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I

NEUROECONOMICS

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REWARD, REPRESENTATION, AND IMPULSIVITY: A THEORETICAL FRAMEWORK FOR THE NEUROSCIENCE OF RISKY DECISION MAKING

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We provide an overview of neuroscience research on risky decision making, organizing findings in an integrative theoretical framework aimed at elucidating mechanisms that drive behavior. The concept of risk has been used to describe a variety of influences on decisions—including both the variance of outcomes and the potential for a negative outcome—each of which may have a distinct influence on neural processing. Armed with these distinctions, we examine neural substrates of reward and valuation, reviewing evidence that the ventromedial prefrontal cortex (vmPFC) computes a common currency signal that allows comparison of rewards across domains (e.g., food and money). This common currency signal is modulated by the variables that shape decision making, such as gains, losses, and their probabilities. We review evidence that subjective feelings about the uncertainty and valence of outcomes (e.g., risk and loss aversion) follow from signals in the insula and that signals associated with uncertainty

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can be distinguished from signals of emotional salience in the amygdala. Representations of options in vmPFC/medial orbitofrontal cortex serve as inputs to a comparison process in anterior cingulate cortex (ACC)/ dorsomedial prefrontal cortex (dmPFC), which reflects decisional conflict. Activation in ACC/ dmPFC is greater when choices conflict with otherwise dominant strategies, such as gist-based simplification versus verbatim-based trading off, triggering cognitive control mechanisms in dorsolateral PFC. When value signals are translated into actions, prefrontal signals influence processing of neurons in the posterior parietal cortex whose activity is consistent with drift-accumulator models of choice. This tentative process model differentiates several independent contributors to risk-taking behavior and identifies levers of behavioral change that could be used to prevent unhealthy decisions.

People often die from risky choices. Choices to initiate smoking, take illicit drugs, eat unhealthy foods, drive recklessly, and drink excessively contribute to cancer, heart disease, and trauma from traffic accidents (Reyna & Farley, 2006). Lifetime prevalence of alcohol dependence alone approaches 20% (Bloom, 2010). Crime is less a phenomenon of psychopathy (although psychopaths commit a disproportionate percentage of crime) than of age-related risk taking, peaking in adolescence and young adulthood. Indeed, homicide is the second leading cause of death among 15- to 19-year-olds, after unintentional injury; firearms are the instrument of death in 85% of cases (Centers for Disease Control and Prevention, 2012). Therefore, the "burden of illness"—the severity and prevalence of harm to oneself and others—produced by risky decision making is enormous (Arnett, 1992; Reyna & Rivers, 2008).

Risky decision making is a major public health problem, as illustrated by this brief litany of statistics, but it is also a problem of law, medicine, social relations, and personal finance. In the domain of personal finance, for example, people commit errors of risk aversion and risk seeking, avoiding riskier investments with better lifetime returns and spending down reserves rather than saving enough for retirement (Benartzi & Thaler, 1995; Rick, Cryder, & Loewenstein, 2008). Although risky behaviors can differ across domains of life, they also correlate with one another, peak at roughly similar ages, and activate a common neural circuitry of risk and reward valuation (Jessor, 1991; D. J. Levy & Glimcher, 2011; Porcelli & Delgado, 2009; Reyna, Chapman, Dougherty, & Confrey, 2012). As we discuss in this chapter, understanding the risk and reward mechanisms in the brain, and their development, is key to unraveling the mystery of irrational risk taking in real life.

Laboratory experiments are an essential element in this understanding. Risky decisions made in the laboratory predict real-world decisions,

although far from perfectly (e.g., Galvan et al., 2006; Galvan, Hare, Voss, Glover, & Casey, 2007; Lejuez, Aklin, Zvolensky, & Pedulla, 2003; Parker & Fischhoff, 2005; Pleskac, 2008; Reyna et al., 2011; Steinberg, Cauffman, Woolard, Graham, & Banich, 2009; Stout, Rock, Campbell, Busemeyer, & Finn, 2005). Alternatively, using real-world behaviors for prediction of different risky behaviors does not solve the problem of imperfect prediction, and typically such relationships are difficult to interpret because they are confounded (i.e., multiple correlated factors contribute to risky behaviors). (Using some real-world risky behaviors to "predict" other risky behaviors is satisfactory for actuarial purposes, but it sheds little light on causation or mechanisms.) Consequently, when the proper methodological controls are used, laboratory tasks provide crucial insight into the mechanisms of risky decision making. The main challenge in developing effective practice and public policy is *understanding* these mechanisms of risky decision making to determine (a) whether they apply in a given context and (b) how they combine to produce behavior.

Progress in this endeavor hinges on distinguishing among related concepts such as impulsivity, reward sensitivity, risk perception, risk preference, sensation seeking, and how rewards are represented and remembered; many tasks used in research conflate these concepts. One thesis of our broader argument is that behavioral evidence that distinguishes these concepts is essential to inform the design of tasks and analyses, and, hence, the interpretation of neuroscience data. Despite generally impressive rigor, the amount of neuroscience data regarding any one question is relatively limited, and what data there are is sometimes not sufficiently theoretically grounded to be interpreted unambiguously. In addition, neuroscience research can be enhanced through a greater emphasis on hypothesis testing and by experimental designs that complement correlational approaches, as found in relevant behavioral research (e.g., see Huettel, Song, & McCarthy, 2009).

Furthermore, although it is possible to wait until more data arrive, research on both brain and behavior will progress more expeditiously if they are brought together. More fundamentally, the functions of the brain cannot be understood without a process analysis of the behavioral tasks they support, and a deep understanding of behavior cannot be achieved in ignorance of the rich new world of neuroscience research. In short, this chapter is a prolegomenon to a fully verified theoretical framework for the neuroscience of risky decision making. The interpretations we offer are preliminary, but they respond to the challenge of developing a functional taxonomy that maps decision behavior onto its underlying processes, both psychological and neuroscientific (Huettel, 2010; Poldrack et al., 2011).

DEFINITIONS AND DISTINCTIONS

There are two main approaches to defining risky decision making: economic and psychological. For economists, risk is about variance of known outcomes (i.e., uncertain events, not uncertainty in the epistemic sense). Within the expected utility framework of economics, *risk preference* (sometimes called *risk attitude*) refers to the shape of the utility function estimated from a series of risky choices, with the choice between a sure versus a risky option of equal expected value being a canonical example (von Winterfeldt & Edwards, 1986). Expected utility is a nonlinear transformation of expected value into subjective value.

For example, consider a choice between (a) winning $1,000 for sure versus (b) a .50 probability of winning $2,000 and .50 probability of winning nothing. Risk-averse people would prefer the sure thing (a) over the risky gamble (b). Comparison of option (a) with option (b) indicates that the second option has more variance in outcomes than the first. (It may be useful to think of the sure option as $.50 \times \$1{,}000 + .50 \times \$1{,}000$, illustrating that outcomes do not vary when they are sure.) When the expected values of each option are equal to one another, as in this example, sure options ($1,000) are mathematically closer to the origin (i.e., closer to zero) than non–zero gamble outcomes ($2,000). Thus, if people prefer the sure option, their "utility" function for money is assumed to have a concave curvature—$2,000 deviates more (in a downward direction) from its objective value than $1,000 does from its objective value.

In neuroscience research on decision making, the values of decision options are talked about in at least two ways: the value of the outcomes ($1,000 and $2,000 in our example) and the value of the whole option, known as *expected value*, which is a weighted average of outcomes and probabilities (i.e., $\$1{,}000 \times 1.0 = \$1{,}000$ or $.5 \times \$2{,}000 + .5 \times 0 = \$1{,}000$) (e.g., D. J. Levy & Glimcher, 2012; Zhang & Hirsch, 2013). When outcomes are positive, they are referred to as *rewards*; expected value captures the idea that probability and reward trade off to produce overall value (e.g., stocks have greater expected value but greater risk than bonds).

Expected value corresponds roughly to people's intuitions about probabilities and outcomes. If people did not weight outcomes by their probabilities, they would feel the same way about a lottery ticket that paid off with a $1,000 prize for sure and one that paid off $1,000 with a .50 probability. Even if a person has never heard of the concept of expected value, she would not prefer a .5 chance (e.g., flipping a coin) to win $1,000 over a sure $1,000. Animals and children who have not yet learned to multiply also make such trade-offs of risk and reward intuitively (Reyna & Brainerd, 1994). Thus, the formal concepts of variance and expected value have some psychological reality.

For economists, none of the outcomes need to be "bad" for an option to be considered risky. The second option (or gamble) in our example could be a .50 chance of winning $1,200 and a .50 chance of winning $800, and it would still be riskier in the economics sense compared with a sure $1,000. For psychologists, however, behavioral choices that lead to the possibility of loss or harm are referred to as *risky* (Fox & Tannenbaum, 2011). As in the economists' definition of *risky*, these bad outcomes (losses or harms) are *uncertain:* Crimes go unpunished, most people who drink never become alcoholics, unprotected sex does not necessarily result in HIV infection, and many pieces of pie do not necessarily produce death or disease. Individuals differ in their willingness to tolerate this uncertainty.

Taking undue risks can be considered pathological and, thus, classified in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV–TR*; e.g., eating disorders or antisocial personality disorder; American Psychiatric Association, 2000) or the *International Statistical Classification of Diseases and Related Health Problems* (10th ed.; ICD–10; World Health Organization, 2010). However, risky choices can also be socially approved, for example, when football players risk serious injury, or they can be an ordinary part of daily life, as in high-risk jobs such as logging or washing windows of skyscrapers. A question that has guided neuroscience research is how the brain processes this variance or uncertainty, in particular, how brain systems support compensatory tradeoffs between a safer, lower value option and a riskier, higher value option (De Martino, Kumaran, Seymour, & Dolan, 2006; Huettel, 2006; Tom, Fox, Trepel, & Poldrack, 2007; Venkatraman, Payne, Bettman, Luce, & Huettel, 2009). Given the uncertainty inherent in risk taking, it can be difficult to draw a clear line between healthy and unhealthy risk taking, but that distinction is the crux of adaptive behavior (Reyna & Farley, 2006).

The psychological definition of risky behavior conflates two aspects of decision making, however, that are separable theoretically and functionally in the brain: risk and loss attitudes (e.g., Yechiam & Telpaz, 2013). The example of the type that we have discussed (a sure gain vs. a gamble of equal expected value) is frequently used to illustrate *risk aversion* because most people prefer the sure option to the gamble of equal expected value. People find the uncertainty in the gamble aversive despite its mathematical equivalence over repeated trials (i.e., if the gamble were enacted repeatedly, the average winnings or expectation would be $1,000). However, risk aversion is not the same as loss aversion.

Loss aversion does not just mean that losses (e.g., losing money, people dying rather than being saved) are negative, but that losses hurt more than gains of similar magnitude feel good (Kahneman & Tversky, 2000; there is also evidence that people pay closer attention to losses; see Yechiam & Telpaz, 2013). So, most people will not accept a bet on the flip of a coin in which

the amount to win is equal to the amount to lose (e.g., win $10 if heads, lose $10 if tails). They usually require about a 2:1 ratio of wins to losses to accept the bet (e.g., win $20 if heads, lose $10 if tails). Loss aversion refers to an asymmetry in the impact of gain versus loss outcomes, rather than risk or uncertainty (Willemsen, Böckenholt, & Johnson, 2011).

In sum, risk aversion can be defined as preferring a sure thing to a roughly mathematically equivalent (or superior) gamble, and risk taking as the opposite preference. Economists focus on people's taste for variance, which classic theories identify with nonlinear functions of outcomes, such as money. In the economists' view, two gambles can differ in risk if one has higher variance in outcomes than another. Psychologists focus on tolerance for uncertain bad outcomes, such as losses, and the implications of risk attitudes for life outcomes. However, risk and loss aversion can be distinguished empirically—and loss aversion can be demonstrated when options are certain. Both economists' and psychologists' definitions of risk attitude have influenced neuroscience research (e.g., Schonberg, Fox, & Poldrack, 2011).

According to either economists' or psychologists' definitions, most people have an aversion to uncertainty. When people demonstrate a high tolerance for uncertain outcomes, whether inside or outside of the laboratory, what are the brain processes that underlie their risk preferences? Do they process risk or reward differently, fail to trade them off properly, or lack the ability to control their attraction to rewards? Moreover, are the nature of thinking and underlying brain processes qualitatively different among those who seek versus avoid risks, especially in ways that go beyond traditional dichotomies between sensation seeking and self-control (Chick & Reyna, 2012; Reyna et al., 2011)? With these definitions and distinctions in mind, we can begin to assemble the neural building blocks of risky decision making.

NEURAL SUBSTRATES OF REWARD AND VALUATION

The neural substrates of reward have been well characterized, building on carefully controlled research with animals (Platt & Huettel, 2008). The reward circuit of the brain consists of the midbrain dopamine areas (the ventral tegmental area and substantia nigra) and the basal ganglia structures to which they project (the ventral striatum, where the nucleus accumbens is located, and the dorsal striatum). Axons from the midbrain areas also project broadly to the prefrontal cortex, but particularly to the ventromedial prefrontal cortex (vmPFC; Galvan, 2012). Dopaminergic activity in these areas has been linked to current and anticipated rewards (e.g., Glimcher, Camerer, Fehr, & Poldrack, 2009).

Rewards are often appetitive in studies of animals (e.g., juice or food), but studies of humans have shown generalization of value signals to include monetary, social, and other rewards (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000). The idea that there is a common currency of rewards in the brain is consistent with economic notions of utility in which the subjective values of different types of rewards all project onto a dimension of reward value (Montague & Berns, 2002; Smith et al., 2010). (We initially focus on rewards, such as appetitive stimuli, but discuss aversive stimuli below.)

To test the common-currency hypothesis, D. J. Levy and Glimcher (2011) mapped neural circuits for reward valuation by scanning subjects who made choices for money, food, and water (see also Chib, Rangel, Shimojo, & O'Doherty, 2009; FitzGerald, Seymour, & Dolan, 2009; Kim et al., 2010). For example, a subject might choose between a sure win of five M&Ms versus a risky option offering a 38% chance of winning 20 M&Ms (and a 62% chance of winning nothing). Subjects also received trade-off trials in which they chose between a sure win of a small amount of money ($0.50) versus some probability of either winning a fixed amount of food or water (or getting nothing).

D. J. Levy and Glimcher (2011) found that risk preferences across reward types were correlated: The level of risk aversion when choosing among monetary options predicted risk aversion for food and water. In other words, a subject who was more risk averse for money was generally also more risk averse for food and water (although substantial variation among individuals was observed). The common areas of neural activation that varied with valuation across domains were the vmPFC and striatum. These results are reminiscent of those of other studies implicating the vmPFC and striatum in representing values in risky choice tasks (Daw, O'Doherty, Dayan, Seymour, & Dolan, 2006; De Martino, Kumaran, Holt, & Dolan, 2009; Huettel, Stowe, Gordon, Warner, & Platt, 2006; Kable & Glimcher, 2007; Knutson, Fong, Bennett, Adams, & Hommer, 2003; I. Levy, Snell, Nelson, Rustichini, & Glimcher, 2010).

Interestingly, vmPFC is close to, or part of, the default mode network, which is more active at rest than during task performance (Raichle & Snyder, 2007). This suggests that task-related activations in vmPFC might correspond to different degrees of deactivation, relative to rest (Rushworth, Noonan, Boorman, Walton, & Behrens, 2011). However, traditional default mode activation is anterior to the posterior region of the vmPFC most often linked to common-currency activation.

In addition to common areas of activation in vmPFC, D. J. Levy and Glimcher (2011) found that distinct neural networks represented monetary and food rewards: the dorsal hypothalamic region responded mainly to the reward value of food, whereas the posterior cingulate cortex responded mainly

to the value of money. Within the vmPFC itself, there were overlapping but also distinct areas of activation for money and food (see also Clithero, Carter, & Huettel, 2009, for similar conclusions from pattern classification of brain activation). However, only the vmPFC represented the value of money and food on what appeared to be a common scale, as predicted by expected utility and neurobiological approaches (Glimcher, Dorris, & Bayer, 2005; Glimcher, 2011; von Neumann & Morgenstern, 1944). That is, the relative levels of activity in vmPFC to food and money rewards reflected the relative values of food and money rewards to that individual and predicted trade-off choices between these different types of rewards.

In contrast to this common-currency hypothesis, Weber, Blais, and Betz (2002) and others have argued that an individual's risk taking in one domain, such as finances, need not be reflected in her risk taking for health or other outcomes. Weber et al.'s Domain-Specific Risk-Taking (DOSPERT) scale assesses risk-taking separately for decisions about monetary gambling and investment, ethical, health/safety, social, and recreational domains (for an updated scale, see Blais & Weber, 2006). As in classic expected-utility approaches, they distinguish between perceptions of risks and benefits (i.e., rewards), and relate those to risk taking.

More specifically, the updated DOSPERT's items include "Having an affair with a married man/woman" (*Ethical*), "Investing 10% of your annual income in a new business venture" (*Financial*), "Engaging in unprotected sex" (*Health/Safety*), "Disagreeing with an authority figure on a major issue" (*Social*), and "Taking a weekend sky-diving class" (*Recreational*). Weber et al. (2002) and other studies show that risk attitudes are not the same across these domains within individuals (which seems to contradict D. J. Levy & Glimcher's, 2011, results, but see below). Content varies across items, which makes them rich descriptors of real-life behavior, but they do not isolate risk preference per se. In other words, each item taps factors unrelated to risk preference, such as moral compunctions or athletic interests, as well as risk preference.

Despite finding domain differences, Weber et al. (2002) reported that sensation seeking (and its various subscales) correlated significantly with risky behavior in all five domains of the DOSPERT scale, with the highest correlation between the thrill-and-adventure-seeking subscale and recreational risk taking ($r = .56$), echoing decades of similar results in which sensation seeking predicts a broad spectrum of risky behaviors (e.g., Arnett, 1990a, 1990b; Hoyle, Fejfar, & Miller, 2000; Zuckerman, 1994). In addition, Weber et al. found that scores on each domain's risk attitudes scale significantly predicted frequency of risky behaviors for the other domains (with the exceptions of the social subscale and social risk taking). Thus, D. J. Levy and Glimcher (2011) used a common procedure to elicit risk attitudes across reward domains, rather than comparing apples to oranges (i.e., noncommensurate financial vs. health

risk preferences), but Weber et al.'s results nevertheless confirm that preferences across reward types that reflect realistic decisions are correlated within individuals. That is, a person who is more risk averse in a given reward type is likely be more risk averse in another reward type. A recent meta-analysis using data from 13 different functional magnetic resonance imaging (fMRI) studies corroborated the conclusion that this common reward representation is located in a subregion of the vmPFC and adjacent medial orbitofrontal cortex (mOFC; D. J. Levy & Glimcher, 2012).

NEURAL SUBSTRATES DIFFERENTIATING GAINS, LOSSES, AND PROBABILITY

Consistent with research that we have reviewed, the vmPFC/mOFC blood oxygen level dependent (BOLD) signal has been shown to be correlated with the reward value of choices in multiple studies (e.g., Rushworth et al., 2011). For example, Plassmann, O'Doherty, and Rangel (2007) presented subjects with a series of pictures of food items and asked them how much they were willing to pay for each item. They used a Becker–DeGroot–Marschak method to ensure that subjects had an incentive to provide their true valuation; if their valuation exceeded a randomly drawn number, they forfeited money and accepted the food instead. The vmPFC/mOFC signal increased with participants' valuation of the food.

The balance of evidence suggests that the vmPFC/mOFC signal responds to losses as well as gains, decreasing proportionately to the magnitude of anticipated losses or negative outcomes (Rushworth et al., 2011; Tom et al., 2007). For example, Plassman, O'Doherty, and Rangel (2010) showed that willingness to pay to avoid eating an unpleasant food inversely correlated with signal in this region. The signal also decreases with other dimensions of choice that lower the overall value of an option, such as lower probability of gains or greater delay to receive gains (Kable & Glimcher, 2007; Peters & Buchel, 2009; Prévost, Pessiglione, Me´te´reau, Cléry-Melin, & Dreher, 2010). Hunt et al. (2012) advanced a plausible model of neuronal activity in this region in which signals initially correspond to the sum of the values of potential choices, but, later in the trial, reflect the value difference between choices. Using magnetoencephalography, Hunt et al. found that such a sequence of signals occurred in vmPFC/mOFC and also superior parietal cortex near the intraparietal sulcus. Thus, conflicting results supporting both the presence of overall value and value-difference signals in vmPFC/mOFC can be reconciled by assuming that these outputs occur at different points in time (Rushworth, Kolling, Sallet, & Mars, 2012; Wunderlich, Rangel, & O'Doherty, 2010).

Therefore, across studies, neural valuation subsumes both the reward value of outcomes and overall expected value, which includes probability (or risk) and outcomes. However, little consensus exists about the neural substrates of probability or risk (Huettel, 2010). Indeed, information about probability or risk has no meaning without information about outcomes; the probability must refer to the probability of some outcome to elicit preferences or choices. (This psychological reality about probability does not imply that probability cannot be varied orthogonally to outcomes; see d'Acremont, Fornari, & Bossaerts, 2013; Reyna et al., 2011).

D'Acremont et al. (2013) varied probability independently of outcome value using a probability learning task (subjects learned the probabilities of stimuli representing various payoffs through repetitive sampling). They found that activation in the medial prefrontal cortex and parietal cortex (angular gyri) increased linearly with the probability of the currently observed stimulus. Connectivity analyses during rest and task revealed that these regions were part of the default mode network. (Outcome values were encoded outside of the default network, in the striatum, when net rewards were realized at the end of the decision phase.)

Thus, contrary to the usual characterization, the default mode network was active during a task requiring attention to external stimuli (see also Spreng, 2012). Neuroimaging studies have identified these areas of the network, parietal cortex and medial prefrontal cortex, as involved in successful recognition (Cabeza, Ciaramelli, Olson, & Moscovitch, 2008; Wagner, Shannon, Kahn, & Buckner, 2005). D'Acremont et al. (2013) argue that a major function of the default mode network is to represent memory strength for traces of past events, consistent with contemporary memory-based theories of decision making (Reyna & Brainerd, 2011; Weber & Johnson, 2009). As we discuss in greater detail below, activation in dorsal anterior cingulate cortex (ACC) was related to *choice* uncertainty (i.e., uncertainty in making a choice, or decisional conflict). When choices were made but outcomes were unknown (the risky phase of the task), the right anterior insula and bilateral caudate were activated; the insula signaled *outcome* uncertainty.

To evaluate the evidence about neural substrates of probability (or risk), Mohr, Biele, and Heerkeren (2010) conducted a quantitative meta-analysis of fMRI experiments on risk processing. Mohr et al. examined how risk processing is influenced by emotions, how it differs between choice and nonchoice situations, and how it changes when losses are possible. Over a range of paradigms, risk was consistently associated with activation in the anterior insula, a brain region known to process aversive emotions, such as anxiety. The anterior insula was predominantly active in the presence of potential losses. The authors interpreted these results as evidence that risk processing is influenced by emotions and as indicating that potential losses modulate risk processing.

Reflecting the nature of the literature, Mohr et al.'s (2010) conclusion spans the concepts of risk and loss aversion, which, as we have already discussed, are distinct (e.g., Kahneman & Tversky, 2000). However, these results raise the interesting question of whether risk and loss aversion share an underlying commonality—a subjective feeling of aversion—despite the fact that this feeling is elicited by different causes (i.e., uncertainty vs. loss, both of which many people find distasteful; Reyna & Brainerd, 2011; Willemsen et al., 2011). This subjective feeling of aversion, in turn, is one avenue through which risk taking can be inhibited (Clark et al., 2008).

An interpretation of Mohr et al.'s (2010) meta-analysis, then, one that is consistent with studies cited by Wood and Bechara in this volume, is that the insula signals the phenomenology of aversion, common to risk and loss aversion. In other words, the insula provides an interoceptive signal of subjective feeling, not only of aversion (or disgust) but also of cravings and other homeostatic signals, as well as more abstract feelings such as admiration, love, and indignation (Chapter 7, this volume). Physiological states activated in the posterior insula seem to be re-represented in the anterior insula, the latter mapping onto subjective feelings. Craig, Chen, Bandy, and Reiman (2000), for example, found that activity in the posterior insula was linearly related to the actual temperature of a stimulus (applied to the hand), but activity in the anterior insula correlated with subjective ratings of temperature. Thus, processing of the magnitudes of potential losses in vmPFC/mOFC should be distinguished from subjective twinges associated with feeling those losses in the insula, which may help people avoid risk (Kuhnen & Knutson, 2005).

The function of the insula should also be distinguished from that of the amygdala, although they are connected reciprocally (the anterior insula is also connected to the ventral striatum, vmPFC, and ACC). As Wood and Bechara (Chapter 7, this volume) explain, "Although the amygdala is responsible for associative learning and emotions, the insula governs the conscious feeling of those emotions" (p. 189). Some patients with amygdala lesions do not show loss aversion, which is consistent with other research showing such patients' impaired processing of negative stimuli (De Martino, Camerer, & Adolphs, 2010; Murray, 2007). However, other patients with amygdala lesions have shown impairments for risky decisions involving gains rather than losses (Weller, Levin, Shiv, & Bechara, 2007). Basten, Biele, Heekeren, and Fiebach (2010) found that ventral striatum activated more in response to expected rewards or benefits in a decision task, whereas amygdala activated more to expected losses (and cost–benefit differences were correlated with an inferred comparison region in the vmPFC). As suggested above, results for vmPFC were consistent with computing expected reward by trading off amygdalar "costs" and ventral striatal "benefits."

Although stimulating the amygdala produces fear, anxiety, and vigilance (Phillips, Drevets, Rauch, & Lane, 2003), the amygdala activates more in response to positive, negative, and unusual or interesting stimuli than in response to neutral stimuli (Cunningham & Brosch, 2012). Therefore, the amygdala may signal emotional salience, with riskiness and losses being among those features of decision options that have functional significance (Cunningham, Van Bavel, & Johnsen, 2008; De Martino et al., 2006; Roiser et al., 2009). De Martino et al. (2010) speculated that an initial negative anticipatory response is generated in the amygdala to potential losses, which influences the striatal computation of the gamble's net value, and consequently leads to loss aversion. Thus, in this view, the amygdala is not the locus of memory or choice, and its effect on loss aversion is indirect. Although patients with amygdala lesions would be less likely to be loss averse, those with an intact ventral striatum and vmPFC/mOFC—and especially insula—might be able to avoid disadvantageous (in the sense of lower expected value) gambles involving losses (Weller et al., 2007).

These areas of the brain are dynamically connected, as seen when people regulate emotional affect. Studies of emotion regulation suggest that activation of the vmPFC and the amygdala are related, and coupling of the amygdala and vmPFC is related to trait anxiety in both task-related and resting-state studies (Burghy et al., 2012). For example, individuals high in trait anxiety exhibit reduced amygdala-vmPFC resting-state connectivity (Kim, Gee, Loucks, Davis, & Whalen, 2011), as though the lack of connectivity reflected habitual inability to down-regulate anxiety (but see Poldrack, 2006, for caveats regarding "reverse inference").

COMPARISON AND CONFLICT

As we have discussed, the evidence suggests that values of gains, losses, and their probabilities are represented in the vmPFC/mOFC, informed by the striatum and the rest of the reward circuit. The options also give rise to subjective feelings and are infused with emotional significance, reflected in the insula and amygdala. At some point in the decision process, the representations of options are input to another comparison process that takes place in dorsal ACC or adjacent (e.g., Hare, Schultz, Camerer, O'Doherty, & Rangel, 2011).

In contrast to signals in the vmPFC/mOFC, the ACC/dmPFC BOLD signal increases when the difference between the values of potential choices decreases. For example, Pochon, Riis, Sanfey, Nystrom, and Cohen (2008) had subjects choose between two attractive faces; activity in the ACC/dmPFC was higher when faces were similar in attractiveness, provoking decisional

conflict. Similarly, De Martino et al. (2006) observed greater ACC/dmPFC activation when subjects made choices that were inconsistent with their dominant preference of framing effects. Increased activation in the dmPFC seems to represent a conflict between the generally preferred gist-based response and a compensatory analytical choice. In other words, the difficulty of a comparison between options varies with signal in the ACC/dmPFC as though this area captures disparity detection (e.g., error monitoring), cognitive conflict, or decisional conflict (e.g., Brown & Braver, 2008; Taren, Venkatraman, & Huettel, 2011; Venkatraman et al., 2009; see Venkatraman & Huettel, 2012). The output of this comparison process then determines activation in the motor system, guiding which response should be made to satisfy the reward goal, and culminating in the response (Cai & Padoa-Schioppa, 2012; Rushworth et al., 2012). Hence, the vmPFC/mOFC can be thought of as "choosing" (or favoring) a reward goal, whereas the ACC/dmPFC adjudicates potentially antagonistic actions or rules for decision making (Brown & Braver, 2008; Rushworth et al., 2012; Venkatraman & Huettel, 2012).

REPRESENTATION AND RESPONSE PREFERENCE

While the vmPFC/mOFC signal increases with the difference in value between possible choices, the BOLD signal in the parietal cortex and some other motor association areas increases as the choice selection becomes more difficult, as indexed by reaction time. The parietal signal, therefore, often has characteristics that are the opposite of the vmPFC/mOFC signal. Like the ACC/dmPFC, the size of the parietal signal (e.g., posterior parietal cortex, the medial intraparietal sulcus) is negatively correlated with the difference in value between choices (Basten et al., 2010; Rushworth et al., 2011). According to Basten et al. (2010), for example, a neural representation of the difference between rewards (ventral striatal benefit signal) and losses (amygdalar cost signal) is evaluated in the vmPFC and then accumulates in the parietal cortex (intraparietal sulcus) until a decision threshold is reached.

Thus, one way to think of the accumulator is as an index of response preference; difficult choices are those for which the difference in value between response options is small. The accumulator can operate within trials, as people process information about risks and rewards, or costs and benefits, with longer response latency and lower confidence for difficult choices. The accumulator can also be thought of as operating across trials, as information becomes acquired through experiencing outcomes of risky decisions. As examples of learning from experience, one can have unprotected sex and either experience pregnancy or not over the course of a year; one can leave a car unlocked each

night for a month and find out each morning whether items in the car have been stolen or not (e.g., Yechiam, Barron, & Erev, 2005). Alternatively, one can read about the probability of pregnancy given unprotected sex over a year on a contraceptive label or look up the rates at which cars are broken into in a neighborhood, learning from description as opposed to experience (Reyna & Adam, 2003; Reyna & Farley, 2006).

Many of the decision paradigms we have cited involve learning about outcomes and probabilities through experience rather than direct description, as in the Iowa Gambling Task (IGT). In the IGT, subjects select cards from "good" and "bad" decks, discovering that some decks yield small rewards and losses but net gains, whereas other decks yield large rewards but still larger losses (e.g., Hochman, Yechiam, & Bechara, 2010). Therefore, subjects learn to anticipate outcomes by virtue of experience, a process that relies heavily on memory for outcomes and their probabilities (Stout et al., 2005). Developmental differences in risk taking in the IGT, for example, virtually disappear when children are provided with ongoing tallies of frequencies of outcomes (Van Duijvenvoorde, Jansen, Bredman, & Huizenga, 2012; see also Reyna & Brainerd, 1994). As we discuss, in addition to the medial temporal lobe (MTL), encompassing the hippocampus and parahippocampal regions, the parietal cortex plays an important role in memory representation and retrieval (Cabeza et al., 2008).

Memory Representations

Memory representations are generally encoded simultaneously in two formats: verbatim (e.g., the number of dollars to be won) and gist (e.g., the categorical or ordinal qualitative essence of the amount to be won, such as "some money" or "a lot of money"; Kühberger & Tanner, 2010; Reyna & Brainerd, 1995, 2011). Verbatim representations support precise processing (e.g., trading off the magnitudes of risk and reward, as in expected value or utility), but gist representations support the fuzzy, impressionistic processing of intuition (Reyna, 2013). Although neuroscience research has focused on representing quantitative valuation of rewards or expected value (even when options are nonnumerical, such as food; Hare et al., 2011), behavioral research demonstrates that qualitative, fuzzy gist representations of decision options, along with retrieval of associated broad mores (e.g., that saving lives, gaining money, or helping family is good), often govern risk preferences (Reyna, 2012; Reyna et al., 2011).

Specifically, consistent with neuroscience research, decision makers appear to estimate expected value or expected utility (e.g., Glimcher, 2011; Hare, Camerer, & Rangel, 2009), a conclusion that, at first blush, contradicts the literature ruling out expected value or utility as viable descriptive theories

of behavior (e.g., Tversky & Kahneman, 1986). However, these views can be reconciled by positing parallel systems that process both verbatim (symbolic, but superficial) values of options and their qualitative gist. Qualitative gist, in turn, is responsible for effects of meaning and context that can be said to "bias" decisions, such as framing effects, used as evidence against expected value and utility theories (Reyna & Brainerd, 2011).

Furthermore, these verbatim–gist parallel systems are required to account for predicted double dissociations and nonmonotonic effects in the behavioral literature, and they are supported by mathematical models tested for goodness-of-fit with behavioral data (e.g., see Brainerd, Reyna, & Howe, 2009; Brainerd, Reyna, & Mojardin, 1999; Reyna, 2012; Reyna & Brainerd, 1995; Singer & Remillard, 2008). Evidence that people encode both verbatim (literal) and gist (simple meaning) representations comes from research on memory, reasoning, and decision making, and has been studied across the life span (e.g., for reviews, see Reyna, 1995, 2012; Reyna & Mills, 2007).

In particular, many experiments have compared verbatim-based true memory to so-called false memory, the latter usually based on memory for the gist of experience (Kim & Cabeza, 2007; Slotnick & Schacter, 2004). While true recollection (verbatim memory is vivid or recollective) has been associated with neural activity in the MTL (hippocampus and posterior parahippopcampal gyrus), medial prefrontal cortex (mPFC), lateral parietal cortex, and posterior cingulate, vague familiarity has been associated with activity in lateral PFC regions, the MTL (anterior parahippocampal gyrus and rhinal cortex), and the superior parietal cortex (e.g., Daselaar, Fleck, & Cabeza, 2006; Spaniol et al., 2009; Yonelinas, Otten, Shaw, & Rugg, 2005; for reviews, see Diana, Yonelinas, & Ranganath, 2007; Eichenbaum, Yonelinas, & Ranganath, 2007; Eichenbaum, Sauvage, Fortin, Komorowski, & Lipton, 2012).

These memory studies provide clues about the neural basis of gist versus verbatim representations and processing that explain risky decision making, as we presently discuss. However, many of the memory studies confound phenomenology—vivid recollection versus vague familiarity—with verbatim or gist representation. Although verbatim representations (e.g., memory for frequencies of outcomes of draws in the IGT) are vivid, gist representations can be either vague (experienced as global similarity or familiarity) or vivid (experienced as "phantom recollection"; Brainerd, Payne, Wright, & Reyna, 2003).

Using a recognition task with pictures, Dennis, Bowman, and Vandekar (2012) separated recollection from familiarity by asking subjects to judge recognition-test pictures as "remember," "know," or "new" ("remember" responses were designated as recollection and "know" responses as familiarity). Test items were previously presented pictures (true), semantically similar but not identical pictures (false or gist-consistent), or unrelated pictures.

Directly comparing true with false recollection revealed that true recollection uniquely involved hippocampus and early visual cortex, consistent with verbatim memory supporting remember responses to presented pictures (remember hits; Brainerd et al., 2003). Gist memory is assumed to be common to both true and false recollection and thus may be reflected in conjunction results for true and false recollection (e.g., precentral gyrus and superior parietal cortex).

Dennis et al. (2012) also assessed whole brain functional connectivity for an MTL region in the anterior parahippocampal gyrus that was activated for true (true remember > true know) and for false recollection (false remember > false know). For true recollection (compared with false recollection), the anterior parahippocampal gyrus showed greater functional connectivity with inferior regions, including the bilateral hippocampus, the ACC, the orbitofrontal cortex, and the occipital cortex. In contrast, false recollection (compared with true recollection) showed greater functional connectivity between the anterior parahippocampal gyrus and superior regions including the bilateral pre- and post-central gyrus, the superior PFC, and the bilateral parietal cortex. This inferior–superior dissociation may support a distinction between bottom-up, lower-order verbatim processes in true recollection and top-down, higher order gist and control processes in false recollection (Cabeza, 2008).

Memory Representations of Risk Preference in Decision Making

In decision making as in other information-processing tasks, people encode verbatim and gist representations of options, and in experiential paradigms (e.g., IGT), they encode both types of representations of the probabilities and outcomes of choices. People extract multiple gist representations of options but typically rely on the simplest (categorical) gist, distinguishing between some quantity versus nothing, outputting a decision if the choice is not contradicted by parallel verbatim processing. For example, the simplest gist of the example presented earlier is (a) winning some money versus (b) either winning some money or winning nothing. Because most people value money, some money is generally preferred to nothing, favoring the sure option.

The same kind of categorical (some–none) gist applies to choices involving losses: Thus, a choice between (c) losing $1,000 for sure versus (d) a .5 probability of losing $2,000 and .5 probability of losing nothing boils down to (c) losing some money versus (d) either losing some money or losing nothing. Because most people value losing nothing more than losing money, they generally prefer the risky option. The shift from risk avoiding for gains to risk seeking for losses is an example of a framing effect (Kahneman & Tversky, 2000). Critical tests have been conducted showing that categorical

gist accounts for a variety of framing effects and ruling out expected utility and prospect theory as explanations for these effects (e.g., Kühberger & Tanner, 2010; Reyna, 2012; Reyna & Brainerd, 1991, 1995).

To take one example, removing the mathematically redundant zero complement of the gamble (.5 probability of winning/losing nothing = 0) removes the categorical some–none contrast and eliminates framing effects, although all of the causal elements of framing effects, according to utility and prospect theory, remain. These and other empirical results, such as larger framing effects when numbers are removed, experimentally manipulating attention to the zero outcome to increase framing effects, and the growth of framing effects from childhood to adulthood, all support a fuzzy-trace theory account of verbatim and gist processing in risky decision making (e.g., Kühberger & Tanner, 2010; Reyna & Brainerd, 1991, 1995; Reyna & Ellis, 1994; Reyna et al., 2011; Reyna & Farley, 2006). Although framing effects themselves divulge the use of categorical gist (per the explanation above), parallel verbatim processing is revealed through people's sensitivity to expected value: People are usually indifferent between options when their expected values are equal—provided that the zero complement is removed—but they favor the option with the larger expected value when options are unequal (e.g., Reyna & Brainerd, 1995; Weller et al., 2007).

Venkatraman et al. (2009) explored the use of these alternative strategies of verbatim trading off of expected value versus categorical some–none gist processing by presenting subjects with a series of five-outcome gambles containing gain and loss outcomes (probabilities are shown in parentheses), such as $80 (.20), $40 (.25), $0 (.20), –$25 (.15), –$70 (.20). However, subjects could improve the gambles, for example, by adding $15 to either the $0 outcome (changing that outcome to $15) or to the –$70 (changing that outcome to –$55). The choices subjects made to change gambles diagnosed their information-processing strategies.

Altogether, Venkatraman et al. (2009) assessed three strategies: increasing the magnitude of the highest gain (Gmax), decreasing the magnitude of the worst loss (Lmin), or improving the probability of winning something by adding money to the middle outcome (e.g., by eliminating the categorical possibility of winning nothing; Pmax). The Gmax and Lmin choices represent a compensatory verbatim analytical strategy consistent with standard models of risky choice, whereas Pmax represents a simplifying gist-based strategy. Thus, the tasks used by Venkatraman et al. (2009), like those used by Kühberger and Tanner (2010); Mills, Reyna, and Estrada (2008); and Reyna (2012), make it possible to discriminate between gist- and verbatim-based strategies in risky decision making.

Activation in the posterior parietal cortex and dorsolateral prefrontal cortex (dlPFC) predicted gist-based, simplifying choices, whereas activation

in the vmPFC and anterior insula predicted verbatim analytical, compensatory choices, maximizing gains (i.e., Gmax) and minimizing losses (i.e., loss aversion or Lmin), respectively (see also, Hedgcock, Denburg, Levin, & Halfmann, 2012). Functional connectivity (psychophysiological interaction) analyses showed positive correlations between the dmPFC and the dlPFC for simplifying choices and between the dmPFC and insula for compensatory choices. Although many scholars assume that compensatory strategies are more adaptive, research has shown that gist-based simplifying strategies are associated with greater development (from childhood to adulthood), greater expertise in adulthood, and better health outcomes (e.g., Reyna et al., 2011; Reyna & Farley, 2006; Reyna & Lloyd, 2006).

Consistent with our earlier discussion of the ACC/dmPFC, activation in this area was greater when subjects made choices that conflicted with their dominant strategy, such as when people who generally preferred the gist-based, simplifying choice made a compensatory choice and vice versa. Greater ACC/dmPFC activation occurs for higher decisional conflict even when subjects do not have to respond overtly on a given trial and when decision and response phases are separated (Pochon et al., 2008; Venkatraman et al., 2009). Further, Venkatraman et al. (2009) showed, using resting state connectivity analyses, that the dmPFC was connected to the dlPFC, the latter associated with response selection, in an anterior to posterior organization (e.g., anterior dmPFC to rostral dlPFC; posterior dmPFC to caudal dlPFC and premotor cortex; Bunge, Hazeltine, Scanlon, Rosen, & Gabrieli, 2002; Taren et al., 2011). Venkatraman and Huettel (2012) argued that the anterior dmPFC regulates activity in the anterior dlPFC when control demands are highly abstract (e.g., choosing among conflicting strategies), but the posterior dmPFC regulates the posterior dlPFC and premotor cortices when control demands merely choose between conflicting motor responses.

Impulsivity/Inhibition of Responses

The dlPFC, often in conjunction with the ACC, has long been associated with cognitive control, which can be manifested as response inhibition, cognitive distraction (distancing), or reappraisal of the meaning of a stimulus (Ochsner & Gross, 2008; Venkatraman & Huettel, 2012; see also Chapter 6, this volume). The dlPFC modulates the value signal encoded in the vmPFC, and dlPFC activity is correlated with successful self-control (e.g., in go/no-go tasks; Casey et al., 2011; or when choosing between healthy and unhealthy foods; Hare et al., 2009).

Several studies have reported a link between higher dlPFC activity and lower risk taking. For example, Gianotti et al. (2009) found a negative correlation between tonic activity in the dlPFC (i.e., cortical hypoactivity) as

measured using electroencephalogram at rest and risk-taking propensity in a laboratory task. These results are consistent with findings from patients with lesions in this area and healthy subjects with "virtual lesions" (created using transmagnetic cranial stimulation, or TMS), who showed increased risk taking relative to controls (Knoch et al., 2006). Conversely, Fecteau et al. (2007) reduced risk taking in a laboratory task by increasing dlPFC activity using transcranial direct current stimulation. Schonberg et al. (2012) interpreted their results showing increasing dlPFC activation as risk mounts in a repeated sampling task as reflecting the increased engagement of self-control, which prompts subjects to terminate the trial.

Finally, Hutcherson, Plassman, Gross, and Rangel (2012) instructed subjects to down-regulate ("distance") or up-regulate ("indulge") their desire for foods (or react naturally) while being imaged. During down-regulation, activation *decreased* in the dlPFC but not in the vmPFC, and the relative contribution of the two value signals to behavior shifted toward the dlPFC. The opposite pattern was observed during up-regulation; activation increased in the vmPFC but not the dlPFC, and the relative contribution to behavior shifted toward the vmPFC. Although the direction of activation seems to contradict other research on cognitive regulation of emotion, the authors interpret the reduction in activation in the dlPFC as indicating value modulation (see Hutcherson et al.'s, 2012, discussion), a finding consistent with recent research on emotional value in vmPFC (Winecoff et al., 2013). However, in both regulation conditions relative to "natural" responding, Hutcherson et al. observed significant activation in the ventrolateral PFC and the posterior parietal cortex, areas also found in other studies. Taken together, these studies suggest that dlPFC (and other areas') activity reflects cognitive control that reigns in risk taking, generally through inhibition or reevaluation of emotional, rewarding, or prepotent responses.

Going beyond the influence of a single area or region such as the dlPFC, Shannon et al. (2011) correlated resting state connectivity with a measure of impulsivity for adolescents incarcerated in a high-security facility. The impulsivity measure included "need for stimulation" (e.g., "I enjoy gambling for large stakes"). Shannon et al. showed that prediction of impulsivity peaked using premotor (i.e., motor planning regions) functional connectivity (i.e., selecting or adding other brain regions of interest degraded prediction of impulsivity). A typical young adult sample displays positive correlations between the premotor area and both the dorsal attention and executive networks, and negative correlations with the default mode network. A similar pattern was observed in less impulsive incarcerated youth as well as normal controls; premotor areas were connected to networks involving attention and executive processes.

In contrast, premotor areas were positively correlated with the default network among the impulsive incarcerated youth, parts of which (e.g., vmPFC),

as we have discussed, are involved in task-related reward valuation and probability learning in normally developing adults. Premotor areas were negatively correlated with attention and control networks. Hence, impulsive incarcerated youth displayed the opposite pattern shown with normal adults. Moreover, functional connectivity varied with age among another sample of normal controls ranging in age from 7 to 31. As age increased, functional connectivity with the premotor area migrated from the default network (significantly decreasing with age) to the attention and control networks (significantly increasing with age). Thus, the relationship of the networks to impulsivity, though stronger in incarcerated youth, was recapitulated by age, as though impulsive offenders were developmentally delayed.

OVERVIEW AND IMPLICATIONS FOR RISKY DECISION MAKING

Our review of the literature on the neuroscience of risky decision making has ranged from reward circuitry to response inhibition. We have reviewed activations in different brain regions, but also the interconnections among areas, for example, between the amygdala and the vmPFC (e.g., the input of the amygdala to vmPFC valuation comparisons). The hypotheses offered about function represent an effort to make sense of neuroscientific findings, but they can only be tentative as the necessary process models remain underspecified.

In particular, the simultaneous and countervailing interactions among different brain processes challenge interpretation (e.g., Shannon et al., 2011, which demonstrates network-level interactions). For example, if the dlPFC downregulates the global valuation of a risky option, then activation of the vmPFC might appear misleadingly small. By misleading, we mean that the causal pathway for risky behavior differs for an individual who has mastered self-control (but for whom self-regulation is imperfect) versus someone who was never tempted to begin with (DeYoung, 2010). Understanding these causal pathways is crucial for designing interventions and public policies to reduce unhealthy risk taking.

For example, the levers for behavioral change include shaping the encoding of the *functionally significant* features of decisions (e.g., consequential financial losses or major health threats; Reyna, 2008). Very little is understood about "salience" and how the amygdala categorizes features of decisions, such as their riskiness, as significant or not. The presence of risk, by itself, is not a contraindication to choosing an option; driving in most cities represents a nonnegligible risk. (Phobias about driving, flying, or otherwise engaging in everyday risks can severely impair adaptive decision making.) Thus, despite the widespread faith in expected utility, decision trees, and other rational approaches to decision making, adaptive risk taking is generally not

characterized by a focus on such details, but, rather, on the "big picture" of what is important.

That is, adaptive decision making involves more than applying a rote formula, as suggested by fuzzy trace theory. Understanding functional significance (i.e., what the individual represents as important about options that involve risk) is a deep and highly contextual judgment. The concept of gist, which dates back to research on summarizing narratives, sheds some light on how people distill the substance of decisions (e.g., Clark & Clark, 1977). Although the representation of the gist of options—their meaning—seems to shape risk preferences, most neuroscience research employs tasks that fail to capture or even assess that meaning. Naturally, researchers who do not measure gist processing will not find it in the brain, omitting a major mode of real-world decision making.

The concept of self-control is also fraught with ambiguity in the current literature, but our review suggests some basic distinctions that may be helpful. As Casey et al. (2011) emphasized, responsiveness to enticing rewards represents a distinct vulnerability for impulsive individuals, and, presumably, for those who take risks (here, impulsivity is defined narrowly as failure to inhibit responses, but see Zuckerman, 1994). Response inhibition in the sense of restraint seems to be discriminable from reward sensitivity (i.e., temptation by valuation). The inability to engage in cognitive distraction or reappraisal of meaning because of low intelligence or poor coping skills is yet another source of vulnerability. The failure to learn the probabilities of events from experience or to detect conflict among decision strategies, and the absence of internal feeling cues, are additional risk factors for maladaptive decision making. How each of these factors operates to promote risk taking (alone or in combination)—and their neural circuitry—is poorly understood.

One impediment to progress in this area is the mistaken belief that laboratory risk-taking tasks that unconfound causal factors do not predict real-life risk taking. Many studies have refuted that misconception, and yet it persists. However, this does not imply that a narrow focus on college students of a certain age with little variation in culture or individual differences is adequate; representative samples of subjects are also needed. Rather than measure large numbers of people using poorly conceived tasks (and powerful statistics), however, progress in the neuroscience of risky decision making requires a greater focus on tasks motivated by process-oriented theory.

To some decision neuroscience researchers, theory harkens back to traditional expected utility models and simple Skinnerian behaviorism. Is risky decision making merely a matter of computing gains (rewards), losses, and probabilities (with perhaps a touch of Spence's notion of behavioral inhibition)? Certainly, at one level, such simple concepts successfully predict

approach and avoidance behavior, including some aspects of risky decision making. However, behaving logically and trading off risk and reward do not seem to be sufficient for adaptive decision making. Children (by about 6 years of age), adolescents, and people with mild autism are less prone to classic judgment-and-decision-making biases, compared with neurodevelopmentally typical adults, but they nevertheless have deficits in real-world risky decision making (Reyna & Brainerd, 2011). Acknowledging "risk as feelings" is also not sufficient to predict or improve risky decision making to date (for a discussion of the shortcomings of this approach, see Sunstein, 2008). Instead, an integrative approach is needed that emphasizes prediction and hypothesis testing, an approach that incorporates motivational, emotional, and cognitive elements but goes beyond them in designing new tasks that capture the richness and relevance of risky decision making.

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