk. *Econometrica*, *47*(2), 363-391.

Kandel, E. R., Schwartz, J. H., Jessell, T. M., Siegelbaum, S., Hudspeth, A. J., & Mack, S. (Eds.). (2021). *Principles of neural science*. New York: McGraw-hill.

Kaping, D., Vinck, M., Hutchison, R. M., Everling, S., & Womelsdorf, T. (2011). Specific contributions of ventromedial, anterior cingulate, and lateral prefrontal cortex for attentional selection and stimulus valuation. *PLoS biology*, *9*(12), e1001224.

Kettenmann, H., Hanisch, U. K., Noda, M., & Verkhratsky, A. (2011). Physiology of microglia. *Physiological reviews*, *91*(2), 461-553.

Khani, S., & Tayek, J. A. (2001). Cortisol increases gluconeogenesis in humans: its role in the metabolic syndrome. *Clinical Science*, *101*(6), 739-747.

Kimura, D. (2002). Sex hormones influence human cognitive pattern. *Neuroendocrinology Letters*, *23*(4), 67-77.

Kirschbaum, C., & Hellhammer, D. H. (1994). Salivary cortisol in psychoneuroendocrine research: recent developments and applications. Psychoneuroendocrinology, 19(4), 313-333.

Klein-Flügge, M. C., Kennerley, S. W., Friston, K., & Bestmann, S. (2016). Neural signatures of value comparison in human cingulate cortex during decisions requiring an effort-reward trade-off. *Journal of Neuroscience*, *36*(39), 10002-10015.

Klibanoff, P., Marinacci, M., & Mukerji, S. (2005). A smooth model of decision making under ambiguity. *Econometrica*, *73*(6), 1849-1892.

Klimecki, O. M. (2015). The plasticity of social emotions. *Social neuroscience*, *10*(5), 466-473.

Knight, E. L., & Mehta, P. H. (2014). Hormones and hierarchies. *The psychology of social status*, 269-301.

Kocher, M. G., Lahno, A. M., & Trautmann, S. T. (2018). Ambiguity aversion is not universal. *European Economic Review*, *101*, 268-283

Kolling, N., Wittmann, M. K., Behrens, T. E., Boorman, E. D., Mars, R. B., & Rushworth, M. F. (2016). Value, search, persistence and model updating in anterior cingulate cortex. *Nature neuroscience*, *19*(10), 1280-1285.

Komorita, S. S. (2019). *Social dilemmas*. Routledge.

Krain, A. L., Wilson, A. M., Arbuckle, R., Castellanos, F. X., & Milham, M. P. (2006). Distinct neural mechanisms of risk and ambiguity: a meta-analysis of decision-making. *Neuroimage*, *32*(1), 477-484.

Krall, S. C., Rottschy, C., Oberwelland, E., Bzdok, D., Fox, P. T., Eickhoff, S. B., ... & Konrad, K. (2015). The role of the right temporoparietal junction in attention and social interaction as revealed by ALE meta-analysis. *Brain Structure and Function*, *220*, 587-604.

Krueger, F., & Meyer-Lindenberg, A. (2019). Toward a model of interpersonal trust drawn from neuroscience, psychology, and economics. *Trends in neurosciences*, *42*(2), 92-101.

Kuhn, T. S. (1962). *The structure of scientific revolutions*. University of Chicago press.

Kurath, J., & Mata, R. (2018). Individual differences in risk taking and endogeneous levels of testosterone, estradiol, and cortisol: A systematic literature search and three independent meta-analyses. *Neuroscience & Biobehavioral Reviews*, *90*, 428-446.

Kusev, P., Purser, H., Heilman, R., Cooke, A. J., Van Schaik, P., Baranova, V., ... & Ayton, P. (2017). Understanding risky behavior: The influence of cognitive, emotional and hormonal factors on decision-making under risk. *Frontiers in psychology*, *8*, 102.

Ladyman, J., Lambert, J., & Wiesner, K. (2013). What is a complex system?. *European Journal for Philosophy of Science*, *3*, 33-67.

Laland, K. N., Sterelny, K., Odling-Smee, J., Hoppitt, W., & Uller, T. (2011). Cause and effect in biology revisited: is Mayr's proximate-ultimate dichotomy still useful?. *science*, *334*(6062), 1512-1516.

Larivière, V., Haustein, S., & Mongeon, P. (2015). The oligopoly of academic publishers in the digital era. *PloS one*, *10*(6), e0127502.

Lee, D. (2008). Game theory and neural basis of social decision making. *Nature neuroscience*, *11*(4), 404-409.

Leslie, A. M., Friedman, O., & German, T. P. (2004). Core mechanisms in 'theory of mind'. *Trends in cognitive sciences*, *8*(12), 528-533.

Levin, E. R. (1999). Cellular functions of the plasma membrane estrogen receptor. *Trends in Endocrinology & Metabolism*, *10*(9), 374-377.

Levin, E. R. (2002). Cellular functions of plasma membrane estrogen receptors. *Steroids*, *67*(6), 471-475.

Levin, E. R. (2009). Plasma membrane estrogen receptors. *Trends in Endocrinology & Metabolism*, *20*(10), 477-482.

Levy, I., Snell, J., Nelson, A. J., Rustichini, A., & Glimcher, P. W. (2010). Neural representation of subjective value under risk and ambiguity. *Journal of neurophysiology*, *103*(2), 1036-1047.

Little, S. G., Swangler, J., & Akin-Little, A. (2017). Defining social skills. *Handbook of social behavior and skills in children*, 9-17.

Mahon, B. Z., & Caramazza, A. (2010). Judging semantic similarity: An event-related fMRI study with auditory word stimuli. *Neuroscience*, *169*(1), 279-286.

Maner, J. K., Miller, S. L., Schmidt, N. B., & Eckel, L. A. (2008). Submitting to defeat: Social anxiety, dominance threat, and decrements in testosterone. *Psychological Science*, *19*(8), 764-768.

Manuck, S. B., Marsland, A. L., Flory, J. D., Gorka, A., Ferrell, R. E., & Hariri, A. R. (2010). Salivary testosterone and a trinucleotide (CAG) length polymorphism in the androgen receptor gene predict amygdala reactivity in men. *Psychoneuroendocrinology*, *35*(1), 94-104.

Markova, D., Richer, L., Pangelinan, M., Schwartz, D. H., Leonard, G., Perron, M., ... & Paus, T. (2016). Age-and sex-related variations in vocal-tract morphology and voice acoustics during adolescence. *Hormones and behavior*, *81*, 84-96.

Mars, R. B., Neubert, F. X., Noonan, M. P., Sallet, J., Toni, I., & Rushworth, M. F. (2012). On the relationship between the “default mode network” and the “social brain”. *Frontiers in human neuroscience*, *6*, 189.

Martens, J. P., Tracy, J. L., & Shariff, A. F. (2012). Status signals: Adaptive benefits of displaying and observing the nonverbal expressions of pride and shame. *Cognition & Emotion*, *26*(3), 390-406.

Maschler, M., Zamir, S., & Solan, E. (2020). *Game theory*. Cambridge University Press.

McCall, C., & Singer, T. (2012). The animal and human neuroendocrinology of social cognition, motivation and behavior. *Nature neuroscience*, *15*(5), 681-688.

McNamee, D., Rangel, A., & O'doherty, J. P. (2013). Category-dependent and category-independent goal-value codes in human ventromedial prefrontal cortex. *Nature neuroscience*, *16*(4), 479-485.

Mehta, P. H., & Prasad, S. (2015). The dual-hormone hypothesis: a brief review and future research agenda. *Current opinion in behavioral sciences*, *3*, 163-168.

Mehta, P. H., DesJardins, N. M. L., van Vugt, M., & Josephs, R. A. (2017). Hormonal underpinnings of status conflict: Testosterone and cortisol are related to decisions and satisfaction in the hawk-dove game. *Hormones and behavior*, *92*, 141-154.

Mehta, P. H., Welker, K. M., Zilioli, S., & Carré, J. M. (2015). Testosterone and cortisol jointly modulate risk-taking. *Psychoneuroendocrinology*, *56*, 88-99.

Melcangi, R. C., & Panzica, G. (2009). Neuroactive steroids: an update of their roles in central and peripheral nervous system. *Psychoneuroendocrinology*, *34*, S1-S8.

Miller, D. J., Duka, T., Stimpson, C. D., Schapiro, S. J., Baze, W. B., McArthur, M. J., ... & Sherwood, C. C. (2012). Prolonged myelination in human neocortical evolution. *Proceedings of the National Academy of Sciences*, *109*(41), 16480-16485.

Mischel, W., Shoda, Y., & Rodriguez, M. L. (1989). Delay of gratification in children. *Science*, *244*(4907), 933-938.

Mishra, S. (2014). Decision-making under risk: Integrating perspectives from biology, economics, and psychology. *Personality and Social Psychology Review*, *18*(3), 280-307.

Mohawk, J. A., & Takahashi, J. S. (2011). Cell autonomy and synchrony of suprachiasmatic nucleus circadian oscillators. *Trends in neurosciences*, *34*(7), 349-358.

Montinari, N., & Rancan, M. (2018). Risk taking on behalf of others: The role of social distance. *Journal of Risk and Uncertainty*, *57*, 81-109.

Mountcastle, V. B. (1998). *Perceptual neuroscience: the cerebral cortex*. Harvard University Press.

Mousavi, S., & Gigerenzer, G. (2014). Risk, uncertainty, and heuristics. *Journal of Business Research*, *67*(8), 1671-1678.

Müller, G. B. (2007). Evo–devo: extending the evolutionary synthesis. *Nature reviews genetics*, *8*(12), 943-949.

Mundy, P., & Newell, L. (2007). Attention, joint attention, and social cognition. *Current directions in psychological science*, *16*(5), 269-274.

Nelson, J. A. (2016). Not-so-strong evidence for gender differences in risk taking. *Feminist Economics*, *22*(2), 114-142.

Nelson, R. J. (2023). *An introduction to behavioral endocrinology*. Sinauer Associates.

Neubert, F. X., Mars, R. B., Sallet, J., & Rushworth, M. F. (2015). Connectivity reveals relationship of brain areas for reward-guided learning and decision making in human and monkey frontal cortex. *Proceedings of the national academy of sciences*, *112*(20), E2695-E2704.

Norman, A. W., & Litwack, G. (1997). General considerations of hormones. *Hormones*, *1*, 2-49.

Nowak, M. A., Page, K. M., & Sigmund, K. (2000). Fairness versus reason in the ultimatum game. Science, 289(5485), 1773-1775.

Nowak, M. A. (2006). Five rules for the evolution of cooperation. *science*, *314*(5805), 1560-1563.

O'Connell, L. A., & Hofmann, H. A. (2012). Evolution of a vertebrate social decision-making network. *Science*, *336*(6085), 1154-1157.

Orsini, C. A., Blaes, S. L., Hernandez, C. M., Betzhold, S. M., Perera, H., Wheeler, A. R., ... & Setlow, B. (2021). Regulation of risky decision making by gonadal hormones in males and females. *Neuropsychopharmacology*, *46*(3), 603-613.

Österlund, M. K., Grandien, K., Keller, E., & Hurd, Y. L. (2000). The human brain has distinct regional expression patterns of estrogen receptor α mRNA isoforms derived from alternative promoters. *Journal of neurochemistry*, *75*(4), 1390-1397.

Pachur, T., Hertwig, R., & Steinmann, F. (2012). How do people judge risks: availability heuristic, affect heuristic, or both?. *Journal of Experimental Psychology: Applied*, *18*(3), 314.

Paletta, P., Bass, N., Aspesi, D., & Choleris, E. (2022). Sex differences in social cognition.

Panksepp, J., & Biven, L. (2012). *The archaeology of mind: Neural origins of human emotion*. WW Norton & Company.

Parker, A. M., De Bruin, W. B., & Fischhoff, B. (2007). Maximizers versus satisficers: Decision-making styles, competence, and outcomes. *Judgment and Decision making*, *2*(6), 342-350.

Pawlowski, B., Atwal, R., & Dunbar, R. I. (2008). Sex differences in everyday risk-taking behavior in humans. *Evolutionary Psychology*, *6*(1), 147470490800600104.

Paxton, J. M., & Greene, J. D. (2010). Moral reasoning: Hints and allegations. Topics in Cognitive Science, 2 (3), 511–527.

Perlman, W. R., Webster, M. J., Herman, M. M., Kleinman, J. E., & Weickert, C. S. (2007). Age-related differences in glucocorticoid receptor mRNA levels in the human brain. *Neurobiology of aging*, *28*(3), 447-458.

Peterburs, J., Voegler, R., Liepelt, R., Schulze, A., Wilhelm, S., Ocklenburg, S., & Straube, T. (2017). Processing of fair and unfair offers in the ultimatum game under social observation. *Scientific Reports*, *7*(1), 1-12.

Peters, E. (2012). Beyond comprehension: The role of numeracy in judgments and decisions. *Current Directions in Psychological Science*, *21*(1), 31-35.

Peterson, M. (2017). *An introduction to decision theory*. Cambridge University Press.

Pfrieger, F. W. (2010). Role of glial cells in the formation and maintenance of synapses. *Brain research reviews*, *63*(1-2), 39-46.

Pfrieger, F. W., & Barres, B. A. (1997). Synaptic efficacy enhanced by glial cells in vitro. *Science*, *277*(5332), 1684-1687.

Poppa, T., & Bechara, A. (2018). The somatic marker hypothesis: revisiting the role of the 'body-loop'in decision-making. *Current opinion in behavioral sciences*, *19*, 61-66.

Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication, and self-regulation (Norton Series on Interpersonal Neurobiology)*. WW Norton & Company.

Porges, S. W. (2021). Polyvagal Theory: A biobehavioral journey to sociality. *Comprehensive Psychoneuroendocrinology*, *7*, 100069.

Pratto, F., Sidanius, J., Stallworth, L. M., & Malle, B. F. (1994). Social dominance orientation: A personality variable predicting social and political attitudes. *Journal of personality and social psychology*, *67*(4), 741.

Proverbio, A. M. (2021). Sex differences in the social brain and in social cognition. *Journal of Neuroscience Research*.

Puy, L., MacLusky, N. J., Becker, L., Karsan, N., Trachtenberg, J., & Brown, T. J. (1995). Immunocytochemical detection of androgen receptor in human temporal cortex: characterization and application of polyclonal androgen receptor antibodies in frozen and paraffin-embedded tissues. *The Journal of steroid biochemistry and molecular biology*, *55*(2), 197-209.

Racey, D., Young, M. E., Garlick, D., Ngoc-Minh Pham, J., & Blaisdell, A. P. (2011). Pigeon and human performance in a multi-armed bandit task in response to changes in variable interval schedules. *Learning & behavior*, *39*, 245-258.

Rad, M. S., Martingano, A. J., & Ginges, J. (2018). Toward a psychology of Homo sapiens: Making psychological science more representative of the human population. *Proceedings of the National Academy of Sciences*, *115*(45), 11401-11405.

Raichle, M. E. (2015). The brain's default mode network. *Annual review of neuroscience*, *38*, 433-447.

Raichle, M. E., MacLeod, A. M., Snyder, A. Z., Powers, W. J., Gusnard, D. A., & Shulman, G. L. (2001). A default mode of brain function. *Proceedings of the national academy of sciences*, *98*(2), 676-682.

Rand, D. G., Tarnita, C. E., Ohtsuki, H., & Nowak, M. A. (2013). Evolution of fairness in the one-shot anonymous Ultimatum Game. *Proceedings of the National Academy of Sciences*, *110*(7), 2581-2586.

Rangel, A., Camerer, C., & Montague, P. R. (2008). A framework for studying the neurobiology of value-based decision making. *Nature reviews neuroscience*, *9*(7), 545-556.

Rangel, A., & Clithero, J. A. (2014). The computation of stimulus values in simple choice. *Neuroeconomics*, 125-148.

Rapoport, A., & Chammah, A. M. (1965). *Prisoner's dilemma: A study in conflict and cooperation* (Vol. 165). University of Michigan press.

Ratcliff, R., Smith, P. L., Brown, S. D., & McKoon, G. (2016). Diffusion decision model: Current issues and history. *Trends in cognitive sciences*, *20*(4), 260-281.

Reed, M. S., Evely, A. C., Cundill, G., Fazey, I., Glass, J., Laing, A., ... & Stringer, L. C. (2010). What is social learning?. *Ecology and society*, *15*(4).

Reimers, L., & Diekhof, E. K. (2015). Testosterone is associated with cooperation during intergroup competition by enhancing parochial altruism. *Frontiers in neuroscience*, *9*, 183.

Reppert, S. M., & Weaver, D. R. (2001). Molecular analysis of mammalian circadian rhythms. *Annual review of physiology*, *63*(1), 647-676.

Rilling, J. K., & Sanfey, A. G. (2011). The neuroscience of social decision-making. *Annual review of psychology*, *62*, 23-48.

Robertson, T. E., Sznycer, D., Delton, A. W., Tooby, J., & Cosmides, L. (2018). The true trigger of shame: Social devaluation is sufficient, wrongdoing is unnecessary. *Evolution and Human Behavior*, *39*(5), 566-573.

Romeo, R. D. (2003). Puberty: a period of both organizational and activational effects of steroid hormones on neurobehavioural development. *Journal of neuroendocrinology*, *15*(12), 1185-1192.

Ronay, R., & Von Hippel, W. (2010). Power, testosterone, and risk-taking. *Journal of Behavioral Decision Making*, *23*(5), 473-482.

Rousseau, D. M., Sitkin, S. B., Burt, R. S., & Camerer, C. (1998). Not so different after all: A cross-discipline view of trust. *Academy of management review*, *23*(3), 393-404.

Rudorf, S., & Hare, T. A. (2014). Interactions between dorsolateral and ventromedial prefrontal cortex underlie context-dependent stimulus valuation in goal-directed choice. *Journal of Neuroscience*, *34*(48), 15988-15996.

Ruff, C. C., & Fehr, E. (2014). The neurobiology of rewards and values in social decision making. *Nature Reviews Neuroscience*, *15*(8), 549-562.

Ruggeri, K., Alí, S., Berge, M. L., Bertoldo, G., Bjørndal, L. D., Cortijos-Bernabeu, A., ... & Folke, T. (2020). Replicating patterns of prospect theory for decision under risk. *Nature human behaviour*, *4*(6), 622-633.

Sanfey, A. G., Rilling, J. K., Aronson, J. A., Nystrom, L. E., & Cohen, J. D. (2003). The neural basis of economic decision-making in the ultimatum game. *Science*, *300*(5626), 1755-1758

Sapolsky, R. M. (2004). Social status and health in humans and other animals. *Annu. Rev. Anthropol.*, *33*, 393-418.

Sarkey, S., Azcoitia, I., Garcia-Segura, L. M., Garcia-Ovejero, D., & DonCarlos, L. L. (2008). Classical androgen receptors in non-classical sites in the brain. *Hormones and behavior*, *53*(5), 753-764.

Sarlo, M., Lotto, L., Palomba, D., Scozzari, S., & Rumiati, R. (2013). Framing the ultimatum game: gender differences and autonomic responses. *International Journal of Psychology*, *48*(3), 263-271.

Solnick, S. J. (2001). Gender differences in the ultimatum game. *Economic Inquiry*, *39*(2), 189-200.

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*(36), 15268-15273.

Schipper, B. C. (2014). Sex hormones and choice under risk. *Available at SSRN 2046324*.

Schipper, B. C. (2015). Sex hormones and competitive bidding. *Management Science*, *61*(2), 249-266.

Schulkin, J., McEwen, B. S., & Gold, P. W. (1994). Allostasis, amygdala, and anticipatory angst. *Neuroscience & Biobehavioral Reviews*, *18*(3), 385-396.

Schulz, K. M., Molenda-Figueira, H. A., & Sisk, C. L. (2009). Back to the future: the organizational–activational hypothesis adapted to puberty and adolescence. *Hormones and behavior*, *55*(5), 597-604.

Shenhav, A., Cohen, J. D., & Botvinick, M. M. (2016). Dorsal anterior cingulate cortex and the value of control. *Nature neuroscience*, *19*(10), 1286-1291.

Si, Y., Wu, X., Li, F., Zhang, L., Duan, K., Li, P., ... & Xu, P. (2019). Different decision-making responses occupy different brain networks for information processing: a study based on EEG and TMS. *Cerebral Cortex*, *29*(10), 4119-4129.

Siegel, M., Engel, A. K., & Donner, T. H. (2011). Cortical network dynamics of perceptual decision-making in the human brain. *Frontiers in human neuroscience*, *5*, 21.

Simon, H. A. (1956). Rational choice and the structure of the environment. *Psychological review*, *63*(2), 129.

Sisk, C. L. (2016). Hormone-dependent adolescent organization of socio-sexual behaviors in mammals. *Current opinion in neurobiology*, *38*, 63-68.

Slovic, P., & Peters, E. (2006). Risk perception and affect. *Current directions in psychological science*, *15*(6), 322-325.

Slovic, P., Peters, E., Finucane, M. L., & MacGregor, D. G. (2005). Affect, risk, and decision making. *Health psychology*, *24*(4S), S35.

Smals, A. G. H., Kloppenborg, P. W. C., & Benraad, T. J. (1976). Circannual cycle in plasma testosterone levels in man. *The Journal of Clinical Endocrinology & Metabolism*, *42*(5), 979-982.

Smith, R. P., Coward, R. M., Kovac, J. R., & Lipshultz, L. I. (2013). The evidence for seasonal variations of testosterone in men. *Maturitas*, *74*(3), 208-212.

Sofroniew, M. V., & Vinters, H. V. (2010). Astrocytes: biology and pathology. *Acta neuropathologica*, *119*, 7-35.

Sporns, O., & Betzel, R. F. (2016). Modular brain networks. *Annual review of psychology*, *67*, 613-640.

Sporns, O., & Kötter, R. (2004). Motifs in brain networks. *PLoS biology*, *2*(11), e369.

Sporns, O., & Zwi, J. D. (2004). The small world of the cerebral cortex. *Neuroinformatics*, *2*, 145-162.

Spunt, R. P., & Adolphs, R. (2017). A new look at domain specificity: insights from social neuroscience. *Nature Reviews Neuroscience*, *18*(9), 559-567.

Stanton, S. J. (2017). The role of testosterone and estrogen in consumer behavior and social & economic decision making: A review. *Hormones and behavior*, *92*, 155-163.

Stanton, S. J., Liening, S. H., & Schultheiss, O. C. (2011). Testosterone is positively associated with risk taking in the Iowa Gambling Task. *Hormones and behavior*, *59*(2), 252-256.

Stanton, S. J., Mullette-Gillman, O. D. A., McLaurin, R. E., Kuhn, C. M., LaBar, K. S., Platt, M. L., & Huettel, S. A. (2011). Low-and high-testosterone individuals exhibit decreased aversion to economic risk. *Psychological science*, *22*(4), 447-453.

Stanton, S. J., & Schultheiss, O. C. (2007). Basal and dynamic relationships between implicit power motivation and estradiol in women. *Hormones and behavior*, *52*(5), 571-580.

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.

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*(4), 412-416.

Stenstrom, E., & Saad, G. (2011). Testosterone, financial risk-taking, and pathological gambling. *Journal of Neuroscience, Psychology, and Economics*, *4*(4), 254.

Strogatz, S. H. (2018). *Nonlinear dynamics and chaos with student solutions manual: With applications to physics, biology, chemistry, and engineering*. Boca Raton, FL. CRC press. doi: https://doi.org/10.1201/9780429492563

Sundin, Z. W., Chopik, W. J., Welker, K. M., Ascigil, E., Brandes, C. M., Chin, K., ... & Tackett, J. L. (2021). Estimating the associations between big five personality traits, testosterone, and cortisol. *Adaptive Human Behavior and Physiology*, 1-34.

Swaab, D. F., Chung, W. C., Kruijver, F. P., Hofman, M. A., & Hestiantoro, A. (2003). Sex differences in the hypothalamus in the different stages of human life. *Neurobiology of aging*, *24*, S1-S16.

Szczepanski, S. M., Pinsk, M. A., Douglas, M. M., Kastner, S., & Saalmann, Y. B. (2013). Functional and structural architecture of the human dorsal frontoparietal attention network. *Proceedings of the National Academy of Sciences*, *110*(39), 15806-15811.

Sznycer, D., & Cohen, A. S. (2021). How pride works. *Evolutionary Human Sciences*, *3*, e10.

Sznycer, D., Sell, A., & Lieberman, D. (2021). Forms and functions of the social emotions. *Current Directions in Psychological Science*, *30*(4), 292-299.

Sznycer, D., Tooby, J., Cosmides, L., Porat, R., Shalvi, S., & Halperin, E. (2016). Shame closely tracks the threat of devaluation by others, even across cultures. *Proceedings of the National Academy of Sciences*, *113*(10), 2625-2630.

Takahashi, T., Sakaguchi, K., Oki, M., Homma, S., & Hasegawa, T. (2006). Testosterone levels and discounting delayed monetary gains and losses in male humans. *Neuroendocrinology letters*, *27*(4), 439-444.

Tenover, J. S., Matsumoto, A. M., Clifton, D. K., & Bremner, W. J. (1988). Age-related alterations in the circadian rhythms of pulsatile luteinizing hormone and testosterone secretion in healthy men. *Journal of Gerontology*, *43*(6), M163-M169.

Ter Horst, J. P., de Kloet, E. R., Schächinger, H., & Oitzl, M. (2012). Relevance of stress and female sex hormones for emotion and cognition. *Cellular and molecular neurobiology*, *32*, 725-735.

Tompkins, M. K., Bjälkebring, P., & Peters, E. (2018). Emotional aspects of risk perceptions. *Psychological perspectives on risk and risk analysis: theory, models, and applications*, 109-130.

Toufexis, D., Rivarola, M. A., Lara, H., & Viau, V. (2014). Stress and the reproductive axis. *Journal of neuroendocrinology*, *26*(9), 573-586.

Trofimova, I. N., & Gaykalova, A. A. (2021). Emotionality vs. other biobehavioural traits: a look at neurochemical biomarkers for their differentiation. *Frontiers in Psychology*, *12*, 781631.

Thomson, J. J. (1976). Killing, letting die, and the trolley problem. *The monist*, *59*(2), 204-217.

Tom, S. M., Fox, C. R., Trepel, C., & Poldrack, R. A. (2007). The neural basis of loss aversion in decision-making under risk. *Science*, *315*(5811), 515-518.

Toth, A. L., & Robinson, G. E. (2007). Evo-devo and the evolution of social behavior. *Trends in Genetics*, *23*(7), 334-341.

Tracy, J. L., Cheng, J. T., Robins, R. W., & Trzesniewski, K. H. (2009). Authentic and hubristic pride: The affective core of self-esteem and narcissism. *Self and identity*, *8*(2-3), 196-213.

Tracy, J. L., Shariff, A. F., & Cheng, J. T. (2010). A naturalist's view of pride. *Emotion Review*, *2*(2), 163-177.

Trautmann, S. T., & Van De Kuilen, G. (2015). Ambiguity attitudes. *The Wiley Blackwell handbook of judgment and decision making*, *2*, 89-116.

Trautmann, S. T., & van de Kuilen, G. (2018). Higher order risk attitudes: A review of experimental evidence. *European Economic Review*, *103*, 108-124.

Tversky, A., & Kahneman, D. (1974). Judgment under Uncertainty: Heuristics and Biases: Biases in judgments reveal some heuristics of thinking under uncertainty. *science*, *185*(4157), 1124-1131.

Tversky, A., & Kahneman, D. (1981). The framing of decisions and the psychology of choice. *science*, *211*(4481), 453-458.

Tversky, A., & Kahneman, D. (1992). Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and uncertainty*, *5*, 297-323.

Urbina, D. A., & Ruiz-Villaverde, A. (2019). A critical review of homo economicus from five approaches. *American Journal of Economics and Sociology*, *78*(1), 63-93.

Van Honk, J., Montoya, E. R., Bos, P. A., Van Vugt, M., & Terburg, D. (2012). New evidence on testosterone and cooperation. *Nature*, *485*(7399), E4-E5.

Van Lange, P. A., Joireman, J., Parks, C. D., & Van Dijk, E. (2013). The psychology of social dilemmas: A review. *Organizational Behavior and Human Decision Processes*, *120*(2), 125-141.

Van Wingen, G. A., Ossewaarde, L., Bäckström, T., Hermans, E. J., & Fernández, G. (2011). Gonadal hormone regulation of the emotion circuitry in humans. *Neuroscience*, *191*, 38-45.

Van't Wout, M., Kahn, R. S., Sanfey, A. G., & Aleman, A. (2006). Affective state and decision-making in the ultimatum game. *Experimental brain research*, *169*, 564-568.

Verbeke, W. J., Belschak, F., Ein-Dor, T., Bagozzi, R. P., & Schippers, M. (2018). Exploring the effect of attachment styles and winning or losing a status contest on testosterone levels. *Frontiers in Psychology*, *9*, 1051.

Vermeer, A. L., Krol, I., Gausterer, C., Wagner, B., Eisenegger, C., & Lamm, C. (2020). Exogenous testosterone increases status-seeking motivation in men with unstable low social status. *Psychoneuroendocrinology*, *113*, 104552.

Von Neumann, J., & Morgenstern, O. (1944/2007). Theory of games and economic behavior. In *Theory of games and economic behavior*. Princeton university press.

Wagels, L., Votinov, M., Kellermann, T., Eisert, A., Beyer, C., & Habel, U. (2018). Exogenous testosterone enhances the reactivity to social provocation in males. *Frontiers in Behavioral Neuroscience*, *12*, 37.

Wakker, P. P. (2010). *Prospect theory: For risk and ambiguity*. Cambridge university press.

Welker, K. M., Norman, R. E., Goetz, S., Moreau, B. J., Kitayama, S., & Carré, J. M. (2017). Preliminary evidence that testosterone's association with aggression depends on self-construal. *Hormones and Behavior*, *92*, 117-127.

Welker, K. M., Roy, A. R., Geniole, S., Kitayama, S., & Carré, J. M. (2019). Taking risks for personal gain: An investigation of self-construal and testosterone responses to competition. *Social Neuroscience*, *14*(1), 99-113.

Wellman, H. M. (2018). Theory of mind: The state of the art. *European Journal of Developmental Psychology*, *15*(6), 728-755.

Wilson, R. C., Geana, A., White, J. M., Ludvig, E. A., & Cohen, J. D. (2014). Humans use directed and random exploration to solve the explore–exploit dilemma. *Journal of Experimental Psychology: General*, *143*(6), 2074.

Wingfield, J. C. (2017). The challenge hypothesis: Where it began and relevance to humans. *Hormones and Behavior*, *92*, 9-12.

Wu, Y., Shen, B., Liao, J., Li, Y., Zilioli, S., & Li, H. (2020). Single dose testosterone administration increases impulsivity in the intertemporal choice task among healthy males. *Hormones and Behavior*, *118*, 104634.

Zavala, E., Wedgwood, K. C., Voliotis, M., Tabak, J., Spiga, F., Lightman, S. L., & Tsaneva-Atanasova, K. (2019). Mathematical modelling of endocrine systems. *Trends in Endocrinology & Metabolism*, *30*(4), 244-257.

Zethraeus, N., Kocoska-Maras, L., Ellingsen, T., Von Schoultz, B. O., Hirschberg, A. L., & Johannesson, M. (2009). A randomized trial of the effect of estrogen and testosterone on economic behavior. *Proceedings of the National Academy of Sciences*, *106*(16), 6535-6538.

Zilioli, S., & Bird, B. M. (2017). Functional significance of men’s testosterone reactivity to social stimuli. *Frontiers in Neuroendocrinology*, *47*, 1-18

Zuloaga, D. G., Puts, D. A., Jordan, C. L., & Breedlove, S. M. (2008). The role of androgen receptors in the masculinization of brain and behavior: what we've learned from the testicular feminization mutation. *Hormones and behavior*, *53*(5), 613-626.

Appendix A.

Demographics Questionnaire

Demographics Questionnaire

Please answer the following questions to the best of your ability. Your responses are strictly confidential and will be used for research purposes only. Your responses will be identified only by a confidential participation number.

1. How many rounds of the game do you think you played in total?
2. How many rounds of the game do you think resulted in gains/losses?
3. What is your age?
3. What is your sex?
4. What is your gender identity?
5. How many hours ago has it been since you consumed caffeine (coffee, tea, soda, chocolate)?
6. How many hours ago has it been since you consumed other drugs (including alcohol, nicotine, cannabis, or others)?
7. What is your weekly estimated physical activity (in hours)?
8. Are you currently taking any hormone supplements (including birth control)?
    a. if you take hormonal contraceptives, is it a combination of estrogen and progestin?
9. Are you currently taking any other prescription or nonprescription medications? (if yes, please list them)
10. What is your weight (please indicate kg or lbs):
11. What is your height:
12. How many years have you lived in Canada?
13. Sleep/week cycle (please indicate am/pm)
    a. What time do you normally wake up on weekdays
    b. What time do you normally wake up weekends?
    c. What time do you normally go to sleep on weekdays?
    d. What time do you normally go to sleep weekends?
    e. What time did you go to sleep last night?
    f. What time did you get up this morning?
    g. If you did not have to wake up because of external circumstances like school or work, when would you most prefer to wake up?
14. Is English your primary language?
15. What is your handedness?
16. Have you participated in another study in this lab this semester?
17. Do you menstruate?
    a. If yes:
        i. Is your cycle regular or irregular?
        ii. What is the average length of your cycle in days?
        iii. How many days has it been since your last period began?

Appendix B.

Chapter 2 ANOVA Summary Tables

Tests of Between-Subjects Effects

Dependent Variable: Total_Bet3s

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	10191.534	1	10191.534	5122.924	.009
	Error	1.989	1	1.989[a]		
condx	Hypothesis	12.876	1	12.876	.154	.762
	Error	83.423	1	83.423[b]		
sex	Hypothesis	1.989	1	1.989	.024	.902
	Error	83.423	1	83.423[b]		
condx * sex	Hypothesis	83.423	1	83.423	4.587	.035
	Error	1745.868	96	18.186[c]		

a. MS(sex)
b. MS(condx * sex)
c. MS(Error)

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	E2	Type III Sum of Squares	df	Mean Square	F	Sig.
E2	Linear	.015	1	.015	.725	.399
E2 * condx	Linear	.133	1	.133	6.556	.014
Error(E2)	Linear	.894	44	.020		

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	T	Type III Sum of Squares	df	Mean Square	F	Sig.
T	Linear	19342.769	1	19342.769	15.633	<.001
T * condx	Linear	11839.145	1	11839.145	9.568	.003
Error(T)	Linear	56917.466	46	1237.336		

a. sex = 1

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
C	Sphericity Assumed	.103	1	.103	32.826	<.001
	Greenhouse-Geisser	.103	1.000	.103	32.826	<.001
	Huynh-Feldt	.103	1.000	.103	32.826	<.001
	Lower-bound	.103	1.000	.103	32.826	<.001
C * condx	Sphericity Assumed	.013	1	.013	4.203	.043
	Greenhouse-Geisser	.013	1.000	.013	4.203	.043
	Huynh-Feldt	.013	1.000	.013	4.203	.043
	Lower-bound	.013	1.000	.013	4.203	.043
C * sex	Sphericity Assumed	.009	1	.009	2.982	.088
	Greenhouse-Geisser	.009	1.000	.009	2.982	.088
	Huynh-Feldt	.009	1.000	.009	2.982	.088
	Lower-bound	.009	1.000	.009	2.982	.088
C * condx * sex	Sphericity Assumed	5.193e-6	1	5.193e-6	.002	.968
	Greenhouse-Geisser	5.193e-6	1.000	5.193e-6	.002	.968
	Huynh-Feldt	5.193e-6	1.000	5.193e-6	.002	.968
	Lower-bound	5.193e-6	1.000	5.193e-6	.002	.968
Error(C)	Sphericity Assumed	.270	86	.003		
	Greenhouse-Geisser	.270	86.000	.003		
	Huynh-Feldt	.270	86.000	.003		
	Lower-bound	.270	86.000	.003		

Appendix C.

Chapter 3 ANOVA Summary Tables

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	T	Type III Sum of Squares	df	Mean Square	F	Sig.
T	Linear	4177.744	1	4177.744	20.343	<.001
T * condition1	Linear	75.086	1	75.086	.366	.549
Error(T)	Linear	7393.098	36	205.364		

a. sex = .00

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	T	Type III Sum of Squares	df	Mean Square	F	Sig.
T	Linear	1896.805	1	1896.805	1.941	.179
T * condition1	Linear	3499.008	1	3499.008	3.581	.073
Error(T)	Linear	19542.130	20	977.107		

a. sex = 1.00

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	E2	Type III Sum of Squares	df	Mean Square	F	Sig.
E2	Linear	.202	1	.202	10.370	.003
E2 * condition1	Linear	5.639e-5	1	5.639e-5	.003	.957
Error(E2)	Linear	.700	36	.019		

a. sex = .00

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	C	Type III Sum of Squares	df	Mean Square	F	Sig.
C	Linear	.036	1	.036	9.699	.004
C * condition1	Linear	.005	1	.005	1.473	.233
Error(C)	Linear	.133	36	.004		

a. sex = .00

Tests of Within-Subjects Contrasts[a]

Measure: MEASURE_1

Source	C	Type III Sum of Squares	df	Mean Square	F	Sig.
C	Linear	.010	1	.010	1.774	.198
C * condition1	Linear	.008	1	.008	1.536	.230
Error(C)	Linear	.111	20	.006		

a. sex = 1.00

Appendix D.

Chapter 4 ANOVA Summary Tables

Tests of Between-Subjects Effects

Dependent Variable: A_given_2

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	952.209	1	952.209	65.005	.079
	Error	14.648	1	14.648[a]		
condition1	Hypothesis	171.432	1	171.432	3882.542	.010
	Error	.044	1	.044[b]		
sex	Hypothesis	14.648	1	14.648	331.749	.035
	Error	.044	1	.044[b]		
condition1 * sex	Hypothesis	.044	1	.044	.004	.948
	Error	628.344	62	10.135[c]		

a. MS(sex)
b. MS(condition1 * sex)
c. MS(Error)

Tests of Between-Subjects Effects

Dependent Variable: T_change_percent

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	.147	1	.147	48.412	.091
	Error	.003	1	.003[a]		
condition1	Hypothesis	.037	1	.037	.101	.804
	Error	.365	1	.365[b]		
sex	Hypothesis	.003	1	.003	.008	.942
	Error	.365	1	.365[b]		
condition1 * sex	Hypothesis	.365	1	.365	10.373	.002
	Error	2.145	61	.035[c]		

a. MS(sex)

b. MS(condition1 * sex)

c. MS(Error)

Tests of Between-Subjects Effects[a]

Dependent Variable: E_change_percent

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.140[b]	1	.140	5.993	.019
Intercept	.178	1	.178	7.643	.008
condition1	.140	1	.140	5.993	.019
Error	.980	42	.023		
Total	1.348	44			
Corrected Total	1.119	43			

a. sex = .00

b. R Squared = .125 (Adjusted R Squared = .104)

Tests of Between-Subjects Effects

Dependent Variable: C_change_percent

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
Intercept	Hypothesis	.135	1	.135	1.148	.478
	Error	.117	1	.117[a]		
condition1	Hypothesis	.001	1	.001	.011	.935
	Error	.122	1	.122[b]		
sex	Hypothesis	.117	1	.117	.957	.507
	Error	.122	1	.122[b]		
condition1 * sex	Hypothesis	.122	1	.122	1.590	.212
	Error	4.622	60	.077[c]		

a. MS(sex)
b. MS(condition1 * sex)
c. MS(Error)