other measures of empathy and social competence (Davis, 1983). Therefore, higher PD scores might have to be interpreted as pathological.

MRI acquisition and pre-processing

The images were analyzed using Statistical Parametric Mapping software (SPM5; Wellcome Department of Imaging Neuroscience, London, UK) running in Matlab 2007b (MathWorks, Natick, MA, USA). We used an extension of SPM, the VBM5.1 toolbox written by Gaser (http://dbm.neuro.uni-jena.de/vbm). Images were normalized and segmented in gray and white matter partitions in the new unified segmentation step (Ashburner & Friston, 2005). In SPM5, prior probability maps that are relevant to tissue segmentation are warped to the individual brains, making the creation of a customized template (known as optimized VBM) unnecessary. The images were resliced with $1 \times 1 \times 1$ mm^3 voxels. Images were multiplied (modulated) by the Jacobian determinants for non-linear warping only, from the normalization step to preserve volume information. The modulated gray matter images were smoothed with a Gaussian kernel of 12-mm full width at half maximum, on which all analyses were performed.

We used an extension of statistical parametric mapping 5 (SPM5; Wellcome Department of Imaging Neuroscience, London, UK), specifically, the VBM5.1 toolbox written by Gaser (http://dbm.neuro.uni-jena.de/vbm) running in Matlab 2007b (MathWorks, Natick, MA, USA) for the analysis. Images were normalized and segmented into gray and white matter partitions in the new unified segmentation step (Ashburner et al., 2005). In SPM5, prior probability maps that are relevant to tissue segmentation are warped to the individual brains, making the creation of a customized template (known as optimized VBM) unnecessary. The images were resliced with $1 \times 1 \times 1$ mm^3 voxels. Images were multiplied (modulated) by the Jacobian determinants for non-linear warping only, from the normalization step to preserve volume information. The modulated gray matter images were smoothed with a Gaussian kernel of 12-mm full width at half maximum, on which all analyses were performed.

Data analyses

Clinical, sociodemographic and neuropsychological data

Correlation analysis

To identify the brain region of gray matter, wherein the subjects showed volume reduction correlated with LSRP scores, a multiple regression was undertaken in SPM5. Age and gender and intracranial volume were included as covariates. in the analysis. Output was in the form of statistical parametric maps (SPMs). First, the main effect of the LSRP was tested using the total score (sum of primary psychopathy, and secondary psychopathy) in a [-1] t-contrast, assuming that an increase in LSRP total scores would be associated with decreased gray matter volumes in this patient population. In order to investigate how each of the two psychopathy subscale scores (primary, and secondary psychopathy) are related with brain volume reduction, we looked at the separate effects of primary, and secondary psychopathy using two different design matrices and performing a [-1] t-contrast with additonal zero for covariates of no interest. Based on the previous studies that we mentioned previously, we expected gray matter changes in the following regions: amygdala and orbitofrontal cortex (OFC). For these regions with an a priori hypothesis, significance was assumed at P= 0.001 (uncorrected), and an additional extent threshold of 100 voxels to suppress small clusters possibly arising by chance. For other gray matter regions, no a priori hypothesis could be established. Therefore, significance was assumed only at P = 0.01, corrected for multiple comparisons at cluster level (P= 0.001, uncorrected), and an extent threshold of 100 continguous voxels.
The locations of voxels were expressed in Talairach coordinates (Talairach and Tournoux, 1988), using the nonlinear transformation procedure developed by Brett (mni2tal: http://www.mrc-cbu.cam.ac.uk/Imaging/Common/mnispace.shtml). We included age and gender as a covariate.

Correlation analysis between significant clusters and IRI.

For each cluster of GM reduction detected by the above-mentioned procedures, we extracted the first eigenvariate which explains most of the volumetric variability in the data the by using the "eigenvariate" function (spm_regions.m) in SPM5. We then used this eigenvariate, derived from the cluster of interest to investigate the relationship between patients' GM data in regions of reduction and their IRI scores in SPSS 12.0.

From each of the clusters where we found significant correlations between cortical volumes and LSRP scores in the above-mentioned correlation analysis, the first eigenvariate that explained most of the volumetric variability of the cluster was extracted. Subsequently, we performed partial correlation analysis between these eigenvariates and IRI. Partial correlations were considered significant at $p<0.05$

(two-tailed).

Result

Demographic information is shown in table 2.

Neuroimaging results

Correlation with LSRP scores

LSRP total scores were correlated with right precuneus (Prc) volume reduction. LSRP primary psychopathy scores correlated with volume reduction of bilateral Prc, right amygdala, and right OFC. Secondary psychopathy scores showed no correlation with brain volume decrease (Figure 1).

Assosiation with IRI scores

Within the above-mentioned significant clusters, right amygdala showed moderate positive correlation with IRI EC scores . Right Prc was moderately positively correlated with IRI total scores (Figure 2). The correlation was still significant after we controlled for age and gender.

Discussion

This is the first study to investigate the correlation of psychopathic traits and brain volume reduction among healthy community samples using VBM. Our results reconfirmed the results of former neuroimaging study by showing abnormality of amygdala and OFC is correlated with psychopathic traits (LSRP primary psychopathy). That is, the more the brain volume of OFC and amygdala decrease, the stronger the psychopathic trait is. And the correlation of the amygdala volume change with IRI EC scores shows that amygdala may be one of the core structure of emotional dysfunction seen in psychopathy. Furthurmore, IRI EC scores ,which is known to show emotional aspects of empathy (Davis, 1980), were positively correlated with the volume change of amygdala. This supports our VBM result that amygdala volume decrease is correlated with the LSRP primary psychopathy score, which is the emotional aspect of psychopathy.

Moreover, precuneus volume decrease showed association with psychopathic traits (LSRP total score and primary psychopathy). Furthermore, only the volume change of right precuneus showed correlation with empathic ability. Precuneus, especially right,

is known to play an important role in perspective taking(Ruby, 2001). One of the important role of Precuneus is to assign first-person perspective (the viewpoint of the observing self) and interpreting an action as being controlled by oneself versus another person (Cavana and Trimble, 2006), which is manipulation of perceived agency. Agency is a crucial aspect for successfully navigating shared representations between self and other (Decety and Lamm, 2006). And this function is also supported by right inferior parietal cortex and medial prefrontal cortex (.Vogeley and Fink, 2003). Using transcranial magnetic stimulation (TMS), Lou et al. (2004) has shown that disruption of medial parietal region circuitry would decrease the efficiency of retrieval of previous judgment of mental self compared with that of others. Furthurmore, lesion to the right parietal lobe (either superior or inferior) is known to damage both cognitive and affective empathy (Shamay-Tsoory, 2004). Anatomically, precuneus has neural connections with anterior cingulated cortex, ventromedial frontal cortex and striatum (which is closely connected with amygdala) (Cavana and Trimble, 2006). These areas are known to play an important role for empathic abilities (Decety and Lamm, 2006). Former VBM study of psychopathy (Tihonen et al., 2008) also shows volume decrease in nearby parietal areas, but explains the volume decrease by mirror neuron system abnormality. We suggest that the volume decrease of precuneus may lead to the disability of controlling agency, which results in unsuccesful empathy. This may explain the association of right precuneus volume and IRI total score.

Disinhibition is reported to occur after OFC injury (Damasio et al., 1994). LSRP primary psychopathy score is known to correlate not only with PCL-R factor1 but also factor 2 (antisocial action), which includes disinhibition (Brinkley et al., 2001). In our study, brain volume of OFC which correlates with LSRP primary psychopathy score, showed no correlation with IRI. This may suggest that OFC is related with the disinhibitive aspect of psychopathy.

As a whole, our result may support the idea that psychopathic trait among healthy community samples partly share the same neural basis with the clinical psychopaths. a But the difference between our result and former VBM studies (Müller et al., 2008; Oliveira-Souza et al., 2008: Tihonen et al., 2008) may be explained by the confounding variables we pointed in the introduction, or the demographic differences. The former studies compared clinical psychopaths and healthy comparisons, while our subjects were healthy community samples which should show less antisocial actions. This may also explain why the secondary psychopathy scale showed no correlation with brain volume change. Still, these scores can correlate with brain activity even in healthy

subjects(Rilling et al., 2007).

Blair (2006) suggests that amygdala and OFC pathology are the central features of psychopathy, but the origins of OFC pathology is unclear. Our result suggests that the OFC pathology is unlikely to be the effect of alcohol or drugs. Still, the precise origin or the intercorrelation of these pathological brain regions are unclear. Our result is basically in line with the Blair's idea of neural basis of psychopathy. But additionaly suggests that the emotional information itself may be distorted before entering amygdala, through the abnormal agency control ability caused by precuneus structural abnormality.

One of our limitation is the relatively small number of subjects, and further study including the successful (unincarcerated) psychopaths as subjects, is needed to investigate the relationship of brain structures (especially precuneus) and psychopathic trait.

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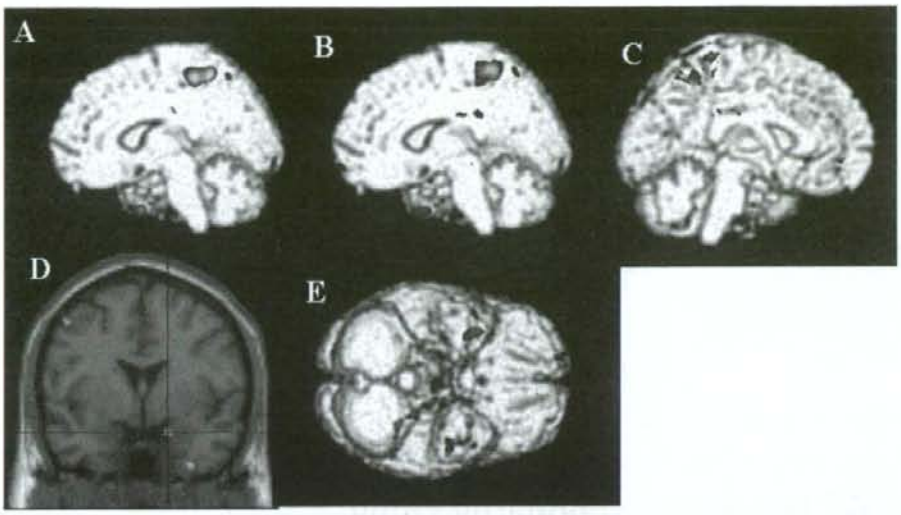
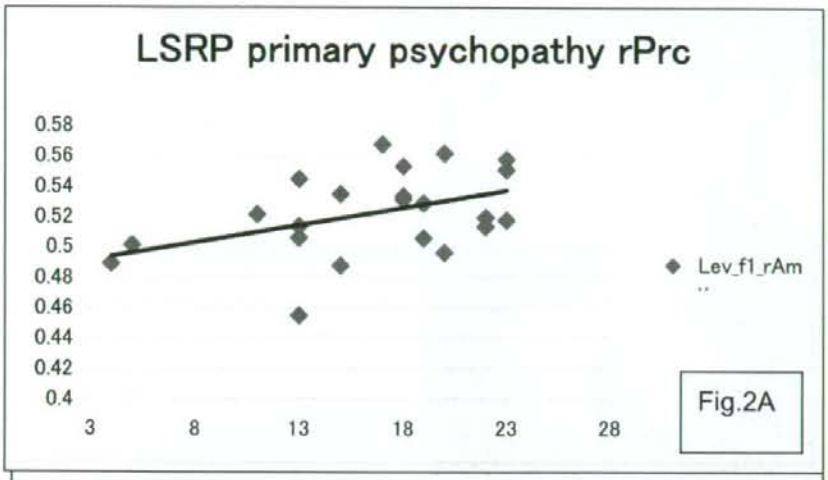
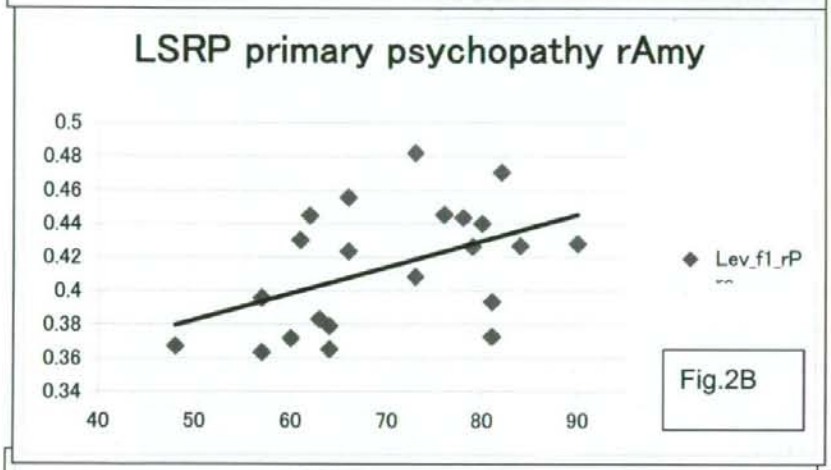


Fig.1. Statistical parametric maps demonstrating the brain regions correlated with psychopathic traits. The left side of the picture is the left side of the brain. A: right precuneus which correlates with LSRP total score. B: right precuneus which correlates with LSRP primary psychopathy. C: left precuneus which correlates with LSRP primary psychopathy. D: right amygdala which correlates with LSRP primary psychopathy. E: right OFC which correlates with LSRP primary psychopathy.





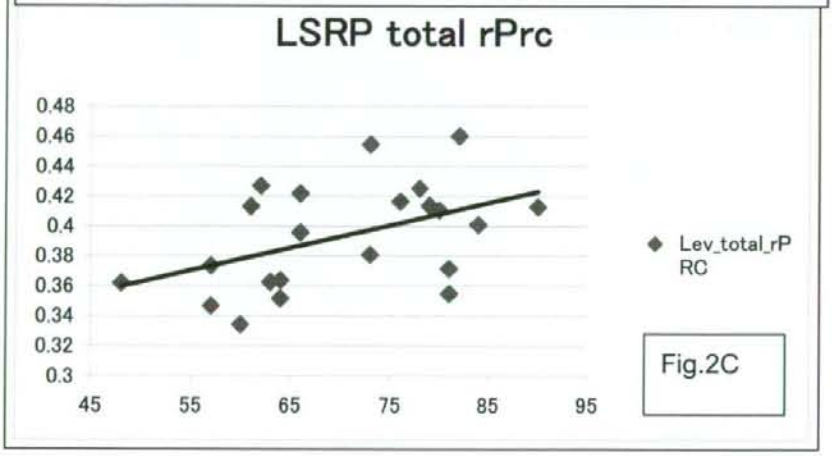


Fig. 2. Correlation of IRI scores and grey matter volume change of areas correlated with psychopathic traits. A: right precuneus. r=0.465. B: right amygdala, r=0.442. C: right precuneus, r=0.471

		healthy control (N=22)	
		Mean	S.D.
Age (years)		21.9	2
Gender (male/female)		8/14	
LSRP	total	54	6.2
	primary psychopathy	32	7.9
	secondary psychopathy	19.7	4.9
IRI	total	70.2	10.9
	fantasy	19.6	3.9
	perspective taking	16	3.8
	empathic concern	16.5	5.4
	personal distress	18	4.4

Table 1. Demographic charachteristics and psychological results.

Title:

Employing delay and probability discounting frameworks for a neuroeconomic understanding of gambling behavior

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Abstract

Although delay and probability discounting theory has been successful in behavioral neuroeconomics, few studies employed the discounting framework for the analysis of gambling behavior. For instance, impaired decision-making in the Iowa Gambling Task (IGT) has been associated with addiction and deficits in the ventromedial prefrontal cortex. Several studies including the somatic marker hypothesis proposed that the deficits on the IGT may be due to insensitivity to future consequences. However, empirical studies regarding the relationship between the impaired decision-making in the IGT and future myopia in intertemporal choice have produced mixed results. On the other hand, studies in psychopharmacology, neuroeconomics, and behavioral psychology state that subject's probabilistic choice in a repeated gambling paradigm is determined by temporal discounting based on molar maximization theory. In this study, we first present our experimental data regarding the relationship between self-reported impulsivity and discounting of delayed and probabilistic gain and loss. Then, we propose that impulsive decision-making in gambling may be attributable to "future myopia" in probability discounting of losses, based on the analysis of the IGT task. The analysis of the relationships demonstrates the important role of a gain-loss asymmetry in probability discounting in risky behavior in gambling. Our present study may help a neuroeconomic understanding of impulsive and risky gambling behavior.

1. Introduction.

1.1 Iowa gambling task

In terms of neuropsychology and neuroeconomics of gambling behavior, pathological gambling is of clinical and theoretical interest. Pathological gambling (PG) is characterized by a loss of control over gambling and continued gambling in spite of associated negative consequences. The disorder is included in the Diagnostic and Statistical Manual of Mental Disorders IV (DSM IV) as an impulse control disorder. A recent study confirmed the validity of the DSM IV criteria by utilizing laboratory gambling experiments and diagnostic interviews (Lakey et al., 2007). Therefore, pathological gambling is a type of impulsive behavior and a type of "anomalies" in economic behavior in that PG may violate the assumption of rationality in economics. DSM IV criteria for PG include repeated unsuccessful efforts to control, or stop gambling, committing crimes to finance gambling, and jeopardizing a job or significant relationships due to gambling. The pathology of PG is characterized by persistent, maladaptive gambling and impaired decision-making, because the gambling behavior persists although the future negative consequences for the gamblers (Lakey et al., 2007).

In order to investigate neuropsychological processing underlying impulsivity in gambling behavior, "Iowa gambling task" (established by Bechara et al, 1994) and its variants have widely been utilized by neurologists and neuroeconomists (Fukui et al., 2005). The Iowa gambling task (IGT) investigates decision making in an ecologically valid gambling task. Actually, Lakey et al. (2007) demonstrated the strong relationship between DSM IV symptoms of PG and maladaptive decision-making in the IGT. A substantial number of studies on the IGT have been published (see Dunn et al., 2006 for a review), and deficient performance has been observed in patients with ventromedial prefrontal lobe (VMPF) damage (Bechara et al., 2000) and patients with types of impulsive behaviors such as substance abuse (Verdejo-García et al., 2007), addiction (Passetti et al., in press), psychopathy (van Honk et al., 2002), and ADHD (Toplak et al., 2005). In the IGT, participants have to choose between four decks of cards. Two of these decks give high rewards constantly, but even higher losses, and these decks are disadvantageous in the statistical expected values of net gains. The two other decks give lower rewards constantly, but even lower losses and these decks are more advantageous in the statistical expected values of net gains. While the IGT renders information on decision making under uncertain reward and loss contingencies, interpretation of task performance is somewhat difficult because of the complexity of the task (Lin et al., 2007; Chiu et al., 2007). The decks of the IGT not only differ in terms of (statisitical) expected outcome (advantageous or disadvantageous), but also in terms of probabilities

of punishment: each pair of advantageous and disadvantageous decks consists of one deck which results in small but likely losses and one deck which results in high but unlikely losses. Furthermore, the IGT includes reward trials and combined reward and loss trials, but no loss trials. Thus, differences in task performance could be due to different underlying motivational and cognitive predispositions. We will illustrate the quantitative characteristics of the IGT in more detail later.

One of the most influential accounts for deficits in the IGT is "future myopia" (insensitivity to future consequences, Bechara et al., 2004) due to a reduction in the operation of "somatic marker" (a feedback effect of peripheral physiological responses to anticipated risk on decision-making) in impulsive patients (Damasio 1996). Damasio (1994) addressed that VM patients act, as is often the case with addicts, in the short term and make decisions which are detrimental in the long run, In psychopharmacological and neuroeconomic studies of intertemporal choice, this tendency (future myopia) has been assessed with the degree to which a subject discounts a delayed outcome (Bickel et al., 2001; Kirby et al., 1999; Reynolds et al., 2004; Ohmura et al., 2005; Petry, 2001b). Specifically, several authors reported that illegal substance misusers, smokers (nicotine addicts), and patients with orbitofrontal cortex lesions discounted delayed rewards more rapidly than healthy controls (Bickel et al., 2001; Kirby et al., 1999; Reynolds et al., 2004; Ohmura et al., 2005; Petry, 2001b, Berlin et al., 2004). Monterosso and colleagues (2001) further demonstrated that strong temporal discounting and deficits on the IGT were associated in cocaine abusers. Therefore, it might be plausible to attribute impaired decision-making in the IGT to future myopia in intertemporal choice (i.e., impulsivity in temporal discounting; in other words, rapid discounting of delayed rewards indicated by a large time-discount rate). This is one of the basic ideas behind Bechara's future myopia theory based on the somatic marker hypothesis (Bechara et al., 1994; 2000; 2002). However, several studies claimed that other hypotheses are more probable and plausible than the future myopia hypothesis, although Bechara's hypothesis is intuitively appealing (Clark et al., 2004; Dom et al., 2007; Fellows, 2004; Kalidindi et al., 2007; Sanfey et al., 2003; Sloman, 2004; Chiu et al., 2007; Lin et al., 2007). The main theoretical basis of these claims is that the roles of immediacy and delay are unclear in the IGT, because each choice at trial t in the IGT is independent of choice at trial t' ($t' \neq t$). In other words, the IGT is not actually an intertemporal choice task, as a recent study of the IGT based on a reinforcement learning theory implied (Kalidindi et al., 2007). Therefore, it is important to establish a theoretical framework which is capable of capturing impulsivity, furure myopia, and disadvantageous gambling behavior in a consistent manner, in order to elucidate psychological and neuroeconomic processing underlying gambling behavior.

In this study, we propose that delay and probability discounting theories often adopted in neuropsychopharmacology and neuroeconomics may be the useful theoretical framework for a better understanding of impulsivity and deficits in the IGT.

1.2 Delay and probability discounting

Impulsive behavior, broadly defined as "actions that are poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable outcomes" (Daruna and Barnes 1993), are frequently observed in drug-dependent subjects (see Bickel and Marsch 2001 for a review) and pathological gamblers. In most neuropsychopharmacological and neuroeconomic studies of intertemporal choice, impulsivity is often operationalized in terms of delay discounting—the tendency to choose smaller, relatively immediate rewards over larger but more delayed rewards (e.g., Kirby et al. 1999; Richards et al. 1999; Passetti et al., 2007; Takahashi, 2005). Recent neurochemical studies on intertemporal choice have demonstrated that low levels of cortisol and salivary alpha-amylase are associated with impulsivity in intertemporal choice (Takahashi, 2004; Takahashi et al., 2007).

In order to mathematically describe delay discounting behavior, standard economic theory has assumed the following exponential discount function:

$$V_D = A\exp(-k_D D) \qquad (1)$$

where V_D is the subjectively discounted value of the reward at delay D, A is the undiscounted value of the reward = $V_D(D = 0)$, D is the delay to the receipt of the reward, and k_D is a free parameter (Samuelson, 1937; Ainslie, 2005; Takahashi, 2005). The larger k_D becomes, the more rapidly a subject discounts the delayed reward (more impulsive intertemporal choice). However, empirical studies in humans and non-human animals reported that delay discounting is better described by the hyperbolic function (Mazur, 1987; Ainslie 2005, Takahashi, 2005):

$$V_D = A/(1+k_D D) \qquad (2)$$

with the same notations as in Equation 1. Again, a larger k_D value corresponds to more rapid temporal discounting. Therefore, in hyperbolic discounting, subjects underestimate their future impulsivity, resulting in preference reversal as time passes (also referred to as "dynamic inconsistency" in economics) (Ainslie, 2005; Takahashi, 2005). It should further be noted that recent studies in personality psychology reported that delay discounting is associated with self-reported impulsivity assessed with