Molecular neuroeconomics of crime and punishment: implications for neurolaw

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Abstract

Criminal behaviors have been associated with risk, time and social preferences in economics (Becker 1968; Davis 1988), criminology (Chamlin & Cochran 1997), and neurolaw (Goodenough & Tucker 2010). This study proposes a molecular neuroeconomic framework for the investigation into crime and punishment. Neuroeconomic parameters (e.g., risk-attitude, probability weighting, time discounting in intertemporal choice, loss aversion, and social discounting) are predicted to be related to criminal behavior. Neurobiological and neuroendocrinological substrates such as serotonin, dopamine, norepinephrine, cortisol (a stress hormone), sex hormones (e.g., testosterone), and oxytocin in brain regions such as the orbitofrontal cortex, the amygdala, and the cingulate may be related to the neuroeconomic parameters governing criminal behaviors. The present framework may help us develop "neurolaw" based on molecular neuroeconomics of criminal and antisocial decision-making processes.

1. INTRODUCTION

Studies in economics and criminology have suggested that economic parameters such as risk attitude, time-discount rate, and altruism may determine the risk of criminal behavior. However, these theoretical considerations have been largely ignored in cognitive neuroscience of antisocial behavior. Past decades have witnessed that utilization of economic theory in other disciplines such as psychiatry, sociology, political science, behavioral ecology, and neuroscience is considerably useful. Therefore, introducing neuroeconomic frameworks is important for a better understanding of criminal behavior and criminals' sensitivity to punishment. Recent neurobiological studies on antisocial behavior demonstrated that several neurobiological substrates such as neurotransmitters (e.g., serotonin, dopamine, and norepinephrine) and hormones (e.g., cortisol and testosterone) in the brain regions such as the orbitofrontal cortex and the limbic structures modulate antisocial behavior. Therefore, combining neuroeconomic theory with these neurobiological finding is helpful for the establishment of molecular neurobiological theory of criminal behavior ("molecular neuroeconomics" of crime and punishment), which may finally contribute to neurolaw (Goodenough & Tucker 2010).

This paper is organized in the following manner. In Section 2, I introduce neuroeconomic theory of risky, impulsive, and antisocial behavior. Also, implications from economics and criminology are introduced. In Section 3, findings in neurobiology regarding the molecular mechanisms of antisocial/criminal behavior are briefly

reviewed. In Section 4, I proposed several predictions from molecular neuroeconomic theory of crime and punishment. Future study directions by utilizing the present molecular neuroeconomic theory of crime and punishment, and how to develop the emerging field of "molecular neurolaw" are also discussed.

2. NEUROECONOMIC THEORY OF RISKY, IMPULSIVE AND ANTISOCIAL DECISION MAKING

Economist Gary Becker proposed his economic theory of crime and punishment after his dissertation defense. In the morning of his dissertation defense, he had to weigh the cost and benefits of legally parking in an inconvenient garage versus illegally parking in a convenient place. After roughly calculating the probability of getting caught and potential punishment and being late for the dissertation defence, Becker rationally opted for the crime (i.e., illegal parking). As can be seen from this example, investigation into decision under risk (probably more irrational in most criminals than Becker's decision) is critical for developing molecular neuroeconomics of crime and punishment. In behavioral economics, in order to explain anomalies in human decision making under risk (e.g., Allais paradox 1953), the prospect theory has been proposed (Kahneman & Tversky 1979) and introduced in recent studies in neuroeconomics, in addition to system dynamics (http://www.systemdynamics.org). In Kahneman-Tversky's prospect theory (Kahneman & Tversky 1979), a subjective value of an uncertain outcome x (x is either positive or negative, gain or loss), which is received at the probability of p, is *v(x) w(p)*, where *v(x)* is a value function for either gain and loss, and *w(p)* is a probability weighting function. Therefore, the prospect theory is a generalization of an expected utility theory in which *w(p)*=p (von Neumann & Morgenstern 1944). Furthermore, if the outcome is delayed, subjective value of the outcome is temporally-discounted. This temporal discounting is mathematically represented with a time-discount function D(t). Then, the subjective value of the potentially uncertain and delayed outcome of the magnitude of x, which can be obtained at probability p and delay t, is *v(x) w(p) D(t)*. Moreover, if the outcome is received by another person at social distance N, the subjective value of the outcome (for a decision-maker herself) is socially-discounted, following the social discount function S(N). Together, the subjective value of a potentially uncertain, delayed, and social outcome is *V(x,p,t,N)=v(x) w(p) D(t) S(N)*. The each functional component in *V(x,p,t,N)* is explained below.

Regarding the functional form of the value function *v(x)*, prospect theory's value function is assumed to be concave for gains, convex for losses, and steeper for losses than for gains. The most popular parametrization of the value function is a power function (Tversky & Kahneman 1992):

$$v(x) = \begin{cases} x^{\alpha} & (x \geq 0) \\ -\lambda(-x)^{\beta} & (x < 0) \end{cases} \quad (1)$$

where α, β > 0 measure the curvature of the value function for gains and losses, and λ is the coefficient of loss aversion (see Figure 1). A recent neuroeconomic study demonstrated that amygdala damage reduced loss aversion (De Martino *et al.* 2010). The probability weighting function has been parameterized as (Prelec 1998; Takahashi 2011):

$$w(p) = \exp[-(-\ln p)^s] \quad (2)$$

where s indicates a distortion in subjective probability (note that s=1 corresponds to linear probability weighting in the expected utility theory, see Figure 2), which has been shown to associate with the anterior cingulate activity (Paulus & Frank 2006) and psychophysical effect of waiting time in repeated gambles (Takahashi 2011a). It is to be noted that subjects with a less concave value function for gain (i.e., larger α) and distorted probability weighting function (i.e., over-weighting of small probabilities) are more risk-taking in uncertain gain.

In order to describe impulsivity and irrationality (time-inconsistency) in temporal discounting, the q-exponential time-discount model for delayed rewards has been studied (Cajueiro 2006; Takahashi 2007; *et al.* 2007a; Takahashi *et al.* 2008ab; Takahashi 2009; Takahashi 2011b):

$$D_{q+}(t) = D_{q+}(0)/\exp_{q+}(k_{q+}t) = D_{q+}(0)/[1+(1-q+)k_{q+}t]^{1/(1-q+)} \quad (3)$$

where $D_{q+}(t)$ is the discount factor for a reward obtained at delay t, q+ is a parameter indicating irrationality in temporal discounting for gain (smaller q+<1 values correspond to more irrational discounting for delayed gains), and k_{q+} is a parameter of impulsivity regarding the reward at delay *t*=0 (i.e., q-exponential discount rate:= $-D_{q+}'(t)/D_{q+}(t)$ at delay t=0). Note that when *q+*=0, equation 3 is the same as a hyperbolic discount function, while *q+→1*, is the same as an exponential discount function (Cajueiro 2006; Takahashi 2009). The shape of the discount function is shown in Figure 3. It is to be noted that steeper temporal discounting indicates more impulsive decision over time. Kable and Glimcher (2007) reported that *v(x) D(t)* is represented as neural activities in brain regions such as the orbitofrontal cotex and the striatum. Furthermore, it is known that delayed gains and losses are distinctly processed in the brain and loss is less steeply temporally-discounted than gains ("sign effect", Xu *et al.* 2009) due to a difference in time perception in waiting gain and loss (Han & Takahashi 2012). Therefore, we should prepare the q-exponential discount function for delayed loss:

$$D_{q-}(t) = D_{q-}(0)/\exp_{q-}(k_{q-}t) = D_{q-}(0)/[1+(1-q-)k_{q-}t]^{1/(1-q-)} \quad (4)$$
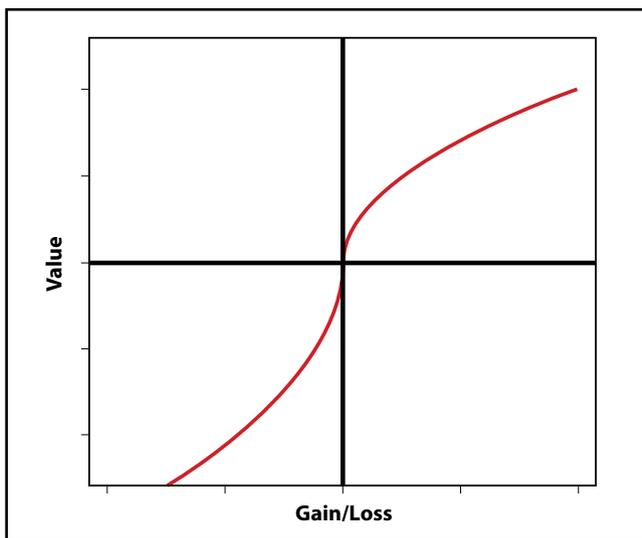
Fig. 1. Value function of Prospect theory. Value function is concave (risk aversion) in gain and convex (risk preference) in loss. Also, value function is more steep in loss.
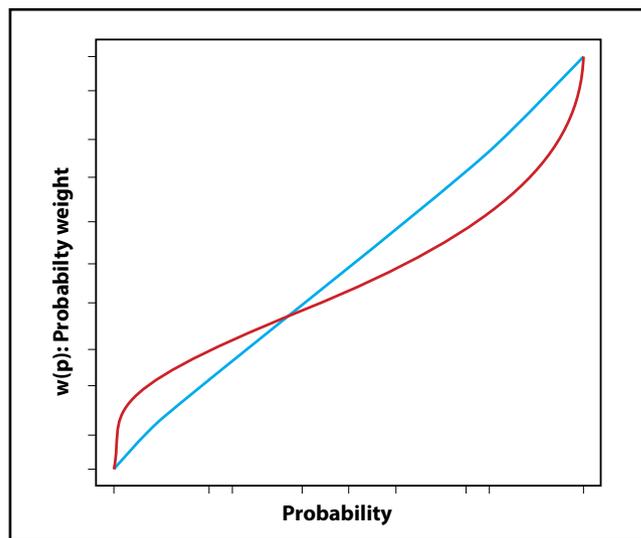
Fig. 2. Probability weighting function. The horizontal axis indicates (objective) probability of winning the outcome. The vertical axis indicates probability weight (subjective probability). The red curve indicates more exaggerated tendency of overweighing small probability and underweighing large probability in decision under risk than the blue curve.

where $D_{q-}(t) > 0$ is the discount factor for a loss at delay t, $q-$ is a parameter indicating irrationality in temporal discounting for loss (smaller $q-<1$ values correspond to more irrational discounting for delayed losses), and k_{q-} is a parameter of impulsivity regarding the loss (i.e., degree of procrastination) at delay $t=0$.

In order to describe antisocial (selfish) decision-making in social decisions on social gain, the following q-exponential social discount function has been proposed (Takahashi 2010):

$$S_{q+}(N)=S_{q+}(0)/\exp_{qs+}(k_{qs+}N)=S_{q+}(0)/[1+(1-q_s+)k_{qs+}N]^{1/(1-q_s+)} \quad (5)$$

where $S_{q+}(N)$ is a social discount factor for a social reward which another person at social distance N receives, k_{qs+} is a social discount rate at social distance N, q_s+ indicates the deviation from exponential social discounting ($q_s+=0$, equation 5 is the same as a hyperbolic discount function, while $q_s+\to1$, is the same as an exponential discount function, see Takahashi 2010). Because loss may be socially-discounted in a distinct manner from gain, we should prepare a social discount function for loss:

$$S_{q-}(N)=S_{q-}(0)/\exp_{qs-}(k_{qs-}N)=S_{q-}(0)/[1+(1-q_s-)k_{qs-}N]^{1/(1-q_s-)} \quad (6)$$

with similar notations to equation 5.

Taken together, it can be said that: (i) risky decision-making is parameterized by α, β, and s, (ii) aversion to loss is parameterized by λ, (iii) impulsive and time-inconsistent decision-making is parameterized by $k_{q+/-}$ and $q+/-$, and (iv) antisocial decision-making is parameterized by $k_{qs+/-}$ and $q_s+/-$. Therefore, prob-
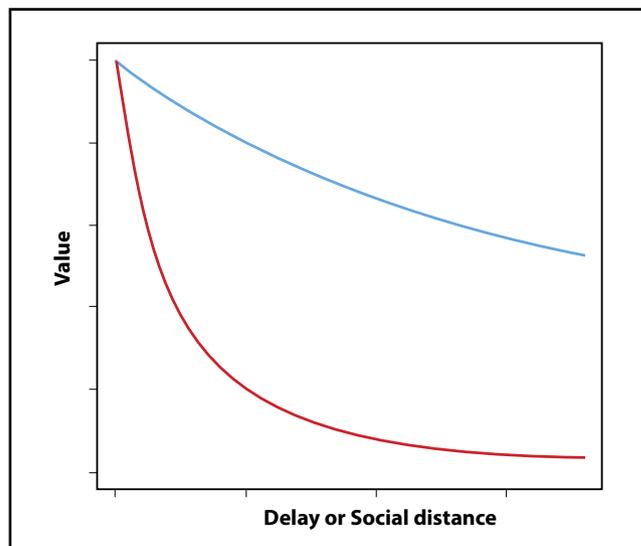
Fig. 3. Delay of Social discount functions (q-exponential functions). The horizontal axis indicates delay until receipt or social distance of receiver from donor. The vertical axis indicates subjective value. Note that the red curve indicates more impulsive (selfish) discounting than the blue curve.

lematic behaviors, potentially associated with criminal behaviors, in both social and non-social domains can be captured by a relatively small number of these neuroeconomic parameters.

Regarding risky decision-making, in Becker's economic theory of crime and punishment (Becker 1968) based on the expected utility theory (von Neumann & Morgenstern 1944), it was hypothesized that criminals may be more risk-taking at least in the realm of punish-

ment. Because recent studies in behavioral economics and neuroeconomics suggest that the prospect theory can capture several important anomalies in decision under risk, better than the expected utility theory, neuroeconomic studies of crime and punishment should employ equation 1 and 2, for analyzing problematic behavior by criminals in decision under risk. Pachur and colleagues (2010) demonstrated, by utilizing the prospect theory, that prisoners were more risk seeking than nonprisoners in lotteries involving losses, but prisoners were less risk seeking in lotteries involving high-probability gains, prisoners had stronger loss aversion than nonprisoners, and prisoners showed a diminished sensitivity to the probability of gains. This study further supports the advantage of the utilization of neuroeconomic theory of decision under risk.

Impulsive decision over time has also been associated with criminal behavior in economic theory. An economist Davis (1988) proposed an economic model of criminal behavior which incorporates temporal discounting. Davis' theory predicts that agents with higher time-discount rates (k parameters in equation 3 and 4) will be likely to commit crime. However, by utilizing a simple hyperbolic time-discounting function (i.e., q+ is fixed at 0 in equation 3), Wilson and Daly (2006) reported that young offenders were not significantly different from the control students in time-discount rates. Therefore, more sophisticated temporal discounting models (e.g., the q-exponential time-discount models, equations 3 and 4) should be adopted in future neuroeconomic studies on the effect of temporal discounting on criminal behavior. Concerning illegal substance use, Becker and Murphy's economic theory of addiction (Becker & Murphy 1988) predicts a positive association between drug addiction and temporal discounting. Subsequently, behavioral and neuroeconomic studies confirmed this prediction (Bickel & Marsch 2001; Ohmura *et al.* 2005; Takahashi *et al.* 2007c; Takahashi 2009; Takahashi *et al.* 2009). Interestingly, rationality in addicts may be associated with nonlinearity of their future temporal cognition (Takahashi 2011b). Therefore, neuroeconomic theory of intertemporal choice may also be useful in future studies on illegal drug use.

With respect to the relationship between crime and social preferences, criminologists Chamlin and Cochran (1997) reported that the cultural value of altruism is inversely related to property and violent crime rates. Neurocognitive studies also implied that psychopathy, which is characterized by a constellation of antisocial behavioral traits, may be associated with altered economic decision-making (Koenigs *et al.* 2010). However, to date, no study utilized the social discounting functions (equation 5 and 6) to examine the relationship between criminal behavior and social preferences. Therefore, future neuroeconomic studies on crime and punishment should investigate parameters in the social discounting functions in criminals.

3 NEUROBIOLOGICAL SUBSTRATES OF RISKY, IMPULSIVE, AND ANTISOCIAL BEHAVIOR

3.1 Brain regions related to criminal behavior

Abnormalities in brain regions such as the orbitofrontal cortex (Laakso *et al.* 2002; Anderson & Kiehl 2012), the amygdala (Blair 2005; Blair 2010), the cingulate cortex (Kiehl *et al.* 2001) have been associated with antisocial behavior and psychopathy. For instance, Blair and colleagues demonstrated that amygdala-orbitofrontal cortex connectivity is reduced during moral judgment in psychopaths (Marsh *et al.* 2011), and psychopathic subjects have a reduction in amygdala and orbitofrontal cortex responses to emotionally provocative stimuli or during emotional learning (Blair 2010). These brain regions have also been associated with economic decision-making. For instance, the orbitofrontal cortex represents subjective value of a delayed reward during intertemporal choice (Kable & Glimcher 2007), the amygdala is related to loss aversion (De Martino *et al.* 2010), and the cingulated is associated with decision under uncertainty (Paulus & Frank 2006; Goñi *et al.* 2010). Therefore, future neuroeconomic studies on crime and punishment should examine the roles of these brain regions by employing the neuroeconomic theory.

3.2 Neurotransmitters related to criminal behavior

Several neuroeconomic studies (Berns *et al.* 2007; Takahashi 2008; Zhong *et al.* 2009) proposed that serotonin and dopamine affect the curvature of the value function in Kahneman-Tversky's prospect theory (equation 1); i.e., risk aversion and loss aversion. Furthermore, both serotonin and dopamine regulate temporal discounting (Takahashi 2009). A reduction in serotonergic functioning was reportedly related to impulsive temporal and social decision-making (Crockett *et al.* 2010). Soderstrom *et al.* (2001) state that serotonin and dopamine distinctly contribute to psychopathy. A recent study found that norepinephrine (noradrenaline) is associated with aggression in prisoners (Chichinadze *et al.* 2010). We have reported that noradrenergic activity is related to temporal discounting (Takahashi *et al.* 2007b; Takahashi *et al.* 2010). Additionally, because risk and time preferences, and loss aversion are predicted to associate with criminal behavior (Becker 1968; Davis 1988; Pachur *et al.* 2010), involvement of serotonergic, noradrenergic, and dopaminergic systems in criminal behavior should more extensively be studied by employing neuroeconomic frameworks in future studies.

3.3 Neuroendocrine modulation of criminal and antisocial behavior

Dysregulation of serotonin in the brain may contribute to the low cortisol (a stress hormone produced in response to the activation of hypothalamic-pituitary-adrenal (HPA) axis) levels (Sobczak *et al.* 2002; Cima

et al. 2008) observed in psychopathy, resulting in a reduced sensitivity to punishment (van Honk *et al.* 2003). Our neuroeconomic studies demonstrated that stress hormones (cortisol and cortisone) modulate temporal discounting (Takahashi 2004; Takahashi *et al.* 2010). Testosterone is a product of the hypothalamic-pituitary-gonadal (HPG) axis and is associated with approach-related behavior, reward sensitivity, and fear reduction (Boissy & Bouissou 1994). Neuroeconomic studies revealed that testosterone is associated with risky decision-making (Goudriaan *et al.* 2010) and antisocial behavior (van Honk & Schutter 2007). In males, testosterone is also nonlinearly associated with temporal discounting (Takahashi *et al.* 2006). Increased testosterone-to-cortisol ratio may be related to psychopathy (van Honk *et al.* 2006; Glenn *et al.* 2011). Chichinadze *et al.* (2010) reported that testosterone is related to aggression in prisoners. Therefore future studies should investigate how these steroid hormones collectively modulate neuroeconomic parameters, resulting in an increased risk of criminal behavior and a decrease in sensitivity to punishment. With respect to social decision, oxytocin has been shown to increase generosity in economic games (Zak *et al.* 2007) but also increase antisocial emotions such as envy (Shamay-Tsoory *et al.* 2009). Therefore, how oxytocin modulates social discount functions should be examined in future studies.

4. IMPLICATIONS FOR NEUROECONOMICS AND NEUROLAW OF CRIME AND PUNISHMENT

This is the first study to propose a possible unified framework for molecular neuroeconomic theory of crime and punishment. Neurobiological substrates such as serotonin, dopamine, norepinephrine, as well as neuroactive hormones may modulate neuroeconomic parameters determining risk, time, and social preferences, which conceivably control the risk of criminal behavior and sensitivity to punishment.

Regarding the extremely severe legal punishment (i.e., capital punishment), what we call "Becker's paradox" is known (Persson *et al.* 2007): Although capital punishment is optimal in Becker's economic theory of crime and punishment (1968), it is rarely observed in the real world, nor effective is capital punishment than thought from Becker's theory of crime and punishment. Although Kahneman-Tversky's prospect theory extends von Neumann-Morgenstern's expected utility theory, the prospect theory cannot readily solve this paradox, because, in prospect theory, small probabilities are assumed to be overweighed. Therefore, criminals following the prospect theory may strongly be afraid of capital punishment even when the probability of the capital punishment is small. In order to solve this paradox, novel non-expected utility theories which further extend Kahneman-Tversky's prospect theory may

be necessary (Dhami & al-Nowaihi 2012). Therefore, future neuroeconomic studies on crime and punishment should develop novel models of decision under risk.

Future studies in molecular neuroeconomics of crime and punishment should employ animal models such as transgenic mice, for a detailed analysis of molecular mechanisms determining the neuroeconomic parameters in the equations above. By utilizing the present neuroeconomic framework, future studies may help establish the discipline of "neurolaw" (Goodenough & Tucker 2010) at the molecular and cellular levels (i.e., "molecular neurolaw"). This approach may lead us to better biomedical treatments for antisocial behavior and conduct disorders. In terms of medical treatment of criminals, structural nature of impairment in adult psychopaths' brains make the disorder incurable after full development, the only time window for intervention is in childhood where reliable diagnostic tools for psychopathy traits are needed. The present theoretical frameworks may be useful for the development of the diagnostic tools.

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