Modeling Decision Making under Risk using Neurochemistry

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Abstract. Challenges to the descriptive validity of the expected utility model have led to the development of non-expected utility models of decision making under risk including prospect theory which incorporates psychological considerations to account for the loss-gain differentiation in risk attitude and overweighting of small probabilities associated with sizable outcomes. We propose a neurochemical extension of prospect theory by linking two evolutionarily ancient neurotransmitters – dopamine and serotonin – to the valuation and saliency of outcomes over gains and losses. This model provides a biological basis for the phenomenon of loss aversion, diminishing valuation sensitivity towards gains and losses, and nonlinear response to probabilistic stimuli. We derive its testable implications linking the decision maker's genetic makeup modulating her dopamine and serotonin tones to her attitude towards the fourfold risks of moderate prospects, moderate hazards, longshot prospects, and longshot hazards. The empirical validity of our model is corroborated by evidence from within-subject correlations of attitude towards fourfold risks as well as evidence from recent published findings on the molecular genetics of risk taking.

Keywords: *risk, decision making, prospect theory, valuation, probability weighting, salience, neurochemistry, dopamine, serotonin, biology, genetics, neuroeconomics*

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1. Introduction

Challenges to the descriptive validity of the expected utility model have led to the development of a number of non-expected utility models of decision making under risk since the eighties (see Starmer 2000, for a survey). The most prominent of these models is prospect theory (PT) by Kahneman and Tversky (1979) which incorporates psychological considerations in positing the loss-gain differentiation in valuation relative to the status quo and the overweighting of small probabilities associated with sizable outcomes. PT can also account for individuals exhibiting the fourfold pattern of risk attitude – being risk averse towards moderate prospects and longshot hazards and risk tolerant towards moderate hazards and longshot prospects. Subsequently, there has been a significant literature on models that generalizes expected utility with several being built around the idea of using decision weights that may deviate from the underlying probabilities to average the utilities that are defined on the underlying outcomes (see Starmer, 2000, for a survey). In their rejoinder, Tversky and Kahneman (1992) offer a cumulative prospect theory (CPT) which incorporates the form of the probability weighting function in Quiggin (1982) separately for gain-oriented and loss-oriented risks.

The emergence of neuroeconomics over the past decade (Camerer, Lowenstein, and Prelec, 2005; Rustichini, 2005) builds on advances in neuroimaging as well as behavioral and experimental economics in understanding economic behavior beyond revealed choice. Specific neuroanatomical regions modulating decision making under risk identified include striatum, frontal cortex and insula (Hsu, et al, 2005; Huettel, Song, and McCarthy, 2005; Huettel et al, 2006; Kuhnen and Knutson, 2005;

Preuschoff, Bossaerts, and Quartz, 2006; Tom, et al, 2007; Preuschoff, Quartz, and Bossaerts, 2008) while amygdala activation has been linked to loss-oriented risks (Yacubian et al, 2006; Martino, Camerer, and Adolphs, 2010). The anterior cingulate cortex has been associated with probability weighting for gain oriented risks (Paulus and Frank, 2006) as well as loss-oriented risks (Berns et al, 2008) while the striatum has been associated with probability weighting in Hsu et al (2009).

The year 2009 saw the publication of a good number of papers on the genetics of economic risk taking. Two of these consist of twin studies involving respectively a Chinese sample (Zhong et al, 2009a) and a Swedish sample (Cesarini et al, 2009a). They find significant heritability of economic risk taking observed through incentivized choice in a controlled laboratory setting. Several studies combine the methodologies of molecular genetics and experimental economics and find association between economic risk taking and specific well-characterized genes (Crisan et al, 2009, Dreber et al, 2009, Kuhnen and Chiao, 2009, Roe et al, 2009, and Zhong et al, 2009b,c).

The nascent literature on the genetics of risk taking motivate the present paper in which we offer a neurochemical model of decision making under risks and derive testable implications linking the decision maker's risk attitude with her genetic makeup. Specifically, we posit a link between two evolutionarily ancient neurotransmitters – dopamine (DA) and serotonin (5HT) – and valuation as well as saliency of economic outcomes. In addition to the loss-gain differentiation in valuation, our model can exhibit nonlinearity in responding to probabilistic stimuli. The empirical validity of our model is corroborated by evidence from within-subject

correlations of attitude towards fourfold risks as well as evidence from recent published findings on the molecular genetics of risk taking.

The rest of the paper is organized as follows. Section 2 presents our neurochemical model of decision making under risks following an exposition of the neurochemistry of valuation and saliency. Section 3 discusses the empirical validity of our model in terms of evidence on correlations of fourfold risk attitudes and on recently findings on the molecular genetics of risk taking. Section 4 discusses further extension of our model and offers some concluding remarks.

2. Neurochemical Model of Decision Making under Risk

We begin with an introductory exposition of neurochemistry and properties of DA and 5HT that pertain to the model being developed in this section. Neurons are excitable cells in the nervous system that process and transmit information by electrochemical signals. They constitute the core components of the brain, the vertebrate spinal cord, and the peripheral nerves. An adult human brain contains about 100 billion neurons (Williams and Herrup, 1988). When neurons communicate with each other, they release signalling chemicals, called neurotransmitters, into a specialized gap called synapse, between adjacent neurons. Neurotransmitters mediate the relay, amplification, and modulation of electrical signals between one neuron and another neuron. Major neurotransmitter systems include the DA system and the 5HT systems among others.

When a neurotransmitter is released across the gap between a transmitting and a receiving neuron, an electrochemical signal is generated between the adjacent

neurons. The strength of the signal is determined, among other things, by the concentration of neurotransmitter and the avidity with which it is recognized by a receptor on the cell surface of the adjacent receiving neuron. Concentration of the signalling molecules can be decreased through recovery of released neurotransmitter by specialized transporter proteins, e.g., dopamine and serotonin transporter proteins, or decreased by specialized enzymes, e.g., monoamine oxidase (MAO), that convert the active neurotransmitter to an inactive form necessary for the system to continue transmitting new information.

2.1. Neurochemistry of valuation and saliency

The idea that one's valuation of outcomes may be related to the underlying DA and 5HT systems has been in the air. The link between valuation over gains and DA is based on pervasive evidence drawn from single neuron recording studies with monkeys, demonstrating that midbrain DA neurons encode reward as well as reward prediction errors, i.e., responding to the incidence of unexpected reward (Schultz, Apicella, and Ljungberg, 1993; Mirenowicz and Schultz, 1996; Schultz, Dayan, and Montague, 1997). More recently, Tobler et al (2005) report a monotonic relation in how the majority of the midbrain DA neurons (75–80%) respond to reward magnitude, such as liquid volume, corroborating its role in coding reward value or utility. Subsequent fMRI studies using human decision making tasks show that the dopaminergic system conveys signals of expected reward (Preuschoff, Bossarts, and Quartz, 2005; Knutson et al, 2005).

DA neurons do not appear to encode for losses or punishment. When reward is omitted after a conditioned inhibitor, it does not produce a negative prediction error or

a depression in DA neurons (Fiorillo, Tobler, and Schultz, 2003). At both the behavioral and the neural levels, Pessiglione et al (2006) find that the administration of DA drugs affects risky decision making under gains but not under losses. In the presence of both gains and losses, D'Ardenne et al (2008) reported that the neural activation in ventral tegmental area reflects positive reward prediction errors modulated by the probability of winning with no significant correlation with loss-oriented events.

It is known that 5HT contributes to the regulation of aversive behavior. Brodie and Shore (1957) first suggest 5-HT's role in behavioral inhibition. Subsequently, Lucki (1998) emphasizes the general principle that 5HT constrains the response of organisms to external arousing stimuli thereby enhancing response to a variety of external stimuli in its absence. Daw et al (2002) further find that due to their low spontaneous firing rate, the inhibitory response of dopaminergic neurons to prediction of no reward (or its omission) is weak, illustrating a 'floor' effect' in which DA signalling bottoms out. They then propose 5HT to be in "opponent partnership" with DA and hypothesize this molecule to mediate aversion-specific responses. Dayan and Huys (2009) suggest the notion that serotonin is an imperfect reflection of dopamine given their observation of a general role for serotonin as a signal associated with predictions and prediction errors for future aversive outcomes. They further observe that the opponency between reward and punishment is fundamentally asymmetric, with, at least in species such as rats and primates, rewards being typically rare and caused by actions of the self, while punishments being typically common and originating in environmental contingencies.

For DA neurons, there are reported findings of their excitation by salient stimuli (e.g., tones and light) that are not inherently reward oriented (see Ungless, 2004 for a review). In a single neuron recording experiment, DA neurons respond to the novelty of an unexpected physical stimulus (Ljungberg, Apicella, and Schultz, 1992). In an fMRI experiment, stratum activation increases with the degree to which an unexpected novel sound interferes, even in the absence of reward (Zink et al, 2006). Taken together, the evidence suggests that DA mediates saliency of an event apart from its role in reward processing, and its response to saliency seems more pronounced for gain-oriented events.

There is evidence that 5HT also encodes information regarding the overall affect salience of an event (e.g., a wakeup call or arousal) and not directly related to reward and punishment or reinforcement learning algorithms. For instance, amygdala activation in response to angry faces is 5HT driven (Hariri et al, 2002). Biased attention towards negative or positive affective stimuli could confer differential vulnerability to emotional disorders such as depression and anxiety. Bias towards negative affective stimulus is one of the characteristics of emotional disorders, while bias towards positive stimuli would be predicted to be protective against emotional disorders. Recent genetic association studies suggest that 5HT modulate the biased attention towards both negative and positive affective stimuli. For example, it was shown that the long allele of serotonin transporter promoter region polymorphism (5-HTTLPR) mediates attention focus towards positive affective stimulus (Fox, Ridgewell, and Ashwin, 2009), and the short allele of 5-HTTLPR was linked to negative affective stimulus (Pérez-Edgar et al, 2009). Taken together, the evidence

suggests that 5HT mediates saliency of an event apart from valuation regarding losses or aversive outcomes, and it appears to focus either more on gain-oriented stimulus or more on loss-oriented stimulus.

2.2. Neurochemical Model

Consider a lottery, *(x, p)*, consisting of a *p* chance of receiving an outcome *x* and a *1 – p* chance of receiving *0*. We refer to gain-oriented (loss-oriented) lotteries as prospects (hazards). In particular, *(G, ½)* with $G > 0$ is an even-chance moderate prospect (MP) and *(G, p)* with *p* small is a longshot prospect (LP). Similarly, for $L < 0$, *(L, ½)* is a moderate hazard (MH) while *(L, q)* with *q* small is a longshot hazard (LH). The PT utility of receiving *(x, p)* is given by $\pi(p)v(x)$ incorporating a probability weighting function π in addition to a utility function *v*. Incorporating psychophysical considerations, Kahneman and Tversky (1979) argue that the utility function *v* ought to exhibit diminishing sensitivity in the magnitude of outcomes, i.e., gains as well as losses, relative to the decision maker's status quo, represented by 0, in which case *v* would be concave over gains, convex over losses, and vanish at *0*. As long as $\pi(½)$ does not exceed ½, PT yields the twofold pattern – *(G/2, 1)* is preferred to *(G, ½)* and yet *(L, ½)* is preferred to *(L/2, 1)* – given its loss-averse utility function. To account for the remaining twofold pattern of risk attitudes, it suffices for π to overweight small probabilities, i.e., $\pi(p) > p$ for small *p*. It follows that *(G, p)* is preferred to *(pG, 1)* if $\pi(p) > v(pG)/v(G$ and *(pL,1)* is preferred to *(L, p)* if $\pi(p) > v(pL)/v(L)$.

Motivated by PT, Quiggin (1982) axiomatized an anticipated utility model, also known as rank-dependent utility, which coincides with PT for prospects with up to two distinct gain outcomes. In their rejoinder, Tversky and Kahneman (1992) incorporated the anticipated utility formulation and offered a cumulative extension of prospect theory with distinct probability weighting functions π^+ and π^- for evaluating prospects and hazards. Naturally, CPT can display the fourfold pattern by requiring each of its probability weighting functions to overweight small probabilities.

Extending the notion in Berns et al (2007) of a biological constraint in the occupancy of DA receptors, Zhong et al (2009b) sketch a neurochemical approach of loss-gain differentiation in risk attitude by linking valuation over gains and losses respectively to the DA tone and 5HT tone, which refers to the low level of background neuron firing in a slow, irregular single spike mode. Polymorphic genes coding for elements of neurotransmission partially regulate tonic level by modulating the available amount of receptors, transporters and enzymes that contribute to background neuron firing. Notice that the DA and 5HT tones may be viewed as the decision maker's neurochemical status quo or reference point. Naturally, a higher tone would make the synaptic signalling system closer to being satiated and thus less sensitive to changes in neurotransmitter levels. This yields the following hypothesis which is illustrated in Figure 1.

Hypothesis V: *Higher DA (5HT) tone associates with a more concave (convex) valuation function over gains (losses).*

It follows that a decision maker with higher DA (5HT) tone will be more (less) risk averse for moderate prospects (hazards) than one with lower DA (5HT) tone.

These implications are tested in relation to reported findings in a number of published studies on the genetics of economic risk taking in the next section.

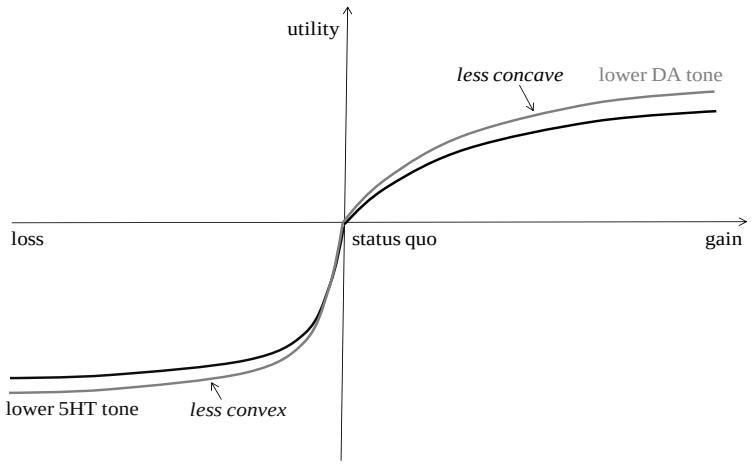


Figure 1. Neurochemical hypothesis on valuation. DA (5HT) tone modulates sensitivity towards incremental gain (loss); the higher the DA (5HT) tone, the lower (higher) the sensitivity towards incremental gain (loss).

In developing a neurochemical model of PT to account for the full fourfold pattern of risk attitude, we follow a suggestion in Trepel et al (2005) that changes in the probability weighting function may be related to the dopaminergic and serotoninergic systems. We posit a link between DA tone and 5HT tone and the π^+ and π^- functions in CPT. In this regard, it seems useful to identify simple parametric functional forms for the π function. Tversky and Kahneman (1992) offer the following one-parameter family of probability weighting function:

(1) $$p^c/[p^c+(1-p)^c]^{1/c},$$

which they estimated in conjunction with a power utility function and found to be initially concave and then convex beyond a crossing point. This finding was replicated in Camerer and Ho (1994). Wu and Gonzalez (1996) find that this inverse

S-shaped property is robust in a more refined study with multiple non-zero outcomes using additional parametric forms besides (1):

(2) $sp^c/[sp^c+(1-p)^c]$ (Lattimore, Baker, and Witte, 1992)

(3) $exp\{-[-\ln p]^a\}$ (Prelec, 1998)

In parallel, Rachlin et al (1991) offer a different intuition for the nonlinear response to probabilistic stimuli via the following special case of (2):

(4) $1/\{1+(1-p)/ps\}$.

They interpret s^{-1} as a hyperbolic bias factor in the probability-odds against oneself $1/p - 1$. Tversky and Kahneman (1992, p. 317) suggested that decision weights in CPT may be sensitive to the level of outcomes and referred to Camerer's (1992) finding that the degree of overweighting of small probabilities depends on the size of outcomes such that large outcomes engender greater curvature than smaller outcomes. Subsequently, Etchart-Vincent (2004) reports that people tend to be more pessimistic when facing large losses than small ones. Rottenstreich and Hsee (2002) interpret the *s* and *c* parameters in (2) as reflecting affect salience and echo the suggestion that they can depend on the underlying outcome *x*.

As an initial attempt at using neurochemistry to model the dependence of the probability weighting function on outcomes, we adopt (4) and posit that the affect salience parameter *s* can depend on *x*. The resulting probability weighting function coincides with that of the weighted utility model (see, e.g., Chew, 1983) which the utility of (x, p) is given by:

(5) $ps(x)v(x)/[ps(x)+1-p]$.

As with the probability weighting function, the salience function *s* does not have a role in the absence of risk. At the same time, when there is risk, *s* can in principle depend on additional contextual factors besides outcome. We discuss this further in the final section as part of our concluding remarks.

To account for the fourfold pattern of risk attitude, we posit a U-shaped salience function with a minimum at the status quo *0* such that the more salient the outcome, be it gain or loss, the higher the corresponding decision weight:

(6) $ps(y)/[ps(y)+1-p] > ps(x)v(x)/[ps(x)+1-p]$ whenever $s(y) > s(x)$.

Intuitively, it seems plausible that salience is minimized at the status quo and as such, the more sizable the outcome, the greater the saliency, and the higher is the overweighting of probability. This disposition to overweight more sizable gains (losses) may be interpreted as reflecting a sense of optimism (pessimism). In particular, $ps(x)v(x)/[ps(x)+1-p]$ approaches p when $s(x)$ is close to $s(0)$ and asymptotically approaches unity as $s(x)$ approach infinity. Consequently, the utility of receiving $x/2$ for sure exceeds the weighted utility of receiving $(x,1/2)$ as long as $[1 + s(0)/s(x)]^{-1} < v(x/2)/v(x)$. For a prospect (G, p), the derivative of its utility with respect to p equals $[s(G)/s(0)]v(G)$ at $p = 0$ whereas the corresponding initial slope of $u(pG)$ equals $u'(0)G$. As long as $v'(0)s(0)(G/s(G))$ is less than upper bound of v, the utility of (G, p) will exceed $v(pG)$ for p sufficiently small. By a similar reasoning, we can see that the decision maker can be risk tolerant for even-chance moderate hazards and concurrently risk averse for longshot hazards involving potentially sizable losses. In the proposition below, μ and λ refer to the upper and lower bounds of v.

Proposition A: Under a loss-averse utility function v and a U-shaped salience function s such that v(0) = 0 and s is minimized at 0, the decision maker exhibits risk aversion towards $(G,½)$ if $v(G/2)/v(G) > [1+s(0)/s(G)]^{-1}$, risk tolerance towards $(L,½)$ if $v(L/2)/v(L) < [1 + s(0)/s(L)]^{-1}$, risk tolerance towards (G, p) with p sufficiently small if $s(G)/G > v'(0)s(0)/\mu$ and risk aversion towards (L, q) with q sufficiently small if $s(L)/|L| > v'(0)s(0)/\lambda$.

For PT, notice that as long as $\pi(½) \leq ½$, $(G/2, 1)$ is always preferred to $(G, ½)$ and $(L, ½)$ is always preferred to $(L/2, 1)$. While neither is necessarily the case for the weighted utility decision maker, we can derive (as in Chew and Tan, 2005) the following sufficient conditions for $(G/2, 1)$ to always be preferred to $(G, ½)$ and $(L, ½)$ to always be preferred to $(L/2, 1)$ respectively:

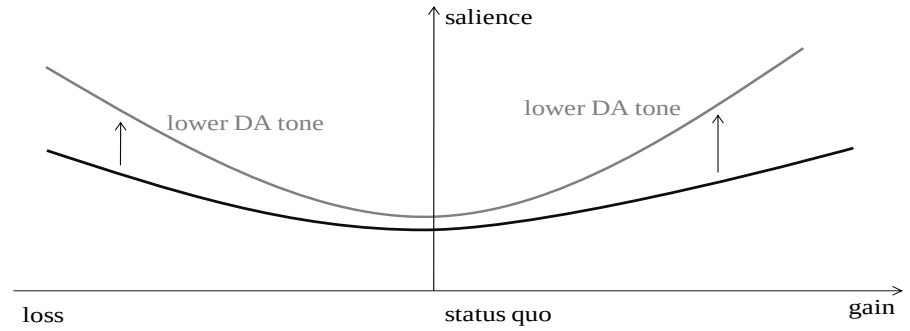
(7) $s'/s \leq -v''/v'$ over gains; $s'/s \geq v''/v'$ over losses

Consider two weighted utility decision makers represented respectively by (s^*, v^*) and (s, v). Say that s^* does not increase faster than s if s^*/s is a nonincreasing function. We rely on the following comparative risk aversion properties shown in Chew (1983).

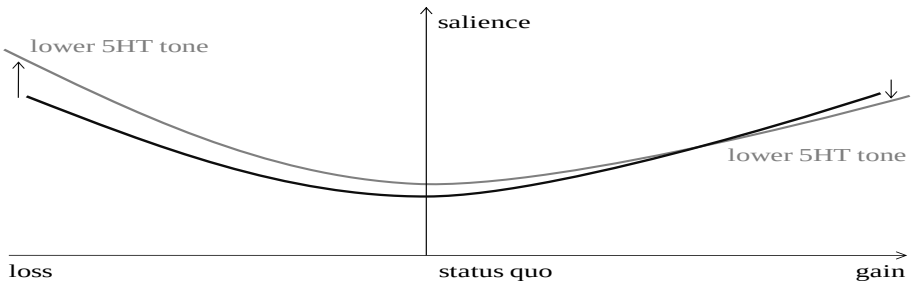
(8) (s^*, v^*) is at least as risk averse as (s, v) if v^* is at least as concave as v and s^* does not increase faster than s.

We state our hypothesis on saliency illustrated in Figures 2A and 2B below.

Hypothesis S: Lower DA tone engenders a salience function that increases faster over gains and decreases faster over losses relative to the case for higher DA tone. Lower 5HT tone engenders a salience function that decreases faster over losses as well as gains relative to the case for higher 5HT tone.



(A) Saliency of outcomes and DA tone



(B) Saliency of outcomes and 5HT tone

Figure 2: Neurochemical hypotheses on saliency. (A) illustrates how DA tone modulates saliency more over gain outcomes while (B) illustrates how lower 5HT tone engenders a salience function that decreases faster over gains as well as losses.

Based on hypotheses V and S, higher DA tone is associated with a more concave utility function and a salience function that increases slower over gains than the case for lower DA tone. Consequently, the decision maker will be more risk averse for moderate prospects as well as longshot prospects in which case the incidence of risk aversion for these two kinds of risks is expected to be positively correlated. A similar reasoning informs us that higher 5HT engenders a more convex utility function and a salience function that increases slower over losses. It follows that the decision maker will be less risk averse.

For longshot risks with a sizable outcome x, the decision maker's risk attitude is determined primarily by the saliency of the outcome x. From Hypothesis S, higher

DA tone induces a salience function that increases slower in the magnitude of the outcome. This leads to the implication, being less averse towards longshot hazards, besides being more averse to longshot prospects. With higher 5HT tone, the associated salience function increases slower with respect to the magnitude of loss. This yields a decision maker who is less averse towards longshot hazards as well as longshot prospects. Summarizing:

Proposition B: Relative to the case of low DA tone, a decision maker with high DA tone will tend to be

D(i) more averse towards moderate prospects.

D(ii) more averse towards longshot prospects.

D(iii) less averse towards longshot hazards.

Relative to case of low 5HT tone, a decision maker with high 5HT tone will tend to be

S(i) less averse towards moderate hazards.

S(ii) less averse towards longshot hazards.

S(iii) less averse towards longshot prospects.

In addition to relating risk attitudes in the fourfold pattern of risks with the DA and 5HT tones, this proposition has implications on within-subject correlation across pairs within the fourfold pattern of risk attitudes. We further develop an implication the correlation between risk attitudes towards MP and MH. While our model posits that DA and 5HT impact decision making under risk separately, there is evidence pointing to the idea that 5HT inhibits DA responses. Fink and Gothert (2007) provide evidence of such inhibition by 5-HT2c receptors, mediating a tonic inhibitory control on both the mesolimbic and the nigro-striatal dopaminergic pathways. Kapur and

Remington (1996) synthesized information from more than 100 published articles about the relationship between DA and 5HT and conclude that the 5HT system inhibits dopaminergic function at the level of the origin of the DA system in the midbrain as well as at the terminal dopaminergic fields in the forebrain. Since high 5HT tone can further inhibit DA tone, i.e., leading to low DA tone, it follows that subjects with high 5HT tone may be more risk tolerant towards MP besides being more risk tolerant over MH.

3. Empirical Validity based on Behavioral and Genetics Evidence

In this section, we discuss the empirical validity of our neurochemical model based on behavioral data from Zhong et al (2009b,c) and evidence from recently published findings on the neurogenetics of risk taking.

3.1. Behavioral Evidence

In our genetic studies of attitude towards four-fold risks (Zhong et al 2009b,c), we recruited a cohort of 350 Chinese subjects in Beijing through internet advertisement, posters and word of mouth to assess their risk attitude and genotype the three polymorphisms. We used the following choice tasks in our studies.

MP: Subjects choose between (*Y60, 1/2)* and *(Y30, 1)*. Those choosing (*Y60, 1/2)* were further asked to choose between the lottery and receiving a higher amount of *Y35* for sure; subjects choosing *Y30* for sure were further asked to choose between (*Y60, 1/2)* and receiving a lower amount of *Y25* for sure.

MH: Subjects choose between *(–Y10, 1/2)* and *(–Y5, 1)*. Those choosing *(–Y10, 1/2)* were further asked to choose between that lottery and losing *Y4* for sure; subjects choosing the sure loss were asked to choose between the lottery and losing *Y6* for sure.

LP: Subjects rank three options: *(Y200, 0.01)*, *(Y20, 0.1)*, and *(Y2, 1)* and are classified as exhibiting longshot preference if *(Y200, 0.01)* is most preferred and *(Y2, 1)* is least preferred.

LH: Subjects are classified as being disposed to insure if they prefer losing *Y2* for sure than losing *Y2,000* with *0.1%* chance.

We did not incentivize the LH task given the amount involved. The reader is referred to Zhong et al, (2009b, c) for more details on our experiments.

	MP	**LP**	**MH**
LP	*Positive: D(i) and D(ii)* 0.160**		
MH	*Positive*† 0.297***	*Positive: S(i) and S(iii)* 0.137*	
LH	*Negative: D(i) and D(iii)* – 0.070	*No implication* 0.034	*Positive: S(i) and S(ii)* 0.031

†5HT inhibiting DA implies positive correlation between MP and MH.

Table 1. Spearman correlation between different pairs of attitude towards fourfold risks. Each cell indicates direction of correlation according to Proposition B and estimated correlation with two-tails significance indicated by * for 5%, ** for 1%, and *** for 0.1%.

Table 1 above displays our data on the correlation of risk attitude between pairs of fourfold risk. They support our model's implications on these correlations except for LH and MH as well as LH and MP which are nominally in the implied directions though they are not significant. This may be due to the fact we did not incentivize the LH task involving a possible hypothetical loss of Y2,000. We also observe highly significant positive correlation between MP and MH as implied by our model regarding interaction between DA and 5HT.

3.2 Evidence from gene association studies

We further discuss the empirical validity of our model by examining a number of recently published association studies (see Table 2) linking specific genes with risk taking observed through incentivized choice. We begin with some introductory molecular genetics.

Study	N	Risk Attitude	Gene
Carpenter et al (2009)	140	Multiple-price list deign	DRD4
Crisan et al (2009)	36	Loss-gain framing	5-HTTLPR
Dreber et al (2009)	94	Portfolio choice	DRD4
Kuhnen & Chiao (2009)	65	Portfolio choice	5-HTTLPR, DRD4
Roe et al (2009)	67	Multiple-price list design	CHRNA4
Roiser et al (2009)	30	Loss-gain framing with fMRI	5-HTTLPR
Zhong et al (2009b)	325	Even-chance risks over gains and losses	Stin2, DAT1
Zhong et al (2009c)	325	Longshot risks over gains and losses	MAOA

Table 2. Summary of studies of molecular genetics of decision making under risk.

Basic Genetics

A gene is the basic unit of heredity in a living organism. Gene is an empirical construct which predates the molecular biology era. Its discovery was based on breeding experiments on plants (first by Gregor Mendel in 1866). At the beginning of the 20th century, Mendel's genes were identified with chromosomes. In 1944, the gene was further identified as DNA and is now known to be represented in sequences of four bases (A, G, C &T) arranged in a linear order, as shown by Watson and Crick in 1953. The DNA molecule has a double helix structure held together by complementary pairing of bases (A = T, G = C) providing an elegant mechanism for molecular replication as well as heritability. The human genome consists of more than

twenty thousand genes distributed on the 23 pairs of chromosomes. A human chromosome is a single DNA double helical molecule. Individuals inherit half of their DNA from each parent. Many genes have various forms, known as alleles representing variations in the sequence of the DNA bases. Every individual has two separate copies of an allele at each locus or location on the chromosome, but each sperm or egg cell contains only one of these alleles. Thus a child has a 50% chance of receiving a particular allele from a particular parent.

In all eukaryotic organisms, genes encode protein sequence in two major steps: the DNA is transcribed to messenger RNA (mRNA) in the cell nucleus; mRNA is translated into proteins in the cytoplasm. The process of producing a biologically functional molecule of mRNA (and culminating in a protein) is known as gene expression. Observable traits and behaviors of interest, referred to as phenotypes, are far downstream from the gene expression. While in some cases a single change in one letter of the DNA alphabet in a single gene alone can lead to a disease (such as Sickle Cell Anemia), the vast majority of phenotypes are complex traits, influenced by multiple genes as well as different environmental factors.

Overall evidence that genes play a role in our ability to understand and manipulate social relationships comes mainly from studying twins. The most common design compares monozygotic (MZ) and dizygotic (DZ) twins. MZ twins share all their genetic material, while DZ twins share approximately 50% of their genes. If we assume the environmental influences are the same for MZ and DZ twins for the phenotype of interest, then heritability is related to the difference in correlations between MZ twins and DZ twins. For details of the twin method, readers are referred

to Neale and Cardon (1992). Twin studies are informative regarding the percentage of variance explained by genes, but not about which specific genes or their number that contribute to the phenotype. For decades, genetic linkage combined with positional cloning has been the workhorse of human genetics producing remarkable success in identifying genes for rare Mendelian disorders (Risch, 2000). The recent completion of the Human Genome Project (HUGO) enables the use of the so-called SNP (single nucleotide polymorphism, a single change in one of the 4 DNA letters) map in testing association between phenotypes and genotype. Humans differ on the average every thousandth base pairs (e.g. A→G). This rich variation explains many differences in human behavioral traits. Another important source of variation in DNA are short-tandem repeat elements – regions of DNA that are variably repeated, e.g., $(GC)_n$. Finally, relatively large regions of DNA (>1kb) that are either duplicated or deleted, so-called copy number variations (CNV), are now recognized as a third source of variation perhaps rivaling that of SNPs in prominence.

Today, there are two general approaches in genetic research of complex traits. One strategy is Genome Wide association studies (GWAS). The power of GWAS is that it is not hypothesis driven and by default engages the entire genome in the analysis. SNP frequencies are compared across cohorts or quantitative phenotypes to ascertain chromosomal regions that partially explain some of the phenotypic variance. A second and also widely used approach is to make use of candidate genes that are known to regulate specific proteins of interest and/or influence related behaviors that make 'biological sense'.

Evidence from Gene Association Studies

In the nascent literature on the neurogenetics of risk taking, researchers have focused on genes that affect neurotransmitter synthesis and reception, hormone regulation and transcription factors. Here we discuss the candidate genes studied in several recently published papers and discuss the extent to which their results support the implications of our model.

Experiments involving moderate risks

DRD4: The dopamine D4 receptor exon 3 polymorphism (DRD4) has been studied in several recent papers on the genetics of risk attitude. The DRD4 exon 3 is characterized by a highly polymorphic VNTR containing a 48 bp repeat (Van Tol et al, 1992). This polymorphism is known for contributing to individual differences in traits including novelty seeking (Ebstein et al, 1996), ADHD (Faraone, Doyle, Mick, and Biederman, 2001), and substance abuse (Laucht, Becker, Blomeyer, and Schmidt, 2007). In terms of its role in regulating DA tone, the presence of the 7-repeat allele makes for a less effective receptor, hence low DA tone (Asghari et al, 1995). Consequently, our neurochemical model would predict that subjects with the 7-repeat allele are more risk tolerant over moderate prospects, i.e., implication D(i) of Proposition B. This is corroborated by the findings in two recent studies (Dreber et al, 2009; Kuhnen and Chiao, 2009). They show that subjects with the 7-repeat allele are more risk tolerant than subjects without the 7-repeat allele in a portfolio choice setting. Carpenter et al (2009) also shows marginal support for the previous two studies.

DAT1: The dopamine transporter DAT (SLC6A3) VNTR (Vandenbergh et al, 1992), located in 3'UTR, modulates gene expression and transporter density in vitro

(VanNess, Owens and Kilts, 2005), midbrain activation (Schott et al, 2006) and in vivo transporter availability (van Dyck et al, 2005), with the 9-repeat allele having more enhancer-like properties than the 10-repeat allele, resulting in low DA tone for the 9-repeat (van Dyck et al, 2005). Zhong et al (2009b) find that subjects with 9-repeat allele are more risk taking in even-chance prospects than those with 10-repeat allele. They do not find its association with risk attitude towards loss-oriented gamble. These findings support implication D(i) in Proposition B.

5-HTTLPR: The serotonin transporter gene (SLC6A4) is characterized by a 44 bp insertion/deletion (5-HTTLPR) in the promoter region (Canli and Lesch, 2007). The reuptake drugs, that inhibit the serotonin transporter, are known to be effective in increasing synaptic serotonin levels (Ansorge et al, 2004), suggesting that the short allele results in low 5HT tone. This notion is consistent with the association of the short allele with depression and anxiety representing clinical conditions that respond well to reuptake inhibitors. The implication S(i) of Proposition B predicts that subjects with the long allele will be more risk tolerant over losses. This is corroborated by the finding in Zhong et al (2009b) that subjects with the long allele (high 5HT tone) are nominally more risk tolerant over losses than subjects with the short allele. In addition, Crisan et al (2009) show that subjects with long allele of 5-HTTLPR choosing the gamble over the sure option more often than short allele in the loss frame, but not in the gain frame, which also supports the hypothesis that long allele would drive subjects to be more risk tolerant in the loss domain. Kuhnen and Chiao (2009) reported subjects with long allele (high 5HT tone) tend to be more risk tolerant than short allele in a portfolio choice setting. This observation may due to the

fact that 5HT inhibits DA responses as discussed above. As a consequence, high 5HT tone would lead to low DA tone, therefore subjects would be more risk taking in gain as D(i) in Proposition B.

STin2: The serotonin transporter gene (SLC6A4) is characterized by a second intronic (STin2) 17 bp variable number of tandem repeat (Lesch et al, 1994). It has been suggested that the VNTR, region may act as a transcriptional regulator of SLC6A4, with 12-repeat allele having stronger enhancer-like properties, hence lower 5HT tone, than 10-repeat allele (Hranilovic et al, 2004). STin2 is found to be significantly associated with risk attitude over losses, but not for risk attitude over gains (Zhong et al, 2009b). Subjects with the 10-repeat allele of STin2 (high 5HT tone) are more risk tolerant over losses than subjects with the 12-repeat allele. This supports proposition implication S(i) of Proposition B.

Experiments involving longshot risks

MAOA: Monoamine oxidase A (MAOA) metabolizes mainly 5HT as well as DA. MAOA is characterized by a promoter region VNTR (Deckert, 1999; Sabol, Hu, and Hamer, 1998). The long 4 allele is more transcriptionally active than the shorter 3, and carriers of this allele are less likely to develop antisocial problems when interacting with specific family environment (Caspi et al, 2002). There is no direct relationship between MAOA polymorphism and 5HT levels suggesting that relationships among gene, brain and behavior may be developmentally mediated (Fowler et al, 2007). Here we take the view that the 3-repeat allele would lead to low 5HT tone, based on the evidence that 3-repeat allele predicts higher amygdala activation during negative emotional arousal (Meyer-Lindenberg et al, 2006), similar

to what is observed about the correlation between the short allele of 5-HTTLPR (low 5HT tone) and amygdala activation in response to negative affect stimulus (Hariri et al, 2002).

Zhong et al (2009c) show that the 3 allele (low 5HT tone) are characterized by a preference for the longshot hazards. Since, as hypothesized, low 5HT tone has a higher saliency the loss domain, leading to the purchase of insurance. For longshot prospects, subjects with the 4 allele (high 5HT tone) tend to prefer purchasing lottery. Since as we hypothesize that high 5HT tone has higher saliency in the gain domain than low 5HT tone leading to the decision maker exhibiting longshot preference. These association results on longshot prospects and longshot hazards support our supports implications S(ii) and S(iii) of Proposition B.

4. **Discussion and Concluding Remarks**

The growth of neuroeconomics has built on insights from behavioral and experimental economics in expanding the scope of observable economics behavior beyond revealed choice (Camerer, Lowenstein, and Prelec, 2005; Rustichini, 2005). In parallel with studies identifying the neuroanatomical substrates of decision making (see, e.g., Kuhnen and Knutson, 2005; Preuschoff et al, 2006; Preuschoff et al, 2008; Seymour et al, 2007; Tom et al, 2007; Yacubian et al, 2006), neuroeconomists have begun building theoretical models to account for the expanded range of observable behavior (Bernheim and Rangel, 2004; Brocas and Carrillo, 2008, 2009; Caplin and Dean, 2008; Glimcher, Dorris, and Bayer, 2005). Notably, Caplin et al (2010) report an fMRI study testing the implication of the Caplin-Dean axiomatization of the DA

reward prediction error function. Contributing to the latter direction, this paper offers a model of decision making under risk by linking by positing a role for DA and 5HT tone in mediating valuation and saliency of outcome stimuli. This yields a neurochemical extension of prospect theory which delivers a loss-averse utility function and a saliency-based probability weighting function in modeling decision making for simple risks with at most a single non-zero outcome. We validate our model empirically through evidence from both within-subject correlations of attitude towards fourfold risks and recently published findings on the molecular genetics of risk taking. In subsequent research, we will further develop our model to encompass a richer domain of lotteries and account for other 'anomalies' of decision making under risks, including Allais type behavior, as well as to design additional tests involving behavioral, gene association, and imaging experiments.

Another direction of extension concerns an increasingly recognized view that decision making under uncertainty does not depend only on probabilities, but also on how uncertainty itself arises. People tend to display ambiguity aversion in preferring to bet on events with known probabilities rather than those for which probabilities are not known (Ellsberg, 1961). This led to the development by Gilboa and Schmeidler (1989) of maxmin expected utility with non-unique priors and its extension in Ghirardato, Maccheroni and Marinacci (2004). Following Keynes (1921), Fox and Tversky (1995) observe that people also tend to bet on uncertainty arising from a source for which they have more knowledge or are familiar with rather than from an unfamiliar source. Moreover, Tversky and Kahneman (1992) state, "The presence of systematic preferences for some sources of uncertainty calls for different weighting

functions for different domains, and suggests that some of these (probability weighting) functions lie entirely above others." This has been studied empirically in Abdellaoui et al (2010). Axiomatic models that can accommodate having a preference over sources of uncertainty include Klibanoff, Marinacci, and Mukerji (2005), Nau (2006), Chew and Sagi (2008), and Ergin and Gul (2009). Applying the Chew-Sagi approach, we can let the salience function in our model depend on the source of uncertainty. To display ambiguity aversion (familiarity bias), the salience function for a known (familiar) source of uncertainty would need to increase faster than the salience function for an unknown (unfamiliar) source. This yields an extension of the model in the current paper to incorporate having preference over distinct sources of uncertainty.

Recent twin studies suggest the importance of both genetic and environmental factors for decision making under risk (Cesarini et al, 2009a; Zhong et al, 2009a). Extensive evidence reveals the importance of environmental factors in determining attitudes towards economic risk, including developmental factors, aging, education, and life experiences (see, e.g., Dohmen et al, 2006; Harrison et al, 2007; Malmendier and Nagel, 2009). At the same time, it is known (see review in Abdolmaleky et al, 2008) that environmental factors across one's lifespan can influence gene expression, specifically genes in the dopaminergic system, so that genes of interest to economic risk taking are potentially methylated. From this perspective, it seems valuable to incorporate epigenetics in future research in modeling risk taking at the neurogenetic level as well as its experimental testing combing the methodologies of neurogenetics and experimental economics.

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