

HUNGER, SATIETY, AND DECISION-MAKING: A REVIEW AND EXTENSION

A Dissertation

Presented to

The Faculty of the C.T. Bauer College of Business

University of Houston

In Partial Fulfillment

Of the Requirements for the Degree

Doctor of Philosophy

By

Rob Austin McKee

June, 2016

HUNGER, SATIETY, AND DECISION-MAKING: A REVIEW AND EXTENSION

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ABSTRACT

Illuminated by diverse fields such as neuroscience, physiology, gastroenterology, gastronomy, nutrition, and economics, the present study explores the roles of hunger and satiety in various decision-making contexts, including temporal discounting, risk propensity, and (un)ethicality. Through this integration of broad research streams, I aspire to achieve some degree of consilience within the literature and to help broaden the scope of organizational behavior by considering robust findings from nontraditionally-related fields. In order to accomplish this, a research study was conducted wherein participants, having fasted overnight, arrived at the experimental setting and were randomly assigned to either a hunger or a satiety condition. I hypothesized that being in a state of hunger (versus satiety) would cause people to 1) discount the future more (or more strongly desire more imminently available rewards), 2) exhibit higher risk propensity, 3) act more unethically (i.e., cheat more), and 4) act less extraordinarily ethically (i.e., give less money to charity). Despite participants' lack of compliance with the experimental manipulation resulting in a lower-than-intended strength of manipulation, Hypothesis 2 was supported. Fluctuating states of hunger and satiety occur naturally as a consequence of the myriad physiological, environmental, and subjective determinants of eating behavior. Because we generally fast overnight and sometimes skip meals, such states are part of our everyday lives. Thus, this study has important practical implications for managerial practice, ranging from managing personal decision-making to managing the decision-making of others, including when to schedule meetings and negotiations around meal times considering the mindset most appropriate (e.g., risk-seeking versus riskaverse) given the outcome of preference.

TABLE OF CONTENTS

1. INTRODUCTION	1
2. VISCERAL STATES	11
(Reverse) Alliesthesia	15
3. VALUE AND REWARD IN DECISION-MAKING	20
Neurological substrates of value and reward	23
Food as reward	29
Neurochemical basis of food as reward	34
4. TEMPORAL DISCOUNTING.	42
Neurological substrates of temporal discounting	52
Visceral states and temporal discounting	56
Hunger and temporal discounting	57
5. RISK PROPENSITY	60
Neurological substrates of risk propensity	61
Visceral states and risk propensity.	62
Hunger and risk propensity	63
6. (UN)ETHICALITY	65
7. METHODS.	70
Participants	71
Recruitment	71
Requirements to participate in this study	73

Additional information regarding the participant recruitment process76
The Study76
Set up
Check-in procedures
Experimental manipulations
Rationale for using the nutritional shakes and bars85
The Survey Instrument85
Introduction to the online survey instrument85
Informed consent
Measures
Temporal Discounting
Risk Propensity
(Un)Ethicality88
Hunger 90
Conclusion of the online survey instrument92
Post-task meal93
The raffle93
8. ANALYSES95
9. RESULTS
10. DISCUSSION
11. CONCLUSION
REFERENCES. 114

LIST OF TABLES

1.	Nutritional Data for Experimental Conditions	86
2.	Correlation Matrix of Key Variables.	.100
3.	Instrumental Variable Regression Estimates	.101
4.	Wald Tests for Equations.	102

LIST OF FIGURES

1.	Hyperbolic discount curves from Ainslie (2012: 7)	48
2.	"Hyperboloid" or "quasihyperbolic" discount curves from Ainslie (2012: 9)	.49
3.	Explanatory Model of the Effect of Hunger on Temporal Discounting, Risk	
	Propensity, and (Un)ethicality	69

Chapter 1

INTRODUCTION

"An empty stomach is not a good political adviser." – Albert Einstein (quoted in Scanes, 2010: 4)

There is a small but growing literature examining topics such as motivation, decision-making, and behavior in light of visceral physiological states such as hunger, thirst, and sexual desire, and related factors such as moods, emotions, fatigue, pain, somnolence, and cravings related to alcohol, cigarette, drug, and food addiction (e.g., Ditto, Pizarro, Epstein, Jacobson, & MacDonald, 2006; Loewenstein, 1996; Loewenstein & O'Donoghue, 2004; Nordgren, van der Pligt, & van Harreveld, 2007, 2008). Such seemingly "extraneous" factors have been shown to affect even expert decision makers in highly consequential realms, such as judicial decisions made during parole board hearings.

In a provocative study, Danziger, Levav, and Avnaim-Pesso (2011) examined the favorability of rulings by judges in parole decisions and parolee requests (see also Danziger, Levav, & Avnaim-Pesso, 2011; Weinshall-Margel & Shapard, 2011). The judges' work days were naturally divided into three individual decision sessions, demarcated by two food breaks, during which the judges ate a meal and were able to

relax. The authors argued that, in doing so, they were also able to reduce cognitive fatigue, which may have concomitantly improved their moods. The authors showed that the percentage of favorable rulings dropped from approximately 65% at the beginning of a given decision session to nearly zero by the end of the session, only to abruptly return to approximately 65% immediately following the ensuing break period. This cycle of waning and waxing decision favorability before and after breaks was observed within and across days in which parole board hearings were conducted. The authors stressed the underappreciated and serious implications of visceral factors like hunger, fatigue, and mood on decision-making processes, which yet remain underexplored in the literature.

Studies like this have helped establish a guidepost for future researchers, though there are many paths forward. The present study forges onward into the realm of organizational behavior, illuminated by neuroscience, physiology, gastroenterology, gastronomy, nutrition, and economics, as well as popular theories of motivation and decision-making. In this study, I explore the role of visceral states, specifically hunger and satiety, in various decision-making, motivational, and behavioral contexts, including temporal discounting (i.e., time preferences), risk propensity, and (un)ethicality.

Through this integration of broad research streams, I aspire to achieve some degree of consilience, or a "'jumping together' of knowledge by the linking of facts and fact-based theory across disciplines to create a common groundwork of explanation" (Wilson, 1998: 8). This concept has been called upon to support the convergence of neuroscience with fields such as economics (Glimcher & Rustichini, 2004) and accounting (Dickhaut, Basu, McCabe, & Waymire, 2010), as well as various theories of motivation and decision-making, including temporal discounting, expectancy theory,

cumulative prospect theory, and needs theory (Steel & Konig, 2006). I call upon this concept to support the convergence of organizational behavior with fields as diverse as neuroscience, economics, gastroenterology, and nutrition.

In order to understand how visceral states such as hunger and satiety affect the processes by which we engage in valuation computations of rewards and, relatedly, how we actually make choices among myriad available rewards, as well as how we respond to rewards we anticipate and/or receive, it is helpful to turn to neuroscience. Neuroscience is "an interdisciplinary field of study [that] seeks to understand behavioral phenomenon in terms of the brain mechanisms and interactions that produce cognitive processes and behavior" (Becker & Cropanzano, 2010: 1055; see also Ochsner & Lieberman, 2001). Delving into this informative literature is a partial yet sincere response to a call by numerous organizational science researchers (e.g., Becker & Cropanzano, 2010; Becker, Cropanzano, & Sanfey, 2011; Powell, 2011; Volk & Kohler, 2012) for greater integration of neuroscience with the organizational sciences, reminiscent of newly minted domains such as neuropsychology, neuroeconomics, and neuromarketing. It is also a response to researchers who discuss and critique the role of neuroscience in management research (e.g., Ashkanasy, Becker, & Waldman, 2014; Lee, Senior, & Butler, 2012; Lindebaum & Jordan, 2014; Powell, 2011; Senior, Lee, & Butler, 2011), because although no neuroimaging (e.g., functional magnetic resonance imaging, or fMRI; for a brief discussion, see Kable, 2011) is conducted in this study, this study still represents movement in that direction in its use of well-supported findings from the field of neuroscience to build theory and generate testable hypotheses. Indeed, such use of findings from existing neuroeconomic studies is recommended as a first step for integrating neuroscience with organizational science research (Volk & Kohler, 2012).

There are several reasons to begin this integration passively rather than by actively and immediately attempting to gather our own fMRI data. First, because these techniques are at the leading edge of technical sophistication, we first need to learn the basics of neuroscience, including the terminology, which can be accomplished by delving into the neuropsychology, neuroeconomics, and neuromarketing literatures. This paper addresses this issue by using language native to these neuroscience-inspired literatures, including the names of relevant brains structures and neurochemicals, providing definitions when needed. Secondly, we may need to convince our own scholars, researchers, and, perhaps more importantly, editors of the value of such approaches. As cognitive psychologists have (perhaps jokingly) asked, "Why should we study the brain in order to understand the mind?" (Kable, 2011). Thus, an important debate is likely necessary at the field level regarding the incorporation of these techniques as has occurred in economics (e.g., Camerer, 2007, 2008; Camerer, Loewenstein, & Prelec, 2005; Glimcher, 2010; McCabe, 2008) and psychology (e.g., Henson, 2005; Page, 2006), and even our own field regarding, for instance, alternatives to null hypothesis significance testing (e.g., McKee & Miller, 2015; Zyphur & Oswald, 2013). Thus, this second reason for initially passive integration with neuroscience represents a goal in the longer term toward which this paper aspires to contribute. Thirdly, we need to show the wider scientific community that we are capable of interpreting and integrating existing neuroscientific findings into our literature as a precursor to collaboration with

experienced neuroscientists. This is an important consideration because it is easy to misinterpret these findings.

This third reason for initially passive integration is of particular concern to the present research endeavor because much focus is given here to interpreting and incorporating neuroscientific findings. To this point, fMRI data are commonly misused when making inferences about psychological processes (Kable, 2011; Powell, 2011). This occurs when assumptions are made concerning the function(s) of a brain region based on past studies and then inferring that because the experimental task(s) activated the same region, the task must elicit some particular psychological process. "For example, one might find that decisions under risk are associated with activity in the amygdala, note that the amygdala has previously been implicated in the emotion of fear, and then conclude that decisions under risk involved fear" (Kable, 2011: 78). There are at least two problems here. First, a one-to-one mapping of a given psychological process to a specific brain region is highly unlikely; second, as a consequence of that unlikelihood, the "reverse inference" reasoning exhibited in this example is simply "not deductively valid." (Poldrack, 2006: 59; see also Aguirre, 2003; Aguirre & D'Esposito, 1999; Ariely & Berns, 2010; Henson, 2006). However, reverse inference can still provide some valuable information to researchers. That is, reverse inference is an imperfect tool that can be used to help generate novel hypotheses that can then be tested experimentally; it should not be used as a means for post hoc explanation of some particular finding (Poldrack, 2006; Poldrack & Wagner, 2004). Despite these cautions, "fMRI can [generally] demonstrate whether two different kinds of decisions use similar or different neural processes, and thus whether they are likely to use similar or different cognitive

processes" (Kable, 2011: 78). Championing the overall trend toward increased integration, Volk and Kohler (2012) argue in support of the external validity and generalizability of neuroeconomic approaches and findings. Thus, only as a basis for theory building and hypothesis generation, this paper confidently uses well-supported findings from the neuroscience literature, being as diligent as possible to point out controversies and debates where they exist, as was done in this paragraph.

For this research endeavor, we also need to understand the effects of hunger and satiety on neurological and physiological processes. This is necessary because our relationship with food is distinguished from that of monetary and material rewards in that food, like water, sex, and shelter, is a primary reinforcer, directly satisfying a physiological or visceral need (e.g., the maintenance of homeostasis or reproduction), whereas monetary, material, and social rewards are secondary reinforcers, which may require conditioned learning because they are only indirectly and abstractly related to physiological needs and survival (Epstein, Salvy, Carr, Dearing, & Bickel, 2010; McClure, Ericson, Laibson, Loewenstein, & Cohen, 2007; Sescousse, Caldú, Segura, & Dreher, 2013). Moreover, food is distinguished from other types of primary rewards in that it both elicits and responds to a cascade of unique hormones (e.g., ghrelin, leptin, insulin), and shifts blood glucose levels (Begg & Woods, 2013; Korakianiti, Hillier, & Clegg, 2014; Page et al., 2011; Patton & Mistlberger, 2013; Schwartz, Woods, Porte, Seeley, & Baskin, 2000). Like neuroscience, gastroenterology (the study of the digestive system and related pathologies), gastronomy (the art of and culture surrounding the selection, cooking, and eating of foods), and nutrition are somewhat esoteric literatures

and, as such, many of the same issues regarding terminology and interpretation of results are relevant.

Particular to food, fluctuating states of hunger and satiety occur naturally as a result of the myriad physiological, environmental, and subjective determinants of eating behavior (de Castro, 1988). States of hunger resulting from fasting may occur for a variety of externally imposed reasons, including medical procedures (Nygren, 2006; Watson & Rinomhota, 2001) or more dire circumstances such as poverty, food shortages, or natural disasters (Lumey et al., 2007; Stein, Susser, Saenger, & Marolla, 1975), as well as for a variety of internally imposed reasons, including dieting (Grigg, Bowman, & Redman, 1996; Story et al., 1991) and religious observances (Frost & Pirani, 1987; Karaagaoglu & Yucecan, 2000; Sarri, Linardakis, Bervanaki, Tzanakis, & Kafatos, 2004; Trepanowski & Bloomer, 2010). Although people essentially self-impose fluctuating states of hunger and satiety, the circadian nature of these states also follows culturallyestablished meal times (i.e., breakfast upon waking, lunch in the afternoon, and dinner in the evening) (Chiva, 1997; Kant & Graubard, 2015; Larson, Branscomb, & Wiley, 2006; Sobal & Nelson, 2003), as well as other diurnal, hebdomadal (i.e., weekly), and seasonal rhythms (Baron, Reid, Kern, & Zee, 2011; de Castro, 1987, 1988, 1991a, 1991b; Scheer, Morris, & Shea, 2013). More germane to the organizational behavior literature, hunger can occur as a result of poor work-life balance (Devine et al., 2006, 2009). More generally, because we commonly fast overnight and sometimes skip meals, states of hunger and satiety are part of our everyday lives (Scheer et al., 2013; Tal & Wansink, 2013; Wansink, Tal, & Shimizu, 2012).

Thus, due to the prevalence of hunger and satiety, this study has important practical implications ranging from managing personal decision-making to managing the decision-making of others, including when to schedule meetings and negotiations around meal times considering the mindset most appropriate (e.g., risk seeking versus risk averse) given the outcome of preference. One of the phenomena studied herein, temporal discounting, involves intertemporal choices, which are "decisions with consequences that play out over time. These choices range from the prosaic – how much food to eat at a meal – to life changing decisions about education, marriage, fertility, health behaviors and savings. Intertemporal preferences also affect policy debates about long-run challenges, such as global warming" (Berns, Laibson, & Loewenstein, 2007: 482). Importantly, these intertemporal choices seem to be influenced by both neurological and metabolic processes (i.e., hunger and satiety). Another phenomenon studied herein, risk propensity, also seems to have neurological and metabolic underpinnings. "Understanding the neural basis of choice under uncertainty is important because it is a fundamental activity at every societal level, with examples as diverse as people saving for retirement, companies pricing insurance, and countries evaluating military, social, and environmental risks" (Hsu, Bhatt, Adolphs, Tranel, & Camerer, 2005). Yet another phenomenon, (un)ethicality, underlies concepts like organizational deviance and fraud. These behaviors have important financial implications (Mazar, Amir, & Ariely, 2008). The 2014 Report to the Nations on Occupational Fraud and Abuse (2014) provides some disquieting statistics, estimating a projected \$3.7 trillion in global occupational fraud loss for the year 2013. Of reported cases, 42% were perpetrated by employees, causing a median loss of \$75,000, 36% were perpetrated by managers, causing a median loss of \$130,000, and 19% were caused by owners/executives, causing a median loss of \$500,000. These financial losses are, of course, in excess of the psychological effects endured by the victims of occupational fraud (Ganzini, McFarland, & Bloom, 1990). Considering these wide-ranging domains, this study considers applied and theoretical issues of great consequence. As such, it is essential to understand the factors that may (perhaps unexpectedly) influence such decisions, both from a personal and an organizational perspective. Considering these implications, this paper makes several important contributions to the organizational science literature in its consideration of hunger and satiety as potent influential states.

In making these contributions, this paper represents a review and extension of research in the area of visceral states and motivation, decision-making, and behavior. The review conducted here is important because most other studies proffering hypotheses similar to those presented here have provided only partial evidence as to why those hypotheses were deductively valid. The literature review conducted for this study delves into the neurobiology and physiology underlying the hypotheses, bridging gaps between many literatures to provide a more objective and nuanced narrative than has been heretofore constructed. This study is somewhat unorthodox for the organizational science community in several key ways. Firstly, as stated above, it calls upon numerous nontraditionally-related disciplines to build theory and hypotheses. Indeed, the reader will find relatively few references to the authors and journals most familiar to the organizational science readership. Secondly, this study goes a step beyond the deductive practice of using theory to derive hypotheses. Rather, it makes use of a diverse collection of findings to cultivate new theory from which the hypotheses are developed. In this

sense, it includes both deductive as well as inductive elements. Finally, this study extends the scope of current organizational behavior literature while simultaneously contributing to the neuroscience, physiology, gastroenterology, gastronomy, nutrition, economics, and decision-making literatures from which much of its substance is derived.

The remainder of the paper is organized as follows: First, I introduce the concept of visceral states and how they may specifically and generally affect motivation, decision-making, and behavior. I then discuss valuation computations of reward including neurological substrates of reward and a special section on food as reward, including its neurochemical basis. Following that I introduce the concept of temporal discounting, including neurological substrates and how visceral states affect this key decision criterion, building up to the hypothesized effects of hunger and satiety on temporal discounting. Using similar argumentation, I then develop hypotheses regarding risk propensity in light of hunger and satiety. That section is followed by theory and hypotheses relating hunger and satiety to (un)ethicality. I then provide description of methodology used for this study, after which analyses and results are discussed. I conclude with a discussion about the study and results.

Chapter 2

VISCERAL STATES

"An empty stomach makes a fierce dog." – Robert Falcon Scott (Scott & Huxley, 1913: 109)

Per Loewenstein (1996: 272), "the defining characteristics of visceral factors are, first, a direct hedonic impact (which is usually negative), and second, an effect on the relative desirability of different goods and actions." That is, visceral factors cause a state of internal tension that may be subjectively pleasant or unpleasant, but that ultimately seeks release, and are salient at a given time only if they are activated at that time (i.e., one is currently hungry, thirsty, sexually aroused, craving heroin, etc.). Interestingly, these visceral states may be relevant not only to the specific rewards or cues that correspond to the instigating state (e.g., desiring food when one is hungry), but also more generally to domains that are unrelated to the instigating state (e.g., one is more or less responsive to monetary rewards when one is hungry or sexually aroused, see Briers, Pandelaere, Dewitte, & Warlop, 2006; Van den Bergh, Dewitte, & Warlop, 2008). This latter consideration will be discussed in detail in upcoming sections of this paper as it provides a central premise for the current research effort. Thus, visceral states may

exhibit both specific and general effects in realms such as motivation, decision-making, and behavior.

Rewards or cues specific to an instigating visceral state are easy to imagine and underlie conventional wisdom that warns us, for instance, against grocery shopping while hungry (Inman, 2015). Within the realm of food, much of this literature focuses on food choices made by calorie-deprived (i.e., fasting) individuals compared to sated individuals, finding that members of the former group seek out more food or more calorie-dense foods when eating or grocery shopping, at least among normal-weight individuals (e.g., Gilbert, Gill, & Wilson, 2002; Goldstone et al., 2009; Kirk & Logue, 1997; Mela, Aaron, & Gatenby, 1996; Nisbett & Kanouse, 1969; Read & van Leeuwen, 1998; Tal & Wansink, 2013; Wansink et al., 2012) or, more broadly, on self-control and impulsiveness regarding food as a reward or reinforcing stimulus (e.g., Epstein et al., 2010; Forzano & Logue, 1992; Kirk & Logue, 1997; Logue & King, 1991). In another domain, it has been found that when comparing sexually-aroused to neutral (i.e., nonaroused) individuals, members of the former group make more morally questionable and riskier decisions regarding sexual acts (Ariely & Loewenstein, 2006; Blanton & Gerrard, 1997; Ditto et al., 2006). There is an important caveat to report, however; most of these types of studies' samples were comprised entirely of male heterosexual undergraduate students, so the results may not be broadly generalizable (for an exception, see Imhoff & Schmidt, 2014). Still, this adds to the evidence showing that whether one is or is not in a given visceral state has important implications for how state-relevant cues and rewards are interpreted and pursued.

Rewards or cues unrelated to an instigating visceral state are perhaps less easy to imagine, though nonetheless important to consider. On one hand, some researchers have contended that decision-making constructs such as temporal discounting (a phenomenon wherein people prefer more imminent rewards to more delayed rewards to such an extent that they are often willing to accept smaller rewards received sooner over larger rewards received later, discussed in detail below) should apply only to cues or rewards directly associated with the visceral factor being considered, which leads to the assumption that, for instance, a hungry person would have the same discount rate toward monetary and other material (i.e., noncaloric) outcomes as would a sated person (Kirk & Logue, 1997; Loewenstein, 1996; Read & van Leeuwen, 1998). On the other hand, in opposition to such assumptions, there is a small literature that extends the examination of the effects of visceral states such as hunger and sexual arousal beyond cognitions and decisions involving food and sex, respectively. For instance, there appears to be a strong connection between the desire for food and the desire for monetary and material rewards (Briers & Laporte, 2013; Briers et al., 2006; Gal, 2012; Reuben, Sapienza, & Zingales, 2010). More broadly, hunger has been shown to impact a variety of cognitive and decision-making processes related to impulsivity, risk-taking, and economic decisions (e.g., de Ridder, Kroese, Adriaanse, & Evers, 2014; Ditto et al., 2006; Levy, Thavikulwat, & Glimcher, 2013; Nordgren et al., 2007, 2008; Symmonds, Emmanuel, Drew, Batterham, & Dolan, 2010).

There is also a small literature demonstrating that exposure to sexual cues influences aspects of economic decision-making related to impulsivity and temporal discounting, desire for monetary and material rewards, risk taking, and spending on

conspicuous and costly consumer goods (Baker Jr. & Maner, 2008; Griskevicius et al., 2007; Roney, 2003; Van den Bergh & Dewitte, 2006; Van den Bergh et al., 2008; Wilson & Daly, 2004). As before, these studies often sample only male heterosexual undergraduate students using visual stimuli (e.g., pictures of attractive women), potentially limiting their generalizability. However, it has been shown that men and women differ in their response to visual sexual stimuli (Hamann, Herman, Nolan, & Wallen, 2004), prompting a study by Festjens, Bruyneel, and Dewitte (2013) which found that women respond to tactile sexual cues (in this case, touching a pair of men's boxer shorts) in such a way as to make them less loss averse regarding food and money outcomes and more reward-seeking regarding economic outcomes. Based on this evidence, it seems the effects of a given visceral state are at least to some degree generalizable to cues and rewards not directly related to that state.

As Claudio in Shakespeare's play *Measure for Measure* opined, "surfeit is the father of much fast" (1603: 1.2.118). Although it is implicit in the preceding discussion, it should perhaps be stated explicitly that satiation seems to attenuate the effects of visceral states (it should probably be noted that there is some technical distinction between satiation and satiety (e.g., De Graaf, De Jong, & Lambers, 1999), though the terms are used synonymously in this text). For instance, Van den Bergh, Dewitte, and Warlop (2008: 94) found that "sexual appetite induces monetary cravings" as evinced by increased temporal discounting of monetary outcomes. Supporting the generalizability of visceral states to ostensibly unrelated cues and rewards, this impatience for reward when sexually aroused extended to nonmonetary rewards such as candy and soda. However, the authors found that "satiation" of sexual appetite attenuated these discount effects. This

satiety effect is also pertinent to hunger; physiological satiety induced by eating affects attitudes toward food (Gilbert et al., 2002; Goldstone et al., 2009; Kirk & Logue, 1997) as well as monetary reinforcers (Briers et al., 2006). This is consistent with findings from studies on goals and motivation which show that, while "motivational states, such as needs, goals, intentions, and concerns are characterized by enhanced accessibility of motivation-related constructs" (Förster, Liberman, & Higgins, 2005: 220), these motivational states and constructs can be suppressed by the fulfillment of the appropriate motivation(s) or goal(s) (Briers et al., 2006; Förster et al., 2005; Liberman & Förster, 2000, 2005; Marsh, Hicks, & Bink, 1998). As a further point that will be applicable later in this narrative, unfulfilled goals may interfere with higher-order executive functioning (Marien, Custers, Hassin, & Aarts, 2012; Masicampo & Baumeister, 2011).

In summary, we must account for the fact that a given visceral state may affect motivation, decision-making, and behavior vis-à-vis cues and rewards corresponding to that specific state, but also cues and rewards seemingly unrelated to that state. I begin this treatise via a discussion of (reverse) alliesthesia, followed by a discussion of classes of rewards, and then a primer on temporal discounting, risk propensity, and (un)ethicality.

(Reverse) Alliesthesia

As Toates (1981: 37) stated, "what is a high incentive in one energy state is a low incentive in another." A sort of counterpoint to visceral states, alliesthesia refers to the phenomenon wherein the sensation (i.e., the hedonic evaluation of pleasure or displeasure) associated with some stimulus varies as a function of the inner state (i.e., the visceral or drive state) of an individual (Berridge, 2000; Cabanac, 1979, 1988; Jiang et al., 2008). In other words, deprivation of some reinforcer can increase the motivation to

obtain or consume said reinforcer. As an example, the sight, smell, taste, and mouthfeel of a spoonful of crème brûlée should be more pleasing to someone who is hungry than to someone who is sated. Thus, the concept of alliesthesia rests on the notion that there is a decreasing marginal utility associated with further exposure to some appetitive stimulus and, moreover, that this utility may even become negative if exposure extends beyond the point of satiation (this would seem to resemble a curvilinear, inverted-U relationship between exposure and hedonic evaluation). Cabanac (1979) argues that such effects are domain specific (i.e., temperature should not affect alliesthesia related to food), though there is some evidence that this may not be the case (for examples relating temperature to food intake, see Briers & Lerouge, 2011; Herman, 1993; Johnson, Mavrogianni, Ucci, Vidal-Puig, & Wardle, 2011).

However, at least within the realm of food and drink, the narrative may be more nuanced. It could be argued that per the theory of alliesthesia, as well as common and perhaps even expert intuition, consumption of a small amount of some food or drink should decrease hunger or thirst as well as subsequent consumptive behaviors (see Wadhwa, Shiv, & Nowlis, 2008). With respect to food and drink consumption, this represents an energy depletion model (discussed in Epstein, Leddy, Temple, & Faith, 2007). However, it also seems reasonable that consumption of a small amount of some food or drink could *increase* hunger or thirst along with subsequent consumptive behaviors (Berridge, 2000; Toates, 1986; Wadhwa et al., 2008). For instance, building upon the notion that "the appetite is sharpened by the first bites" (José Rizal (1861-1896), quoted in Rattiner, 2002: 32), Brendl, Markman, and Messner (2003: 467) argue that "the physiological need to eat can be stimulated by first letting people taste a very small

quantity of food to prompt an appetizing effect" (see also Cornell, Rodin, & Weingarten, 1989; Rodin, 1985; Yeomans, 1996, 1998). This effect is similar to the priming effect described for food and addictive drugs by de Wit (1996), as well as by Pavlov as early as 1897 (Pavlov, 1897). The fact that hunger increases during the early stages of eating a meal does not necessarily mean that more food is consumed, but merely that subjective ratings of hunger increase (Yeomans, 1996, 1998; Yeomans, Gray, Mitchell, & True, 1997). However, this effect may extend beyond the subjective realm as will be discussed with respect to cephalic phase responses later in the section on food and reward.

Integrating lines of inquiry associated with alliesthesia and the appetizer effect, Wadhwa et al. (2008) explored whether sampling a small quantity of a high incentive value consumptive cue (i.e., a good-tasting food or drink) would further increase consumption of that particular cue as well as consumption more generally. Thus, "just as drive states affect the incentive value of relevant rewarding stimuli, encountering stimuli high in incentive value could intensify motivational states and thus enhance the desire to engage in subsequent consumption-related behaviors" (Wadhwa et al., 2008: 403). The first portion of this statement represents the concept of alliesthesia. The authors term the phenomenon represented in the second portion of the statement "reverse alliesthesia," and relate it to cue-specific, drive-specific, and general motivational effects. With cuespecific effects, sampling a spoonful of crème brûlée would only increase the desire for more crème brûlée, whereas with drive-specific effects, sampling a spoonful of crème brûlée would more broadly increase the desire for more dessert or food (i.e., it would increase overall hunger). With general motivational effects, limited consumption of some high incentive cue like the spoonful of crème brûlée would be expected to increase desire

for a broad range of reward cues, including those not related to food (e.g., a massage or vacation). Moreover, the authors showed that aversive consumption cues (e.g., an unpleasant smell) might suppress subsequent reward-seeking behaviors.

Reverse alliesthesia clearly parallels much of the above-described literature on visceral states. However, it differs in at least two important ways. First, this literature does not typically consider the existence of preactivated visceral states. In fact, some studies cited as evidence of reverse alliesthesia went so far as to examine the effect of hedonic cues despite satiation of the relevant drive state (e.g., eating pizza or ice cream when one's appetite is already sated, see Cornell et al., 1989). This is not to say that satiety is a neutral state that has no effect on reward-seeking motivation and behavior within the context of reverse alliesthesia. Indeed, in the study cited above, Wadhwa et al. (2008) were able to reduce future food consumption related to an induced state of hunger by "satiating" participants with a small surprise monetary reward during an intervening task in the experiment.

The second difference is that with reverse alliesthesia, exposure to some stimuli that would, given sufficient quantity, typically reduce (i.e., satiate) a related drive state, actually increases the drive state when the quantity is limited and, moreover, may activate or increase the potency of unrelated drive states. In this case, a high incentive value consumptive cue actually can instigate specific and general drive states. It is this distinction that makes reverse alliesthesia a valuable contribution to this discussion. In this regard, it is interesting to note that, having mentioned a spoonful of crème brûlée several times in the preceding paragraph, the literature on reverse alliesthesia and the appetizer effect suggests that the reader would now be expected to exhibit temporarily

greater sensitivity to a wide range of reward cues, perhaps even despite having eaten recently, provided the reader enjoys crème brûlée, of course. This enhanced sensitivity may be evinced through key decision-making constructs such as temporal discounting and risk propensity, which will be examined after a brief discussion of value and reward in decision-making.

Chapter 3

VALUE AND REWARD IN DECISION-MAKING

An important question arises as to why phenomena such as visceral states and reverse alliesthesia are relevant not only to the specific rewards or cues that correspond to the instigating state, but also more generally to domains that are unrelated to the instigating state. It could reasonably be argued that need- or goal-directed behavior toward an object or outcome should only be increased if the object or outcome is capable of satisfying an active need or goal and, furthermore, that in light of such an active need or goal, attention toward all other objects and outcomes should be suppressed (Yam, Reynolds, & Hirsh, 2014). This "devaluation effect" involved in suppressing attention toward objects and outcomes unrelated (i.e., neither instrumental nor counterproductive) to a drive state seems theoretically sound and has some empirical support (Brendl et al., 2003). However, outcome types, degrees of relatedness, and levels of abstraction must certainly be considered when evaluating a set of potential alternatives relative to a given drive state. As an example, a visceral state such as hunger almost certainly alters motivation, decision-making, and behavior in such a way as to promote caloric intake, perhaps causing the devaluation of rewards and cues such as money. However, food and money are related in that they are exchangeable; money is a conditioned and more abstract means to achieving the same end. Thus, it is conceivable that the incentive

salience of money would be increased in a state of hunger. Regarding the evaluation of and selection among potential alternatives, especially when considering contextual factors such as visceral states, a large body of research has begun to illuminate the intricacies of decision-making processes.

"In the simplest terms, human decision-making can be framed as an energetic problem that pits an organism's investment for each choice against the immediate and long-term returns expected" (Montague, King-Casas, & Cohen, 2006: 418). In this paper, I am concerned with economic choice, in which the options available in different situations can vary on a multitude of dimensions, which necessitates the neural representation and computation of value (Padoa-Schioppa, 2011). In other words, in order to make decisions, the brain must represent the set of available choices and calculate the respective desirability of each. In this regard, it is helpful to distinguish between two closely related terms: reward and value. Reward refers to the "immediate advantage accrued from the outcome of a decision (e.g., food, sex, or water)" (Montague et al., 2006: 419). Reward occurs in the temporal present, is relatively easy to measure and quantify, and is usually experimentally operationalized as some positive reinforcer. The converse of reward is punishment, some immediate disadvantage accrued from the outcome of a decision. Value, on the other hand, is an estimate of or expectation regarding the degree of reward or punishment that will result from the decision, both now as well as in the future. Thus, value is computed at the time of choice and is subject to myriad contextual and circumstantial determinants that can be either external (e.g., the commodity, quantity, delay, risk, ambiguity, and cost) or internal (e.g., motivational state, impatience, attitudes toward risk and ambiguity) to the individual (Padoa-Schioppa, 2011). Per this framework, the commodity term refers to a unitary amount of some specified good independent of all other contextual and circumstantial determinants. These concepts are important because one aim of the current study is to manipulate an internal motivational state (i.e., hunger) experimentally to see what effect it has on select external determinants (e.g., delay, risk) of valuation computations. I will present a series of equations relating reward to value in the upcoming section on temporal discounting.

Within this discussion and as a preface to upcoming sections, it is also helpful to distinguish between two types of reinforcers: primary and secondary. Whereas primary reinforcers directly satisfy some evolved appetitive mechanism (i.e., a visceral state such as hunger, thirst, or sexual desire), secondary reinforcers "may evoke limbic activity only indirectly, mediated by more abstract symbolic and/or associative processes that may be more susceptible to contextual framing effects" (McClure et al., 2007: 5803). That is to say, rewards such as money, a secondary reinforcer, may be more subject to framing effects than rewards such as food, a primary reinforcer. Berridge (2007: 393) briefly discusses reinforcement terminology more broadly. In correspondence with the idea of primary and secondary reinforcers, researchers have found, for instance, higher temporal discounting (i.e., an increased preference for more imminent rewards even when they are smaller in magnitude than more delayed rewards) for heroin than money (Giordano et al., 2002; Madden, Bickel, & Jacobs, 1999), as well as higher discounting of alcohol and food relative to money, regardless of reward amount (Odum, Baumann, & Rimington, 2006; Odum & Rainaud, 2003), lending credibility to the idea that rewards that are directly consumable are discounted more steeply than rewards that are not directly consumable (i.e., monetary rewards). Perhaps unsurprisingly, college students appeared

to discount a host of consumables (e.g., beer, candy, and soda) more steeply than money (Estle, Green, Myerson, & Holt, 2007).

Within this discussion it is also helpful to distinguish between "three dissociable psychological components of reward: 'liking' (hedonic impact), 'wanting' (incentive salience), and learning (predictive associations and cognitions)" (Berridge, Robinson, & Aldridge, 2009: 65). The term 'dissociable' is important because recent research indicates that these phenomena are at least to some degree psychologically and neurobiologically (i.e., in terms of neuroanatomy and neurotransmitters) distinct (e.g., Peciña & Berridge, 2005; Richard, Castro, DiFeliceantonio, Robinson, & Berridge, 2013; Robinson & Flagel, 2009; Tindell, Berridge, Zhang, Peciña, & Aldridge, 2005). Regarding psychological distinctions, 'liking' refers to pleasure or palatability (especially with respect to food), whereas 'wanting' refers to incentive salience or appetite (Berridge, 1996). As discussed by Castro and Berridge (2014), in the strictest sense, 'liking' is an objective positive hedonic reaction (based on, for instance, quantifying discrete orofacial affective reactions, such as tongue protrusions, in response to different tastes). This phenomenon is referred to as hedonic impact and is not necessarily accompanied by a subjective sense of pleasure. Similarly, 'wanting' refers to motivational aspects (both in terms of neural and behavioral responses) of incentive salience (discussed in greater detail below), which is not necessarily accompanied by subjective feelings of desire.

Neurological substrates of value and reward

Regarding the neurobiological distinctions mentioned in the last paragraph, brain dopamine and endogenous opioids are the primary neurotransmitters of inquiry, though other neurotransmitters such as endocannabinoids and GABA perform related functions

in similar neural regions (e.g., Jager & Witkamp, 2014; Mahler, Smith, & Berridge, 2007; Reynolds & Berridge, 2002; Söderpalm & Berridge, 2000). Additionally, hormones such as testosterone and oxytocin are shown to influence economic decision-making and behavior (Apicella et al., 2008; Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005). However, I focus on the former two neurotransmitters (i.e., dopamine and endogenous opioids) in this discussion for brevity and because they seem to have garnered the most attention to date within this literature. Also regarding neurobiological distinctions, despite the fundamental contrasts delineating broad categories of reward (i.e., primary versus secondary) as well as the psychological components of reward discussed above, "there is striking consistency in the set of neural structures that respond to rewards across a broad range of domains, from primary ones such as food, to more abstract ones such as money and social rewards (including reciprocity, fairness, and cooperation)" (Montague et al., 2006: 438). Several aspects of this paragraph independently require further explication, though it must be stressed that the reward and valuation system(s) are highly integrated. The following discussion is organized in reverse order to this paragraph.

There is strong support for a "common reward circuit" based upon the neuroimaging literature (Sescousse et al., 2013). The ventral striatum (including the nucleus accumbens), orbitofrontal cortex, and ventromedial prefrontal cortex have been found consistently to respond to a wide variety of rewarding stimuli, though most studies focus on food-based, erotic, and monetary rewards (e.g., Breiter & Rosen, 1999; Hoebel, 1985; Knutson, Fong, Adams, Varner, & Hommer, 2001; McClure, Berns, & Montague, 2003; Schultz, Dayan, & Montague, 1997; Fliessbach et al., 2007). Other relevant neural regions include the ventral tegmental area and substantia nigra, which send dopaminergic

projections to these areas (Cohen, Haesler, Vong, Lowell, & Uchida, 2012; Dickson et al., 2011; Fields, Hjelmstad, Margolis, & Nicola, 2007; Hjelmstad, Xia, Margolis, & Fields, 2013; Skibicka, Hansson, Alvarez-Crespo, Friberg, & Dickson, 2011). These core areas may respond to different aspects of rewarding stimuli and certain types of stimuli (or the context(s) in which the stimuli are presented) may differentially activate these core areas. As discussed by Montague et al. (2006: 420), the striatum and orbitofrontal cortex are responsive to rewards that change, accumulate, or are learned over time (Berns, McClure, Pagnoni, & Montague, 2001; Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Elliott, Newman, Longe, & Deakin, 2003; Galvan et al., 2005; Knutson, Fong, Bennett, Adams, & Hommer, 2003; Koepp et al., 1998; Sugrue, Corrado, & Newsome, 2005), whereas the ventromedial prefrontal cortex is responsive to reward value (Knutson, Fong, et al., 2001; O'Doherty et al., 2003). Via meta-analysis, Sescousse et al. (2013: 681) supported the notion that abstract secondary rewards such as money are "represented in evolutionary more recent brain regions" such as the anterior portion of the orbitofrontal cortex. However, their analysis ultimately "confirmed the existence of a core set of brain regions processing reward outcomes in an indiscriminate fashion, in line with the idea of a centralized 'reward circuit'" (Sescousse et al., 2013: 686).

Introducing language that will be important to the methodology of this study, the Behavioral Activation System (BAS), together with the Behavioral Inhibition System (BIS), provide the foundation for a critical theory of personality based on physiological mechanisms respectively associated with approach and avoidance motivation, sensitivity to reward and punishment, and positive and negative affect (Carver & White, 1994; Gray, 1981). These two systems have been extended beyond their trait-based origins to explore

state-based activation, as would be the case with visceral states such as hunger and the BAS (Yam, Reynolds, et al., 2014). "The neurobiology of the BAS is related to the commonly identified dopaminergic reward circuitry. It involves the projections from the substantia nigra and the ventral tegmental area (VTA) to the dorsal and ventral striatum, and also their corresponding cortical projections to the prefrontal cortex" (Barros-Loscertales et al., 2010: 18). To be clear, the BAS is understood to be equivalent to the mesolimbic dopamine system (i.e. common neural reward circuitry) discussed throughout much of this paper (Barros-Loscertales et al., 2010; Berridge, 1996; Schultz et al., 1997; Wise, 2004).

Regarding mesolimbic dopamine, one commonality between many neural regions implicated in reward is their heavy innervation by dopaminergic circuitry (McClure, Laibson, Loewenstein, & Cohen, 2004). "Dopamine has long been associated with the idea of reward but historically was assumed to mediate directly the reward value of an event" (Montague et al., 2006: 425–426; see Wise, 1980). However, there has been a paradigm shift within the psychopharmacological field away from the notion that brain dopamine, particularly within mesolimbic dopamine projections to the nucleus accumbens, directly mediates the pleasurable aspects of primary rewards (Salamone, 2007). While dopamine release *may* occur in response to reward itself, especially delivery of an unexpected reward, release is generally observed to be greater in response to some cue that reliably predicts subsequent reward, especially when that cue is unexpected (Montague et al., 2006). Contemporary literature on dopamine "emphasizes aspects of instrumental learning, Pavlovian conditioning and Pavlovian-instrumental interactions, reward prediction, incentive salience and behavioral activation" (Salamone, 2007: 389),

though controversy exists as to precisely which functions the neurotransmitter regulates (Barbano & Cador, 2007; Berridge, 2007; Flagel et al., 2011). However, it is increasingly accepted that, despite widespread belief, even among some scientific communities, dopamine does not cause pleasure (i.e., the experience of 'liking' some reward stimuli) (Salamone, 2007). Rather, "dopamine mediates only a 'wanting' component, by mediating the dynamic attribution of incentive salience to reward-related stimuli, causing them and their associated reward to become motivationally 'wanted'" (Berridge, 2007: 408). Endogenous opioids seem to rule the realm of 'liking' and pleasure.

Building on the idea of a "common reward circuit" (Sescousse et al., 2013), areas such as the nucleus accumbens are also hotspots for endogenous opioids which may serve to enhance hedonic experience (i.e., pleasure) (Berridge et al., 2009). Endogenous opioids may contribute to both the 'liking' and 'wanting' phenomena, at least within the heretofore unmentioned ventral pallidum, which is activated by a variety of rewards such as cocaine, sex, food, and money (Berridge et al., 2009). However, opioids are generally more implicated in 'liking' rather than 'wanting' a reward because they mediate the evaluation of a reward as pleasant or palatable (Barbano & Cador, 2007; Berridge, 1996; Drewnowski, Krahn, Demitrack, Nairn, & Gosnell, 1992; Glass, Billington, & Levine, 1999; Kelley et al., 2002; Kelley, Baldo, Pratt, & Will, 2005). For instance, opioids in the nucleus accumbens seem to increase the hedonic impact of sweetness (e.g., Peciña & Berridge, 2005).

The role of dopamine is partially explained by the activation-sensorimotor hypothesis, per which dopamine mediates "general functions of action generation, effort, movement, and general arousal or behavioral activation" (Berridge, 2007: 392; see also

Dommett et al., 2005; Horvitz, 2002; Robbins & Everitt, 1982; Salamone, Cousins, & Bucher, 1994; Stricker & Zigmond, 1976). However, the roles of neurotransmitters like dopamine and endogenous opioids in reward are perhaps best captured by the literature on incentive salience (i.e., 'wanting') and hedonic impact (i.e., 'liking') (Berridge, 2007; Berridge & Robinson, 1998; Berridge et al., 2009). The principal proposition of this literature is that "reward is a composite construct that contains multiple component types: wanting, learning, and liking" (Berridge, 2007: 408). Incentive salience helps transform neural representation and valuation of some reward (conditioned or unconditioned stimulus) into motivation and behavior, promoting approach and consumption of desired rewards. In other words, incentive salience "most often acts to add incentive value to learned Pavlovian conditioned stimuli that predict a wide variety of learned rewards" (Berridge, 2007: 408; see also Berridge & Robinson, 1998; Dayan & Balleine, 2002; Elliott et al., 2003; Everitt & Robbins, 2005; Hyman & Malenka, 2001; Insel, 2003; Kelley et al., 2005; McClure, Daw, & Read Montague, 2003; Volkow et al., 2002). In doing so, incentive salience maps 'liked' rewards to 'wanted' rewards (McClure, Daw, et al., 2003), meaning that most of the time we like what we want, but it may be better to say that we learn (i.e., associative learning or Pavlovian-style operant conditioning) to want what we like (Flagel et al., 2011). However, it is easy to imagine wanting a reward that ultimately is disliked; every birthday gift is wanted while it is still wrapped, though some will almost certainly disappoint the recipient. Importantly, incentive salience is distinct from and can potentially undermine more cognitive (i.e., explicit, declarative) goals and expectations, resulting in irrational 'wanting' for what is not cognitively wanted, as is often the case with addiction (Berridge et al., 2009). More germane to our purposes, while cue-triggered 'wanting' can involve specific rewards, it may also elicit a more generalized 'wanting' for other rewards, spurring motivation to seek out and consume many types of stimuli. This idea directly mirrors the prior discussion about the generalizability of visceral states but provides a psychological and neurobiological basis. Next, I discuss how aspects of food and eating interact with concepts of reward.

Food as Reward

Our relationship with food is a multifaceted phenomenon (Begg & Woods, 2013; Berthoud, 2011; Kong & Singh, 2008; Schwartz et al., 2000). Regarding the processes associated with eating and digestion, "there exists a complex interplay between immunomodulators, neurotransmitters and neuroendocrine factors that underlie gastrointestinal sensing mechanisms and enable orchestration of appropriate host responses" (Grundy, 2006: 76). The gastrointestinal tract involves multiple organs, stages, and functions, and is rich with "sensory" innervation, projections from which are coordinated and integrated by the central nervous system with behavioral responses, such as the regulation of food intake (Grundy, 2006; Schwartz et al., 2000). While eating is a conscious act (though as we will illustrate, guided by unconscious forces), the process of digestion is largely unconscious and without the sensations that may be implied by use of the word 'sensory.' "Imaging studies show a comprehensive neuronal network controlling human feeding behavior that includes parts of the cortex, hypothalamus, thalamus, and the limbic system. This network connects the regions of the brain that control the perception of sensations and flavors (the cortex); that influence the reinforcing value of food (the limbic system); and that influence appetite, body weight, and energy balance (the thalamus and hypothalamus)" (Epstein et al., 2007: 892). Neurotransmitters

such as dopamine and endogenous opioids, as well as hormones (i.e., "neuroendocrine factors") such as ghrelin and leptin, play important roles in this process, though their effects may trespass into seemingly unrelated realms, such as temporal discounting and risk propensity, which underlies their importance to this paper. These neurotransmitters and hormones will be discussed after a brief synopsis of food as reward.

Food is a primary reinforcer (Epstein et al., 2010; McClure et al., 2007; Sescousse et al., 2013), activating, both generally and specifically, portions of the common neural decision-making and reward circuitry (Castro & Berridge, 2014; Rangel, 2013; Richard et al., 2013; Sescousse et al., 2013). For instance, at least one common brain region associated with representing aspects of reward value of various stimuli, the aforementioned orbitofrontal cortex, is activated in response to taste, smell, the mouthfeel of fat, as well as more abstract reinforcers like winning and losing money (Kringelbach, 2005; Rolls, 2004). The nucleus accumbens and striatal circuitry also play important roles in eating behavior and food reward (Castro & Berridge, 2014; Richard et al., 2013). Broadly, activity in the reward areas of the brain "changes in response to the taste, smell, thought and sight of food" (Goldstone et al., 2009: 1625; see also Beaver et al., 2006; DelParigi, Chen, Salbe, Reiman, & Tataranni, 2005; Gordon et al., 2000; Gottfried, O'Doherty, & Dolan, 2002; Hinton et al., 2004; Holsen et al., 2005; Morris & Dolan, 2001; O'Doherty, 2007; Porubská, Veit, Preissl, Fritsche, & Birbaumer, 2006; Simmons, Martin, & Barsalou, 2005; Small et al., 2007; St-Onge, Sy, Heymsfield, & Hirsch, 2005; Tataranni & DelParigi, 2003; Wang et al., 2004), as well as to "changes with the rewarding properties of the food stimuli, e.g. high-calorie vs. low-calorie foods, foods self-reported as preferentially craved, more appetizing or disgusting, or with food

aversion during repeated consumption of the same food" (Goldstone et al., 2009: 1625; Beaver et al., 2006; Calder et al., 2007; Farooqi et al., 2007; Gordon et al., 2000; Hinton et al., 2004; Killgore et al., 2003; Kringelbach, O'Doherty, Rolls, & Andrews, 2003; O'Doherty et al., 2000; Rolls & McCabe, 2007; Rothemund et al., 2007; Small, Zatorre, Dagher, Evans, & Jones-Gotman, 2001; Stoeckel et al., 2008). Even pictures of food activate the reward system in hungry people, especially when comparing obese to nonobese people (e.g., Bruce et al., 2010; Frank et al., 2010; Martin et al., 2010; Schienle, Schäfer, Hermann, & Vaitl, 2009; Stoeckel et al., 2008).

There are two broad classes of mechanisms regulating feeding behavior: homeostatic (i.e., metabolic) and hedonic (i.e., reward-driven) (Appelhans, 2009; Berthoud, 2011; Epstein et al., 2007; Goldstone et al., 2009; Saper, Chou, & Elmquist, 2002). "Hedonic feeding, which can be triggered by visual or olfactory food cues, involves brain regions that play a role in reward and motivation, while homeostatic feeding is thought to be under the control of circulating hormones acting primarily on the hypothalamus" (Malik, McGlone, Bedrossian, & Dagher, 2008: 400). If homeostatic mechanisms were the sole regulator of feeding behavior, overconsumption and obesity would be far less prevalent as we would likely not eat beyond our homeostatic needs were consumption of palatable food not inherently rewarding. However, beyond the physiological effects associated with consuming the nutritive value of the food are the sensations of eating including taste, mouth feel, and smell, which would be difficult to separate from the remainder of the digestive processes. Eating often occurs simply due to the rewarding properties of appetitive food stimuli. This hedonic feeding occurs in the absence of nutritional or caloric deficiency (Appelhans, 2009). However, because food is intrinsically reinforcing, there are links between these homeostatic and hedonic systems to ensure adequate nutritional intake (Saper et al., 2002). It seems each system helps modulate the functioning of the other (Berthoud, 2011) and both systems may respond to similar signals. For instance, the hormones leptin and ghrelin are implicated in homeostatic feeding via their effects on the central nervous system, but are also implicated in hedonic feeding via their effects on neural dopaminergic circuitry (and neural opioidergic circuitry, in the case of leptin) (Saper et al., 2002), as will be discussed in greater detail below. Overall, "eating can be triggered by metabolic need, hedonic drive, or an interaction between the two, and there are several neural circuits that represent this interface. Importantly, metabolic signals of energy status can modulate processing of cognitive and reward functions in corticolimbic systems (bottom-up processing), which influence regulatory processes to restore energy status to the optimal level. Yet the cognitive and emotional brain can also override homeostatic regulation (top-down processing), to yield an energy imbalanced state" (Berthoud, 2011: 893).

People generally select and react to foods based on taste, smell, and mouthfeel rather than macronutrient (i.e., carbohydrate, fat, and protein) content (Levine, Kotz, & Gosnell, 2003). However, macronutrient content is an important factor in taste; high fat content or sugar content (particularly in the form of simple sugars (e.g., fructose, sucrose)) is often present in good tasting food, especially when dietary fat and simple sugars are presented together, such as in sweetened high-fat dairy products like ice cream (Drewnowski & Greenwood, 1983). In modern times, such foods are often preferentially overconsumed (Drewnowski, 1995), which makes sense considering our evolutionary history as hunter-gatherers during much of which sweet foods such as fruit and fatty

foods such as animal meat were often limited, requiring foraging or hunting (Appelhans, 2009). Motivation to engage in such potentially exhausting behaviors undoubtedly benefited from our finding sweet and fatty foods highly rewarding. Regarding the distinction between 'liking' and 'wanting,' "from an evolutionary standpoint, it is highly advantageous to have independent neurotransmitter systems to regulate the different aspects of a motivated behavior. As palatable items are often associated with high contents of fat and carbohydrates, a system capable of signaling these high-caloric foods to the organism would improve fitness. Likewise, a system mediating anticipatory responses will favor behavioral adaptation to a changing environment and, hence, increase reproduction and survival" (Barbano & Cador, 2007: 503). Consuming these foods beyond current homeostatic needs allows some energy to be stored as fat which can act as a buffer against malnutrition or starvation should food becomes scarce. "Thus, the ability of palatable food to promote overconsumption by overriding satiety signals was preserved in our species" (Appelhans, 2009: 640; see also Berthoud, 2007; Pinel, Assanand, & Lehman, 2000).

A converse to overconsumption, food deprivation (i.e., a decrease in overall caloric intake), whether voluntary or involuntary, can increase the reinforcing value of food as well as the motivation to consume and, thus, actual consumption (Drobes et al., 2001; Raynor & Epstein, 2003; Siep et al., 2009), though certain eating disorders like anorexia nervosa may negate these effects (Wierenga et al., 2014). These effects of food deprivation can appear in as little as four hours if the person is anticipating a forthcoming meal; this is why food deprivation and restriction (i.e., limited access to certain food(s) as a result of self-imposed or environmental constraints) may paradoxically reduce the

effectiveness of common obesity treatments of which they are the core components (Raynor & Epstein, 2003; Siep et al., 2009). Indeed, food deprivation (i.e., fasting) by skipping breakfast makes the brain's reward center more responsive to high-calorie (versus low-calorie) foods (Goldstone et al., 2009). The idea that food deprivation can increase impulsivity (or decrease self-control) regarding food reinforcers (Kirk & Logue, 1997; Logue & King, 1991) has already been discussed in this paper, though it was framed as an issue of hunger rather than deprivation. Using that language, it can be said that the brain responds to appetitive cues such as taste differently depending on whether one is hungry or sated (Haase, Cerf-Ducastel, & Murphy, 2009; Haase, Green, & Murphy, 2011).

Neurochemical basis of food as reward

"The neurochemical systems mediating food reward include dopaminergic, endocannabinoid, serotonergic, opioid and orexin pathways" (Goldstone et al., 2009: 1632; see also Berthoud & Morrison, 2008; Cardinal & Everitt, 2004; Kringelbach, 2004; Volkow & Wise, 2005). However, the focus here is primarily on opioids and dopamine. Orexin pathways will also be discussed. The opioid system may be largely responsible for the hedonic value of sweet and fatty foods (e.g., Bertino, Beauchamp, & Engelman, 1991; Drewnowski et al., 1992) and perhaps even food reward more generally (Kelley et al., 2002). "It has been proposed that the opioid system would regulate food intake through the specific modulation of food hedonic properties or palatability" (Barbano & Cador, 2007: 501; see Berridge, 1996; Glass et al., 1999; Kelley et al., 2002). For instance, administrations of opioid antagonists (which decrease the effects of endogenous opioids) and opioid agonists (which increase the effects of endogenous opioids) have

been shown to preferentially and respectively decrease and increase the perceived pleasantness and consumption of highly palatable (i.e., sweet and/or fatty) foods (Yeomans & Gray, 1997, 2002), without functional changes in subjects' ability to perceive taste (Drewnowski et al., 1992). Neuroanatomically, at least part of the story involves opioid receptors within the ventral tegmental area. These receptors can effectively activate dopamine neurons within the same region (Hjelmstad et al., 2013; Margolis, Hjelmstad, Fujita, & Fields, 2014). Such activation of the ventral tegmental area may promote dopamine release in the striatum and nucleus accumbens (Dickson et al., 2011), both of which were mentioned earlier as key components of the core reward circuitry. The ventral tegmental area is noteworthy in that neurons within this region play a critical role in the motivational and rewarding actions of palatable food and drugs of abuse (Berridge, 2009; Everitt & Robbins, 2005; Fields et al., 2007). Thus, endogenous opioids seem to regulate the evaluation of food palatability and pleasantness (i.e., the 'liking' of food) and, thereby, food-motivated behaviors in general.

As is the case with other types of reinforcers, dopamine also plays an important role in the motivational and reward components of food and aspects of eating behavior (Baldo, Sadeghian, Basso, & Kelley, 2002; Kelley et al., 2005; Salamone & Correa, 2002). Neuroimaging studies reveal that dopamine is released during the anticipation and consumption of food (Hardman, Herbert, Brunstrom, Munafò, & Rogers, 2012; Small, Jones-Gotman, & Dagher, 2003; Volkow et al., 2002, 2003). Additionally, dopamine agonists and antagonists respectively increase and decrease the reinforcing value of food (Wise, 2006). However, dopamine seems to be involved primarily in the approach, anticipation, and 'wanting' aspects of food and eating, especially when preceded by cues,

rather than modulating the consummatory and hedonic aspects of eating, though this effect may be limited to the anticipation of "palatable" (i.e., good tasting) food stimuli (Barbano & Cador, 2007). As mentioned earlier, the nucleus accumbens is an especially important dopaminergic region. Connections from the nucleus accumbens to hypothalamic feeding centers provide a pathway through which palatable food can influence feeding behavior by overriding satiety signals" (Appelhans, 2009: 641; see also Berthoud, 2007). Levels of dopamine are also "modulated by the process of food consumption via the neurotransmitter acetylcholine. Food consumption raises the dopamine above the baseline level. This rise depends on the animals' hunger, which when satisfied increases the level of acetylcholine, which causes the dopamine level to fall back to the baseline" (Vig. Gupta, & Basu, 2011: 373). Though perhaps an oversimplification, it is essentially suspected that hunger should raise dopamine levels and, thereby, initiate a state of general and specific 'wanting' in the presence of a broad array of reward cues, while satiety may decrease dopamine levels and, thereby, lessen general and specific 'wanting.'

Regarding the endocrine system, meal timing reciprocally affects and synchronizes certain circadian processes, particularly those associated with the synthesis and secretion of certain hormones (e.g., corticosterone, ghrelin, leptin, insulin, glucagon, and glucagon-like peptide 1) (Patton & Mistlberger, 2013). That is to say, food is distinguished from other reinforcers in that it both elicits and responds to a cascade of unique hormones, such as ghrelin, leptin, and insulin, and shifts blood glucose levels (Begg & Woods, 2013; Korakianiti et al., 2014; Page et al., 2011; Patton & Mistlberger, 2013; Schwartz et al., 2000). Of these hormones, evidence suggests leptin and ghrelin

"modulate the expression of food anticipatory rhythms" (Patton & Mistlberger, 2013: 1), though in opposing directions (i.e., leptin suppresses appetite while ghrelin augments appetite, see Wren & Bloom, 2007), and modulate activity in neural regions (e.g., the hippocampus) involved in decision-making and memory (Diano et al., 2006; Harvey, Solovyova, & Irving, 2006).

The appetizer effect mentioned earlier is mediated at least in part by cephalic phase responses (CPRs). The purpose of CPRs is to "help maintain homeostasis, i.e., to minimize disturbances of the internal milieu resulting from food intake" (Smeets, Erkner, & De Graaf, 2010: 645; see also Zafra, Molina, & Puerto, 2006). Like food-related dopamine release, CPRs are triggered by the sight, smell, taste, or mere anticipation of appetitive food stimuli. For instance, it has recently been shown that umami (one of the five basic tastes, described as "savory, meaty and broth-like," see Yamaguchi & Ninomiya, 2000: 921) increases the appetizer effect (Masic & Yeomans, 2014). CPRs involve a host of preprandial and/or preabsorptive physiological processes, such as the release of insulin in cephalic phase insulin release (CPIR), increased salivation, increased gastric acid secretion, increased gastrointestinal motility, and thermogenesis (Begg & Woods, 2013; Powley, 1977; Smeets et al., 2010; Zafra et al., 2006). For instance, the release of insulin associated with CPIR may decrease blood glucose in some people, as well as increase initial appetite and meal size through various physiological mechanisms (Rodin, 1985; Smeets et al., 2010). Additionally, the magnitude of CPIR is positively correlated with palatability (Bellisle, Louis-Sylvestre, Demozay, Blazy, & Le Magnen, 1985; Lucas, Bellisle, & Di Maio, 1987; Teff & Engelman, 1996). However, the appetizer effect mediated by CPIR may be relatively short-lived (i.e., present for fewer

than 15 minutes, see Korakianiti et al., 2014; van Kleef, Shimizu, & Wansink, 2013; Zafra et al., 2006). It should be noted that while hormones such as insulin, ghrelin, leptin are part of CPRs, their secretion is not limited to CPRs. In other words, the release of these hormones may be promoted or inhibited pre-, intra-, post-, or inter-prandially (i.e., before, during, after, or between meals) or during periods of acute or chronic fasting, depending on numerous physiological and contextual factors (Begg & Woods, 2013). The hormones ghrelin and leptin are discussed below, both generally and with respect to reward. Broadly, "rising ghrelin levels in concert with falling leptin levels may serve as a critical signal to induce hunger during fasting" (Saper et al., 2002: 201).

Ghrelin is a powerful circulating orexigen (a term used for substances that increase or stimulate the appetite) which correlates with hunger scores among healthy subjects (Cummings, Frayo, Marmonier, Aubert, & Chapelot, 2004) and is known to contribute to preprandial hunger (Wren & Bloom, 2007). Moreover, ghrelin is increasingly implicated in food motivation and reward (Dickson et al., 2011; Perello et al., 2010; Skibicka & Dickson, 2011; Skibicka et al., 2011). Thus, ghrelin contributes to both homeostatic and hedonic feeding (Malik et al., 2008). In line with the idea of an appetizer effect, evidence indicates one type of CPR involves increasing circulating levels of ghrelin in anticipation of a meal (Cummings et al., 2001; Sugino et al., 2002), peaking approximately half an hour before the expected meal (Drazen, Vahl, D'Alessio, Seeley, & Woods, 2006), enhancing sensitivity to both taste (Shin et al., 2010) and smell (Tong et al., 2011), and ultimately resulting in more food being consumed (Tschöp, Smiley, & Heiman, 2000). Postprandially, ghrelin is suppressed in proportion to ingested

calories, though this effect does not predict the ensuing inter-meal interval (i.e., the time between meals or until a person's next spontaneous meal request) (Callahan et al., 2004).

Circulating ghrelin may act directly on dopaminergic circuitry (Dickson et al., 2011; Skibicka & Dickson, 2011; Skibicka et al., 2011). For instance, activity in dopaminergic regions such as the ventral tegmental area and dorsal striatum, which are functionally related to the ventral striatum and similarly implicated in food-based reward, is modulated by ghrelin (Egecioglu et al., 2010; Malik et al., 2008; Naleid, Grace, Cummings, & Levine, 2005). As mentioned above, activation of the ventral tegmental area (e.g., via increased circulating ghrelin) may promote dopamine release in portions of the core reward circuitry (Dickson et al., 2011). "Ghrelin therefore appears to modulate the response to food cues of a neural network involved in the regulation of feeding and, most importantly, in the appetitive response to food cues. This appetitive response has several components: attention, anticipation of pleasure, motivation to eat (hunger), consumption, and memory for associated cues" (Malik et al., 2008: 405). However, this line of research regarding reward circuits targeted by ghrelin has led to a surprising finding: beyond the effects of ghrelin in enhancing the rewarding properties of food (Egecioglu et al., 2010; Perello et al., 2010; Skibicka et al., 2011; Skibicka, Hansson, Egecioglu, & Dickson, 2012), ghrelin is also implicated in enhancing the rewarding properties of addictive drugs (Jerlhag et al., 2009; Kaur & Ryabinin, 2010; Tessari et al., 2007; Wellman, Davis, & Nation, 2005). Dickson et al. (2011: 80–81) discuss the "emerging concept that this system operates at the interface between neurobiological circuits involved in appetite and reward, increasing the incentive motivational value of both natural rewards (such as food) and artificial rewards (such as drugs of abuse)."

These effects of ghrelin provide yet another explanation for how the consequences of hunger may be extended beyond the desire for food to the desire for seemingly unrelated cues and rewards (Skibicka & Dickson, 2011).

Much of the discussion has focused on the visceral state of hunger rather than on its antipode, satiety. At least with respect to food and eating behaviors, satiety should not be considered a neutral state (as was discussed earlier in research regarding sexual arousal). That is to say, the state of satiety may exhibit some degree of influence on the core neural reward system that opposes the effects of hunger. For example, the circulating anorexigen leptin is involved in the regulation of appetite, both short- and long-term, and is released by adipocytes in proportion to lipid stores (Ahima, Saper, Flier, & Elmquist, 2000; Figlewicz, 2003; Morton et al., 2005; Patton & Mistlberger, 2013). Higher circulating leptin may decrease food palatability and even the perception of sweet tastes, as well as overall appetite, promoting meal termination and increasing postprandial satiation (Farooqi et al., 2007; Isganaitis & Lustig, 2005; Ninomiya et al., 2001; Raynaud et al., 1999; Rosenbaum, Sy, Pavlovich, Leibel, & Hirsch, 2008; Smeets et al., 2010). Importantly, leptin may inhibit dopaminergic projections to the striatum (Cota, Barrera, & Seeley, 2006). For instance, Farooqi et al. (2007) and Rosenbaum et al. (2008) similarly showed that leptin modulated activity in the ventral striatum, decreasing the motivational salience (i.e., the 'wanting') of appetitive food stimuli. Shaley, Yap, and Shaham (2001) showed that, despite the generalized effect of food deprivation on drugseeking behaviors (Carroll & Meisch, 1984), leptin attenuates acute food deprivationinduced relapse to heroin-seeking behaviors, suggesting hormones signaling metabolic status such as insulin, leptin, and ghrelin "may act by modulating dopaminergic or

opioidergic function" (Figlewicz & Benoit, 2009: R14). This hints at the possibility that the effects of satiety may be extended beyond decreased desire for food to decreased desire for seemingly unrelated cues and rewards.

Chapter 4

TEMPORAL DISCOUNTING

Temporal discounting (i.e., delay discounting, time preferences) is a principal economic theory relating the concepts of reward and value. It refers to the tendency when choosing among alternatives to progressively discount or undervalue future outcomes as they become increasingly temporally delayed (i.e., as they occur further in the future) (Frederick, Loewenstein, & O'Donoghue, 2002). In other words, the subjective value of an outcome is inversely proportional to the delay preceding the outcome (Harrison & McKay, 2012; Kirby, 1997). Thus, people generally prefer more imminent rewards to more delayed rewards to such an extent that they are often willing to accept smaller rewards received sooner over larger rewards received later (contrarily, people prefer aversive outcomes in lesser degrees, later rather than sooner). These two comparative classes of reward outcomes are customarily referred to using the terms smaller sooner, or "SS," and larger later, or "LL." Individuals are assumed to have relatively stable discount rates over time, with empirical evidence indicating high test-retest reliability over time frames of up to a year (Baker, Johnson, & Bickel, 2003; Beck & Triplett, 2009; Black & Rosen, 2011; Harrison & McKay, 2012; Kirby, 2009; Ohmura, Takahashi, Kitamura, & Wehr, 2006; Simpson & Vuchinich, 2000; Takahashi, Furukawa, Miyakawa, Maesato, & Higuchi, 2007). Additionally, evidence indicates that individual discount rates correlate

across commodities (Bickel et al., 2011; Odum, 2011). Within the evaluation computation framework presented above, this represents the external contextual element of delay (see Padoa-Schioppa, 2011).

Some argue that "high short-term discount rates are related to an underlying individual trait" (Reuben et al., 2010: 125); others identify the more colloquial trait, arguing that "delay discounting and trait impulsivity load onto the same psychological factor" (Kayser, Allen, Navarro-Cebrian, Mitchell, & Fields, 2012; see also Meda et al., 2009; Odum, 2011), though it may be more accurate to conceptualize temporal discounting as a subset of impulsivity, as the latter term is notoriously less precise (Dalley & Roiser, 2012). Drawing corollaries with research on stress (see Sapolsky, 2004), some go so far as to label chronically excessive discount rates as a "trans-disease process" (Bickel, Jarmolowicz, Mueller, Koffarnus, & Gatchalian, 2012; Bickel & Mueller, 2009), implicated in myriad health-related disorders, ranging from the abuse of alcohol, cigarettes, cocaine, methamphetamine, and opioids, to behaviors such as gambling and binge eating, to psychopathologies such as attention deficit hyperactivity disorder, schizophrenia, depression, and disruptive behavior disorder (see Koffarnus, Jarmolowicz, Mueller, & Bickel, 2013). More normatively, "early onset of sexual activity in teenagers is associated with discount rate, as is failing to engage in a wide variety of prohealth behaviors such as eating breakfast, wearing sunscreen, wearing a seatbelt, exercising vigorously, checking blood pressure, following physician advice, or having a recent mammogram, Pap smear, prostrate examination, dental visit, cholesterol test, or flu shot" (Koffarnus et al., 2013: 35; see also Axon, Bradford, & Egan, 2009; Bradford,

2010; Chesson et al., 2006). At this point, the importance of discovering factors that may influence discount rates should be adequately established.

Per the usual conceptualization of temporal discounting, the net present value (i.e., discounted value) of some future outcome is compared with some present outcome. "In simple models [the future outcome] is multiplied by a discount factor, which is a particular realization of the discount function for a given time delay, to derive the notionally equivalent [present outcome] that should make a decision maker indifferent between [the future and present outcomes]" (Doyle, 2013: 117). Another conceptualization focuses "on the rate parameter implied by a given discounting model (e.g., in the exponential model, the rate parameter is the continuously compounded interest rate that would make a [present outcome] now into [a future outcome] at [sometime in the future]) (Doyle, 2013: 117). Although perhaps not psychologically equivalent, the two conceptualizations presented above would both lead to the same choices (i.e., preference ordering) among available alternatives (Doyle, 2013). In this paper, Equation 1 reflects this latter conceptualization, whereas the Equations 2-5 reflect the former conceptualization.

Although perhaps dozens of delay discounting models exist, most have a similar structure, allowing one to infer a discount rate or decision parameter from separable time and reward components, each affected by functions that reflect subjective perceptions. Discount rates are determined via some mathematical operation by which (subjective valuations of) the time and reward components are combined. As summarized by Doyle (2013: 132):

(1)

"where s(.) and S(.) are functions that return subjective perceptions of the [reward] and time aspects, respectively, and \otimes is an operation by which these become combined." It should be noted that money most often is used, either implicitly or explicitly, as the reward in studies using discounting models. It should also be noted that the use of the \otimes symbol here is not intended to indicate the Kronecker product related to mathematical matrix operations.

Regarding specific models, there is general support for (quasi)hyperbolic versus other (e.g., exponential) models of discounting (e.g., Ainslie, 2012; Ainslie & Haslam, 1992; Green & Myerson, 2004; Kirby, 1997, 2006; Madden et al., 1999; Mazur, 1987; Ohmura et al., 2006; Zarr, Alexander, & Brown, 2014). Indeed, "the quasihyperbolic discount function... to date still remains the most widely utilized approximation of a hyperbolic discount function in discrete time" (Musau, 2014: 21), The quasihyperbolic discount function has been found to "provide a good fit to experimental data and to shed light on a wide range of behaviors, such as retirement saving, credit-card borrowing, and procrastination" (McClure et al., 2004: 506; see Angeletos, Laibson, Repetto, Tobacman, & Weinberg, 2001; O'Donoghue & Rabin, 1999). Per Ainslie (2012: 6), "the decline in rewarding effect with delay is described better by a discount function that is inversely proportional to delay (hyperbolic discount curve) than by a function that declines by a constant proportion of remaining value for each unit of delay (exponential discount curve)."

There is a significant advantage of (quasi)hyperbolic discount models over normative (i.e., exponential) models. Despite the relative stability of individual discount rates over time, people do not necessarily have consistent *preferences* over time. Consistent preferences over time without new information being introduced would yield exponential discounting (Koopmans, 1960; Samuelson, 1937), per which subjective perceptions of reward would be logarithmic and subjective perceptions of time would be linear (Doyle, 2013). However, this "standard valuation model in economics" has serious limitations in that it does not permit "expectable changes of preference," which may be especially prevalent as future rewards become temporally proximal (Ainslie, 2012: 5; Strotz, 1955). These concerns are at least partially addressed by the use of hyperbolic (e.g., Ainslie, 1975, 1992) and, more popularly, quasihyperbolic (e.g., Laibson, 1994, 1997) discount models.

What the hyperbolic models account for that the exponential does not is the tendency for people, when choosing among some pair of time-lagged, differentially-valued outcomes, to favor an LL (i.e., larger later) reward over an SS (i.e., smaller sooner) reward when both are temporally distal, but to change preferences to favor the SS reward as it becomes temporally proximal (see Figure 1, borrowed from Ainslie (2012: 7)). Discoveries of such preference reversals have been used before to question and redress inadequacies in popular notions of rational economic behavior (e.g., Grether & Plott, 1979; Kahneman, 2013; Tversky, Slovic, & Kahneman, 1990).

The most widely used equation for hyperbolic discounting is that of Mazur (1987):

$$V_i = \frac{A_i}{Z + (k \times D_i)}$$

(2)

where V_i is the present value or utility of the reward (or reinforcer, in Mazur's language), A_i is (monotonically related to) the amount of reward received at payout (i.e., the value of the reward if received immediately), D_i represents delay or the amount of time between the present time and receipt of the reward, Z is a constant (usually set at a value of 1) used to prevent V_i from approaching infinity as D_i approaches zero, and k is a "free parameter that can vary to account for individual differences among subjects or procedural differences among experiments" (Mazur, 1987: 57–58), which some researchers interpret as an individual's sensitivity to delay (e.g., Steel & Konig, 2006) or degree of impatience (e.g., Ainslie, 2012). However, this equation has since been modified to account for the emergence of two ideas that have hence become allied in much of the literature: β spikes and viscerality.

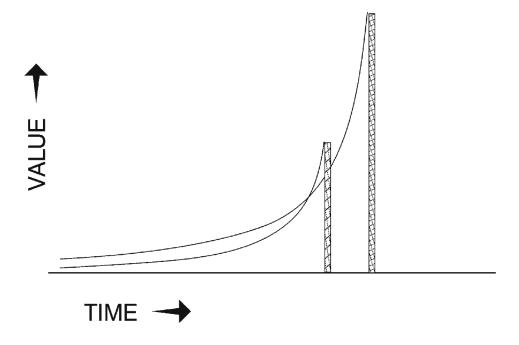


Figure 1. Hyperbolic discount curves from Ainslie (2012: 7)

The idea of β spikes in temporal discounting came largely from the work of Laibson (1994, 1997; see also Elster, 1979; Phelps & Pollak, 1968), who proposed a quasihyperbolic discounting function. McClure, Laibson, Loewenstein, and Cohen (2004) discuss and simplify this "beta-delta" discounting equation as follows (notation has been changed to be consistent with that of Mazur (1987)):

$$V_i = A_i \times \beta \times \delta^{D_i} \tag{3}$$

Here, β (technically the inverse of β , see Doyle, 2013) is a premium placed on imminent rewards relative to future rewards. Thus, $\beta = 1$ when reward is immediate and 0 $< \beta < 1$ at all other times. "When $\beta < 1$, all future rewards are uniformly downweighted relative to immediate rewards" (McClure et al., 2004: 504). "The purpose of β is to help

tick the box of unexpectedly steep discounting at short durations, which is the hallmark of actual behavioral data, and which first motivated the use of hyperbolic discounting. Thereafter, the model assumes that discounting follows the standard normative model [i.e., the exponential model]" (Doyle, 2013: 124). The δ parameter is just the discount rate from the standard exponential model that follows the β parameter, per which a given delay is treated equivalently regardless of whether it occurs sooner or later; $\delta \leq 1$. The discount curves that result from such a model are shown in Figure 2, borrowed from Ainslie (2012: 9). The curves are principally exponential, excepting the β spike shown immediately prior to the receipt of the (visceral) SS reward, which effectively, though temporarily, elevates its value greater than that of the previously favored (nonvisceral) LL reward. The connection between β spikes and viscerality will be discussed shortly.

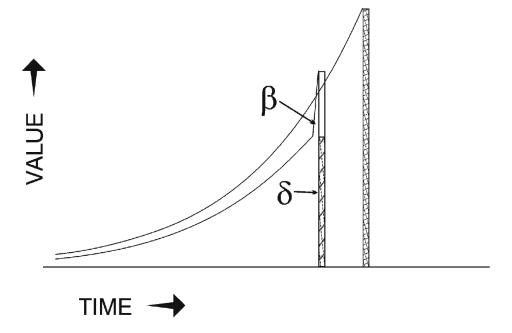
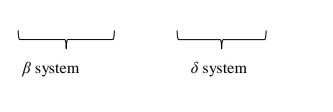


Figure 2. "Hyperboloid" or "quasihyperbolic" discount curves from Ainslie (2012: 9)

(4)

Building upon this, McClure, Ericson, Laibson, Loewenstein, and Cohen (2007: 5802) advanced the hyperbolic discount function described by Laibson (1997) as well as their own prior work (McClure et al., 2004), ultimately presenting a generalization of the quasihyperbolic model wherein the β spike discussed above has been "softened into a second, steep exponential discount curve from the moment of expected reward, which sums with the shallower standard discount curve" (Ainslie, 2012: 9). The equation is as follows:

$$V(t) = \omega \sum_{\tau=0}^{\infty} \beta^{\tau} u(c_{t+\tau}) + (1 - \omega) \sum_{\tau=0}^{\infty} \delta^{\tau} u(c_{t+\tau})$$



where V(t) is the present value or utility of a stream of consumables $(c1, c2, \ldots)$, ω is a weighting factor, u is the present value or utility function, τ is the delay until consumption, t is the present time, and β and δ are discount parameters as described above. Thus, the β system is associated with immediately available rewards, whereas the δ system is associated with all intertemporal choices. In this version, $\beta < \delta$. "This function has the important property that short-run discount rates are higher than long-run aggregate discount rates, because the relative magnitude of the (impatient) β system is greater in the short run than in the long run" (McClure et al., 2007: 5802).

Ainslie (2012) discusses and simplifies the McClure et al. (2007) equation as follows (notation has been changed to be consistent with that of Mazur (1987)):

$$V_i = A_i \times \{(\omega \times \beta^{D_i}) + [(1 - \omega) \times \delta^{D_i}]\}$$

$$\beta$$
 system δ system

(5)

The history of evolution of discounting models outlined above, particularly (quasi)hyperbolic models, eventually toward a more general model of temporal discounting that "(a) preserves the overall structure of hyperbolic discounting, but (b) is linked to neuroscience findings in such a way as to easily incorporate many contextual effects on discounting, and (c) links to psychological and process models of discounting" (van den Bos & McClure, 2013: 58–59), is important to the narrative here because recent literature argues that pure hyperbolic or exponential discount models may have greater utility and/or validity in at least some contexts (Ainslie, 2012; Andersen, Harrison, Lau, & Rutström, 2014). The exponential model is of particular interest, as a recent study found "robust evidence of almost-constant discounting," meaning only very limited evidence for quasihyperbolic discounting was found (Andersen et al., 2014: 22). The authors went on to ponder what (sub)samples, domains, and tasks might elicit hyperbolic behavior. They responded by considering "impulsive choices over foods and alcohol, drugs, sexual habits, driving behavior, gambling, perhaps to individuals and families close to the poverty level, and perhaps to younger people: a myriad of real behaviors and contexts with real welfare consequences," urging researchers to "systematically apply rigorous methods to those settings" (2014: 32). Thus, within the context of this study, it may be that the quasihyperbolic model is only applicable when under the influence of an

aroused visceral state such as hunger, with the exponential model applicable at all other times. Fortunately, both the quasihyperbolic and exponential models can be tested using the same measures and data.

The idea of the something akin to the β -spike function increasing the subjective utility of immediately available rewards despite the otherwise general preference for LL rewards represented by the δ function fits "seductively into the long tradition of twofaculty models that date back to Plato's chariot of the soul, pulled by the well-behaved horse of reason and the unruly horse of passion" (Ainslie, 2012: 9; see Plato, 2005: section 253). Although some authors have contended that a single decision-making system underlies intertemporal inconsistency (e.g., Herrnstein, 1997; Montague & Berns, 2002; Rachlin, 2000), others have argued for the presence of two systems (e.g., Loewenstein, 1996; Metcalfe & Mischel, 1999). In this regard, Thaler and Shefrin (1981: 392) refer to the individual as simultaneously a "farsighted planner" and a "myopic doer," while Stanovich and West (2000; see also Kahneman, 2013) describe individual cognition as comprising both a System 1 component (associative, holistic, automatic, quick, cognitively-effortless) and a System 2 component (rule-based, analytic, controlled, slow, cognitively-effortful), while Camerer (2005) discusses controlled and automatic processes. Recent research has shown that there is a neurological basis for such distinctions (e.g., McClure et al., 2007, 2004; Tanaka et al., 2004).

Neurological Substrates of Temporal Discounting

Some of the neural structures involved in the valuation of rewards support higher level, evaluative cognitive processes, whereas others support more automatic, primitive, and specialized processes. In this regard, the quasihyperbolic discount function (see Equation 3) has been used to support the existence of two distinct neural systems that differentially value immediate and delayed rewards. McClure et al. (2004) hypothesized that the β system (see Equations 4 and 5) would represent limbic structures, which are replete with dopaminergic innervation. Dopamine plays an important though complicated role in valuing rewards (e.g., Kayser et al., 2012), impulsivity (e.g., Dalley & Roiser, 2012), and even the motivational components of eating (e.g., Hardman et al., 2012), which is especially germane to the current study. McClure et al. (2004) further hypothesized that the δ system (again, see Equations 4 and 5) would represent structures including and associated with the lateral prefrontal cortex, supporting higher-level cognitive functioning. Using fMRI techniques, they showed that only immediate rewards (in the form of gift certificates sent via email) disproportionately activated so-called β areas, including the ventral striatum, medial orbitofrontal cortex, and medial prefrontal cortex. "As predicted, these are classic limbic structures and closely associated paralimbic cortical projections. These areas are all also heavily innervated by the midbrain dopamine system and have been shown to be responsive to reward expectation and delivery by the use of direct neuronal recordings in nonhuman species and brainimaging techniques in humans" (McClure et al., 2004: 505). "Together with the responses to the near threshold stimulus itself and the subjective value coding in temporal discounting, dopamine responses reflect the animal's subjective perception and valuation of the stimuli beyond purely physical reward properties. As decisions are ultimately made according to subjective reward values, the dopamine responses may provide rather direct and parsimonious, and therefore evolutionary beneficial and selected, inputs to neuronal decision processes" (Schultz, 2013: 232). It is perhaps worth noting that increases in dopamine in certain brain regions, particularly the frontal cortex, may actually decrease temporal discounting (Kayser et al., 2012).

On the other hand, consistent with predictions, *all* intertemporal decisions activated the lateral prefrontal cortex and associated parietal areas. This finding is consistent with neurophysiological and neuroimaging studies that have linked these areas with higher-level cognitive functioning such as deliberation, planning, and numerical computation, as well as the deferral of gratification (e.g., Dehaene, Dehaene-Lambertz, & Cohen, 1998; Miller & Cohen, 2001; Smith & Jonides, 1999). Tanaka et al. (2004) found that reward prediction for immediate and future timescales differentially recruited similar neural regions. Perhaps most impressively, McClure et al. (2004) showed that actual decisions between immediate and delayed rewards were determined by the relative activation of the β and δ systems, with δ areas significantly more active relative to β areas when delayed options were chosen, while β areas were similarly activated for all decisions involving immediate rewards regardless of the choice made, though there was a slight trend toward greater β area activation when the immediate option was chosen.

In a follow-up study, Equation 4 was essentially realized by the decomposition of a more general quasihyperbolic discount function into separate continuous exponential discount functions to more accurately reflect the β and δ systems (McClure et al., 2007). McClure et al. (2007) altered the experiment such that a "primary" reward (in this case, fruit juice and water, which were found to be approximately equally rewarding for subjects who had been instructed to abstain from fluid intake for three hours prior to the study) was used rather than a monetary-based reward (which allowed the authors to control both when the reward was delivered and when it was consumed) with time delays

of minutes instead of weeks. Even despite the different modality of rewards as well as timescales used in the two studies, results confirmed that the β system was more active when an immediate reward was present and that the δ system was similarly active across choices regardless of delay, as well as that the relative activation of the β and δ systems predicted actual choices made by participants.

Interestingly, in the 2007 study by McClure et al., the most delayed LL reward was delivered sooner than the most immediate SS reward in the 2004 study, because the soonest a participant could actually receive a gift certificate in the 2004 study was when they accessed it via email on a computer after the experimental session had ended, and the soonest a participant could actually receive some good in exchange for the gift certificate was at least one day. Nonetheless, β system activation was only seen in the latter case, per the authors' predictions. To account for this pattern of results, the authors considered the possibility that the β system may respond the earliest available reward rather than the absolute duration of delay until a reward is received (see also Berridge, 2007; Tindell et al., 2005). However, a second experiment failed to confirm this hypothesis; even a five-minute delay in receiving the fluid reward was enough to inhibit activation of the β system. The authors conjectured this may have resulted from differential processing of primary and secondary reinforcers (see also Estle et al., 2007; Odum et al., 2006; Odum & Rainaud, 2003). Other researchers have found similar shortterm discount rates for primary and monetary rewards (Reuben et al., 2010). However, an avenue of inquiry of the current research effort is whether an activated 'wanting' for primary rewards such as appetitive food stimuli (i.e., these visceral state of hunger) could alter discount rates for other rewards such as money.

Visceral States and Temporal Discounting

As Ainslie (2012: 4) points out, quasihyperbolic discount models require people "to rely on an additional source of motivation that does not depend on current prospects," which he discusses largely in terms of viscerality. However, despite the popularity and utility of quasihyperbolic discount models, there are limitations associated with their use and explanatory power with respect to visceral states. Per Ainslie (2012), viscerality is an elusive concept. Impulses may involve mundane activities, such as procrastination, and some impulses may take longer than arousal can be sustained, such buying a house. Additionally, arousal may be self-initiated without external stimuli, as would be the case in fantasizing. Issues also exist regarding the translation of results from stimuli and arousal in the laboratory to real-world conditions. However, in spite of these limitations, Ainslie (2012: 10) concedes that the "idea that viscerality is the source of β spikes that in turn explain impulsiveness has some support in psychological research as well as common experience." As McClure et al. (2004: 506) note, their results regarding the β and δ systems help "explain why many factors other than temporal proximity, such as the sight or smell or touch of a desired object, are associated with impulsive behavior. If impatient behavior is driven by limbic activation, it follows that any factor that produces such activation may have effects similar to that of immediacy." This assertion may help account for the fact that, as discussed before, a given visceral state may affect motivation, decision-making, and behavior vis-à-vis cues and rewards corresponding to that specific state, but also cues and rewards ostensibly unrelated to that state.

Our myopic nature in the face of SS rewards is reflected by the literature on visceral physiological states (e.g., Loewenstein, 1996; Loewenstein & O'Donoghue,

2004), which include hunger, thirst, and sexual desire, and related factors such as fatigue, pain, and cravings related to alcohol, cigarette, drug, and food addiction. Much of this literature directly addresses discount rates. For instance, substance abusers generally exhibit greater delay discounting than nonabusers, though discount rates may vary depending on the type of substance abused (e.g., cocaine, heroin, alcohol, nicotine), even among nondeprived individuals (i.e., individuals not actively craving) (Kirby & Petry, 2004; Mitchell, 1999). However, a state of deprivation seems to augment the discounting effect. For example, opioid deprivation among opioid-dependent individuals has been shown to elicit increased discounting of both heroin and monetary rewards (Giordano et al., 2002). Similarly, nicotine deprivation has been shown to increase delay discounting in smokers for both cigarette and monetary rewards (Field, Santarcangelo, Sumnall, Goudie, & Cole, 2006). A recent meta-analysis found strong evidence regarding an overall tendency for individuals engaging in addictive behaviors to exhibit increased temporal discounting (MacKillop et al., 2011). Venturing into other disciplines, sexual arousal also increases discounting, at least among men. For instance, Van den Bergh, Dewitte, and Warlop (2008: 94) found that inducing sexual appetite among men increased temporal discounting of money, candy, and soda. Wilson and Daly (2004) similarly found that exposing males to pictures of attractive female faces induced increased discounting.

Hunger and Temporal Discounting

Thus, it seems that, broadly, visceral states may elicit a type of generalized temporal myopia. However, there appears to be a dearth of research examining the role of hunger and satiety within this realm of decision-making beyond what was mentioned in

the introduction, that hunger leads to more impulsive grocery shopping. More broadly, however, a handful of studies has shown that hunger and satiety impact a variety of cognitive and decision-making processes (e.g., de Ridder et al., 2014; Ditto et al., 2006; Levy et al., 2013; Nordgren et al., 2007, 2008; Symmonds et al., 2010). Briers et al. (2006) explore conceptually-related hypotheses to those presented here, though their argumentation does not delve so deeply as to why hunger should reciprocally affect the desire for other types of rewards such as money, nor do they consider temporal discounting. However, they did show a reciprocal relationship between the desire for food and the desire for money. There is a body of research showing that obese women with and without binge eating disorder exhibit more myopic decision-making in delay discounting tasks as well as on the Iowa Gambling Task, which assesses the ability weigh short-term and long-term prospects in the face of uncertainty (Davis, Patte, Curtis, & Reid, 2010; for discussion of Iowa Gambling Task, see Bechara, Damasio, & Anderson, 1994; Colombetti, 2008). At least one study has been shown that low blood glucose levels are associated with increased discounting (Wang & Dvorak, 2010). It is informative to explore why hunger and satiety, specifically, may be associated with more or less discounting.

The literature reviewed above seems to imply that "brain dopamine activation (e.g., by drugs, natural appetites, or stress) might provide a mechanism [for increased discounting]: cue-triggered discounting would arise by amplification of 'wanting' for an immediately cued reward, which was available right away... and the amplification would not apply to the same extent to a delayed reward signaled by other cues... If so, mesolimbic dopamine activation could, thus, especially precipitate giving into immediate

gratification, at least in situations influenced by cue-triggered 'wanting'" (Berridge, 2007: 420). I have supported the notion that hunger, via homeostatic or hedonic mechanisms, may activate both general and food-specific aspects of the neural reward system, leading to generalized and food-specific 'wanting of reward-related stimuli, which may increase the incentive salience of myriad immediately available reward outcomes (i.e., relative discounting of future reward outcomes). This argumentation is outlined in Figure 3. Thus, the following hypothesis is proposed:

Hypothesis 1: Hunger (versus satiety) will be associated with increased temporal discounting of monetary rewards.

Chapter 5

RISK PROPENSITY

As noted above, value is computed at the time of choice and is subject to myriad contextual and circumstantial determinants that can be either external (e.g., the commodity, quantity, delay, risk, ambiguity, and cost) or internal (e.g., motivational state, impatience, attitudes toward risk and ambiguity) to the individual (Padoa-Schioppa, 2011). We have just discussed mechanisms by which hunger and satiety may affect decisions wherein the degree of temporal delay is manipulated. It may also be informative to explore how hunger and satiety may affect decisions wherein degree of risk the primary determinant of concern. Thus, within the evaluation computation framework presented above, I currently explore the external contextual element of risk (see Padoa-Schioppa, 2011). Although temporal discounting and risk-taking are conceptually related, both considered to be aspects of impulsive decision-making (Reynolds, Ortengren, Richards, & de Wit, 2006), they are discrete constructs (Andreoni & Sprenger, 2012a; Green & Myerson, 2004). Like discount rates, individuals are assumed to have relatively stable risk preferences (Andersen, Harrison, Lau, & Rutström, 2008a; Gilaie-Dotan et al., 2014; Harrison, Johnson, McInnes, & Rutström, 2005). Overall, understanding how general and specific cues and rewards may affect risk propensity in the presence of some instigating visceral state has broad implications for

highly consequential realms such as monetary decision-making, which affects "financial, marketing, and political domains" (Knutson, Wimmer, Kuhnen, & Winkielman, 2008: 509). In order to understand how this may occur, I explore the neurological substrates of risk propensity, followed by how visceral states such as hunger underlie these effects. The discussion here is simplified relative to the previous hypothesis regarding temporal discounting because much of the foundational argumentation is the same.

Neurological Substrates of Risk Propensity

A factor common to the below-cited studies is a failure to illuminate why visceral states should affect risk propensity. As previously discussed, the nucleus accumbens is part of the common neural reward circuitry. Activation of the nucleus accumbens increases in anticipation of both monetary (Breiter, Aharon, Kahneman, Dale, & Shizgal, 2001; Knutson, Adams, Fong, & Hommer, 2001; Preuschoff, Bossaerts, & Quartz, 2006) and nonmonetary rewards (Hamann et al., 2004; Sabatinelli, Bradley, Lang, Costa, & Versace, 2007). A study by Kuhnen and Knutson (2005: 763) found that "nucleus accumbens activation preceded risky choices as well as risk-seeking mistakes, while anterior insula activation preceded riskless choices as well as risk-aversion mistakes." As an aside, in contrast to the nucleus accumbens, activation of the anterior insula has been found in response to both monetary (Kuhnen & Knutson, 2005) and nonmonetary punishment (i.e., presentation of aversive stimuli) (Nitschke, Sarinopoulos, Mackiewicz, Schaefer, & Davidson, 2006; Simmons, Matthews, Stein, & Paulus, 2004). Regarding the focal nucleus accumbens, a later study found that the behavioral shift toward higher risk options was partially mediated by nucleus accumbens activation (Knutson et al., 2008). "Together, these results suggest that even incidental reward cues can act on anticipatory

affect to alter financial risk taking" (Knutson et al., 2008: 512). In affective-based, layman's terms, we might say that excitement regarding potential gains across a variety of reward domains promotes specific and generalized risk-taking, whereas anxiety regarding potential loss across a variety of reward domains promotes specific and generalized risk aversion (Knutson, Taylor, Kaufman, Peterson, & Glover, 2005; Kuhnen & Knutson, 2005; Paulus, Rogalsky, Simmons, Feinstein, & Stein, 2003). This perspective is, of course, very similar to prospect theory (Fennema & Wakker, 1997; Kahneman, 2013; Tversky & Kahneman, 1992).

Visceral States and Risk Propensity

Despite the relative stability of individual risk preferences over time, preferences are still amenable to several contextual factors. Our tendency to exhibit greater tolerance for risk when aroused is reflected by the literature on visceral physiological states. A subset of this literature directly addresses risk-taking. Many of the findings mirror those of temporal discounting discussed above. For instance, sexual arousal may lead to more risky sexual decisions, at least among heterosexual undergraduate males (Ariely & Loewenstein, 2006; Ditto et al., 2006). Beyond this domain-specific effect, it has been shown that financial risk-taking is increased after showing participants erotic pictures (again, heterosexual undergraduate males) (Knutson et al., 2008). Experimental evidence also indicates that sunshine and good weather may promote risk-taking via effects on mood (Bassi, Colacito, & Fulghieri, 2013). Visceral states such as thirst may also influence risk propensity (Yamada, Tymula, Louie, & Glimcher, 2013).

Hunger and Risk Propensity

Thus, it seems that, broadly, visceral states may elicit a type of generalized tolerance of risk. However, there appears to be a dearth of research examining the role of hunger and satiety within this realm of decision-making, though a few studies have probed the issue. In one study, exposure to visual and olfactory appetitive food cues (i.e., the presence of cookies in the room) versus a mere description of the food item made participants less sensitive to risk information (Ditto et al., 2006). Another study showed that anorexic, bulimic, and obese women exhibit similar patterns of impairment in decision-making relative to healthy women on the Iowa Gambling Task (Brogan, Hevey, & Pignatti, 2010). A highly relevant study found increasing monetary risk aversion as a function of postprandial satiety (measured via a decline in plasma acyl-ghrelin) (Symmonds et al., 2010). However, interestingly, increased risk-seeking resulted from "a lower than anticipated impact of the meal" (Symmonds et al., 2010: 5), ostensibly meaning satiety had not been induced. Perhaps the researchers inadvertently stimulated something akin to the appetizer effect. A more recent study found that, overall, people become more risk tolerant regarding food, water, and monetary outcomes as hunger and thirst increase (Levy et al., 2013). "More interesting, however, and contrary to [their] initial predictions, were [their] within-subjects observations. [They] found that when sated, individual human subjects showed very diverse risk attitudes, ranging from being highly risk-averse to being weakly risk tolerant. When deprived these risk attitudes converged towards a similar level, for all reward types, which could be described as weakly risk averse" (Levy et al., 2013: 9). However, the question remains: why should hunger affect risk propensity? Returning to physiological considerations, increases in circulating ghrelin as a result of hedonic or homeostatic hunger are related to increased activity in the ventral tegmental area, which seems to increase dopaminergic activity in the nucleus accumbens (Dickson et al., 2011; Egecioglu et al., 2010; Malik et al., 2008; Naleid et al., 2005; Skibicka & Dickson, 2011; Skibicka et al., 2011). As reviewed above, increased activity in the nucleus accumbens is associated with greater risk-taking (Knutson et al., 2008; Kuhnen & Knutson, 2005). Thus, weighing all the evidence and ceding that interaction between metabolic state and risk may be more nuanced than currently indicated, it seems reasonable to expect risk propensity to increase as a function of hunger. The results of this line of inquiry "support the notion that incidental reward cues influence financial risk taking" (Knutson et al., 2008: 509), especially in the presence of some activated visceral state such as hunger. This argumentation is outlined in Figure 3. Thus, the following hypothesis is proposed:

Hypothesis 2: Hunger (versus satiety) will be associated with increased risk propensity regarding monetary rewards.

Chapter 6

(UN)ETHICALITY

"Hunger makes a thief of any man." – Villager in the novel *The Good Earth* by Pearl S. Buck (1931: 135)

Behavioral ethics is a broad topic, referring to "individual behavior that is subject to or judged according to generally accepted moral norms of behavior" (Treviño, Weaver, & Reynolds, 2006: 952). It subsumes literature related to ethical decision-making (O'Fallon & Butterfield, 2005), ethical conduct (Treviño & Weaver, 2003) (Treviño & Weaver, 2003), and the normalization of (un)ethical behavior (Ashforth & Anand, 2003). It also overlaps with literature related to organizational citizenship and organizational deviance. The crucial distinction is that (un)ethical behavior is defined relative to societal norms whereas organizational extrarole and deviant behavior focus on organizational norms (Treviño et al., 2006). Importantly, behavior does not necessarily have to be intentional to qualify as ethical or unethical (Tenbrunsel & Smith-Crowe, 2008). "Behavioral ethics researchers have, for the most part, studied three types of related outcomes: unethical behavior that is contrary to accepted moral norms in society (e.g., lying, cheating, stealing); routine ethical behavior that meets the minimum moral standards of society (e.g., honesty, treating people with respect); and extraordinary

ethical behavior that goes beyond society's moral minima (e.g., charitable giving, whistleblowing)" (Treviño, den Nieuwenboer, & Kish-Gephart, 2014: 636–637). Relatedly, types of occupational fraud generally include asset misappropriations (e.g., theft or fraudulent disbursements of cash or other assets), corruption (e.g., conflicts of interest, bribery, illegal gratuities, extortion), and financial statement fraud (e.g., asset/revenue overstatements and understatements), with most cases committed by individuals working in accounting, operations, sales, executive/upper management, customer service, purchasing, and finance (Holtfreter, 2005). However, on the good side, over 40% of fraud cases are detected by a tip, with employees' whistleblowing, a form of extraordinary ethicality, accounting for nearly half of all tips (Association of Certified Fraud Examiners, Inc., 2014). As mentioned in the introduction, these behaviors have important financial repercussions beyond any psychological effects endured by the victims.

Recent comprehensive reviews of ethical and unethical behavior in organizations have made almost no mention of physiological states within the context of ethical decision-making (Tenbrunsel & Smith-Crowe, 2008; Treviño et al., 2014, 2006), at least partly because so little literature actually exists. Acknowledgment of the role of physiological states in ethical decision-making was limited to one of these reviews (Treviño et al., 2014) discussing the role of sleep deprivation in promoting unethical behavior (Barnes, Schaubroeck, Huth, & Ghumman, 2011; Christian & Ellis, 2011). Much of the underlying argumentation presented in these sleep deprivation studies was based on the ideas of self-control and ego-depletion, which have been studied by other researchers within the realm of unethical behavior and dishonesty (Gino, Schweitzer,

Mead, & Ariely, 2011; Mead, Baumeister, Gino, Schweitzer, & Ariely, 2009). These studies have suggested and found generally negative effects of deprivation states on ethical behavior (or, conversely, positive effects of deprivation on unethical behavior).

Highly germane to the present study, Yam, Reynolds, and Hirsh (2014: 125) argued that "deprivation of a physiological need would produce a domain-specific effect on unethical behavior" and supported this notion by showing that hungry participants were more willing to cheat on an experimental task when the reward involved food and less likely to cheat when the reward involved nonfood items. Thus, they contend that, when hungry, the incentive salience of food reward is increased, which is fully supported by the literature reviewed thus far in this paper. However, they concomitantly contend that the incentive salience of all nonfood reward is decreased when hungry, which seemingly contradicts much of the literature reviewed in this paper.

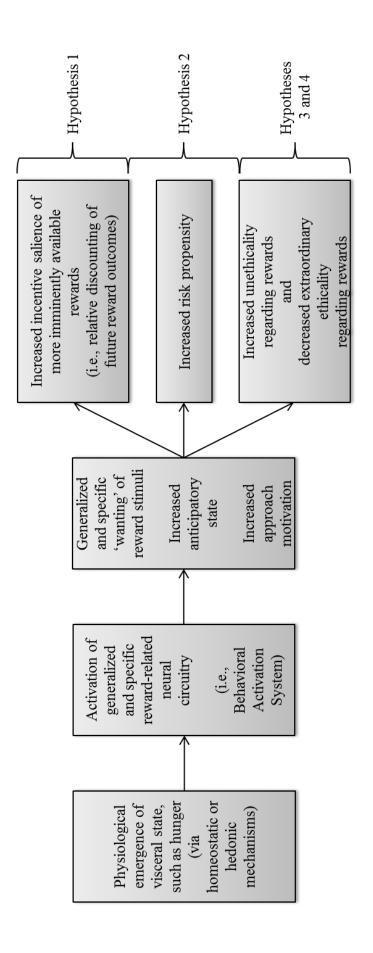
The authors' primary argument for these domain-specific results is that the effects of physiological deprivation on increased need-related unethical behavior and decreased need-unrelated unethical behavior are mediated by the Behavioral Approach System (BAS), "a brain circuit associated with the approach and pursuit of potential rewards, acting as the seat of approach motivation" (Yam, Reynolds, et al., 2014: 124), which concerns the same mesolimbic dopamine system (i.e. common neural reward circuitry) discussed throughout much of this paper (Barros-Loscertales et al., 2010; Berridge, 1996; Schultz et al., 1997; Wise, 2004). Their argumentation is in line with the "devaluation effect" discussed earlier, wherein attention is suppressed toward objects and outcomes unrelated to a given drive state (Brendl et al., 2003), as well as the assertion that "immediately experienced visceral factors have a disproportionate effect on behavior and

tend to 'crowd out' virtually all goals other than that of mitigating the visceral factor" (Loewenstein, 1996: 272). While the study is clearly well-conducted and the results represent an important advance in our understanding of this phenomenon, I am left to address an important contradiction with the arguments presented heretofore in this paper regarding the generalizability of visceral states such as hunger to seemingly unrelated cues and rewards.

However, a closer look at the types of nonfood rewards used in the Yam, Reynolds, and Hirsh (2014) study may help resolve the conflict. In the study, hungry participants were less likely to cheat for nonfood rewards that included drinks, \$10 worth of gift cards to an electronics store, and gifts such as notebooks and pens. For some reason, the researchers elected not to use money as a nonfood reward. The strong connection between the desire for food and desire for money has been supported theoretically in this paper as well as empirically by other researchers (e.g., Briers & Laporte, 2013; Briers et al., 2006; Gal, 2012; Reuben et al., 2010). Thus, I feel confident hypothesizing that hunger should have an effect on (un)ethical decisions regarding monetary outcomes in such a way as to increase cheating (Yam, Chen, & Reynolds, 2014; Yam, Reynolds, et al., 2014) and decrease charitable giving (Gino & Margolis, 2011). This argumentation is outlined in Figure 3. Thus, the following hypotheses are proposed:

Hypothesis 3: Hunger (versus satiety) will be associated with increased unethicality (i.e., cheating) regarding monetary rewards.

Hypothesis 4: Hunger (versus satiety) will be associated with decreased extraordinary ethicality (i.e., charitable donations) regarding monetary rewards.



Please note that this model is not tested. It is intended solely as an illustrative tool to frame the general argumentation and hypotheses.

Figure 3. Explanatory Model of the Effect of Hunger on Temporal Discounting, Risk Propensity, and (Un)ethicality

Chapter 7

METHODS

In order to test the above hypotheses, a research study was conducted wherein participants, having fasted overnight, arrived at the experimental setting and were randomly assigned to either a hunger condition (hereafter referred to as Condition H) or a satiety condition (hereafter referred to as Condition S), differentiated by the quantity of nutriment provided. The experimental manipulation involved participants taste-testing a variety of foods in order to obscure the otherwise discernible intent of the study and was designed to conform as closely as possible to randomized, single-blinded, and placebocontrolled clinical experimental protocol (Hulley, Cummings, Browner, Grady, & Newman, 2013). Once the experimental manipulations were completed, participants performed the various experimental tasks via an online survey instrument. Participants were then provided a meal as reciprocation for having fasted overnight, as well as the opportunity to win monetary prizes in a raffle. In order to get participants to take the survey more seriously, the values of monetary prizes in the raffle were based on participants' responses to survey items asking them to make decisions regarding monetary outcomes. Processes associated with conducting the study are described in detail below.

Participants

Participants were 319 undergraduate students. The sample was 41% male. The average age of participants was 22.5 years (SD = 5.01) with an average of 3.45 years of work experience (SD = 3.45). The average self-reported GPA of participants was 3.36 (SD = 0.40). The sample was approximately 39% White, 33% Asian (nonIndian), 11% Indian, 8% Black, 5% American Indian, and 1% Pacific Islander. Additionally, 32% of the participants were Hispanic.

Recruitment. Participants were recruited via announcements in undergraduate classes at the Bauer College of Business at the University of Houston during the fall semester of 2015. An instructor of MANA 3335, Introduction to Management and Organizational Behavior, was contacted via email and face-to-face meeting and approved efforts to recruit students from her classes. She taught two large sections that semester, each with an enrollment capacity of 215 students. Both sections were at capacity. The classes were both on Mondays and Wednesdays, one from 8:30am-10am and the other from 11:30am-1pm. The study was conducted during one full class period (i.e., during the normal class meeting time) for each section of MANA 3335 involved in the study. She awarded 10 points of extra credit to her students for participating. No persons having access to or influence on students' grades (i.e., professors, instructors, or teaching assistants) were involved in the recruitment for or conduct of the study beyond permitting me access to classes in order to recruit, reminding students about the study via email and Blackboard announcements, and awarding extra credit to students in exchange for their participation, when applicable. Persons not eligible or otherwise unable to participate in the study were permitted by the professor an alternative means of earning the 10 points of extra credit awarded for participating in the study. Procedures related to awarding extra credit are discussed shortly. I collaborated with the University of Houston Institutional Review Board (IRB) to ensure this study met their criteria. The study was approved by the IRB prior to commencement.

Interested parties were asked during the recruitment process to preregister for the study by sending their names and a brief statement regarding intent to participate to a special email address I created specifically for that purpose. This stratagem served four purposes. Firstly, it allowed me to estimate the amount of study-related materials needed on-site for each iteration of the study. Students were informed that preregistering would assure that enough food was on hand that they need not leave hungry. Students were also told that they would not be penalized for failing to show up, but that study materials and food might be needlessly wasted. Students were told they could show up and participate in the study even if they didn't register, but that space might be limited. Thus, the ultimate goal of preregistering participants was to help prevent wasting resources such as food and money. Secondly and relatedly, asking potential participants to preregister should have increased the likelihood that interested parties actually showed up to the study because they had precommitted to doing so (Elster, 2000). Thirdly, for similar reasons, preregistering participants should have increased participant compliance with the study requirements outlined below. Fourthly, having participant email addresses allowed me to organize and disseminate relevant information (e.g., date, time, and location of the individual experimental sessions). For instance, the email addresses were used to send out emails reminding preregistered students about the study a week before and a day before the study date. As mentioned above, I also requested that the participating professor make

announcements about the study via Blackboard a week before and a day before the study date. Names and email addresses collected during the recruitment process are not traceable to survey results because no identifying information was collected during the study, so I was not able to confirm who actually showed up to participate in the study. This is not to say however that participation was anonymous.

Participants were informed during the recruitment process as well as at the beginning of the online survey instrument via the informed consent document (discussed below) that participation was confidential. Although no identifying information was collected during the study (not even IP addresses were collected), a mechanism (a raffle ticket number, discussed in greater detail below) was in place that allowed me and the students to report a unique numerical identifier to the students' professor in order to facilitate awarding extra credit to students in exchange for their participation. Although I did not have access to the names of students who participated and the students' professor did not have access to the survey results, the numerical identifier could conceivably be used to link individual participants to their survey responses. However, proper data safety protocols were utilized, such that the students' names and data will never be stored in the same physical location or on the same hard drive. Additionally, all data associated with the study is being maintained on a password-protected hard drive only accessible by me.

Requirements to participate in this study. Potential participants were provided the following summary and list of requirements for participating in this study during the recruitment process as well as the morning of the study. The use of second-person pronouns below reflects the fact that this language was directed at potential participants.

You are cordially invited to participate in a research study in which you will taste several food items and answer a series of questions on an online survey. The study will take place during your normal class time. In exchange for your participation, you will receive lunch (pizza and soda) after the study, 10 points of extra credit from your professor, and the opportunity to win a share of \$200 in monetary prizes in a raffle after the study. However, in order to participate, you must meet the requirements enumerated below:

- 1. You must fast overnight, consuming nothing but common beverages such as water and coffee during the 12-hour period immediately preceding the study. In other words, you should refrain from eating any food and from drinking any calorie-dense beverages such as liquid meal replacements, protein shakes, and milk after dinner the night before the study. Most importantly, please do not eat breakfast the morning of the study. Additionally, please refrain from consuming any beverages for at least 2 hours prior to the study.
- 2. You must not have dairy, wheat, soy, or nut allergies. The foods you will taste during the study contain ingredients derived from these sources. Also, you are not required to eat the provided lunch of pizza and soda after the study, but if you choose to, please be mindful of any food allergies you may have such foods (e.g., dairy, wheat, egg).
- 3. You must not have any medical conditions that would present a risk from fasting overnight, such as diabetes. If you are unsure whether you should participate, please speak with a qualified medical practitioner.

- 4. You must bring a laptop or tablet computer from which you can access the internet in order to take the online survey instrument.
- 5. Please also bring a pen, pencil, and eraser.

Persons not eligible to participate because they do not meet the above requirements will be permitted by your professor an alternative means of earning the 10 points of extra credit awarded for participating in the study. If you do not bring the necessary supplies (listed in items 4-6 immediately above), you will likely not be able to participate in the study because the researcher cannot provide those materials to you. Also, if you are late to the study, you will likely not be able to participate in the study. Persons who do not bring the required materials or show up late will forfeit the opportunities to participate in the study, earn extra credit for participating in the study, participate in the raffle, and eat lunch after the study. However, they will still be permitted the same alternative means of earning 10 points of extra credit by the professor. For further details, please contact your professor.

Your participation in this study will be confidential. Although no identifying information is collected during the study (not even IP addresses will be collected), a mechanism (a raffle ticket number, discussed in greater detail below) will be in place that will allow the researcher and you to report a unique numerical identifier to your professor in order to facilitate awarding extra credit in exchange for your participation. Although the researcher will not have access to your name once the study is complete and the students' professor will not have access to the survey results, the numerical identifier could conceivably be used to link individual participants to their survey responses. However, proper data safety protocols will be utilized, such that students' names and

survey data will never be stored in the same physical location or on the same hard drive.

Additionally, all data associated with the study will be maintained on a passwordprotected hard drive only accessible by the primary researcher.

No deception is used in conducting this study.

Additional information regarding the participant recruitment process. It should be noted that participants were not told to abstain entirely from common beverages, including caffeinated beverages, except during the two-hour window immediately prior to the study. The reasoning for this was based on two rationales. Firstly, liquids are processed by the stomach faster than are solids (Lartigue et al., 1994; Maurer, 2012; Urbain & Davidcharkes, 1995). Per the literature on gastric emptying, it seems abstaining from liquids for two hours before the study would be sufficient. Secondly, abstaining from caffeine can affect cognitive performance, decision-making, behavior, and mood (Chambers, 2009; Einöther & Giesbrecht, 2013; Frankish, 2011; Häusser, Schlemmer, Kaiser, Kalis, & Mojzisch, 2014; James, 2005, 2014; James & Gregg, 2004; James & Rogers, 2005; Maridakis, Herring, & O'Connor, 2009). Thus, it was important to have no explicit restriction on caffeinated beverages so that people who typically consumed caffeine were not suffering adverse cognitive effects as a result of having abstained.

The Study

Set up. Regarding the setting for the experimental sessions, the experimental manipulations and tasks for each of the two conditions were conducted in separate but similar rooms (i.e., one or more rooms for each condition). I, along with a team of volunteers (hereafter collectively referred to as the researcher(s)), prepared the rooms

prior to the stated arrival time for participants (i.e., their normal class time). At each available seat within each room, the researcher(s) placed the food items used for the experimental manipulations and a preformatted sheet of paper. All of this is discussed in great detail below. As a preview, the preformatted sheets of paper were used by participants at the end of the survey to write down their responses to a preselected set of survey items and responses to the manipulation checks, as well as their raffle ticket numbers. The survey responses they were instructed to write down were presented in a summary page at the end of the survey for their convenience. The researcher(s) arranging the two rooms with the food items used for the experimental manipulations took appropriate food safety precautions such as thoroughly washing hands, wearing gloves and hairnets, and using clean serving utensils and containers, most of which were new and used only for the purposes of this study.

Check-in procedures. On the day of the study, an area outside the two rooms (e.g., a table stationed in the corridor) was used for checking in participants, as per the procedures described below:

- Participants reported the class from which they were recruited in order to ensure that only persons who were actively recruited were permitted to participate in the study.
- Participants received a raffle ticket. Raffle tickets were distributed from a standard sequentially-numbered roll. The raffle ticket numbers served three purposes: 1) they were used to facilitate the selection of raffle winners, 2) they permitted linking handwritten survey responses to those provided via the online survey, as raffle ticket numbers were entered on both, and 3) they were reported

back to students' professor in order to facilitate awarding extra credit to those who participated in the study. All three of these purposes are discussed in greater detail below. It is important to note that the researcher(s) did not collect participants' names or student ID numbers and thus cannot personally identify them or link them to their survey responses. Both the participants and I reported raffle ticket numbers to the professor from whose classes participants were recruited. This device allowed the professor to determine and verify those students who completed the requirements for extra credit. However, the professor did not have access to survey responses and, thus, could not link individual students to survey responses via the raffle ticket numbers.

- Participants were randomly assigned to one of the two conditions, H or S, via a list of randomly generated numbers. The randomly generated numbers that comprised the list were designated to participants as they arrived (i.e., consecutively), with even numbers assigned to Condition H and odd numbers assigned to Condition S. Numbers on the list were crossed off as they were designated to participants. These numbers served no other purpose.
- The researcher(s) ensured that participants brought a laptop or tablet from which the survey could be accessed, as well as some writing utensil(s).
- The researcher(s) reminded participants that they should not participate if they had dairy, wheat, soy, or nut allergies.

After each participant had completed the above-described check-in procedure, he or she was instructed to enter the room in which his or her experimental condition was being hosted (as determined by the list of randomly generated numbers) and to have a

seat at any available location. Once the maximum allowable number of participants (determined by the number of available seats in the rooms) had checked in and was seated in the two rooms, instructions for accessing the online survey were posted in each room. All further instruction were provided via the online survey instrument discussed below. Participants were instructed to raise their hands if they had any questions during the study. As mentioned, at least one researcher was present in each room.

Experimental manipulations. As stated above, in order to test the above hypotheses, a research study was proposed in which participants, having fasted overnight, arrived at the experimental setting and were randomly assigned to either a hunger condition (Condition H) or a satiety condition (Condition S), differentiated by the quantity of nutriment provided. Critically, it was assumed all participants arrived experiencing some degree of homeostatic hunger as a result of having fasted overnight (Appelhans, 2009; Berthoud, 2011; Epstein et al., 2007; Goldstone et al., 2009; Saper et al., 2002). Thus, the intended outcome of the experimental manipulation was to maintain participants in Condition H in a state of hunger and transition participants in Condition S to a state of satiety. The simplest approach would have been to provide Condition S nutriment while withholding nutriment from Condition H. However, this approach presents at least one obvious confound in that Condition S would have experienced a markedly different pretask intervention by receiving a food reward. Additionally, due to the explicit requirement for overnight fasting by participants, both conditions might have been able to ascertain the intent of study easily, leading to demand effects influencing their survey responses (Orne, 1969; Zizzo, 2010). Considering these issues, it was

necessary that both conditions experience a similar pretask intervention while simultaneously obscuring the intent of the study.

In order to demonstrate causality unsullied by confounding variables, it was my aim to exemplify the spirit, if not the precise form, of a randomized, single-blinded, placebo-controlled clinical trial. This entailed a parallel, between-groups design wherein one group received an intervention and the comparison group received essentially a nonactive treatment (Hulley et al., 2013). However, considering the goal of the experimental manipulation, which was to have participants in Condition H in a state of hunger and participants in Condition S in a state of satiety, it was required that I be somewhat craftier than providing a clichéd placebo pill to the control group. As all participants should have arrived in a state of hunger, it was necessary to transfer one group to a state of satiety (Condition S) while leaving the other in a state of hunger (Condition H), while providing both groups a similar pretask experience.

There are at least two ways this goal could have been accomplished for this study. The first method involves providing each group the same quantity of different forms of nutriment; for instance, Condition S might receive a high-calorie nutritional shake while Condition H receives a zero-calorie shake. However, this method seems unfeasible because low- and zero-calorie foods are often markedly different from normal or high-calorie foods in terms of taste, smell, and mouthfeel. In other words, such a manipulation may have introduced a confound based solely on the rewarding properties of the food substances because one group received foodstuffs that tasted good while the other group received foodstuffs that tasted (at least relatively) bad. Such a manipulation would also have given both conditions a similar degree of gastrointestinal distention, which is an

important factor in inducing satiety (Wang et al., 2008). The second method to achieve the goal involves providing both groups with differing quantities of the same form of nutriment; for instance, Condition S might receive a large serving of a nutritional shake while Condition H receives a small serving. However, such an obvious manipulation may have led subjects, perhaps particularly in Condition H, to ascertain the purpose of the experiment, introducing demand effects. Thus, a further tweak was necessary, per which both groups would receive differing quantities of more than one foodstuff, transferring their focus from the total quantity of nutriment provided to the variety of flavors provided. It is hoped that this tweak caused participants to believe that the experiment concerned perceptions of taste rather than manipulations regarding hunger and satiety. Further inculcating this belief among participants was accomplished by explicitly asking them to rate the flavors provided as part of the survey instrument.

Thus, during the course of this research experiment, participants were provided nutriment in the form of nutritional shakes and nutritional bars, the former of which, at least, has precedent in related research (e.g., Gailliot et al., 2007; Gailliot & Baumeister, 2007; Mishra & Mishra, 2010). A popular high-calorie, high-protein, gluten-free, lactose-free, nonGMO, Kosher-certified, Halal-certified, no-artificial-sweetener brand of meal replacement shakes was used (see http://www.drinkenu.com/). A popular high-calorie, high-protein, high-fiber, nonGMO, certified organic, no-artificial-sweetener brand of meal replacement bars was used (see http://shop.theprobar.com/Products/PROBAR-Meal). In order to mask the true intent of the study, all participants were provided two different flavors of the nutritional shake, chocolate and vanilla, as well as three different flavors of the nutritional bar, "Superfood Slam," "Peanut Butter Chocolate Chip," and

"Wholeberry Blast." These brands and flavors were chosen both because they meet the nutritional requirements of the study (discussed below) and because they are very highly rated by consumers on Amazon.com. However, all labeling was removed so that participants were not able to identify the brands of nutriment provided. Participants were told during the recruitment process as well as before the study that individuals allergic to dairy proteins, wheat, soy, or nuts should not participate in the study.

Regarding the nutritional shakes, both conditions received the two flavors of shakes in identical 8 ounce opaque cups with lids and were instructed during the experiment to consume the entire contents of both cups they received. Containers with chocolate-flavored shake were labelled with a ¾ inch round yellow sticker and containers with vanilla-flavored shake were labeled with a ¾ inch round orange sticker. The unopened shakes were each 11 ounces. Each participant in Condition H received one ½ ounce serving (approximately 1/24 of a full shake, or one tablespoon) of the chocolate-flavored shake and one ½ ounce serving of the vanilla-flavored shake. Each participant in Condition S received one 5.5 ounce serving (approximately ½ of a full shake) of the chocolate-flavored shake and one 5.5 ounce serving of the vanilla-flavored shake.

Regarding the nutritional bars, both conditions received the three flavors of bars in identical 4 ounce translucent plastic disposable portion cups with lids and were instructed during the experiment to consume the entire contents of all three containers they receive. Containers with "Superfood Slam" flavored bars were labeled with a ¾ inch round red sticker, containers with "Peanut Butter Chocolate Chip" flavored bars were labeled with a ¾ inch round green sticker, and containers with "Wholeberry Blast" were labeled with a ¾ inch round blue sticker. The unopened bars were each 3 ounces. Each

participant in Condition H received one 1/8 ounce serving (approximately 1/24 of a full bar) of each of the three flavored bars. Each participant in Condition S received one 1.5 ounce serving (approximately ½ of a full bar) of each of the three flavored bars.

Thus, the manipulations and pretask experiences of Conditions H and S were designed to be identical in all regards, except the quantity of nutriment consumed. Moreover, steps were taken to shift participants focus from the manipulated variable (i.e., the quantity of nutriment) to the variety of flavors of nutriment they were asked to taste by asking them to subjectively rate each flavor of nutriment (discussed below). Despite efforts to make the conditions indistinguishable to participants, it is hoped that the experimental manipulation would engender important differences between the two groups. In Condition H, participants performing the experimental task should have experienced homeostatic hunger as a result of having fasted overnight, as well as hedonic hunger as a result of 1) having received and consumed a small quantity of appetitive food stimuli (i.e., the nutritional shakes and bars) and 2) anticipating the post-study meal of pizza and soda (Appelhans, 2009; Berthoud, 2011; Epstein et al., 2007; Goldstone et al., 2009; Saper et al., 2002). In Condition S, participants performing experimental task should have experienced a state of satiety, having consumed the provided calorie-dense meal (i.e., the nutritional shakes and bars). The approximate nutritional intake for all foods consumed in both conditions is shown in Table 1 below. For condition H, the caloric load of 88 calories provided in the experiment is roughly equivalent to 0.6 ounces of potato chips, or approximately 7 to 8 chips.

TABLE 1

Conditions	I acitibao
Nutritional Data for Experimental	S acitibao

ving Size al Shake al Bar from fat fat	11 fl. oz. (330 mL) 4.5 oz. (127 g) Amount per Serving % of Daily Value 1060 405	1 fl. oz. (30 mL) 0.6 oz. (16 g) Amount per Serving % of] 88	30 mL) (16 g) % of Daily Value
al Shake al Bar from fat fat	(127 g) g % of Daily Value	1 fl. oz. (0.6 oz. Amount per Serving 88	(30 mL) (16 g) % of Daily Value
al Bar From fat fat	. (127 g) g % of Daily Value	0.6 oz. Amount per Serving 88	(16 g) % of Daily Value
from fat fat ol	ig % of Daily Value	Amount per Serving	% of Daily Value
from fat fat ol		88	
from fat fat ol			
. fât ol		34	
fât ol	%69	4 g	%9
lo.	41%	18	3%
lo.		g 0	
		0 mg	
Sodium Sodium	13%	24 mg	1%
Total carbohydrate 129.5 g	43%	11 g	4%
Dietary fiber 10 g	42%	1 g	3%
Sugars 47.5 g		4 g	
Protein 40 g	71%	39	%9

Percent Daily Values are based on 2,000 calorie diet. Values are approximate.

Rationale for using the nutritional shakes and bars. The nutritional shakes and bars were chosen as the satiety-inducing meal for two primary reasons. First, such foods are highly palatable (see the previous discussion on sweet and fatty foods; see also Bertino et al., 1991; Drewnowski, 1995; Drewnowski & Greenwood, 1983; Drewnowski et al., 1992) and should help ensure compliance with experimental protocols. Secondly, macronutrient content is an important consideration for inducing satiety (de Castro, 1987; Gerstein, Woodward-Lopez, Evans, Kelsey, & Drewnowski, 2004; Rolls, Hetherington, & Burley, 1988); this food and drink combination provides ample amounts of carbohydrates, proteins, and fats, at least for Condition S. Moreover, these foods are very high in protein, the most satiating macro-nutrient (Astrup, 2005; Bertenshaw, Lluch, & Yeomans, 2008; Gerstein et al., 2004; Rolls et al., 1988; Vandewater & Vickers, 1996; Weigle et al., 2005). Gastric distention is also an important factor in satiety (Wang et al., 2008), which is why participants in Condition S are required to consume a greater quantity of nutriment than Condition H.

The Survey Instrument

Before beginning the survey instrument, each participant completed the check-in procedures and was seated in the rooms specified by his or her given experimental condition. They had all materials necessary to complete the study: laptops or tablets, and writing utensil(s) they brought with them, as well as the nutritional shakes and bars, and preformatted answer sheets placed at each available seat within the experimental session rooms.

Introduction to the online survey instrument. In order to standardize and equalize participant experiences across the two conditions, all instructions and the

informed consent were provided via text in the online survey instrument. Participants were discouraged from rushing though the survey by explicitly stating there was no incentive to finish early because everyone must finish before any participants were allowed to eat pizza and before the raffle begins. However, participants were informed that they were welcome to leave after completing the survey, but that by doing so they forfeited the post-task meal and opportunity to participate in the raffle. Participants were encouraged to silence their phones. Participants were encouraged to close all browser windows and apps unrelated to the study on their laptops or tablets. Participants were discouraged from talking to each other for the duration of the study, though talking seemed unlikely because of the presence of the researcher(s) in the room. Regarding this latter point, there was at least one researcher in each room at all times. The researcher(s) served two primary purposes during the experimental sessions. Firstly, they provided participants instructions on how to access the online survey instrument. Secondly, they monitored the sessions, which should have passively discouraged interaction among the participants, and unobtrusively answered any questions posed by individual participants during the sessions.

Informed consent. Informed consent was gathered via cover letter on the online survey instrument. Participants were required to sign the informed consent document to acknowledge that they read and understand the document. As part of the informed consent, participants were assured that no deception was to be used throughout the course of the experiment.

Measures

Immediately preceding the experimental tasks described below, participants were informed that the values of individual monetary prizes awarded during the raffle would be determined by their responses to survey items that asked them to make decisions about monetary outcomes and that payments would be made in cash by the researcher(s). It is important that the decisions involved real monetary outcomes so that participants took the experimental tasks more seriously. Although hypothetical and real rewards activate common valuation areas (Kang, Rangel, Camus, & Camerer, 2011), "the use of real monetary rewards is important both because it creates objective incentives for accurate responding, and because discount rates for hypothetical rewards are typically much lower than those for real rewards, possibly creating floor effects that would make group differences more difficult to discern" (Kirby & Petry, 2004: 468; see also Johnson & Bickel, 2002; Kang et al., 2011; Kirby, 1997; Lagorio & Madden, 2005; Madden et al., 2004; Madden, Begotka, Raiff, & Kastern, 2003).

Temporal Discounting. There are numerous methods of eliciting discount rates and risk propensity, and much debate and controversy surround the issues of functional forms, measurement, and analysis (Andersen, Harrison, Lau, & Rutström, 2008b; Andersen et al., 2014; Andreoni, Kuhn, & Sprenger, 2015; Andreoni & Sprenger, 2012a, 2012b; Hardisty, Thompson, Krantz, & Weber, 2013; Smith & Hantula, 2008). The task for eliciting time preferences were based on the procedures outlined by Andersen and colleagues (Andersen et al., 2008b, 2014), often referred to as double multiple price lists (DMPL) (Andreoni et al., 2015; Bradford, Courtemanche, Heutel, McAlvanah, & Ruhm, 2014; Charness, Gneezy, & Imas, 2013). In this task, participants made 40 decisions

between two options labeled "A" and "B." As a sample, say Option A pays \$20 today, and Option B pays \$25 one month from now. This example represents a classic scenario involving SS and LL rewards referenced in the review section on temporal discounting. The amounts and payment dates varied across the 40 decisions. As a note, the papers by Andersen and colleagues (Andersen et al., 2008b, 2014) present monetary values in kroner rather than dollars. Thus, monetary values have been transformed via the exchange rate (approximately 5.5:1, kroner to dollar) at the time the 2014 paper was published. This conversion rate is also relevant to the monetary values used in assessing risk preferences.

Risk Propensity. The task for eliciting risk preferences was also based on the procedures outlined by Andersen and colleagues (Andersen et al., 2008b, 2014), who adapted popular procedures used by other researchers (Hey & Orme, 1994; Holt & Laury, 2002, 2005). In this task, participants were again asked to make 40 decisions between two options labeled "A" and "B." As an example, say Option A pays \$12 if the outcome (number of pips on the uppermost face) of a roll of a 10-sided die is 1 and \$8 if the outcome is 2-10. Option B pays \$18 if the outcome of the die roll is 1 and two dollars if the outcome is 2-10. Although participants were not explicitly told so, it is apparent that Option B is the riskier option because the expected value of Option A is \$8.40, whereas the expected value of Option B is \$3.60. However, the maximum potential gain is larger in Option B. The amounts and probabilities varied across the 40 decisions.

(Un)Ethicality. As discussed in reviews by Treviño and colleagues (2014, 2006), unethicality includes behaviors such as lying, cheating, and stealing. Conversely, ethicality includes behaviors such as being honest and treating people with respect,

whereas extraordinary ethicality includes behaviors such as charitable giving. In line with previous research, I probed the unethicality-ethicality spectrum via a task that allowed participants to cheat or be honest and probed extraordinary ethicality via participants' willingness to donate to charity. Many studies examining (un)ethicality operationalize the construct as participants' (dis)honesty in self-reporting scores on, for instance, problemsolving, multiple-choice, perceptual, and/or anagram tasks. These tasks generally last several minutes, with each correct answer worth a small amount of money (e.g., \$0.10) (Barnes et al., 2011; Gino & Ariely, 2012; Gino & Margolis, 2011; Gino & Pierce, 2009; Gino et al., 2011; Mazar et al., 2008; Schweitzer, Ordóñez, & Douma, 2004; Yam, Chen, et al., 2014; Yam, Reynolds, et al., 2014). At least one study also used participants' adherence to less-than-strictly imposed time limits on such tasks as a mechanism for detecting cheating; in this case, "spending more than the prespecified amount of time to complete the task was used as a measure of unethical behavior" (Yam, Reynolds, et al., 2014: 128). In the current study, I allow participants to act unethically by overstating the number of answers they get correct as well as by exceeding a less-than-strictly imposed time limit.

More explicitly, for this task, participants were told they were to solve as many (up to 60) simple math problems (involving addition and subtraction of single- and double-digit numbers, as well as multiplication of single-digit by double-digit numbers, e.g., 42+17) as possible in two minutes and that a few people would be drawn at random to receive \$1 per correct answer. The full set of 60 problems was presented in subsets of 15 questions on four separate pages, each displaying a timer counting down from 30 seconds to zero. Thus, each timer effectively indicated when the 30-second period

allotted for that page had expired. However, participants could continue to solve math problems beyond each 30-second window under the guise that the software could not or would not automatically stop the task and progress to the final screen. This device allowed participants to "cheat" up to four times by exceeding the stated time limit on each page. It should be noted that participants were allowed to stop this task at any time during the task by clicking a special button available on the bottom of each page. One of the entries on the summary report provided at the conclusion of the online survey instrument (mentioned above and discussed below) displayed the number of questions each participant answered correctly on this task. This set-up provided subjects a final opportunity to cheat by overreporting their scores on the task.

For the extraordinary ethicality portion of the task, participants were asked to provide the percentage (from 0% to 100%) of any monetary prizes they may have won in the raffle that they would have been willing to donate to a charity, nonprofit, or cause of their choice, along with the name of said organization. A list of popular organizations was provided to help facilitate this process, covering a sufficiently wide range of sociopolitical issues. A previous study (Gino & Margolis, 2011) gave participants the opportunity to donate to only one cause, National Public Radio (NPR). Providing only one outlet for donations seems restrictive because some participants may be unfamiliar or otherwise opposed (for social or political reasons) to any given cause or charity.

Hunger. Assessing subjective ratings of hunger and satiety were crucial in showing that the experimental manipulations were successful. Thus, an online version of the standard 100mm visual analogue scale will be used for assessing subjective ratings of hunger (Flint, Raben, Blundell, & Astrup, 2000). In this questionnaire, subjects are asked

questions regarding how hungry they feel, how satisfied they feel, how full they feel, how much they think they can eat, and whether they would like to eat sweet, salty, savory, or fatty foods. Variants of the scale have been used by numerous researchers (Horner, Byrne, & King, 2014; Masic & Yeomans, 2014; Porubská et al., 2006; Sarker, Franks, & Caffrey, 2013; Siep et al., 2009) and it has been validated for use via electronic format (Brunger et al., 2015). The scale was administered as part of the online survey instrument. Due to random assignment of participants, I did not feel it was necessary to administer the survey before the experimental sessions. However, it was necessary to ensure that subjects had indeed complied with the requirement for overnight fasting. Thus, at the end of the survey, participants were asked to estimate the time of their last preexperiment caloric intake. Noncompliant subjects (e.g., subjects who had eaten breakfast) could then be removed from the dataset. However, this approach may be overly conservative, as many studies concerning hunger have only required participants to fast for 3 to 5 hours prior to the study (e.g., Aarøe & Petersen, 2013; Frank et al., 2010; Tal & Wansink, 2013; Yam, Reynolds, et al., 2014), though some studies have required overnight fasting (e.g., Goldstone et al., 2009; Haase et al., 2009, 2011; Siep et al., 2009; Wansink et al., 2012; Wierenga et al., 2014)

It should be noted that a more objective measure of hunger would be ideal, such as measuring circulating ghrelin. However, there are numerous factors that make collecting blood samples from participants unfeasible for this study, including cost, timing, and issues related to proving the necessity of collecting blood samples in petitioning the IRB for approval of the study. Moreover, there are issues related to the collection and analysis of ghrelin, specifically. Ghrelin is highly unstable with a short

half-life and is affected by conditions of collection and storage, the pH, and the method of measurement, necessitating stringent standardization of the preparation of samples to ensure reliable measurement (Hosoda & Kangawa, 2012). Because circulating ghrelin is highly correlated with self-reported hunger (Cummings et al., 2004), the visual analogue scale discussed above was used.

Conclusion of the online survey instrument. As mentioned above, participants were provided a preformatted sheet of paper that was to be used at the end of the survey to write down their responses to a preselected set of survey items (as well as their responses to the manipulation checks at the beginning of the survey), along with their raffle ticket numbers. The survey responses they were to write down were presented in a summary page at the end of the survey for their convenience. The summary report comprised six of their responses to items on the survey instrument. Five items were taken from their responses to the risk propensity task and one item from the un-ethicality cheating task. Participants were reminded that they would receive one dollar per correct answer for the un-ethicality cheating task. Responses to the temporal discounting task were not used because doing so would have required establishing a system by which delayed rewards could be paid out, which likely would have violated confidentiality because these delay discounting responses would have had to be traced back to the individual to facilitate payment at a future date in cases wherein delayed responses were chosen. So, upon viewing the summary screen, subjects were instructed to write down the responses displayed on the screen, along with their raffle ticket number, on the sheet of paper to be turned into the researcher(s) in order to facilitate the selection of prizes for the raffle winners. Participants were explicitly encouraged to write legibly. At the end of the

survey, all of the filled-in preformatted sheets of paper were collected in a large box, in which they were tumbled so that they were randomized, and from which they were drawn during the raffle.

Post-task meal. At the conclusion of the online survey and prior to the start of the raffle, participants were allowed to consume a provided meal of pizza and soda *ad libitum*. While participants were eating, the raffle was conducted as described below.

The raffle. As mentioned above, participants were informed immediately before the experimental tasks that the values of individual monetary prizes awarded during the raffle would be determined by their responses to survey items that asked them to make decisions about monetary outcomes and that payments would be made in cash by the researcher(s). The raffle was conducted by drawing the filled-in preformatted sheets of paper from the large box in which they had been tumbled. The raffle number written on each sheet of paper drawn from the box was read aloud. Participants with raffle ticket numbers matching those written on the papers drawn from the box were instructed to come to the front of the room. Those participants then rolled a common six-sided die. The number of pips on the uppermost face of the die determined which of the six survey responses written on the preformatted sheet of paper was used to determine their cash reward. The researcher(s) had copies of the actual survey items from which the report on the summary screen was derived. For example, if the uppermost face of the six-sided die displayed one pip after the participant rolled the die, the first answer choice they wrote down was used to determine their cash reward. In this case, it would have been a risk propensity task portraying a scenario involving different payouts depending on the outcome of rolling a 10-sided die. The researcher(s) would have then referred to a copy

of that particular survey item to see how the options (i.e., dollar amounts and probabilities) were structured. For an example, refer to the discussion of the risk propensity measure above. Participants then would have rolled a 10-sided die to determine their payout based on the dollar amount specified by the outcome of the die roll. If the uppermost face of the die displayed six pips, the self-report of the number of correct answers on the un-ethicality cheating task would be used to determine the payout at a rate of \$1 per correct answer.

Chapter 8

ANALYSES

I estimated the model using Stata's (StataCorp, 2015) structural equation modeling function. I essentially ran an instrumental variable regression, using the experimental condition as an instrument, hunger score as an endogenous regressor, the hypothesized outcome variables (discount score, risk score, unethicality score, and ethicality score) as dependent variables, and exogenous covariates (age, gender, GPA, years of work experience, ethnicity, hours since last meal, and size of last meal) as controls in both stages. Supporting my use of instrumental variable regression, the Hausman (1978) endogeneity test was significant for the covariance between the risk score and hunger score, indicating a common cause (i.e., the condition) of risk and hunger, meaning that hunger score was endogenous and should be instrumented. Additionally, hunger score is necessarily endogenous with respect to the experimental condition because the purpose of the experimental condition was to establish participants' subjective perceptions of hunger. The decision to use the experimental condition as an instrumental variable rather than as the primary independent variable was made because the researcher(s) witnessed a general lack of compliance with the experimental manipulation during the experimental sessions (more on this below). Thus, it was thought that hunger score might be a better predictor of the outcome variables, though hunger score still must be treated as endogenous with respect to condition. In the first stage of the instrumental variable regression, the experimental condition (i.e., the instrument) and all control variables (age, gender, GPA, years of work experience, ethnicity, hours since last meal, and size of last meal) were used to predict participants' hunger scores. In the second stage, their hunger scores and all control variables were used to predict the dependent variables (discount score, risk score, unethicality score, and ethicality score).

I used a robust (i.e., sandwich) estimator (Huber, 1967; White, 1980, 1982) to obtain the variance-covariance matrix of the estimates, including the reported standard errors, in order to account for possible violations of the assumption regarding multivariate normality. Additionally, I used maximum likelihood with missing values as the method to obtain estimated parameters. The covariance structure of all exogenous variables, including the dependent variables, was left unstructured, allowing the disturbances to correlate. The reader may note that fit indices (e.g., chi-square test of fit) and R^2 are not included in the results. Fit indices are not appropriate here because when there is "only one instrument, the model [is] just-identified and a test of fit cannot be conducted (though the Hausman endogeneity test can still be done)" (Antonakis, Bendahan, Jacquart, & Lalive, 2010: 1102).

 R^2 is not reported here because it has no real statistical meaning in the context of instrumental variable regression. In the model used here to estimate the parameters, the regressor representing the experimental condition was entered as an instrument. "However, since our goal is to estimate the structural model, the actual values, not the instruments for the endogenous right-hand-side variables, are used to determine the model sum of squares (MSS). The model's residuals are computed over a set of

regressors different from those used to fit the model. This means a constant-only model of the dependent variables is not nested within the [model], even though the [model] estimates an intercept, and the residual sum of squares (RSS) is no longer constrained to be smaller than the total sum of squares (TSS). When RSS exceeds TSS, the MSS and the R^2 will be negative" (Sribney, Wiggins, & Drikker, 2015). Indeed, R^2 for risk score in the second stage of the model was negative.

Instead of reporting R^2 , Bentler-Raykov squared multiple correlation coefficients are reported (Bentler & Raykov, 2000). Similar to R^2 , it quantifies the explained variance (i.e., the relatedness or fit of data for a dependent variable with the model's linear prediction), and in a recursive model the two statistics would represent different versions of the same number. However, because of the abovementioned problems inherent to using R^2 in nonrecursive models, it is recommended that researchers use Bentler-Raykov squared multiple correlation coefficients for nonrecursive systems involving endogenous variables with reciprocal causations (StataCorp, 2015). These coefficients are presented in Table 3.

Additionally, statistics are reported for equation-by-equation Wald tests (see Table 4). The null hypothesis for these Wald tests is that the coefficients for the observed variables (other than the intercepts) are zero (StataCorp, 2015).

Chapter 9

RESULTS

In the first stage of the instrumental regression model, participants' hunger scores largely depended on their having been in the hunger condition. Although the experimental condition statistically predicted participant hunger scores (β = 60.79, p < .01 in stage 1 of the instrumental variable regression procedure, see Table 2) and there was a significant difference in hunger scores between the experimental groups (mean hunger scores were 23.06 [SD = 90.62] for the satiety condition and 81.36 for the hunger condition [SD = 62.09], F(1, 317) = 45.17, p < .01), the experimental manipulation was not as strong as intended. Ideally, the satiety condition would have reported a negative hunger score, indicating a state of satiety rather than low hunger. I believe this weaker-than-intended manipulation resulted from participants largely failing to comply with the experimental manipulation. Participants were observed throwing away much of the food and beverage provided to them during the experimental sessions, with at least some proclaiming that the food was not palatable. This circumstance should be kept in mind when considering the results discussed below.

In the second stage of the instrumental regression model, participants' subjective perceptions of hunger (i.e., their hunger scores) were used to predict the outcome variables of concern (discount score, risk score, unethicality score, and ethicality score).

Hypothesis 1, contending that hunger (versus satiety) would be associated with increased temporal discounting of monetary rewards, was not supported ($\beta = .01$, nonsignificant in stage 2 of the instrumental variable regression procedure, see Table 2). However, Hypothesis 2, contending that hunger (versus satiety) would be associated with increased risk propensity regarding of monetary rewards, was supported ($\beta = .03$, p < .05 in stage 2 of the instrumental variable regression procedure, see Table 2). Hypothesis 3, contending that hunger (versus satiety) would be associated with increased unethicality (i.e., cheating) regarding monetary rewards, was not supported ($\beta = <.01$, nonsignificant in stage 2 of the instrumental variable regression procedure, see Table 2). It is interesting to note here that the value for unethicality was particularly low with little variance (M = .28, SD = 2.02, see Table 1), indicating that participants generally did not cheat on the task. Thus, there may have been methodological deficiencies inherent to the task beyond the problems acknowledged above regarding participants' compliance with the experimental manipulation. Additionally, Hypothesis 4, contending that hunger (versus satiety) would be associated with decreased ethicality (i.e., charitable donations) regarding monetary reward, was not supported ($\beta = .02$, nonsignificant in stage 2 of the instrumental variable regression procedure, see Table 2).

TABLE 2
Correlation Matrix of Key Variables

				5				J											
	M	as	1	2	3	4	2	9	7	8	6	10	11	12	13	14	15	16 17	7 18
1. Hunger condition $(= 1; else = 0)$.51	.50																	
2. Discount score	24.20	24.20 11.33	.01																
3. Risk Score	20.47 6.71	6.71	.1 4	08															
4. Unethicality score	.28	2.02	.02	80.	01														
5. Ethicality score	31.28 29.21	29.21	.02	12*	9.	01													
6. Age	22.48 5.01	5.01	00.	60:	.10		.15*												
7. Male $(= 1; else = 0)$.41	.49	02	.10			.14	.13*											
8. GPA	3.36	.40	00.	07			.05		20*										
9. Years of work experience	3.45	3.45	01	90.		.05	.10		.03	10									
10. Hispanic $(= 1; else = 0)$.32	.47	.03	03			08	.05	80:	14*	9.								
11. American Indian $(= 1; else = 0)$.05	.22	.05	02			.01	.13*		11		.21*							
12. Asian $(= 1; else = 0)$.33	.47	04	.02	05		60:	01	07	.16*	-X-	43*	13*						
13. Black $(= 1; else = 0)$	80.	.28	.01	.10	.03		03	60:		12*		09	02	14*					
14. Pacific Islander $(= 1; else = 0)$.01	.10	.03	01	90:	.02	90:	9.		.03		00.	.13*		60:				
15. White $(= 1; else = 0)$.39	.49	*	14*	.05		.03	02		01		.33*	03		17*	01			
16. Indian $(= 1; else = 0)$.11	.31	08	04	01	.02	90	03		02		24*	08		11	03	28*		
17. Hours since last meal	13.96 3.07	3.07	02	04	12*	90:-	.03	12*		.05		80.	90:	14*	07	80:	.07	.03	
18. Size of last meal	2.92	.81	09	90:	40	.04	.05	90:		07	01	.03	07	05	08		03	.07	.23*
19. Hunger	52.67	52.67 82.66	.35*	.02	.04	.08	09	10	.10	00.		.13*	.02	05	.07	.03	00.	06 .12	.12* .04
Note: $n = 319$. * $n < 0.05$.																			

TABLE 3
Instrumental Variable Regression Estimates

Variables:	(1)			(2)	
DVs in columns;	Hunger	Discount	Risk	Unethicality	Ethicality
IVs in rows	score	score	score	score	score
Hunger condition (= 1; else = 0)	60.79**				
	(7.35)				
Hunger score		.01	.03*	.01	.02
		(.65)	(2.31)	(.13)	(.38)
Age	-2.90**	.08	.22	01	1.09*
	(-2.71)	(.46)	(1.84)	(27)	(2.20)
Male $(= 1; else = 0)$	18.31*	1.52	.65	.14	-9.13**
	(2.22)	(1.10)	(.83)	(.52)	(-2.62)
GPA	-1.27	76	1.71	.24	4.83
	(10)	(42)	(1.77)	(.67)	(1.04)
Years of work experience	2.44	.21	08	.03	.22
	(1.65)	(.86)	(54)	(.60)	(.36)
Hispanic (= 1 ; else = 0)	27.82**	11	.60	34	-4.81
	(2.74)	(06)	(.61)	(94)	(-1.14)
American Indian (= 1; else = 0)	-5.15	-1.73	.57	21	1.97
	(33)	(70)	(.36)	(-1.12)	(.23)
Asian (= 1; else = 0)	7.41	57	54	23	6.60
	(70)	(31)	(51)	(59)	(1.44)
Black (= 1 ; else = 0)	30.64*	2.44	45	47	49
	(2.10)	(.94)	(27)	(-1.28)	(08)
Pacific Islander (= 1; else = 0)	51	-1.89	4.08	.67	14.40
	(02)	(34)	(1.25)	(1.10)	(.71)
White $(= 1; else = 0)$	-12.60	-3.30*	.52	.11	6.67
	(-1.31)	(-2.12)	(.54)	(.42)	(1.60)
Hours since last meal	2.81	14	35**	04	.29
	(1.92)	(67)	(-3.03)	(65)	(.48)
Size of last meal	4.44	.76	29	09	2.35
	(.87)	(.88)	(60)	(82)	(1.09)
Constant	14.35	24.05**	13.69**	.42	-21.80
	(.26)	(2.88)	(2.76)	(.24)	(95)
Bentler-Raykov squared					
multiple correlation coefficient	.20	.04	.03	.02	.07

Note: Estimates are unstandardized. Stage 1 utilizes Hunger condition as an instrumental variable to predict the endogenous variable Hunger score. Stage 2 utilizes Hunger score as a regressor to predict the DVs hypothezed in the paper.

Robust z-statistics in parentheses.

DV = dependent variables; IV = independent variables.

n = 319, ** p<0.01, * p<0.05.

TABLE 4
Wald Tests for Equations

		_	
Observed Variables	chi-square	df	р
Hunger score	92.07	13	.00
Discount score	16.18	13	.24
Risk score	24.14	13	.03
Unethicality score	8.26	13	.83
Ethicality score	33.38	13	.00

Note: This table shows equation-by-equation Wald tests that all coefficients excluding the intercepts are zero.

Chapter 10

DISCUSSION

"Lunch? Aw, you gotta be kidding. Lunch is for wimps." – fictional Wall Street stockbroker Gordon Gekko from the film Wall Street (Stone, 1987)

If visceral factors such as hunger influence financial decision-making and, perhaps, promote risk propensity, then perhaps encouraging young stockbrokers to skip lunch so that they literally and figuratively are hungrier may have unforeseen and deleterious effects on the economy, especially considering brokers are already associated with elevated risk-taking (Noll et al., 2012). The present study sought to determine whether the visceral states of hunger and satiety affect decision-making regarding nonfood (i.e., monetary) rewards seemingly unrelated to the instigating visceral states (i.e., hunger and satiety). In order to accomplish this objective, a research study was conducted wherein participants, having fasted overnight, arrived at the experimental setting and were randomly assigned to either a hunger or a satiety condition, differentiated by the quantity of food provided (see Table 1). I hypothesized that being in a state of hunger (versus satiety) would cause people to: 1) engage in more temporal discounting of monetary rewards (or more strongly desire more imminently available monetary rewards), 2) exhibit higher risk propensity regarding monetary rewards, 3) act

more unethically regarding monetary rewards (i.e., cheat to gain a larger monetary prize), and 4) act less extraordinarily ethically regarding monetary rewards (i.e., give less money to charity).

Unfortunately, support for these hypotheses may have been obscured by participants' lack of compliance with the experimental manipulation. The researcher(s) witnessed participants throwing away large portions of the nutritional bars and shakes provided to them, despite being instructed to consume all of the food and drink items presented during the sessions. Participants were overheard remarking that the food items were unpalatable, particularly the nutritional shakes. Some researchers even reported that they witnessed participants grimacing while consuming some of the items. Such reports are surprising given the generally positive reviews posted on Amazon.com for the products used in the study. It is also possible that some participants did not want to become full from eating the nutritional bars and shakes given the impending opportunity to consume pizza and soda ad libitum immediately following the experimental sessions. Overall, it seems that participants' lack of compliance with the experimental manipulation resulted in a statistically significant but weaker-than-intended manipulation effect after which subjects in both experimental conditions reported positive subjective perceptions of hunger. It was intended that subjects assigned to Condition S would report negative hunger scores (i.e., they would be satiated) after the experimental manipulation was completed.

It is possible that I inadvertently introduced one or more confounds to the study related to neurological, psychological, or emotional states as a result of participants' exposure to the unexpectedly aversive nutritional shakes and bars used during the study. The intent was that participants would like the food items (i.e., find the food items to be palatable), though that was apparently not the case. The mesolimbic dopamine system comprising the core reward circuitry responds differently to aversive stimuli than it does to appetitive stimuli (Roitman, Wheeler, Wightman, & Carelli, 2008). For instance, dopamine neurons in the ventral tegmental area may be inhibited by aversive stimuli, while nondopamine neurons in the same region may be excited by aversive stimuli (Ungless, Magill, & Bolam, 2004). Dopaminergic projections to the nucleus accumbens may be excited by appetitive stimuli, but not by aversive stimuli, which seem selectively to excite dopaminergic projections to another area, the medial prefrontal cortex (Lammel, Ion, Roeper, & Malenka, 2011). As I discussed throughout much of the hypotheses development, the activation state of this reward circuitry has important implications for decision-making.

Beyond these neurologically defined effects, there is a more psychological connection between food and emotion (Jiang, King, & Prinyawiwatkul, 2014). Emotional states related to sadness, disgust, and anger may be evoked by disliked foods (de Wijk, Kooijman, Verhoeven, Holthuysen, & de Graaf, 2012). Emotional states have been shown to affect (economic) decision-making generally (Lerner, Li, Valdesolo, & Kassam, 2015; Lerner, Small, & Loewenstein, 2004; So et al., 2015). Emotions have also been shown to affect, more specifically, risk-taking and risk perceptions across several domains (Kugler, Connolly, & Ordóñez, 2012; Lerner & Keltner, 2001; Lupton, 2013; Podoynitsyna, Van der Bij, & Song, 2012; Rosenboim, Benzion, Shahrabani, & Shavit, 2012). For example, fear may cause people to express pessimistic risk estimates and risk-aversive choices, while anger may cause people to express optimistic risk estimates and

risk-seeking choices (Lerner & Keltner, 2001), at least for lottery-based risk tasks (Kugler et al., 2012), which are relevant to the current study. Hence, if participants felt angry as a result of being asked to consume aversive food stimuli, their risk perceptions may have been biased toward increased risk-seeking. I am not insinuating that participants felt any degree of anger as a result of consuming the food items, nor that one group would have felt any more or less of some conceivably aroused emotion than the other group. I am simply including this discussion to account for the possibility that some confound related to unintentional and unmeasured aroused neurological or emotional states may have been introduced.

Thus, it is possible that by including aversive food stimuli in the study, I unintentionally elicited neurological or psychological states that may have, for one reason or another, differed between the two experimental conditions as a function of the quantity of aversive stimuli provided or consumed via the experimental manipulation. Randomization of participants within the experimental conditions would not have accounted for any such differences. Thus, beyond the effects of the weaker-than-intended strength of manipulation, it is possible that any such induced neurological or psychological states may have occluded the studies objectives, resulting in only mediocre differences between the experimental groups on three of the four hypotheses. However, as a worst-case scenario, it is also conceivable that the one significant result regarding hunger (versus satiety) and risk propensity was an artifact of confounding cognitive or emotional states. One way to have avoided this situation would have been to include some measure(s) of distaste, emotion, or mood in the study so that any such differences

between the two experimental conditions could have been controlled for via the statistical analyses.

Although it may be difficult to extrapolate theoretical or practical implications in light of weak results and null results, and in light of potential inadvertent confounding effects, it is refreshing to see that despite these unfortunate and unforeseen issues, risk propensity was still significantly predicted by participants' degree of hunger in the second stage of the instrumental variable regression. If visceral states such as hunger and satiety have a measurable effect on monetary risk propensity alone, without affecting the other outcome variables of concern (i.e., temporal discounting and (un)ethicality) in any meaningful way, then that finding is still noteworthy because of the novelty of the concept and because of the importance of risk-taking in our individual and collective lives.

Taking risks is ubiquitous; as stated, it is "a fundamental activity at every societal level, with examples as diverse as people saving for retirement, companies pricing insurance, and countries evaluating military, social, and environmental risks" (Hsu et al., 2005). If financial risk taking is affected by something as simple as whether or not someone is in a state of hunger or satiety, then that presently unknown or underappreciated truth would have enormous implications for organizations around the world. For instance, if an employer were asking employees to contribute to a 401(k), pension, or other such retirement plans where employees must weigh value and reward, or risk and temporal perspectives, it seems that making such decisions after lunch may be best. At an individual investment level, states like hunger and satiety may affect how someone interprets risk when deciding to invest in stocks, bonds, and portfolios. These

visceral states may also affect how investors and venture capitalists assess risk when investing in start-ups and other entrepreneurial enterprises. Similarly, if employees in the insurance industry are making decisions regarding the pricing of insurance policy premiums, their perceptions of risk would likely matter to and have implications for both the organization and its customers. Thus, their supervisors would be wise to consider their degree of hunger.

States such as hunger and satiety may also affect whether organizations choose to expand into new areas of business, acquire or merge with another company, or negotiate contracts with another company. As an example, if a firm's managers or employees are entering into negotiations with another firm or entity, and the firm's management knows that visceral states such as hunger and satiety can affect the decisions regarding financial risk made by members of the firm, then a sound understanding of visceral states would be both highly beneficial and immediately actionable to the firm regarding the individuals it sends to the negotiation(s). However, in this example, there is yet another perspective to consider; the firm should also realize that members of the firm with which it is negotiating are similarly affected by these visceral states. Thus, steps could be taken by the firm to place both parties (i.e., members of one's own firm and members of the other firm) in the visceral state (i.e., hunger or satiety) most appropriate to the risk perspective (i.e., risk-taking or risk-averse) that would best support the outcome of preference, whatever that may be.

If we viewed buying a home as a risky financial decision, it would make sense for real estate agents to try to induce a state of hunger within potential home buyers. Indeed, it is often reported that real estate agents will use appetitive stimuli such as the artificial smell of freshly baked cookies to entice or otherwise influence potential home buyers. Beyond the quaint notion of making the house smell like a home, perhaps there is some effect of the smell on visceral states such that potential home buyers are made to feel hungrier, inducing them to want reward stimuli in a broader sense, compelling them to behave in a riskier manner and to commit to buying the house. If that were the case, it might be best to go shopping for a home only while full, or, better yet, with one's nose plugged so as to avoid the potential for induced hedonic hunger even when homeostatic hunger may be low due to having eaten recently. I hope my facetiousness is not lost on the reader.

If these results were generalizable beyond financial risks to, say, risk-taking regarding personal safety and the safety of others, then that would have significant implications for fields as diverse as doctors and nurses, military personnel and commanders, construction workers, and truck and bus drivers. Such organizations would want to ensure that any such employees were afforded the opportunity to eat, regardless of how busy they were. It should be mentioned that discussing any effects beyond the influence of hunger and satiety on personal financial decision making is conjecture at this point, but nonetheless interesting to consider.

So, "above and beyond contributing to rational choice, anticipatory neural activation may also promote irrational choice. Thus, financial decision-making may require a delicate balance—recruitment of distinct circuits may be necessary for taking or avoiding risks, but excessive activation of one mechanism or the other may lead to mistakes" (Kuhnen & Knutson, 2005: 767). Affording greater attention to one's own hunger and satiety, or the hunger and satiety of those with whom one interacts or

supervises, seems a fairly simple and straightforward way to mitigate or promote greater or lesser risk-taking behaviors, at least with regard to financial decisions. Moreover, affording greater attention to one's own hunger and satiety, or the hunger and satiety of those with whom one interacts or supervises, implies actionable steps to negate potentially undesired effects. These actionable steps are, namely, to feed or avoid feeding one's self or others when one wants to curtail or promote one's own or others financial risk-taking, respectively.

In the future, I intend to replicate this study with some crucial alterations. There are at least two versions I would consider. The first version would repeat the experimental study as it is presented here, attempting to transition the experimental group from a state of hunger to a state of satiety, while leaving the control group in a state of hunger, but presenting more palatable food participants for consumption during the experimental sessions. The foods still would be high in macronutrient and caloric content, but more appetitive; such foods might include desserts and savory meat items. The second version of the study I would consider attempts to elicit hunger within the experimental group by presenting food-related appetitive stimuli such as pictures and smells of food items, while the control group is presented pictures and smells of pleasant nonfood items such as essential oils or flowers. Thus, this version would not attempt to induce satiety within the experimental group, but rather hunger. This version might not require both groups to fast before the study. Rather, I could test whether the appetitive food cues were more effective at engendering hunger, as well as the outcomes of concern, for persons who were more or less subjectively hungry at the start of the study. For cost

considerations, I would likely elect not to include the post-task meal in future iterations of the study.

Chapter 11

CONCLUSION

Visceral states such as hunger and satiety are underappreciated and understudied in the organizational sciences literature, though nonetheless important to consider. This paper hypothesizes that hunger and satiety may play an important role in decision-making involving temporal discounting, risk propensity, ethicality, and unethicality in financial realms. Despite a weaker-than-intended experimental manipulation and the possible inadvertent introduction of neurological or psychological confounds, the study conducted here has supported the notion that hunger and satiety may respectively promote and inhibit risk-taking regarding monetary outcomes. Although the null effects present in this study do not negate the unsupported hypotheses involving temporal discounting and (un)ethicality, any further discussion of these concepts beyond the literature review and development of hypotheses would be conjecture.

The sole supported finding regarding risk propensity is highly relevant to practitioners because it provides actionable directives related to selecting appropriate meal times for one's self and others, as described in the previous section. The finding is also highly relevant to academicians because it gives great impetus and direction to follow-up studies in terms of theoretical underpinnings and methodology, including what to do and, more importantly, what *not* to do in an experimental study. Follow-up studies

that overcome the methodological limitations of the current study are needed to support the results presented in this paper and to retest the heretofore unsupported hypotheses.

REFERENCES

- Aarøe, L., & Petersen, M. B. 2013. Hunger Games: Fluctuations in Blood Glucose Levels Influence Support for Social Welfare. *Psychological Science*, 24(12): 2550–2556.
- Aguirre, G. K. 2003. Functional imaging in behavioral neurology and cognitive neuropsychology. In T. E. Feinberg & M. J. Farah (Eds.), *Behavioral Neurology and Cognitive Neuropsychology*: 35–46. McGraw-Hill.
- Aguirre, G. K., & D'Esposito, M. 1999. Experimental design for brain fMRI. In C. T. W. Moonen & P. A. Bandettini (Eds.), *Functional MRI*: 369–380. Berlin: Spring Verlag.
- Ahima, R. S., Saper, C. B., Flier, J. S., & Elmquist, J. K. 2000. Leptin regulation of neuroendocrine systems. *Frontiers in Neuroendocrinology*, 21(3): 263–307.
- Ainslie, G. 1975. Specious reward: A behavioral theory of impulsiveness and impulse control. *Psychological Bulletin*, 82(4): 463–496.
- Ainslie, G. 1992. *Picoeconomics: The strategic interaction of successive motivational states within the person*. New York: Cambridge University Press.
- Ainslie, G. 2012. Pure hyperbolic discount curves predict "eyes open" self-control. *Theory and Decision*, 73(1): 3–34.
- Ainslie, G., & Haslam, N. 1992. Hyperbolic discounting. In G. Loewenstein & J. Elster (Eds.), *Choice over time*: 57–92. New York: Russell Sage Foundation.
- Andersen, S., Harrison, G. W., Lau, M. I., & Rutström, E. E. 2008a. Lost in state space: Are preferences stable? *International Economic Review*, 49(3): 1091–1112.
- Andersen, S., Harrison, G. W., Lau, M. I., & Rutström, E. E. 2008b. Eliciting risk and time preferences. *Econometrica*, 76(3): 583–618.
- Andersen, S., Harrison, G. W., Lau, M. I., & Rutström, E. E. 2014. Discounting behavior: A reconsideration. *European Economic Review*, 71: 15–33.
- Andreoni, J., Kuhn, M., & Sprenger, C. 2015. *Measuring Time Preferences: A Comparison of Experimental Methods*: 1–48. National Bureau of Economic Research.
- Andreoni, J., & Sprenger, C. 2012a. Risk Preferences Are Not Time Preferences. *The American Economic Review*, 102(7): 3357–3376.
- Andreoni, J., & Sprenger, C. 2012b. Estimating time preferences from convex budgets. *American Economic Review*, 102(7): 3333–3356.
- Angeletos, G.-M., Laibson, D., Repetto, A., Tobacman, J., & Weinberg, S. 2001. The Hyperbolic Consumption Model: Calibration, Simulation, and Empirical Evaluation. *The Journal of Economic Perspectives*, 15(3): 47–68.
- Antonakis, J., Bendahan, S., Jacquart, P., & Lalive, R. 2010. On making causal claims: A review and recommendations. *The Leadership Quarterly*, 21(6): 1086–1120.
- Apicella, C. L., Dreber, A., Campbell, B., Gray, P. B., Hoffman, M., et al. 2008. Testosterone and financial risk preferences. *Evolution and Human Behavior*, 29(6): 384–390.
- Appelhans, B. M. 2009. Neurobehavioral Inhibition of Reward-driven Feeding: Implications for Dieting and Obesity. *Obesity*, 17(4): 640–647.
- Ariely, D., & Berns, G. S. 2010. Neuromarketing: the hope and hype of neuroimaging in business. *Nature Reviews Neuroscience*, 11(4): 284–292.

- Ariely, D., & Loewenstein, G. 2006. The heat of the moment: the effect of sexual arousal on sexual decision making. *Journal of Behavioral Decision Making*, 19(2): 87–98.
- Ashforth, B. E., & Anand, V. 2003. The normalization of corruption in organizations. *Research in Organizational Behavior*, 25: 1–52.
- Ashkanasy, N. M., Becker, W. J., & Waldman, D. A. 2014. Neuroscience and organizational behavior: Avoiding both neuro-euphoria and neuro-phobia. *Journal of Organizational Behavior*, 35(7): 909–919.
- Association of Certified Fraud Examiners, Inc. 2014. 2014 Report to the Nations on Occupational Fraud and Abuse: 1–80.
- Astrup, A. 2005. The satiating power of protein—a key to obesity prevention? *The American Journal of Clinical Nutrition*, 82(1): 1–2.
- Axon, R. N., Bradford, W. D., & Egan, B. M. 2009. The role of individual time preferences in health behaviors among hypertensive adults: A pilot study. *Journal of the American Society of Hypertension*, 3(1): 35–41.
- Baker, F., Johnson, M. W., & Bickel, W. K. 2003. Delay discounting in current and never-before cigarette smokers: Similarities and differences across commodity, sign, and magnitude. *Journal of Abnormal Psychology*, 112(3): 382–392.
- 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.
- Baldo, B. A., Sadeghian, K., Basso, A. M., & Kelley, A. E. 2002. Effects of selective dopamine D1 or D2 receptor blockade within nucleus accumbens subregions on ingestive behavior and associated motor activity. *Behavioural Brain Research*, 137(1): 165–177.
- Barbano, M. F., & Cador, M. 2007. Opioids for hedonic experience and dopamine to get ready for it. *Psychopharmacology*, 191(3): 497–506.
- Barnes, C. M., Schaubroeck, J., Huth, M., & Ghumman, S. 2011. Lack of sleep and unethical conduct. *Organizational Behavior and Human Decision Processes*, 115(2): 169–180.
- Baron, K. G., Reid, K. J., Kern, A. S., & Zee, P. C. 2011. Role of Sleep Timing in Caloric Intake and BMI. *Obesity*, 19(7): 1374–1381.
- Barros-Loscertales, A., Ventura-Campos, N., Sanjuan-Tomas, A., Belloch, V., Parcet, M.-A., et al. 2010. Behavioral activation system modulation on brain activation during appetitive and aversive stimulus processing. *Social Cognitive and Affective Neuroscience*, 5(1): 18–28.
- Bassi, A., Colacito, R., & Fulghieri, P. 2013. 'O Sole Mio: An Experimental Analysis of Weather and Risk Attitudes in Financial Decisions. *Review of Financial Studies*, 26(7): 1824–1852.
- Beaver, J. D., Lawrence, A. D., van Ditzhuijzen, J., Davis, M. H., Woods, A., et al. 2006. Individual differences in reward drive predict neural responses to images of food. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 26(19): 5160–5166.
- Bechara, A., Damasio, A. R., Damasio, H., & Anderson, S. W. 1994. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*, 50(1–3): 7–15.

- Beck, R. C., & Triplett, M. F. 2009. Test–retest reliability of a group-administered paper–pencil measure of delay discounting. *Experimental and Clinical Psychopharmacology*, 17(5): 345–355.
- Becker, W. J., & Cropanzano, R. 2010. Organizational neuroscience: The promise and prospects of an emerging discipline. *Journal of Organizational Behavior*, 31: 1055–1059.
- Becker, W. J., Cropanzano, R., & Sanfey, A. G. 2011. Organizational Neuroscience: Taking Organizational Theory Inside the Neural Black Box. *Journal of Management*, 37(4): 933–961.
- Begg, D. P., & Woods, S. C. 2013. The endocrinology of food intake. *Nature Reviews Endocrinology*, 9(10): 584–597.
- Bellisle, F., Louis-Sylvestre, J., Demozay, F., Blazy, D., & Le Magnen, J. 1985. Cephalic phase of insulin secretion and food stimulation in humans: A new perspective. *American Journal of Physiology-Endocrinology And Metabolism*, 249(6): E639–E645.
- Bentler, P. M., & Raykov, T. 2000. On measures of explained variance in nonrecursive structural equation models. *Journal of Applied Psychology*, 85(1): 125–131.
- Berns, G. S., Laibson, D., & Loewenstein, G. 2007. Intertemporal choice toward an integrative framework. *Trends in Cognitive Sciences*, 11(11): 482–488.
- Berns, G. S., McClure, S. M., Pagnoni, G., & Montague, P. R. 2001. Predictability modulates human brain response to reward. *The Journal of Neuroscience*, 21(8): 2793–2798.
- Berridge, K. C. 1996. Food reward: Brain substrates of wanting and liking. *Neuroscience & Biobehavioral Reviews*, 20(1): 1–25.
- Berridge, K. C. 2000. Reward learning: Reinforcement, incentives, and expectations. In D. L. Medlin (Ed.), *Psychology of Learning and Motivation*, vol. 40: 223–278. San Diego: Academic Press.
- Berridge, K. C. 2007. The debate over dopamine's role in reward: the case for incentive salience. *Psychopharmacology*, 191(3): 391–431.
- Berridge, K. C. 2009. "Liking" and "wanting" food rewards: brain substrates and roles in eating disorders. *Physiology & Behavior*, 97(5): 537–550.
- Berridge, K. C., & Robinson, T. E. 1998. What is the role of dopamine in reward: hedonic impact, reward learning, or incentive salience? *Brain Research Reviews*, 28(3): 309–369.
- Berridge, K. C., Robinson, T. E., & Aldridge, J. W. 2009. Dissecting components of reward: "liking", "wanting", and learning. *Current Opinion in Pharmacology*, 9(1): 65–73.
- Bertenshaw, E. J., Lluch, A., & Yeomans, M. R. 2008. Satiating effects of protein but not carbohydrate consumed in a between-meal beverage context. *Physiology & Behavior*, 93(3): 427–436.
- Berthoud, H.-R. 2007. Interactions between the "cognitive" and "metabolic" brain in the control of food intake. *Physiology & Behavior*, 91(5): 486–498.
- Berthoud, H.-R. 2011. Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Current Opinion in Neurobiology*, 21(6): 888–896.
- Berthoud, H.-R., & Morrison, C. 2008. The brain, appetite, and obesity. *Annual Review of Psychology*, 59: 55–92.

- Bertino, M., Beauchamp, G. K., & Engelman, K. 1991. Naltrexone, an opioid blocker, alters taste perception and nutrient intake in humans. *The American Journal of Physiology*, 261(1 Pt 2): R59-63.
- Bickel, W. K., Jarmolowicz, D. P., Mueller, E. T., Koffarnus, M. N., & Gatchalian, K. M. 2012. Excessive discounting of delayed reinforcers as a trans-disease process contributing to addiction and other disease-related vulnerabilities: Emerging evidence. *Pharmacology & Therapeutics*, 134(3): 287–297.
- Bickel, W. K., Landes, R. D., Christensen, D. R., Jackson, L., Jones, B. A., et al. 2011. Single- and cross-commodity discounting among cocaine addicts: the commodity and its temporal location determine discounting rate. *Psychopharmacology*, 217(2): 177–187.
- Bickel, W. K., & Mueller, E. T. 2009. Toward the study of trans-disease processes: A novel approach with special reference to the study of co-morbidity. *Journal of Dual Diagnosis*, 5(2): 131–138.
- Black, A. C., & Rosen, M. I. 2011. A money management-based substance use treatment increases valuation of future rewards. *Addictive Behaviors*, 36(1–2): 125–128.
- Blanton, H., & Gerrard, M. 1997. Effect of sexual motivation on men's risk perception for sexually transmitted disease: There must be 50 ways to justify a lover. *Health Psychology*, 16(4): 374–379.
- Bradford, D., Courtemanche, C., Heutel, G., McAlvanah, P., & Ruhm, C. 2014. *Time Preferences and Consumer Behavior, NBER Working Paper No. 20320*: 1–54. National Bureau of Economic Research.
- Bradford, W. D. 2010. The association between individual time preferences and health maintenance habits. *Medical Decision Making*, 30(1): 99–112.
- Breiter, H. C., Aharon, I., Kahneman, D., Dale, A., & Shizgal, P. 2001. Functional Imaging of Neural Responses to Expectancy and Experience of Monetary Gains and Losses. *Neuron*, 30(2): 619–639.
- Breiter, H. C., & Rosen, B. R. 1999. Functional Magnetic Resonance Imaging of Brain Reward Circuitry in the Human. *Annals of the New York Academy of Sciences*, 877(1 ADVANCING FRO): 523–547.
- Brendl, C. M., Markman, A. B., & Messner, C. 2003. The Devaluation Effect: Activating a Need Devalues Unrelated Objects. *Journal of Consumer Research*, 29(4): 463–473.
- Briers, B., & Laporte, S. 2013. A Wallet Full of Calories: The Effect of Financial Dissatisfaction on the Desire for Food Energy. *Journal of Marketing Research*, 50(6): 767–781.
- Briers, B., & Lerouge, D. 2011. The Effects of Temperature Cues on Food Intake. *Advances in Consumer Research*, 39: 658–659.
- Briers, B., Pandelaere, M., Dewitte, S., & Warlop, L. 2006. Hungry for Money: The Desire for Caloric Resources Increases the Desire for Financial Resources and Vice Versa. *Psychological Science*, 17(11): 939–943.
- Brogan, A., Hevey, D., & Pignatti, R. 2010. Anorexia, bulimia, and obesity: Shared decision making deficits on the Iowa Gambling Task (IGT). *Journal of the International Neuropsychological Society*, 16(4): 711–715.
- Bruce, A. S., Holsen, L. M., Chambers, R. J., Martin, L. E., Brooks, W. M., et al. 2010. Obese children show hyperactivation to food pictures in brain networks linked to

- motivation, reward and cognitive control. *International Journal of Obesity*, 34(10): 1494–1500.
- Brunger, L., Smith, A., Re, R., Wickham, M., Philippides, A., et al. 2015. Validation of an iPad visual analogue rating system for assessing appetite and satiety. *Appetite*, 84: 259–263.
- Buck, P. S. 1931. *The Good Earth*. Pocket Books, Inc.
- Cabanac, M. 1979. Sensory Pleasure. *The Quarterly Review of Biology*, 54(1): 1–29.
- Cabanac, M. 1988. Alliesthesia. *Sensory Systems: II*: 1–1. Boston, MA: Birkhäuser Boston.
- Calder, A. J., Beaver, J. D., Davis, M. H., van Ditzhuijzen, J., Keane, J., et al. 2007. Disgust sensitivity predicts the insula and pallidal response to pictures of disgusting foods. *The European Journal of Neuroscience*, 25(11): 3422–3428.
- Callahan, H. S., Cummings, D. E., Pepe, M. S., Breen, P. A., Matthys, C. C., et al. 2004. Postprandial Suppression of Plasma Ghrelin Level Is Proportional to Ingested Caloric Load but Does Not Predict Intermeal Interval in Humans. *The Journal of Clinical Endocrinology & Metabolism*, 89(3): 1319–1324.
- Camerer, C. F. 2007. Neuroeconomics: Using Neuroscience to Make Economic Predictions*. *The Economic Journal*, 117(519): C26–C42.
- Camerer, C. F. 2008. The potential of neuroeconomics. *Economics and Philosophy*, 24(3): 369–379.
- Camerer, C., Loewenstein, G., & Prelec, D. 2005. Neuroeconomics: How Neuroscience Can Inform Economics. *Journal of Economic Literature*, 43(1): 9–64.
- Cardinal, R. N., & Everitt, B. J. 2004. Neural and psychological mechanisms underlying appetitive learning: Links to drug addiction. *Current Opinion in Neurobiology*, 14(2): 156–162.
- Carroll, M. E., & Meisch, R. A. 1984. Increased drug-reinforced behavior due to food deprivation. *Advances in Behavioral Pharmacology*, 4: 47–88.
- Carver, C. S., & White, T. L. 1994. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS Scales. *Journal of Personality and Social Psychology*, 67(2): 319–333.
- Castro, D. C., & Berridge, K. C. 2014. Advances in the neurobiological bases for food "liking" versus "wanting." *Physiology & Behavior*, 136: 22–30.
- Chambers, K. P. (Ed.). 2009. *Caffeine and health research*. New York: Nova Biomedical Books.
- Charness, G., Gneezy, U., & Imas, A. 2013. Experimental methods: Eliciting risk preferences. *Journal of Economic Behavior & Organization*, 87: 43–51.
- Chesson, H. W., Leichliter, J. S., Zimet, G. D., Rosenthal, S. L., Bernstein, D. I., et al. 2006. Discount rates and risky sexual behaviors among teenagers and young adults. *Journal of Risk and Uncertainty*, 32(3): 217–230.
- Chiva, M. 1997. Cultural aspects of meals and meal frequency. *British Journal of Nutrition*, 77(S1): S21–S28.
- Christian, M. S., & Ellis, A. P. J. 2011. Examining the Effects of Sleep Deprivation on Workplace Deviance: A Self-Regulatory Perspective. *Academy of Management Journal*, 54(5): 913–934.

- Cohen, J. Y., Haesler, S., Vong, L., Lowell, B. B., & Uchida, N. 2012. Neuron-type-specific signals for reward and punishment in the ventral tegmental area. *Nature*, 482(7383): 85–88.
- Colombetti, G. 2008. The somatic marker hypotheses, and what the Iowa Gambling Task does and does not show. *The British Journal for the Philosophy of Science*, 59(1): 51–71.
- Cornell, C. E., Rodin, J., & Weingarten, H. 1989. Stimulus-induced eating when satiated. *Physiology & Behavior*, 45(4): 695–704.
- Cota, D., Barrera, J. G., & Seeley, R. J. 2006. Leptin in energy balance and reward: two faces of the same coin? *Neuron*, 51(6): 678–680.
- Cummings, D. E., Frayo, R. S., Marmonier, C., Aubert, R., & Chapelot, D. 2004. Plasma ghrelin levels and hunger scores in humans initiating meals voluntarily without time-and food-related cues. *American Journal of Physiology-Endocrinology and Metabolism*, 287(2): E297–E304.
- Cummings, D. E., Purnell, J. Q., Frayo, R. S., Schmidova, K., Wisse, B. E., et al. 2001. A preprandial rise in plasma ghrelin levels suggests a role in meal initiation in humans. *Diabetes*, 50(8): 1714–1719.
- Dalley, J. W., & Roiser, J. P. 2012. Dopamine, serotonin and impulsivity. *Neuroscience*, 215: 42–58.
- Danziger, S., Levav, J., & Avnaim-Pesso, L. 2011. Reply to Weinshall-Margel and Shapard: Extraneous factors in judicial decisions persist. *Proceedings of the National Academy of Sciences*, 108(42): E834–E834.
- Danziger, S., Levav, J., Avnaim-Pesso, L., & Kahneman, D. 2011. Extraneous factors in judicial decisions. *Proceedings of the National Academy of Sciences of the United States of America*, 108(17): 6889–6892.
- Davis, C., Patte, K., Curtis, C., & Reid, C. 2010. Immediate pleasures and future consequences. A neuropsychological study of binge eating and obesity. *Appetite*, 54(1): 208–213.
- Dayan, P., & Balleine, B. W. 2002. Reward, motivation, and reinforcement learning. *Neuron*, 36(2): 285–298.
- de Castro, J. M. 1987. Circadian rhythms of the spontaneous meal pattern, macronutrient intake, and mood of humans. *Physiology & Behavior*, 40(4): 437–446.
- de Castro, J. M. 1988. Physiological, environmental, and subjective determinants of food intake in humans: A meal pattern analysis. *Physiology & Behavior*, 44(4–5): 651–659.
- de Castro, J. M. 1991a. Seasonal rhythms of human nutrient intake and meal pattern. *Physiology & Behavior*, 50(1): 243–248.
- de Castro, J. M. 1991b. Weekly rhythms of spontaneous nutrient intake and meal pattern of humans. *Physiology & Behavior*, 50(4): 729–738.
- De Graaf, C., De Jong, L. S., & Lambers, A. C. 1999. Palatability Affects Satiation But Not Satiety. *Physiology & Behavior*, 66(4): 681–688.
- de Ridder, D., Kroese, F., Adriaanse, M., & Evers, C. 2014. Always Gamble on an Empty Stomach: Hunger Is Associated with Advantageous Decision Making. (T. Boraud, Ed.)*PLoS ONE*, 9(10): e111081.
- de Wijk, R. A., Kooijman, V., Verhoeven, R. H. G., Holthuysen, N. T. E., & de Graaf, C. 2012. Autonomic nervous system responses on and facial expressions to the sight,

- smell, and taste of liked and disliked foods. *Food Quality and Preference*, 26(2): 196–203.
- de Wit, H. 1996. Priming effects with drugs and other reinforcers. *Experimental and Clinical Psychopharmacology*, 4(1): 5–10.
- Dehaene, S., Dehaene-Lambertz, G., & Cohen, L. 1998. Abstract representations of numbers in the animal and human brain. *Trends in Neurosciences*, 21(8): 355–361.
- Delgado, M. R., Nystrom, L. E., Fissell, C., Noll, D. C., & Fiez, J. A. 2000. Tracking the hemodynamic responses to reward and punishment in the striatum. *Journal of Neurophysiology*, 84(6): 3072–3077.
- DelParigi, A., Chen, K., Salbe, A. D., Reiman, E. M., & Tataranni, P. A. 2005. Sensory experience of food and obesity: a positron emission tomography study of the brain regions affected by tasting a liquid meal after a prolonged fast. *NeuroImage*, 24(2): 436–443.
- Devine, C. M., Farrell, T. J., Blake, C. E., Jastran, M., Wethington, E., et al. 2009. Work conditions and the food choice coping strategies of employed parents. *Journal of Nutrition Education and Behavior*, 41(5): 365–370.
- Devine, C. M., Jastran, M., Jabs, J., Wethington, E., Farell, T. J., et al. 2006. "A lot of sacrifices:" Work–family spillover and the food choice coping strategies of low-wage employed parents. *Social Science & Medicine*, 63(10): 2591–2603.
- Diano, S., Farr, S. A., Benoit, S. C., McNay, E. C., da Silva, I., et al. 2006. Ghrelin controls hippocampal spine synapse density and memory performance. *Nature Neuroscience*, 9(3): 381–388.
- Dickhaut, J., Basu, S., McCabe, K., & Waymire, G. 2010. Neuroaccounting: Consilience between the Biologically Evolved Brain and Culturally Evolved Accounting Principles. *Accounting Horizons*, 24(2): 221–255.
- Dickson, S. L., Egecioglu, E., Landgren, S., Skibicka, K. P., Engel, J. A., et al. 2011. The role of the central ghrelin system in reward from food and chemical drugs. *Molecular and Cellular Endocrinology*, 340(1): 80–87.
- Ditto, P. H., Pizarro, D. A., Epstein, E. B., Jacobson, J. A., & MacDonald, T. K. 2006. Visceral influences on risk-taking behavior. *Journal of Behavioral Decision Making*, 19(2): 99–113.
- Dommett, E., Coizet, V., Blaha, C. D., Martindale, J., Lefebvre, V., et al. 2005. How visual stimuli activate dopaminergic neurons at short latency. *Science*, 307(5714): 1476–1479.
- Doyle, J. R. 2013. Survey of time preference, delay discounting models. *Judgement and Decision Making*, 8(2): 116–135.
- Drazen, D. L., Vahl, T. P., D'Alessio, D. A., Seeley, R. J., & Woods, S. C. 2006. Effects of a fixed meal pattern on ghrelin secretion: Evidence for a learned response independent of nutrient status. *Endocrinology*, 147(1): 23–30.
- Drewnowski, A. 1995. Energy intake and sensory properties of food. *The American Journal of Clinical Nutrition*, 62(5): 1081S–1085S.
- Drewnowski, A., & Greenwood, M. 1983. Cream and sugar: Human preferences for high-fat foods. *Physiology & Behavior*, 30(4): 629–633.

- Drewnowski, A., Krahn, D., Demitrack, M., Nairn, K., & Gosnell, B. 1992. Taste responses and preferences for sweet high-fat foods: Evidence for opioid involvement \(\times \). **Physiology & Behavior**, 51(2): 371–379.
- Drobes, D. J., Miller, E. J., Hillman, C. H., Bradley, M. M., Cuthbert, B. N., et al. 2001. Food deprivation and emotional reactions to food cues: Implications for eating disorders. *Biological Psychology*, 57(1): 153–177.
- Egecioglu, E., Jerlhag, E., Salomé, N., Skibicka, K. P., Haage, D., et al. 2010. Ghrelin increases intake of rewarding food in rodents: Ghrelin and food reward. *Addiction Biology*, 15(3): 304–311.
- Einöther, S. J. L., & Giesbrecht, T. 2013. Caffeine as an attention enhancer: reviewing existing assumptions. *Psychopharmacology*, 225(2): 251–274.
- Elliott, R., Newman, J. L., Longe, O. A., & Deakin, J. W. 2003. Differential response patterns in the striatum and orbitofrontal cortex to financial reward in humans: a parametric functional magnetic resonance imaging study. *The Journal of Neuroscience*, 23(1): 303–307.
- Elster, J. 1979. *Ulysses and the sirens: Studies in rationality and irrationality*, vol. 1. Cambridge: Cambridge University Press.
- Elster, J. 2000. *Ulysses unbound: Studies in rationality, precommitment, and constraints*. Cambridge: Cambridge University Press.
- Epstein, L. H., Leddy, J. J., Temple, J. L., & Faith, M. S. 2007. Food reinforcement and eating: A multilevel analysis. *Psychological Bulletin*, 133(5): 884–906.
- Epstein, L. H., Salvy, S. J., Carr, K. A., Dearing, K. K., & Bickel, W. K. 2010. Food reinforcement, delay discounting and obesity. *Physiology & Behavior*, 100(5): 438–445.
- Estle, S. J., Green, L., Myerson, J., & Holt, D. D. 2007. Discounting of Monetary and Directly Consumable Rewards. *Psychological Science*, 18(1): 58–63.
- Everitt, B. J., & Robbins, T. W. 2005. Neural systems of reinforcement for drug addiction: from actions to habits to compulsion. *Nature Neuroscience*, 8(11): 1481–1489.
- Farooqi, I. S., Bullmore, E., Keogh, J., Gillard, J., O'Rahilly, S., et al. 2007. Leptin regulates striatal regions and human eating behavior. *Science*, 317(5843): 1355.
- Fennema, H., & Wakker, P. P. 1997. Original and cumulative prospect theory: A discussion of empirical differences. *Journal of Behavioral Decision Making*, 10: 53–64.
- Festjens, A., Bruyneel, S., & Dewitte, S. 2013. What a feeling! Touching sexually laden stimuli makes women seek rewards. *Journal of Consumer Psychology*, 24(3): 387–393.
- Field, M., Santarcangelo, M., Sumnall, H., Goudie, A., & Cole, J. 2006. Delay discounting and the behavioural economics of cigarette purchases in smokers: The effects of nicotine deprivation. *Psychopharmacology*, 186(2): 255–263.
- Fields, H. L., Hjelmstad, G. O., Margolis, E. B., & Nicola, S. M. 2007. Ventral Tegmental Area Neurons in Learned Appetitive Behavior and Positive Reinforcement. *Annual Review of Neuroscience*, 30(1): 289–316.
- Figlewicz, D. P. 2003. Adiposity signals and food reward: expanding the CNS roles of insulin and leptin. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, 284(4): R882–R892.

- Figlewicz, D. P., & Benoit, S. C. 2009. Insulin, leptin, and food reward: update 2008. American Journal of Physiology. Regulatory, Integrative and Comparative Physiology, 296(1): R9–R19.
- Flagel, S. B., Clark, J. J., Robinson, T. E., Mayo, L., Czuj, A., et al. 2011. A selective role for dopamine in stimulus–reward learning. *Nature*, 469(7328): 53–57.
- Fliessbach, K., Weber, B., Trautner, P., Dohmen, T., Sunde, U., et al. 2007. Social Comparison Affects Reward-Related Brain Activity in the Human Ventral Striatum. *Science*, 318(5854): 1305–1308.
- Flint, A., Raben, A., Blundell, J. E., & Astrup, A. 2000. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity and Related Metabolic Disorders: Journal of the International Association for the Study of Obesity*, 24(1): 38–48.
- Förster, J., Liberman, N., & Higgins, E. T. 2005. Accessibility from active and fulfilled goals. *Journal of Experimental Social Psychology*, 41(3): 220–239.
- Forzano, L. B., & Logue, A. W. 1992. Predictors of adult humans' self-control and impulsiveness for food reinforcers. *Appetite*, 19(1): 33–47.
- Frank, S., Laharnar, N., Kullmann, S., Veit, R., Canova, C., et al. 2010. Processing of food pictures: Influence of hunger, gender and calorie content. *Brain Research*, 1350: 159–166.
- Frankish, R. P. 2011. The Effect of Caffeine Deprivation on Decisions about Future Events in Humans. University of Waikato.
- Frederick, S., Loewenstein, G., & O'Donoghue, T. 2002. Time Discounting and Time Preference: A Critical Review. *Journal of Economic Literature*, 40(2): 351–401.
- Frost, G., & Pirani, S. 1987. Meal frequency and nutritional intake during Ramadan: a pilot study. *Human Nutrition*. *Applied Nutrition*, 41(1): 47–50.
- Gailliot, M. T., & Baumeister, R. F. 2007. The Physiology of Willpower: Linking Blood Glucose to Self-Control. *Personality and Social Psychology Review*, 11(4): 303–327.
- Gailliot, M. T., Baumeister, R. F., DeWall, C. N., Maner, J. K., Plant, E. A., et al. 2007. Self-control relies on glucose as a limited energy source: Willpower is more than a metaphor. *Journal of Personality and Social Psychology*, 92(2): 325–336.
- Gal, D. 2012. A Mouth-Watering Prospect: Salivation to Material Reward. *Journal of Consumer Research*, 38(6): 1022–1029.
- Galvan, A., Hare, T. A., Davidson, M., Spicer, J., Glover, G., et al. 2005. The role of ventral frontostriatal circuitry in reward-based learning in humans. *The Journal of Neuroscience*, 25(38): 8650–8656.
- Ganzini, L., McFarland, B., & Bloom, J. 1990. Victims of fraud: Comparing victims of white collar and violent crime. *Journal of the American Academy of Psychiatry and the Law Online*, 18(1): 55–63.
- Gerstein, D. E., Woodward-Lopez, G., Evans, A. E., Kelsey, K., & Drewnowski, A. 2004. Clarifying concepts about macronutrients' effects on satiation and satiety. *Journal of the American Dietetic Association*, 104(7): 1151–1153.
- Gilaie-Dotan, S., Tymula, A., Cooper, N., Kable, J. W., Glimcher, P. W., et al. 2014. Neuroanatomy Predicts Individual Risk Attitudes. *The Journal of Neuroscience*, 34(37): 12394–12401.

- Gilbert, D. T., Gill, M. J., & Wilson, T. D. 2002. The Future Is Now: Temporal Correction in Affective Forecasting. *Organizational Behavior and Human Decision Processes*, 88(1): 430–444.
- Gino, F., & Ariely, D. 2012. The dark side of creativity: original thinkers can be more dishonest. *Journal of Personality and Social Psychology*, 102(3): 445–459.
- Gino, F., & Margolis, J. D. 2011. Bringing ethics into focus: How regulatory focus and risk preferences influence (Un)ethical behavior. *Organizational Behavior and Human Decision Processes*, 115(2): 145–156.
- Gino, F., & Pierce, L. 2009. The abundance effect: Unethical behavior in the presence of wealth. *Organizational Behavior and Human Decision Processes*, 109(2): 142–155.
- Gino, F., Schweitzer, M. E., Mead, N. L., & Ariely, D. 2011. Unable to resist temptation: How self-control depletion promotes unethical behavior. *Organizational Behavior and Human Decision Processes*, 115(2): 191–203.
- Giordano, L., Bickel, W., Loewenstein, G., Jacobs, E., Marsch, L., et al. 2002. Mild opioid deprivation increases the degree that opioid-dependent outpatients discount delayed heroin and money. *Psychopharmacology*, 163(2): 174–182.
- Glass, M. J., Billington, C. J., & Levine, A. S. 1999. Opioids and food intake: distributed functional neural pathways? *Neuropeptides*, 33(5): 360–368.
- Glimcher, P. W. 2010. *Foundations of neuroeconomic analysis*. New York: Oxford University Press. http://site.ebrary.com/id/10432478.
- Glimcher, P. W., & Rustichini, A. 2004. Neuroeconomics: The Consilience of Brain and Decision. *Science*, 306(5695): 447–452.
- Goldstone, A. P., Prechtl de Hernandez, C. G., Beaver, J. D., Muhammed, K., Croese, C., et al. 2009. Fasting biases brain reward systems towards high-calorie foods. *European Journal of Neuroscience*, 30(8): 1625–1635.
- Gordon, C. M., Dougherty, D. D., Rauch, S. L., Emans, S. J., Grace, E., et al. 2000. Neuroanatomy of human appetitive function: A positron emission tomography investigation. *The International Journal of Eating Disorders*, 27(2): 163–171.
- Gottfried, J. A., O'Doherty, J., & Dolan, R. J. 2002. Appetitive and aversive olfactory learning in humans studied using event-related functional magnetic resonance imaging. *The Journal of Neuroscience*, 22(24): 10829–10837.
- Gray, J. A. 1981. A critique of Eysenck's theory of personality. In H. J. Eysenck (Ed.), *A model for personality*: 246–276. Berlin: Springer.
- Green, L., & Myerson, J. 2004. A Discounting Framework for Choice With Delayed and Probabilistic Rewards. *Psychological Bulletin*, 130(5): 769–792.
- Grether, D. M., & Plott, C. R. 1979. Economic theory of choice and the preference reversal phenomenon. *The American Economic Review*, 69(4): 623–638.
- Grigg, M., Bowman, J., & Redman, S. 1996. Disordered eating and unhealthy weight reduction practices among adolescent females. *Preventive Medicine*, 25(6): 748–756.
- Griskevicius, V., Tybur, J. M., Sundie, J. M., Cialdini, R. B., Miller, G. F., et al. 2007. Blatant benevolence and conspicuous consumption: When romantic motives elicit strategic costly signals. *Journal of Personality and Social Psychology*, 93(1): 85–102.

- Grundy, D. 2006. Signalling the state of the digestive tract. *Autonomic Neuroscience*, 125(1–2): 76–80.
- Haase, L., Cerf-Ducastel, B., & Murphy, C. 2009. Cortical activation in response to pure taste stimuli during the physiological states of hunger and satiety. *NeuroImage*, 44(3): 1008–1021.
- Haase, L., Green, E., & Murphy, C. 2011. Males and females show differential brain activation to taste when hungry and sated in gustatory and reward areas. *Appetite*, 57(2): 421–434.
- Hamann, S., Herman, R. A., Nolan, C. L., & Wallen, K. 2004. Men and women differ in amygdala response to visual sexual stimuli. *Nature Neuroscience*, 7(4): 411–416.
- Hardisty, D. J., Thompson, K. F., Krantz, D. H., & Weber, E. U. 2013. How to measure time preferences: An experimental comparison of three methods. *Judgment & Decision Making*, 8(3): 236–249.
- Hardman, C. A., Herbert, V. M. B., Brunstrom, J. M., Munafò, M. R., & Rogers, P. J. 2012. Dopamine and food reward: Effects of acute tyrosine/phenylalanine depletion on appetite. *Physiology & Behavior*, 105(5): 1202–1207.
- Harrison, G. W., Johnson, E., McInnes, M. M., & Rutström, E. E. 2005. Temporal stability of estimates of risk aversion. *Applied Financial Economics Letters*, 1(1): 31–35.
- Harrison, J., & McKay, R. 2012. Delay Discounting Rates Are Temporally Stable in an Equivalent Present Value Procedure Using Theoretical and Area Under the Curve Analyses. *Psychological Record*, 62(2): 307–320.
- Harvey, J., Solovyova, N., & Irving, A. 2006. Leptin and its role in hippocampal synaptic plasticity. *Progress in Lipid Research*, 45(5): 369–378.
- Hausman, J. A. 1978. Specification tests in econometrics. *Econometrica: Journal of the Econometric Society*, 1251–1271.
- Häusser, J. A., Schlemmer, A., Kaiser, S., Kalis, A., & Mojzisch, A. 2014. The effects of caffeine on option generation and subsequent choice. *Psychopharmacology*, 231(18): 3719–3727.
- Henson, R. 2005. What can functional neuroimaging tell the experimental psychologist? *The Quarterly Journal of Experimental Psychology*, 58(2): 193–233.
- Henson, R. 2006. Forward inference using functional neuroimaging: Dissociations versus associations. *Trends in Cognitive Sciences*, 10(2): 64–69.
- Herman, C. P. 1993. Effects of heat on appetite. In B. M. Marriott (Ed.), *Nutritional* needs in hot environments: Applications for military personnel in field operations: 187–214. Washington, DC: National Adademy Press.
- Herrnstein, R. J. 1997. *The matching law: papers in psychology and economics*. (H. Rachlin & D. I. Laibson, Eds.). New York: Russell Sage Foundation.
- Hey, J. D., & Orme, C. 1994. Investigating Generalizations of Expected Utility Theory Using Experimental Data. *Econometrica*, 62(6): 1291–1326.
- Hinton, E. C., Parkinson, J. A., Holland, A. J., Arana, F. S., Roberts, A. C., et al. 2004. Neural contributions to the motivational control of appetite in humans. *The European Journal of Neuroscience*, 20(5): 1411–1418.
- Hjelmstad, G. O., Xia, Y., Margolis, E. B., & Fields, H. L. 2013. Opioid Modulation of Ventral Pallidal Afferents to Ventral Tegmental Area Neurons. *Journal of Neuroscience*, 33(15): 6454–6459.

- Hoebel, B. G. 1985. Brain neurotransmitters in food and drug reward. *The American Journal of Clinical Nutrition*, 42(5 Suppl): 1133–1150.
- Holsen, L. M., Zarcone, J. R., Thompson, T. I., Brooks, W. M., Anderson, M. F., et al. 2005. Neural mechanisms underlying food motivation in children and adolescents. *NeuroImage*, 27(3): 669–676.
- Holt, C. A., & Laury, S. K. 2002. Risk Aversion and Incentive Effects. *American Economic Review*, 92(5): 1644–1655.
- Holt, C. A., & Laury, S. K. 2005. Risk Aversion and Incentive Effects: New Data without Order Effects. *The American Economic Review*, 95(3): 902–904.
- Holtfreter, K. 2005. Is occupational fraud "typical" white-collar crime? A comparison of individual and organizational characteristics. *Journal of Criminal Justice*, 33(4): 353–365.
- Horner, K. M., Byrne, N. M., & King, N. A. 2014. Reproducibility of subjective appetite ratings and ad libitum test meal energy intake in overweight and obese males. *Appetite*, 81: 116–122.
- Horvitz, J. C. 2002. Dopamine gating of glutamatergic sensorimotor and incentive motivational input signals to the striatum. *Behavioural Brain Research*, 137(1): 65–74.
- Hosoda, H., & Kangawa, K. 2012. Standard Sample Collections for Blood Ghrelin Measurements. *Methods in Enzymology*, vol. 514: 113–126. Elsevier.
- Hsu, M., Bhatt, M., Adolphs, R., Tranel, D., & Camerer, C. F. 2005. Neural Systems Responding to Degrees of Uncertainty in Human Decision-Making. *Science*, 310(5754): 1680–1683.
- Huber, P. J. 1967. The behavior of maximum likelihood estimates under nonstandard conditions. *Proceedings of the fifth Berkeley symposium on mathematical statistics and probability*, 1: 221–233.
- Hulley, S. B., Cummings, S. R., Browner, W. S., Grady, D. G., & Newman, T. B. 2013. *Designing clinical research* (4th ed.). Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins.
- Hyman, S. E., & Malenka, R. C. 2001. Addiction and the brain: The neurobiology of compulsion and its persistence. *Nature Reviews Neuroscience*, 2(10): 695–703.
- Imhoff, R., & Schmidt, A. F. 2014. Sexual Disinhibition Under Sexual Arousal: Evidence for Domain Specificity in Men and Women. *Archives of Sexual Behavior*, 43(6): 1123–1136.
- Inman, M. 2015. Grocery shopping while full VS while hungry. *The Oatmeal*. http://theoatmeal.com/pl/minor_differences/grocery_shopping.
- Insel, T. R. 2003. Is social attachment an addictive disorder? *Physiology & Behavior*, 79(3): 351–357.
- Isganaitis, E., & Lustig, R. H. 2005. Fast food, central nervous system insulin resistance, and obesity. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 25(12): 2451–2462.
- Jager, G., & Witkamp, R. F. 2014. The endocannabinoid system and appetite: relevance for food reward. *Nutrition Research Reviews*, 27(1): 172–185.
- James, J. E. 2005. Caffeine-induced enhancement of cognitive performance: Confounding due to reversal of withdrawal effects. *Australian Journal of Psychology*, 57(3): 197–200.

- James, J. E. 2014. Caffeine and cognitive performance: Persistent methodological challenges in caffeine research. *Pharmacology Biochemistry and Behavior*, 124: 117–122.
- James, J. E., & Gregg, M. E. 2004. Effects of dietary caffeine on mood when rested and sleep restricted. *Human Psychopharmacology: Clinical and Experimental*, 19(5): 333–341.
- James, J. E., & Rogers, P. J. 2005. Effects of caffeine on performance and mood: withdrawal reversal is the most plausible explanation. *Psychopharmacology*, 182(1): 1–8.
- Jerlhag, E., Egecioglu, E., Landgren, S., Salomé, N., Heilig, M., et al. 2009. Requirement of central ghrelin signaling for alcohol reward. *Proceedings of the National Academy of Sciences*, 106(27): 11318–11323.
- Jiang, T., Soussignan, R., Rigaud, D., Martin, S., Royet, J.-P., et al. 2008. Alliesthesia to food cues: Heterogeneity across stimuli and sensory modalities. *Physiology & Behavior*, 95(3): 464–470.
- Jiang, Y., King, J. M., & Prinyawiwatkul, W. 2014. A review of measurement and relationships between food, eating behavior and emotion. *Trends in Food Science & Technology*, 36(1): 15–28.
- Johnson, F., Mavrogianni, A., Ucci, M., Vidal-Puig, A., & Wardle, J. 2011. Could increased time spent in a thermal comfort zone contribute to population increases in obesity? *Obesity Reviews*, 12(7): 543–551.
- Johnson, M. W., & Bickel, W. K. 2002. Within-subject comparison of real and hypothetical money rewards in delay discounting. *Journal of the Experimental Analysis of Behavior*, 77(2): 129–146.
- Kable, J. W. 2011. The cognitive neuroscience toolkit for the neuroeconomist: A functional overview. *Journal of Neuroscience*, *Psychology*, *and Economics*, 4(2): 63–84.
- Kahneman, D. 2013. *Thinking, fast and slow* (1st pbk. ed). New York: Farrar, Straus and Giroux.
- Kang, M. J., Rangel, A., Camus, M., & Camerer, C. F. 2011. Hypothetical and Real Choice Differentially Activate Common Valuation Areas. *Journal of Neuroscience*, 31(2): 461–468.
- Kant, A. K., & Graubard, B. I. 2015. 40-Year Trends in Meal and Snack Eating Behaviors of American Adults. *Journal of the Academy of Nutrition and Dietetics*, 115(1): 50–63.
- Karaagaoglu, N., & Yucecan, S. 2000. Some behavioural changes observed among fasting subjects, their nutritional habits and energy expenditure in Ramadan. *International Journal of Food Sciences & Nutrition*, 51(2): 125.
- Kaur, S., & Ryabinin, A. E. 2010. Ghrelin receptor antagonism decreases alcohol consumption and activation of perioculomotor urocortin-containing neurons. *Alcoholism: Clinical and Experimental Research*, 34(9): 1525–1534.
- Kayser, A. S., Allen, D. C., Navarro-Cebrian, A., Mitchell, J. M., & Fields, H. L. 2012. Dopamine, Corticostriatal Connectivity, and Intertemporal Choice. *Journal of Neuroscience*, 32(27): 9402–9409.

- Kelley, A. E., Bakshi, V. P., Haber, S. N., Steininger, T. L., Will, M. J., et al. 2002. Opioid modulation of taste hedonics within the ventral striatum. *Physiology & Behavior*, 76(3): 365–377.
- Kelley, A. E., Baldo, B. A., Pratt, W. E., & Will, M. J. 2005. Corticostriatal-hypothalamic circuitry and food motivation: integration of energy, action and reward. *Physiology & Behavior*, 86(5): 773–795.
- Killgore, W. D. ., Young, A. D., Femia, L. A., Bogorodzki, P., Rogowska, J., et al. 2003. Cortical and limbic activation during viewing of high- versus low-calorie foods. *NeuroImage*, 19(4): 1381–1394.
- Kirby, K. N. 1997. Bidding on the future: Evidence against normative discounting of delayed rewards. *Journal of Experimental Psychology: General*, 126(1): 54–70.
- Kirby, K. N. 2006. The present values of delayed rewards are approximately additive. *Behavioural Processes*, 72(3): 273–282.
- Kirby, K. N. 2009. One-year temporal stability of delay-discount rates. *Psychonomic Bulletin & Review*, 16(3): 457–462.
- Kirby, K. N., & Petry, N. M. 2004. Heroin and cocaine abusers have higher discount rates for delayed rewards than alcoholics or non-drug-using controls. *Addiction*, 99(4): 461–471.
- Kirk, J. M., & Logue, A. W. 1997. Effects of Deprivation Level on Humans' Self-Control for Food Reinforcers. *Appetite*, 28(3): 215–226.
- Knutson, B., Adams, C. M., Fong, G. W., & Hommer, D. 2001. Anticipation of increasing monetary reward selectively recruits nucleus accumbens. *The Journal of Neuroscience*, 21(16): RC159.
- Knutson, B., Fong, G. W., Adams, C. M., Varner, J. L., & Hommer, D. 2001. Dissociation of reward anticipation and outcome with event-related fMRI. *Neuroreport*, 12(17): 3683–3687.
- Knutson, B., Fong, G. W., Bennett, S. M., Adams, C. M., & Hommer, D. 2003. A region of mesial prefrontal cortex tracks monetarily rewarding outcomes: characterization with rapid event-related fMRI. *Neuroimage*, 18(2): 263–272.
- Knutson, B., Taylor, J., Kaufman, M., Peterson, R., & Glover, G. 2005. Distributed neural representation of expected value. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 25(19): 4806–4812.
- Knutson, B., Wimmer, G. E., Kuhnen, C. M., & Winkielman, P. 2008. Nucleus accumbens activation mediates the influence of reward cues on financial risk taking. *Brain Imaging NeuroReport*, 19(5): 509–513.
- Koepp, M. J., Gunn, R. N., Lawrence, A. D., Cunningham, V. J., Dagher, A., et al. 1998. Evidence for striatal dopamine release during a video game. *Nature*, 393(6682): 266–268.
- Koffarnus, M. N., Jarmolowicz, D. P., Mueller, E. T., & Bickel, W. K. 2013. Changing delay discounting in the light of the competing neurobehavioral decision systems theory: a review. *Journal of the Experimental Analysis of Behavior*, 99(1): 32–57.
- Kong, F., & Singh, R. P. 2008. Disintegration of Solid Foods in Human Stomach. *Journal of Food Science*, 73(5): R67–R80.
- Koopmans, T. C. 1960. Stationary ordinal utility and impatience. *Econometrica*, 28(2): 287–309.

- Korakianiti, A., Hillier, S. E., & Clegg, M. E. 2014. The impact of the cephalic phase on postprandial blood glucose and satiety. *E-SPEN Journal*, 9(6): e220–e222.
- Kosfeld, M., Heinrichs, M., Zak, P. J., Fischbacher, U., & Fehr, E. 2005. Oxytocin increases trust in humans. *Nature*, 435(7042): 673–676.
- Kringelbach, M. L. 2004. Food for thought: hedonic experience beyond homeostasis in the human brain. *Neuroscience*, 126(4): 807–819.
- Kringelbach, M. L. 2005. The human orbitofrontal cortex: linking reward to hedonic experience. *Nature Reviews Neuroscience*, 6(9): 691–702.
- Kringelbach, M. L., O'Doherty, J., Rolls, E. T., & Andrews, C. 2003. Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cerebral Cortex (New York, N.Y.: 1991)*, 13(10): 1064–1071.
- Kugler, T., Connolly, T., & Ordóñez, L. D. 2012. Emotion, Decision, and Risk: Betting on Gambles versus Betting on People. *Journal of Behavioral Decision Making*, 25(2): 123–134.
- Kuhnen, C. M., & Knutson, B. 2005. The Neural Basis of Financial Risk Taking. *Neuron*, 47(5): 763–770.
- Lagorio, C. H., & Madden, G. J. 2005. Delay discounting of real and hypothetical rewards III: Steady-state assessments, forced-choice trials, and all real rewards. *Behavioural Processes*, 69(2): 173–187.
- Laibson, D. I. 1994. *Hyperbolic discounting and consumption*. Massachusetts Institute of Technology.
- Laibson, D. I. 1997. Golden eggs and hyperbolic discounting. *The Quarterly Journal of Economics*, 112(2): 443–477.
- Lammel, S., Ion, D. I., Roeper, J., & Malenka, R. C. 2011. Projection-Specific Modulation of Dopamine Neuron Synapses by Aversive and Rewarding Stimuli. *Neuron*, 70(5): 855–862.
- Larson, R. W., Branscomb, K. R., & Wiley, A. R. 2006. Forms and functions of family mealtimes: Multidisciplinary perspectives. *New Directions for Child and Adolescent Development*, 2006(111): 1–15.
- Lartigue, S., Bizais, Y., Bruley des Varannes, S., Murat, A., Pouliquen, B., et al. 1994. Inter- and intrasubject variability of solid and liquid gastric emptying parameters: A scintigraphic study in healthy subjects and diabetic patients. *Digestive Diseases and Sciences*, 39(1): 109–115.
- Lee, N., Senior, C., & Butler, M. J. R. 2012. The Domain of Organizational Cognitive Neuroscience: Theoretical and Empirical Challenges. *Journal of Management*, 38(4): 921–931.
- Lerner, J. S., & Keltner, D. 2001. Fear, anger, and risk. *Journal of Personality and Social Psychology*, 81(1): 146–159.
- Lerner, J. S., Li, Y., Valdesolo, P., & Kassam, K. S. 2015. Emotion and Decision Making. *Annual Review of Psychology*, 66(1): 799–823.
- Lerner, J. S., Small, D. A., & Loewenstein, G. 2004. Heart Strings and Purse Strings: Carryover Effects of Emotions on Economic Decisions. *Psychological Science*, 15(5): 337–341.
- Levine, A. S., Kotz, C. M., & Gosnell, B. A. 2003. Sugars and fats: The neurobiology of preference. *The Journal of Nutrition*, 133(3): 831S–834S.

- Levy, D. J., Thavikulwat, A. C., & Glimcher, P. W. 2013. State Dependent Valuation: The Effect of Deprivation on Risk Preferences. *PLoS ONE*, 8(1): e53978.
- Liberman, N., & Förster, J. 2000. Expression after suppression: A motivational explanation of postsuppressional rebound. *Journal of Personality and Social Psychology*, 79(2): 190–203.
- Liberman, N., & Förster, J. 2005. Motivation and construct accessibility. In J. P. Forgas,
 K. D. Williams, & S. M. Laham (Eds.), *Social motivation: Conscious and unconscious processes*: 228–245. Cambridge: Cambridge University Press.
- Lindebaum, D., & Jordan, P. J. 2014. A critique on neuroscientific methodologies in organizational behavior and management studies. *Journal of Organizational Behavior*, 35(7): 898–908.
- Loewenstein, G. 1996. Out of Control: Visceral Influences on Behavior. *Organizational Behavior and Human Decision Processes*, 65(3): 272–292.
- Loewenstein, G. F., & O'Donoghue, T. 2004. Animal Spirits: Affective and Deliberative Processes in Economic Behavior. CAE working paper #0414. *SSRN Electronic Journal*, 1–54.
- Logue, A. W., & King, G. R. 1991. Self-control and impulsiveness in adult humans when food is the reinforcer. *Appetite*, 17(2): 105–120.
- Lucas, F., Bellisle, F., & Di Maio, A. 1987. Spontaneous insulin fluctuations and the preabsorptive insulin response to food ingestion in humans. *Physiology & Behavior*, 40(5): 631–636.
- Lumey, L. H., Stein, A. D., Kahn, H. S., van der Pal-de, K. M., Blauw, G. J., et al. 2007. Cohort profile: the Dutch Hunger Winter families study. *International Journal of Epidemiology*, 36(6): 1196–1204.
- Lupton, D. 2013. Risk and emotion: towards an alternative theoretical perspective. *Health, Risk & Society*, 15(8): 634–647.
- MacKillop, J., Amlung, M. T., Few, L. R., Ray, L. A., Sweet, L. H., et al. 2011. Delayed reward discounting and addictive behavior: a meta-analysis. *Psychopharmacology*, 216(3): 305–321.
- Madden, G. J., Begotka, A. M., Raiff, B. R., & Kastern, L. L. 2003. Delay discounting of real and hypothetical rewards. *Experimental and Clinical Psychopharmacology*, 11(2): 139–145.
- Madden, G. J., Bickel, W. K., & Jacobs, E. A. 1999. Discounting of delayed rewards in opioid-dependent outpatients: Exponential or hyperbolic discounting functions? *Experimental and Clinical Psychopharmacology*, 7(3): 284–293.
- Madden, G. J., Raiff, B. R., Lagorio, C. H., Begotka, A. M., Mueller, A. M., et al. 2004. Delay Discounting of Potentially Real and Hypothetical Rewards: II. Between and Within-Subject Comparisons. *Experimental and Clinical Psychopharmacology*, 12(4): 251–261.
- Mahler, S. V., Smith, K. S., & Berridge, K. C. 2007. Endocannabinoid Hedonic Hotspot for Sensory Pleasure: Anandamide in Nucleus Accumbens Shell Enhances "Liking" of a Sweet Reward. *Neuropsychopharmacology*, 32(11): 2267–2278.
- Malik, S., McGlone, F., Bedrossian, D., & Dagher, A. 2008. Ghrelin Modulates Brain Activity in Areas that Control Appetitive Behavior. *Cell Metabolism*, 7(5): 400–409.

- Margolis, E. B., Hjelmstad, G. O., Fujita, W., & Fields, H. L. 2014. Direct Bidirectional Opioid Control of Midbrain Dopamine Neurons. *Journal of Neuroscience*, 34(44): 14707–14716.
- Maridakis, V., Herring, M. P., & O'Connor, P. J. 2009. Sensitivity to Change in Cognitive Performance and Mood Measures of Energy and Fatigue in Response to Differing Doses of Caffeine or Breakfast. *International Journal of Neuroscience*, 119(7): 975–994.
- Marien, H., Custers, R., Hassin, R. R., & Aarts, H. 2012. Unconscious goal activation and the hijacking of the executive function. *Journal of Personality and Social Psychology*, 103(3): 399–415.
- Marsh, R. L., Hicks, J. L., & Bink, M. L. 1998. Activation of completed, uncompleted, and partially completed intentions. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, 24(2): 350–361.
- Martin, L. E., Holsen, L. M., Chambers, R. J., Bruce, A. S., Brooks, W. M., et al. 2010. Neural Mechanisms Associated With Food Motivation in Obese and Healthy Weight Adults. *Obesity*, 18(2): 254–260.
- Masic, U., & Yeomans, M. R. 2014. Umami flavor enhances appetite but also increases satiety. *American Journal of Clinical Nutrition*, 100(2): 532–538.
- Masicampo, E. J., & Baumeister, R. F. 2011. Unfulfilled goals interfere with tasks that require executive functions. *Journal of Experimental Social Psychology*, 47(2): 300–311.
- Maurer, A. H. 2012. Advancing Gastric Emptying Studies: Standardization and New Parameters to Assess Gastric Motility and Function. *Seminars in Nuclear Medicine*, 42(2): 101–112.
- Mazar, N., Amir, O., & Ariely, D. 2008. The Dishonesty of Honest People: A Theory of Self-Concept Maintenance. *Journal of Marketing Research*, 45(6): 633–644.
- Mazur, J. E. 1987. An adjusting procedure for studying delayed reinforcement. In M. L. Commons, J. E. Mazur, J. A. Nevin, & H. Rachlin (Eds.), *Quantitative analyses of behavior V: The effect of delay and of intervening events on reinforcement value*. Hillsdale, N.J.: Erlbaum. http://books.google.com/books?id=1q5mAgAAQBAJ.
- McCabe, K. A. 2008. Neuroeconomics and the economic sciences. *Economics and Philosophy*, 24(3): 345–368.
- McClure, S. M., Berns, G. S., & Montague, P. R. 2003. Temporal Prediction Errors in a Passive Learning Task Activate Human Striatum. *Neuron*, 38(2): 339–346.
- McClure, S. M., Daw, N. D., & Read Montague, P. 2003. A computational substrate for incentive salience. *Trends in Neurosciences*, 26(8): 423–428.
- McClure, S. M., Ericson, K. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. 2007. Time Discounting for Primary Rewards. *Journal of Neuroscience*, 27(21): 5796–5804.
- McClure, S. M., Laibson, D. I., Loewenstein, G., & Cohen, J. D. 2004. Separate neural systems value immediate and delayed monetary rewards. *Science*, 306(5695): 503–507.
- McKee, R. A., & Miller, C. C. 2015. Institutionalizing Bayesianism Within the Organizational Sciences: A Practical Guide Featuring Comments From Eminent Scholars. *Journal of Management*, 41(2): 471–490.

- Mead, N. L., Baumeister, R. F., Gino, F., Schweitzer, M. E., & Ariely, D. 2009. Too tired to tell the truth: Self-control resource depletion and dishonesty. *Journal of Experimental Social Psychology*, 45(3): 594–597.
- Meda, S. A., Stevens, M. C., Potenza, M. N., Pittman, B., Gueorguieva, R., et al. 2009. Investigating the behavioral and self-report constructs of impulsivity domains using principal component analysis. *Behavioural Pharmacology*, 20(5–6): 390–399.
- Mela, D. J., Aaron, J. I., & Gatenby, S. J. 1996. Relationships of Consumer Characteristics and Food Deprivation to Food Purchasing Behavior. *Physiology & Behavior*, 60(5): 1331–1335.
- Metcalfe, J., & Mischel, W. 1999. A hot/cool-system analysis of delay of gratification: Dynamics of willpower. *Psychological Review*, 106(1): 3–19.
- Miller, E. K., & Cohen, J. D. 2001. An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, 24(1): 167–202.
- Mishra, A., & Mishra, H. 2010. We Are What We Consume: The Influence of Food Consumption on Impulsive Choice. *Journal of Marketing Research*, 47(6): 1129–1137.
- Mitchell, S. H. 1999. Measures of impulsivity in cigarette smokers and non-smokers. *Psychopharmacology*, 146(4): 455–464.
- Montague, P. R., & Berns, G. S. 2002. Neural economics and the biological substrates of valuation. *Neuron*, 36(2): 265–284.
- Montague, P. R., King-Casas, B., & Cohen, J. D. 2006. Imaging valuation models in human choice. *Annual Review of Neuroscience*, 29(1): 417–448.
- Morris, J. S., & Dolan, R. J. 2001. Involvement of human amygdala and orbitofrontal cortex in hunger-enhanced memory for food stimuli. *The Journal of Neuroscience*, 21(14): 5304–5310.
- Morton, G. J., Blevins, J. E., Williams, D. L., Niswender, K. D., Gelling, R. W., et al. 2005. Leptin action in the forebrain regulates the hindbrain response to satiety signals. *The Journal of Clinical Investigation*, 115(3): 703–710.
- Musau, A. 2014. Hyperbolic discount curves: a reply to Ainslie. *Theory and Decision*, 76(1): 9–30.
- Naleid, A. M., Grace, M. K., Cummings, D. E., & Levine, A. S. 2005. Ghrelin induces feeding in the mesolimbic reward pathway between the ventral tegmental area and the nucleus accumbens. *Peptides*, 26(11): 2274–2279.
- Ninomiya, Y., Shigemura, N., Yasumatsu, K., Ohta, R., Sugimoto, K., et al. 2001. Leptin and sweet taste. *Vitamins and Hormones*, 64: 221–248.
- Nisbett, R. E., & Kanouse, D. E. 1969. Obesity, food deprivation, and supermarket shopping behavior. *Journal of Personality and Social Psychology*, 12(4): 289–294.
- Nitschke, J. B., Sarinopoulos, I., Mackiewicz, K. L., Schaefer, H. S., & Davidson, R. J. 2006. Functional neuroanatomy of aversion and its anticipation. *NeuroImage*, 29(1): 106–116.
- Noll, J. D., Endrass, P. P. D., Scherrer, P., Rossegger, P. P. D., Urbaniok, P. P. D., et al. 2012. A comparison of professional traders and psychopaths in a simulated non-zero sum game. *Catalyst: A social justice forum*, 2: 1–13.

- Nordgren, L. F., van der Pligt, J., & van Harreveld, F. 2007. Evaluating Eve: Visceral states influence the evaluation of impulsive behavior. *Journal of Personality and Social Psychology*, 93(1): 75–84.
- Nordgren, L. F., van der Pligt, J., & van Harreveld, F. 2008. The instability of health cognitions: Visceral states influence self-efficacy and related health beliefs. *Health Psychology*, 27(6): 722–727.
- Nygren, J. 2006. The metabolic effects of fasting and surgery. *Best Practice & Research Clinical Anaesthesiology*, 20(3): 429–438.
- Ochsner, K. N., & Lieberman, M. D. 2001. The emergence of social cognitive neuroscience. *American Psychologist*, 56(9): 717–734.
- O'Doherty, J. P. 2007. Lights, camembert, action! The role of human orbitofrontal cortex in encoding stimuli, rewards, and choices. *Annals of the New York Academy of Sciences*, 1121: 254–272.
- O'Doherty, J., Rolls, E. T., Francis, S., Bowtell, R., McGlone, F., et al. 2000. Sensory-specific satiety-related olfactory activation of the human orbitofrontal cortex. *Neuroreport*, 11(2): 399–403.
- O'Doherty, J., Winston, J., Critchley, H., Perrett, D., Burt, D. M., et al. 2003. Beauty in a smile: the role of medial orbitofrontal cortex in facial attractiveness. *Neuropsychologia*, 41(2): 147–155.
- O'Donoghue, T., & Rabin, M. 1999. Doing It Now or Later. *The American Economic Review*, 89(1): 103–124.
- Odum, A. L. 2011. Delay discounting: Trait variable? *Behavioural Processes*, 87(1): 1–9.
- Odum, A. L., Baumann, A. A. L., & Rimington, D. D. 2006. Discounting of delayed hypothetical money and food: Effects of amount. *Behavioural Processes*, 73(3): 278–284.
- Odum, A. L., & Rainaud, C. P. 2003. Discounting of delayed hypothetical money, alcohol, and food. *Behavioural Processes*, 64(3): 305–313.
- O'Fallon, M. J., & Butterfield, K. D. 2005. A review of the empirical ethical decision-making literature: 1996–2003. *Journal of Business Ethics*, 59(4): 375–413.
- Ohmura, Y., Takahashi, T., Kitamura, N., & Wehr, P. 2006. Three-month stability of delay and probability discounting measures. *Experimental and Clinical Psychopharmacology*, 14(3): 318–328.
- Orne, M. T. 1969. Demand characteristics and the concept of quasi-controls. In R. Rosenthal & R. L. Rosnow (Eds.), *Artifacts in Behavioral Research*: 143–179. New York: Academic Press.
- Padoa-Schioppa, C. 2011. Neurobiology of Economic Choice: A Good-Based Model. *Annual Review of Neuroscience*, 34(1): 333–359.
- Page, K. A., Seo, D., Belfort-DeAguiar, R., Lacadie, C., Dzuira, J., et al. 2011. Circulating glucose levels modulate neural control of desire for high-calorie foods in humans. *Journal of Clinical Investigation*, 121(10): 4161–4169.
- Page, M. P. A. 2006. What Can't Functional Neuroimaging Tell the Cognitive Psychologist? *Cortex*, 42(3): 428–443.
- Patton, D. F., & Mistlberger, R. E. 2013. Circadian adaptations to meal timing: neuroendocrine mechanisms. *Frontiers in Neuroscience*, 7: 1–14.

- Paulus, M. P., Rogalsky, C., Simmons, A., Feinstein, J. S., & Stein, M. B. 2003. Increased activation in the right insula during risk-taking decision making is related to harm avoidance and neuroticism. *NeuroImage*, 19(4): 1439–1448.
- Pavlov, I. P. 1897. Lectures on the work of the principal digestive glands. *St. Petersburg: Kushnereff*.
- Peciña, S., & Berridge, K. C. 2005. Hedonic hot spot in nucleus accumbens shell: Where do μ-opioids cause increased hedonic impact of sweetness? *The Journal of Neuroscience*, 25(50): 11777–11786.
- Perello, M., Sakata, I., Birnbaum, S., Chuang, J.-C., Osborne-Lawrence, S., et al. 2010. Ghrelin increases the rewarding value of high-fat diet in an orexin-dependent manner. *Biological Psychiatry*, 67(9): 880–886.
- Phelps, E. S., & Pollak, R. A. 1968. On Second-Best National Saving and Game-Equilibrium Growth. *The Review of Economic Studies*, 35(2): 185–199.
- Pinel, J. P., Assanand, S., & Lehman, D. R. 2000. Hunger, eating, and ill health. *American Psychologist*, 55(10): 1105–1116.
- Plato. 2005. *Phaedrus*. (C. J. Rowe, Tran.). London; New York: Penguin Books.
- Podoynitsyna, K., Van der Bij, H., & Song, M. 2012. The Role of Mixed Emotions in the Risk Perception of Novice and Serial Entrepreneurs. *Entrepreneurship Theory and Practice*, 36(1): 115–140.
- Poldrack, R. 2006. Can cognitive processes be inferred from neuroimaging data? *Trends in Cognitive Sciences*, 10(2): 59–63.
- Poldrack, R. A., & Wagner, A. D. 2004. What can neuroimaging tell us about the mind? Insights from prefrontal cortex. *Current Directions in Psychological Science*, 13(5): 177–181.
- Porubská, K., Veit, R., Preissl, H., Fritsche, A., & Birbaumer, N. 2006. Subjective feeling of appetite modulates brain activity: An fMRI study. *NeuroImage*, 32(3): 1273–1280.
- Powell, T. C. 2011. Neurostrategy. *Strategic Management Journal*, 32(13): 1484–1499.
- Powley, T. L. 1977. The ventromedial hypothalamic syndrome, satiety, and a cephalic phase hypothesis. *Psychological Review*, 84(1): 89–126.
- Preuschoff, K., Bossaerts, P., & Quartz, S. R. 2006. Neural differentiation of expected reward and risk in human subcortical structures. *Neuron*, 51(3): 381–390.
- Rachlin, H. 2000. *The science of self-control*. Cambridge, Mass.: Harvard University Press. http://search.ebscohost.com/login.aspx?direct=true&scope=site&db=nlebk&db=n labk&AN=282084.
- Rangel, A. 2013. Regulation of dietary choice by the decision-making circuitry. *Nature Neuroscience*, 16(12): 1717–1724.
- Rattiner, S. L. 2002. *Food and Drink: A Book of Quotations*. Mineola, NY.: Dover Publications. https://books.google.com/books?id=MJfDAgAAQBAJ.
- Raynaud, E., Brun, J.-F., Perez-Martin, A., Sagnes, C., Boularan, A.-M., et al. 1999. Serum leptin is associated with the perception of palatability during a standardized high-carbohydrate breakfast test. *Clinical Science*, 96: 343–348.
- Raynor, H. A., & Epstein, L. H. 2003. The relative-reinforcing value of food under differing levels of food deprivation and restriction. *Appetite*, 40(1): 15–24.

- Read, D., & van Leeuwen, B. 1998. Predicting Hunger: The Effects of Appetite and Delay on Choice. *Organizational Behavior and Human Decision Processes*, 76(2): 189–205.
- Reuben, E., Sapienza, P., & Zingales, L. 2010. Time discounting for primary and monetary rewards. *Economics Letters*, 106(2): 125–127.
- Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. 2006. Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences*, 40(2): 305–315.
- Reynolds, S. M., & Berridge, K. C. 2002. Positive and negative motivation in nucleus accumbens shell: bivalent rostrocaudal gradients for GABA-elicited eating, taste "liking"/"disliking" reactions, place preference/avoidance, and fear. *The Journal of Neuroscience*, 22(16): 7308–7320.
- Richard, J. M., Castro, D. C., DiFeliceantonio, A. G., Robinson, M. J. F., & Berridge, K. C. 2013. Mapping brain circuits of reward and motivation: In the footsteps of Ann Kelley. *Neuroscience & Biobehavioral Reviews*, 37(9): 1919–1931.
- Robbins, T. W., & Everitt, B. J. 1982. Functional studies of the central catecholamines. *International Review of Neurobiology*, 23: 303–365.
- Robinson, T. E., & Flagel, S. B. 2009. Dissociating the Predictive and Incentive Motivational Properties of Reward-Related Cues Through the Study of Individual Differences. *Biological Psychiatry*, 65(10): 869–873.
- Rodin, J. 1985. Insulin levels, hunger, and food intake: An example of feedback loops in body weight regulation. *Health Psychology*, 4(1): 1–24.
- Roitman, M. F., Wheeler, R. A., Wightman, R. M., & Carelli, R. M. 2008. Real-time chemical responses in the nucleus accumbens differentiate rewarding and aversive stimuli. *Nature Neuroscience*, 11(12): 1376–1377.
- Rolls, B. J., Hetherington, M., & Burley, V. J. 1988. The specificity of satiety: The influence of foods of different macronutrient content on the development of satiety. *Physiology & Behavior*, 43(2): 145–153.
- Rolls, E. T. 2004. The functions of the orbitofrontal cortex. *Brain and Cognition*, 55(1): 11–29.
- Rolls, E. T., & McCabe, C. 2007. Enhanced affective brain representations of chocolate in cravers vs. non-cravers. *The European Journal of Neuroscience*, 26(4): 1067–1076.
- Roney, J. R. 2003. Effects of Visual Exposure to the Opposite Sex: Cognitive Aspects of Mate Attraction in Human Males. *Personality and Social Psychology Bulletin*, 29(3): 393–404.
- Rosenbaum, M., Sy, M., Pavlovich, K., Leibel, R. L., & Hirsch, J. 2008. Leptin reverses weight loss-induced changes in regional neural activity responses to visual food stimuli. *The Journal of Clinical Investigation*, 118(7): 2583.
- Rosenboim, M., Benzion, U., Shahrabani, S., & Shavit, T. 2012. Emotions, Risk Perceptions, and Precautionary Behavior Under the Threat of Terror Attacks: A Field Study Among Israeli College Students: Reactions to Terrorism. *Journal of Behavioral Decision Making*, 25(3): 248–256.
- Rothemund, Y., Preuschhof, C., Bohner, G., Bauknecht, H.-C., Klingebiel, R., et al. 2007. Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *NeuroImage*, 37(2): 410–421.

- Sabatinelli, D., Bradley, M. M., Lang, P. J., Costa, V. D., & Versace, F. 2007. Pleasure rather than salience activates human nucleus accumbens and medial prefrontal cortex. *Journal of Neurophysiology*, 98(3): 1374–1379.
- Salamone, J. D. 2007. Functions of mesolimbic dopamine: changing concepts and shifting paradigms. *Psychopharmacology*, 191(3): 389–389.
- Salamone, J. D., & Correa, M. 2002. Motivational views of reinforcement: Implications for understanding the behavioral functions of nucleus accumbens dopamine. *Behavioural Brain Research*, 137(1): 3–25.
- Salamone, J. D., Cousins, M. S., & Bucher, S. 1994. Anhedonia or anergia? Effects of haloperidol and nucleus accumbens dopamine depletion on instrumental response selection in a T-maze cost/benefit procedure. *Behavioural Brain Research*, 65(2): 221–229.
- Samuelson, P. A. 1937. A note on measurement of utility. *The Review of Economic Studies*, 4(2): 155–161.
- Saper, C. B., Chou, T. C., & Elmquist, J. K. 2002. The Need to Feed: Homeostatic and Hedonic Control of Eating. *Neuron*, 36(2): 199–211.
- Sapolsky, R. M. 2004. Why zebras don't get ulcers: The acclaimed guide to stress, stress-related diseases, and coping (3rd ed.). New York: Holt Paperbacks.
- Sarker, M. R., Franks, S., & Caffrey, J. 2013. Direction of post-prandial ghrelin response associated with cortisol response, perceived stress and anxiety, and self-reported coping and hunger in obese women. *Behavioural Brain Research*, 257: 197–200.
- Sarri, K. O., Linardakis, M. K., Bervanaki, F. N., Tzanakis, N. E., & Kafatos, A. G. 2004. Greek Orthodox fasting rituals: a hidden characteristic of the Mediterranean diet of Crete. *British Journal of Nutrition*, 92(2): 277–284.
- Scanes, C. 2010. *Fundamentals of Animal Science*. Cengage Learning. https://books.google.com/books?id=1RIFAAAAQBAJ.
- Scheer, F. A. J. L., Morris, C. J., & Shea, S. A. 2013. The internal circadian clock increases hunger and appetite in the evening independent of food intake and other behaviors. *Obesity*, 21(3): 421–423.
- Schienle, A., Schäfer, A., Hermann, A., & Vaitl, D. 2009. Binge-Eating Disorder: Reward Sensitivity and Brain Activation to Images of Food. *Biological Psychiatry*, 65(8): 654–661.
- Schultz, W. 2013. Updating dopamine reward signals. *Current Opinion in Neurobiology*, 23(2): 229–238.
- Schultz, W., Dayan, P., & Montague, P. R. 1997. A neural substrate of prediction and reward. *Science*, 275(5306): 1593–1599.
- Schwartz, M. W., Woods, S. C., Porte, D., Seeley, R. J., & Baskin, D. G. 2000. Central nervous system control of food intake. *Nature*, 404(6778): 661–671.
- Schweitzer, M. E., Ordóñez, L., & Douma, B. 2004. Goal setting as a motivator of unethical behavior. *Academy of Management Journal*, 47(3): 422–432.
- Scott, R. F., & Huxley, L. 1913. *Scott's Last Expedition, Volume I*. Dodd, Mead, & Co. https://books.google.com/books?id=oXoVAAAAYAAJ.
- Senior, C., Lee, N., & Butler, M. 2011. PERSPECTIVE—Organizational Cognitive Neuroscience. *Organization Science*, 22(3): 804–815.

- Sescousse, G., Caldú, X., Segura, B., & Dreher, J.-C. 2013. Processing of primary and secondary rewards: A quantitative meta-analysis and review of human functional neuroimaging studies. *Neuroscience & Biobehavioral Reviews*, 37(4): 681–696.
- Shakespeare, W. 1603. *Measure for measure*.
- Shalev, U., Yap, J., & Shaham, Y. 2001. Leptin attenuates acute food deprivation-induced relapse to heroin seeking. *The Journal of Neuroscience*, 21(4): 1–5.
- Shin, Y.-K., Martin, B., Kim, W., White, C. M., Ji, S., et al. 2010. Ghrelin is produced in taste cells and ghrelin receptor null mice show reduced taste responsivity to salty (NaCl) and sour (citric acid) tastants. *PLoS ONE*, 5(9): 1–13.
- Siep, N., Roefs, A., Roebroeck, A., Havermans, R., Bonte, M. L., et al. 2009. Hunger is the best spice: An fMRI study of the effects of attention, hunger and calorie content on food reward processing in the amygdala and orbitofrontal cortex. *Behavioural Brain Research*, 198(1): 149–158.
- Simmons, A., Matthews, S. C., Stein, M. B., & Paulus, M. P. 2004. Anticipation of emotionally aversive visual stimuli activates right insula. *Neuroreport*, 15(14): 2261–2265.
- Simmons, W. K., Martin, A., & Barsalou, L. W. 2005. Pictures of appetizing foods activate gustatory cortices for taste and reward. *Cerebral Cortex*, 15(10): 1602–1608
- Simpson, C. A., & Vuchinich, R. E. 2000. Reliability of a measure of temporal discounting. *The Psychological Record*, 50(1): 3–16.
- Skibicka, K. P., & Dickson, S. L. 2011. Ghrelin and food reward: The story of potential underlying substrates. *Peptides*, 32(11): 2265–2273.
- Skibicka, K. P., Hansson, C., Alvarez-Crespo, M., Friberg, P. A., & Dickson, S. L. 2011. Ghrelin directly targets the ventral tegmental area to increase food motivation. *Neuroscience*, 180: 129–137.
- Skibicka, K. P., Hansson, C., Egecioglu, E., & Dickson, S. L. 2012. Role of ghrelin in food reward: impact of ghrelin on sucrose self-administration and mesolimbic dopamine and acetylcholine receptor gene expression. *Addiction Biology*, 17(1): 95–107.
- Small, D. M., Bender, G., Veldhuizen, M. G., Rudenga, K., Nachtigal, D., et al. 2007. The role of the human orbitofrontal cortex in taste and flavor processing. *Annals of the New York Academy of Sciences*, 1121: 136–151.
- Small, D. M., Jones-Gotman, M., & Dagher, A. 2003. Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. *NeuroImage*, 19(4): 1709–1715.
- Small, D. M., Zatorre, R. J., Dagher, A., Evans, A. C., & Jones-Gotman, M. 2001. Changes in brain activity related to eating chocolate: from pleasure to aversion. *Brain: A Journal of Neurology*, 124(Pt 9): 1720–1733.
- Smeets, P. A., Erkner, A., & De Graaf, C. 2010. Cephalic phase responses and appetite. *Nutrition Reviews*, 68(11): 643–655.
- Smith, C. L., & Hantula, D. A. 2008. Methodological considerations in the study of delay discounting in intertemporal choice: A comparison of tasks and modes. *Behavior Research Methods*, 40(4): 940–953.
- Smith, E. E., & Jonides, J. 1999. Storage and executive processes in the frontal lobes. *Science*, 283(5408): 1657–1661.

- So, J., Achar, C., Han, D., Agrawal, N., Duhachek, A., et al. 2015. The psychology of appraisal: Specific emotions and decision-making. *Journal of Consumer Psychology*, 25(3): 359–371.
- Sobal, J., & Nelson, M. K. 2003. Commensal eating patterns: A community study. *Appetite*, 41(2): 181–190.
- Söderpalm, A. H., & Berridge, K. C. 2000. The hedonic impact and intake of food are increased by midazolam microinjection in the parabrachial nucleus. *Brain Research*, 877(2): 288–297.
- Sribney, W., Wiggins, V., & Drikker, D. 2015. *Negative and missing R-squared for 2SLS/IV*. StataCorp. http://www.stata.com/support/faqs/statistics/two-stage-least-squares/.
- Stanovich, K. E., & West, R. F. 2000. Individual differences in reasoning: Implications for the rationality debate. *Behavioral and Brain Sciences*, 23(5): 645–726.
- StataCorp. 2015. *Stata Statistical Software: Release 14*. College Station, TX.: StataCorp LP.
- Steel, P., & Konig, C. J. 2006. Integrating Theories of Motivation. *Academy of Management Review*, 31(4): 889–913.
- Stein, Z., Susser, M., Saenger, G., & Marolla, F. 1975. Famine and human development: The Dutch hunger winter of 1944-1945. New York: Oxford University Press.
- Stoeckel, L. E., Weller, R. E., Cook, E. W., Twieg, D. B., Knowlton, R. C., et al. 2008. Widespread reward-system activation in obese women in response to pictures of high-calorie foods. *Neuroimage*, 41(2): 636–647.
- Stone, O. 1987. Wall Street. Twentieth Century Fox.
- St-Onge, M.-P., Sy, M., Heymsfield, S. B., & Hirsch, J. 2005. Human cortical specialization for food: a functional magnetic resonance imaging investigation. *The Journal of Nutrition*, 135(5): 1014–1018.
- Story, M., Rosenwinkel, K., Himes, J. H., Resnick, M., Harris, L. J., et al. 1991. Demographic and risk factors associated with chronic dieting in adolescents. *American Journal of Diseases of Children*, 145(9): 994–998.
- Stricker, E. M., & Zigmond, M. J. 1976. Brain catecholamines and the lateral hypothalamic syndrome. In D. Novin, W. Wyrwicka, & G. Bray (Eds.), *Hunger; Basic Mechanisms and Clinical Implications*: 19–32. New York: Raven.
- Strotz, R. H. 1955. Myopia and Inconsistency in Dynamic Utility Maximization. *The Review of Economic Studies*, 23(3): 165–180.
- Sugino, T., Yamaura, J., Yamagishi, M., Ogura, A., Hayashi, R., et al. 2002. A transient surge of ghrelin secretion before feeding is modified by different feeding regimens in sheep. *Biochemical and Biophysical Research Communications*, 298(5): 785–788.
- Sugrue, L. P., Corrado, G. S., & Newsome, W. T. 2005. Choosing the greater of two goods: neural currencies for valuation and decision making. *Nature Reviews Neuroscience*, 6(5): 363–375.
- Symmonds, M., Emmanuel, J. J., Drew, M. E., Batterham, R. L., & Dolan, R. J. 2010. Metabolic State Alters Economic Decision Making under Risk in Humans. (L. Santos, Ed.)*PLoS ONE*, 5(6): e11090.

- Takahashi, T., Furukawa, A., Miyakawa, T., Maesato, H., & Higuchi, S. 2007. Two-month stability of hyperbolic discount rates for delayed monetary gains in abstinent inpatient alcoholics. *Neuroendocrinology Letters*, 28(2): 131–136.
- Tal, A., & Wansink, B. 2013. Fattening Fasting: Hungry Grocery Shoppers Buy More Calories, Not More Food. *JAMA Internal Medicine*, 173(12): 1146.
- Tanaka, S. C., Doya, K., Okada, G., Ueda, K., Okamoto, Y., et al. 2004. Prediction of immediate and future rewards differentially recruits cortico-basal ganglia loops. *Nature Neuroscience*, 7(8): 887–893.
- Tataranni, P. A., & DelParigi, A. 2003. Functional neuroimaging: a new generation of human brain studies in obesity research. *Obesity Reviews*, 4(4): 229–238.
- Teff, K. L., & Engelman, K. 1996. Palatability and dietary restraint: effect on cephalic phase insulin release in women. *Physiology & Behavior*, 60(2): 567–573.
- Tenbrunsel, A. E., & Smith-Crowe, K. 2008. Ethical Decision Making: Where We've Been and Where We're Going. *The Academy of Management Annals*, 2(1): 545–607.
- Tessari, M., Catalano, A., Pellitteri, M., Di Francesco, C., Marini, F., et al. 2007. Correlation between serum ghrelin levels and cocaine-seeking behaviour triggered by cocaine-associated conditioned stimuli in rats. *Addiction Biology*, 12(1): 22–29.
- Thaler, R. H., & Shefrin, H. M. 1981. An Economic Theory of Self-Control. *Journal of Political Economy*, 89(2): 392–406.
- Tindell, A. J., Berridge, K. C., Zhang, J., Peciña, S., & Aldridge, J. W. 2005. Ventral pallidal neurons code incentive motivation: amplification by mesolimbic sensitization and amphetamine. *European Journal of Neuroscience*, 22(10): 2617–2634.
- Toates, F. M. 1981. The control of ingestive behaviour by internal and external stimuli A theoretical review. *Appetite*, 2(1): 35–50.
- Toates, F. M. 1986. *Motivational systems*. Cambridge [Cambridgeshire]; New York: Cambridge University Press.
- Tong, J., Mannea, E., Aimé, P., Pfluger, P. T., Yi, C.-X., et al. 2011. Ghrelin enhances olfactory sensitivity and exploratory sniffing in rodents and humans. *The Journal of Neuroscience*, 31(15): 5841–5846.
- Trepanowski, J. F., & Bloomer, R. J. 2010. The impact of religious fasting on human health. *Nutrition Journal*, 9(57): 1–9.
- Treviño, L. K., den Nieuwenboer, N. A., & Kish-Gephart, J. J. 2014. (Un)Ethical Behavior in Organizations. *Annual Review of Psychology*, 65(1): 635–660.
- Treviño, L. K., & Weaver, G. R. 2003. *Managing ethics in business organizations:* Social scientific perspectives. Stanford, CA: Stanford Business Books.
- Treviño, L. K., Weaver, G. R., & Reynolds, S. J. 2006. Behavioral Ethics in Organizations: A Review. *Journal of Management*, 32(6): 951–990.
- Tschöp, M., Smiley, D. L., & Heiman, M. L. 2000. Ghrelin induces adiposity in rodents. *Nature*, 407(6806): 908–913.
- Tversky, A., & Kahneman, D. 1992. Advances in prospect theory: Cumulative representation of uncertainty. *Journal of Risk and Uncertainty*, 5(4): 297–323.
- Tversky, A., Slovic, P., & Kahneman, D. 1990. The causes of preference reversal. *The American Economic Review*, 80(1): 204–217.

- Ungless, M. A., Magill, P. J., & Bolam, J. P. 2004. Uniform inhibition of dopamine neurons in the ventral tegmental area by aversive stimuli. *Science*, 303(5666): 2040–2042.
- Urbain, J., & Davidcharkes, N. 1995. Recent advances in gastric emptying scintigraphy. *Seminars in Nuclear Medicine*, 25(4): 318–325.
- Van den Bergh, B., & Dewitte, S. 2006. Digit ratio (2D: 4D) moderates the impact of sexual cues on men's decisions in ultimatum games. *Proceedings of the Royal Society B: Biological Sciences*, 273(1597): 2091–2095.
- Van den Bergh, B., Dewitte, S., & Warlop, L. 2008. Bikinis Instigate Generalized Impatience in Intertemporal Choice. *Journal of Consumer Research*, 35(1): 85–97.
- van den Bos, W., & McClure, S. M. 2013. Towards a general model of temporal discounting. *Journal of the Experimental Analysis of Behavior*, 99(1): 58–73.
- van Kleef, E., Shimizu, M., & Wansink, B. 2013. Just a bite: Considerably smaller snack portions satisfy delayed hunger and craving. *Food Quality and Preference*, 27(1): 96–100.
- Vandewater, K., & Vickers, Z. 1996. Higher-protein foods produce greater sensory-specific satiety. *Physiology & Behavior*, 59(3): 579–583.
- Vig, L., Gupta, A., & Basu, A. 2011. A Neurocomputational Model for the Relation Between Hunger, Dopamine and Action Rate. *Journal of Intelligent Systems*, 20(4). http://doi.org/10.1515/JISYS.2011.019.
- Volk, S., & Kohler, T. 2012. Brains and Games: Applying Neuroeconomics to Organizational Research. *Organizational Research Methods*, 15(4): 522–552.
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Logan, J., Jayne, M., et al. 2002. "Nonhedonic" food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse*, 44(3): 175–180.
- Volkow, N. D., Wang, G.-J., Maynard, L., Jayne, M., Fowler, J. S., et al. 2003. Brain dopamine is associated with eating behaviors in humans. *International Journal of Eating Disorders*, 33(2): 136–142.
- Volkow, N. D., & Wise, R. A. 2005. How can drug addiction help us understand obesity? *Nature Neuroscience*, 8(5): 555–560.
- Wadhwa, M., Shiv, B., & Nowlis, S. M. 2008. A Bite to Whet the Reward Appetite: The Influence of Sampling on Reward-Seeking Behaviors. *Journal of Marketing Research*, 45(4): 403–413.
- Wang, G.-J., Tomasi, D., Backus, W., Wang, R., Telang, F., et al. 2008. Gastric distention activates satiety circuitry in the human brain. *NeuroImage*, 39(4): 1824–1831.
- Wang, G.-J., Volkow, N. D., Telang, F., Jayne, M., Ma, J., et al. 2004. Exposure to appetitive food stimuli markedly activates the human brain. *NeuroImage*, 21(4): 1790–1797.
- Wang, X. T., & Dvorak, R. D. 2010. Sweet Future: Fluctuating Blood Glucose Levels Affect Future Discounting. *Psychological Science*, 21(2): 183–188.
- Wansink, B., Tal, A., & Shimizu, M. 2012. First Foods Most: After 18-Hour Fast, People Drawn to Starches First and Vegetables Last. *Archives of Internal Medicine*, 172(12). http://doi.org/10.1001/archinternmed.2012.1278.

- Watson, K., & Rinomhota, S. 2001. Preoperative fasting: we need a new consensus. *Nursing Times*, 98(15): 36–37.
- Weigle, D. S., Breen, P. A., Matthys, C. C., Callahan, H. S., Meeuws, K. E., et al. 2005. A high-protein diet induces sustained reductions in appetite, ad libitum caloric intake, and body weight despite compensatory changes in diurnal plasma leptin and ghrelin concentrations. *The American Journal of Clinical Nutrition*, 82(1): 41–48.
- Weinshall-Margel, K., & Shapard, J. 2011. Overlooked factors in the analysis of parole decisions. *Proceedings of the National Academy of Sciences*, 108(42): E833–E833.
- Wellman, P. J., Davis, K. W., & Nation, J. R. 2005. Augmentation of cocaine hyperactivity in rats by systemic ghrelin. *Regulatory Peptides*, 125(1): 151–154.
- White, H. 1980. A heteroskedasticity-consistent covariance matrix estimator and a direct test for heteroskedasticity. *Econometrica: Journal of the Econometric Society*, 817–838.
- White, H. 1982. Maximum likelihood estimation of misspecified models. *Econometrica: Journal of the Econometric Society*, 1–25.
- Wierenga, C. E., Bischoff-Grethe, A., Melrose, A. J., Irvine, Z., Torres, L., et al. 2014. Hunger Does Not Motivate Reward in Women Remitted from Anorexia Nervosa. *Biological Psychiatry*. http://doi.org/10.1016/j.biopsych.2014.09.024.
- Wilson, E. O. 1998. Consilience: The unity of knowledge. New York: Knopf.
- Wilson, M., & Daly, M. 2004. Do pretty women inspire men to discount the future? *Proceedings of the Royal Society of London, Series B: Biological Sciences*, 271(4): 177–179.
- Wise, R. A. 1980. The dopamine synapse and the notion of "pleasure centers" in the brain. *Trends in Neurosciences*, 3(4): 91–95.
- Wise, R. A. 2004. Dopamine, learning and motivation. *Nature Reviews Neuroscience*, 5(6): 483–494.
- Wise, R. A. 2006. Role of Brain Dopamine in Food Reward and Reinforcement. *Philosophical Transactions: Biological Sciences*, 361(1471): 1149–1158.
- Wren, A. M., & Bloom, S. R. 2007. Gut Hormones and Appetite Control. *Gastroenterology*, 132(6): 2116–2130.
- Yam, K. C., Chen, X.-P., & Reynolds, S. J. 2014. Ego depletion and its paradoxical effects on ethical decision making. *Organizational Behavior and Human Decision Processes*, 124(2): 204–214.
- Yam, K. C., Reynolds, S. J., & Hirsh, J. B. 2014. The hungry thief: Physiological deprivation and its effects on unethical behavior. *Organizational Behavior and Human Decision Processes*, 125(2): 123–133.
- Yamada, H., Tymula, A., Louie, K., & Glimcher, P. W. 2013. Thirst-dependent risk preferences in monkeys identify a primitive form of wealth. *Proceedings of the National Academy of Sciences*, 110(39): 15788–15793.
- Yamaguchi, S., & Ninomiya, K. 2000. Umami and food palatability. *The Journal of Nutrition*, 130(4): 921S–926S.
- Yeomans, M. R. 1996. Palatability and the Micro-structure of Feeding in Humans: the Appetizer Effect. *Appetite*, 27(2): 119–133.

- Yeomans, M. R. 1998. Taste, palatability and the control of appetite. *Proceedings of the Nutrition Society*, 57(4): 609–615.
- Yeomans, M. R., & Gray, R. W. 1997. Effects of naltrexone on food intake and changes in subjective appetite during eating: evidence for opioid involvement in the appetizer effect. *Physiology & Behavior*, 62(1): 15–21.
- Yeomans, M. R., & Gray, R. W. 2002. Opioid peptides and the control of human ingestive behaviour. *Neuroscience & Biobehavioral Reviews*, 26(6): 713–728.
- Yeomans, M. R., Gray, R. W., Mitchell, C. J., & True, S. 1997. Independent Effects of Palatability and Within-meal Pauses on Intake and Appetite Ratings in Human Volunteers. *Appetite*, 29(1): 61–76.
- Zafra, M. A., Molina, F., & Puerto, A. 2006. The neural/cephalic phase reflexes in the physiology of nutrition. *Neuroscience & Biobehavioral Reviews*, 30(7): 1032–1044.
- Zarr, N., Alexander, W. H., & Brown, J. W. 2014. Discounting of reward sequences: a test of competing formal models of hyperbolic discounting. *Frontiers in Psychology*, 5(178): 1–9.
- Zizzo, D. J. 2010. Experimenter demand effects in economic experiments. *Experimental Economics*, 13(1): 75–98.
- Zyphur, M. J., & Oswald, F. L. 2013. Bayesian estimation and inference: A user's guide. *Journal of Management*, 1–32.