The effects of reward and punishment on response inhibition in non-clinical psychopathy

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Abstract

Response inhibition is an important control mechanism in reacting effectively to sudden changes in the environment, and a deficit in this mechanism is thought to be a main feature of various impulse control disorders, including psychopathy. This study investigated the effects of reward and punishment on the inhibitory capabilities of non-clinical participants with both high and low levels of psychopathy. Forty participants performed a stop signal task under three conditions in a mixed factorial design: A no reward or punishment (N) condition, a low magnitude reward and punishment (L) condition, and a high magnitude reward and punishment (H) condition. Participants with low psychopathy were more inhibited during both reward and punishment conditions as compared to the no reward/punishment condition. On the other hand, participants with high psychopathy showed increased response inhibition only during the L condition. The presence of reward and/or punishment, regardless of magnitude,

increases response inhibition in participants with low psychopathy, whereas high levels of reward and/or punishment do not affect response inhibition in high psychopathy participants. These results suggest that a deficit in response inhibition under incentive conditions could constitute a dimensional feature or aspect of clinical and non-clinical psychopathy.

Key words: response inhibition, psychopathy, non-clinical, stop-signal paradigm, SSRT, reward, punishment.

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1. Introduction

Response inhibition is the ability to inhibit planned or ongoing actions, and represents an important control mechanism for effectively reacting to sudden changes in the environment. A deficit in this inhibitory capability can induce people to behave impulsively and to react inappropriately. A strong association between response disinhibition and certain mental disorders such as substance abuse or personality disorder is well known (e.g., Miller, Flory, Lynam, & Leukefeld, 2003; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001). For example, Cleckley (1976) reported that a lack of self-control characterizes psychopathic behavior.

Psychopathy is defined by a constellation of affective, interpersonal, and behavioral characteristics, including egocentricity, impulsivity, irresponsibility, shallow

emotions, lack of empathy, guilt, or remorse, pathological lying, manipulativeness, and "the persistent violation of social norms and expectations" (Hare, 1998, pp. 188). Hare and Neumann (2008) suggested that psychopathy might be a trait that is continuously distributed within the general population. Some taxometric studies have indicated that psychopathy is indeed a dimensional construct, whether assessed by self-report (Marcus, John, & Edens, 2004) or via clinical ratings using the Psychopathy Checklist—Revised (PCL-R; Hare, 1991, 2003; see Edens, Marcus, Lilienfeld, & Poythress, 2006; Guay, Ruscio, Knight, & Hare, 2007).

Previous research indicates that response disinhibition is characteristic of clinical psychopathy under reward and punishment conditions (Newman & Kosson, 1986). Newman and Kosson (1986) found that non-psychopathic participants showed a greater decrease in commission errors on a response inhibition Go/No-go task, in a reward and punishment situation as compared to a punishment-only condition. In contrast, clinical

psychopathy did not alter behavior during the task, regardless of condition. In addition, psychopathic traits appear to be positively associated with sensitivity to reward and negatively associated with sensitivity to punishment (Ross et al., 2007). The presence of reward and/or punishment has a differential influence on response inhibition, depending on degree of psychopathy.

A previous study suggested that the presence of incentive has an influence on response inhibition in people who are prone to risk-taking (Rodríguez-Fornells, Lorenzo-Seva, & Andres-Pueyo, 2002). Rodríguez-Fornells et al. (2002) concluded that cautious participants, who show less willingness to take risks, become increasingly cautious in the presence of reward or punishment, while risk-taking participants do not alter their behavior as a function of the presence or absence of incentives. However, no study has investigated whether response inhibition in *non-clinical* psychopathy is altered by the presence of reward and/or punishment. Furthermore, no study has

examined the effects of reward or punishment magnitude on response inhibition. We investigated the inhibitory capabilities of non-clinical individuals with high and low levels of psychopathy using a stop-signal paradigm (SSP; Logan, 1994; Logan & Cowan, 1984), under three reward and punishment conditions: No reward and punishment (N), low magnitude of reward and punishment (L), and high magnitude of reward and punishment (H).

The SSP provides a useful experimental measure of inhibitory abilities in both normal and clinical samples. In the SSP, participants are engaged in a reaction time task and are occasionally and unpredictably presented with a signal (e.g., a tone or light) that instructs them to inhibit their response to the stimulus. The SSP consists of both non-stop and stop trials, and in the non-stop trials, participants are required to react to certain stimuli as quickly as possible. When a stop signal is presented, participants have to inhibit their ongoing behavior to the best of their ability. This stop signal can occur at

one of several time delays following the presentation of the stimulus. Unlike a Go/NoGo task, SSP stimuli are not divided into non-stop stimuli and stop stimuli from the very beginning. Thus, the SSP is more suitable as a measure of one's ability to inhibit or stop ongoing reactions (Masui & Nomura, in press).

The purpose of present study was to examine whether the presence of reward and punishment influences the response inhibition capacities of non-clinical psychopathic individuals. We conducted an SSP task in combination with the provision of rewards and punishments, and our sample included participants with low and high levels of psychopathy. Earlier findings led us to the following hypotheses regarding participants' SSP performance: When participants are required to inhibit responses, low psychopathy participants should show increased inhibitory control under the L and H conditions as compared to the N condition. On the other hand, high psychopathy participants should not show improved response inhibition under incentive conditions.

2. Method

2.1 Participants

Forty participants were recruited to participate in the present experiment from a total initial sample of 145 (106 male, 39 female) Japanese university students who completed the Japanese version of the Levenson Self-Report Psychopathy (LSRP) scale (Levenson, Kiehl, & Fitzpatrick, 1995). We recruited those participants who scored particularly high and low on the LSRP scale: The 20 students who scored highest on the LSRP scale were assigned to the high psychopathy group, and the 20 who scored lowest on the LSRP scale were assigned to the low psychopathy group. All participants were right-handed, in order to standardize the response selection format on the SSP. Half of the participants in both groups were female. Initially, female participants belonging to the high and low psychopathy groups were selected. We then recruited male participants,

such that mean LSRP scale scores were comparable to those of the female participants. The data of two participants who could not gain any points during the reward and punishment condition (task details below) were excluded from further analysis.

The means ages were 19.00 for the high psychopathy group (*SD* = 0.58) and 19.79 for the low psychopathy group (*SD* = 3.77).

2.2 Psychopathy assessment

The LSRP scale is a 26-item questionnaire designed to measure psychopathic traits in a healthy population. Each item is a statement that is rated on a four-point Likert-type scale (from strongly disagree to strongly agree). The LSRP scale has two subscales: The primary and secondary psychopathy subscales. The primary psychopathy scale consists of 16 items measuring manipulation, egocentricity, and lack of empathy and remorse, whereas the secondary scale consists of 10 items measuring impulsivity, quick-

temperedness, and poor behavioral control. The LSRP scale has moderate reliability and convergent validity with alternative measures of psychopathy (Brinkley, Schmitt, Smith, & Newman, 2001; Lynam, Whiteside, & Jones, 1999). A Japanese version of the LSRP scale was developed through back translation of the items (Sugiura & Sato, 2005), and demonstrates the same factor structure as the original, along with adequate test–retest reliability and construct validity (Osumi, Kanayama, Sugiura, & Ohira, 2007). Coefficient alphas for this study were .74 for the total LSRP scale, .70 for the primary psychopathy scale, and .58 for the secondary psychopathy scale, values approximately equivalent to those provided by Levenson et al. (1995).

The scores on the LPSP scale were 50-69 (M = 58.21, SD = 5.43) for the high psychopathy group, and 36-48 (M = 41.95, SD = 3.69) for the low psychopathy group. The primary psychopathy scale scores were 28-46 (M = 35.89, SD = 4.46) for the high psychopathy group and 19-32 (M = 25.37, SD = 3.20) for the low group. For the

secondary psychopathy scale, the scores were 17-30 (M = 22.32, SD = 3.20) for the high psychopathy group and 13-21 (M = 16.58, SD = 2.17) for the low group. There were significant differences in total LSRP scale scores (t (36) = -10.80, $p < .001$, d = 3.50), primary psychopathy scale scores (t (36) = -8.36, $p < .001$, d = 2.71), and secondary psychopathy scale scores (t (36) = -6.47, $p < .001$, d = 2.10) between the two groups.

2.3 Procedure

We used the SSP to measure participants' ability to inhibit actions. As was done in Rodríguez-Fornells et al. (2002), the stimuli used in this study were the uppercase letters V, M, W, and N, viewed on a computer screen at a distance of about 60 cm. The stop signal was a red triangle that appeared above the letters, for a duration of 150 ms.

The letters V and M were assigned to one hand, and W and N were assigned to the other. Each trial began with a white fixation point presented at the center of the screen for 500 ms. This point then disappeared, and 400 ms later a letter stimulus was

presented at the center of the screen for 1.1 s. The screen then went blank for 700 ms. Participants responded by pressing a key corresponding to the hand the letter was assigned to, which was ‘Z’ (for the left hand) or ‘/’ (for the right hand) on the keyboard. The mapping of letters onto the keys was counterbalanced across participants.

All participants were tested in four separate sessions, one after the other. In the first session, the participants performed a choice RT task for two blocks of 100 trials each. In this task, participants merely had to respond with the appropriate hand to the corresponding letter. This task was used to determine the baseline RT of each participant, in order to accurately set the stop-signal delays. The mean RT (MRT) for the second block of the choice RT task was selected as each participant’s baseline. Stop-signal delays were standardized using this MRT time as follows: MRT of 500 ms, MRT of 350 ms, MRT of 250 ms, and MRT of 100 ms (for details, see Solanto et al., 2001).

In the second session, all participants performed the SSP under the N condition.

Participants were instructed not to respond to the primary-task stimulus when the red triangle symbol appeared. They were told that the stop signal would occur in such a way that they would sometimes be able to stop their response, and sometimes not. Participants were instructed to respond as quickly as possible while also maintaining a high level of accuracy. Moreover, they were advised not to delay their response in anticipation of the stop signal. The stop signal was emitted during 50% of the trials. Each stop-signal delay was equiprobable. The sequence of letters, non-stop/stop trials, and stop-signal delay was random, and different random orders were administered to each participant during each session.

The third and fourth sessions were used to create varied reward and punishment conditions by using different payoffs (L and H conditions). These sessions were identical to the previous ones, except that participants were rewarded or punished by awarding or deducting points, depending on their performance. Participants were given

the following task instructions about the reward/punishment feedback under the L condition:

"If your reaction to non-stop stimuli (without the triangle) is faster than your mean reaction time during the choice reaction time task, you will earn 10 points. However, if your reaction time is slower than your mean reaction time on the choice reaction time task, you will be awarded no points, and if the response is completed with the wrong hand or if you fail to respond, you will lose 10 points from your earnings.

If you refrain from responding during the stop-signal trials (when the triangle appears), you will not receive any points but will not lose any points either. However, if you do not inhibit your response, you will lose 10 points. Please earn as many points as you possibly can."

Under the H condition, participants could earn up to 100 points as a reward, or they could lose 100 points as a punishment.

Participants were also told that they would be rewarded depending on their total points. They in fact received the same remuneration (which was equivalent to 200 yen) regardless of the points acquired. The N, L, and H conditions all included 160 trials, and the order of the L and H conditions was counterbalanced across participants.

We measured the behavioral performance of all participants using RTs, probability of response, and the stop-signal reaction times (SSRTs) for each condition. The SSRT, a measure of inhibitory process latency, is a valid index of impulsivity. A slower SSRT is considered to reflect greater impulsivity, as demonstrated by studies on attention deficit and hyperactivity disorder (Solanto et al., 2001), substance abuse disorders (Monterosso, Aron, Cordova, Xu, & London, 2005), pathological gambling, and alcohol dependence (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2006). In the present study, we employed the methods used in earlier work to estimate SSRTs (Solanto et al., 2001).

First, the probability of response in the presence of the stop signal [*p* (respond |

signal)] was calculated for each of the four stop-signal delays (100, 250, 350, and 500 ms). Each of these calculations included a correction for nonresponse due to failures to respond (omission errors) on non-stop trials, as follows:

p (respond | signal) = (x - correct rejections / x - xy)

where x, the number of stop trials under each condition, is 80; correct rejections are the number of correct inhibitions of responses on stop trials; and y is the probability of omission errors on non-stop trials.

The RTs on non-stop trials were then rank-ordered. The reaction time at the "nth" percentile is identified, where "n" corresponds to p (respond | signal). This "nth" RT is the latency from the onset of the go stimuli, which approximates the latency between the go stimuli and the conclusion of the stopping process. To estimate SSRT, stop-signal delay is subtracted from "nth" RT.

2.4 Data Analysis

We compared behavioral performance during the SSP across the three conditions. Because the MRT of the choice RT task in the high psychopathy group was marginally slower than that in the low psychopathy group (t (36) = -1.92, p = .06, d = .62), we conducted an analysis of co-variance (ANCOVA) for RTs, probability of response inhibition, and SSRTs using psychopathy as the between-subject factor, condition as the within-subjects factor, and MRTs on the choice RT task as a covariate. Furthermore, to clarify the specific effects of the two psychopathy subscales, we further conducted ANCOVAs, entering MRTs on the choice RT task and primary or secondary pasychopathy scale scores as covariates.

3. Results

3.1 Behavioral Results

Table 1 shows the mean RTs and probabilities of response inhibition for each condition. There were no significant RT differences between the groups ($F(1, 35) = 1.57$, $p = .22$, $\eta_p^2 = .04$) or conditions ($F(2, 70) = 1.29$, $p = .28$, $\eta_p^2 = .04$), and the interaction between group and condition was not significant, $F(2, 70) = 0.52$, $p = .60$, $\eta_p^2 = .02$. Similarly, there were no significant differences in terms of response inhibition probability across the groups ($F(1, 35) = 0.52$, $p = .48$, $\eta_p^2 = .02$), conditions ($F(2, 70) = 1.24$, $p = .30$, $\eta_p^2 = .03$), or the interaction between group and condition ($F(2, 70) = 1.03$, $p = .36$, $\eta_p^2 = .03$).

=========Table 1 about here=========

Figure 1 shows mean SSRTs for participants with high and low psychopathy under all three conditions. An ANCOVA analysis revealed a significant interaction between the groups and conditions, $F(2, 70) = 3.29$, $p < .05$, $\eta_p^2 = .09$. A post-hoc test showed that for low psychopathy participants, the SSRTs under the H condition were

significantly faster than those under the N condition ($p < .05$). Furthermore, the SSRTs under the L condition were marginally faster than those under the N condition ($p = .05$). On the other hand, the SSRTs for high psychopathy participants were significantly faster under the L condition, as compared to the other conditions (N; $p < .01$, H; $p < .05$).

=========Figure 1 about here=========

When primary psychopathy was controlled during an analysis of SSRTs, the interaction between the groups and conditions was significant, $F(2, 68) = 3.69$, $p < .05$, $\eta_p^2 = .10$. The SSRTs for high psychopathy participants were significantly faster under the L condition as compared to the other conditions (N; $p < .01$, H; $p < .05$). On the other hand, when secondary psychopathy was controlled, the interaction between the groups and conditions was not significant, $F(2, 68) = 1.60$, $p = .21$, $\eta_p^2 = .05$.

4. Discussion

The present study examined the effects of reward and punishment on response inhibition in non-clinical psychopathy. We used a response inhibition task, the SSP, along with two different magnitudes of reward and punishment feedback. Participants first performed the SSP under the control (N) condition. They then performed the task under both low (L) and high magnitude (H) conditions.

Although there were no significant differences between the participants with low and high psychopathy in terms of basic RTs or the probability of response inhibition, there was a significant difference for SSRTs, an index of response inhibition. In the incentive L and H conditions, the SSRTs of low psychopathy participants were faster than during the no incentive condition. On the other hand, and interestingly, for high psychopathy participants, SSRTs under the L condition were faster than those under the N condition, but there was no difference between SSRTs under the H and N conditions.

This result remained robust when participants' primary psychopathy was controlled. These findings suggest that the presence of any magnitude of reward and punishment improves the response inhibition capacity of low psychopathy participants, but that the presence of high levels of reward and punishment has no such effect on the response inhibition capacity of high psychopathy participants. In addition, a deficit in response inhibition in high psychopathy individuals under high magnitude reward and punishment conditions appears to be influenced by the degree of secondary psychopathic traits present.

Some earlier findings are consistent with the results of the present study, such that reward and punishment does affect response inhibition in psychopathic or risk-taking participants (Newman & Kosson, 1986; Rodríguez-Fornells et al., 2002). For example, Newman and Kosson (1986) found that non-psychopathic participants demonstrated fewer commission errors during a Go/No-go task under a reward and punishment

condition, as compared to a punishment only condition. In contrast, psychopathic participants did not alter their behavior during the task, regardless of condition. Similarly, Rodríguez-Fornells et al. (2002) found that low risk-taking participants showed increased caution in the presence of reward and punishment during response inhibition, whereas risk-taking participants did not show a similar change in behavior.

According to motivational theories of impulsive and disinhibitory disorders, impulsive or disinhibited participants have more problems modulating reward-seeking responses or withholding an appetitive response under certain motivational circumstances (Gorestein & Newman, 1980; Gray, 1987; Patterson & Newman, 1993). On the basis of this idea, the present findings that high psychopathy participants did not improve their behavior in the H condition could be interpreted in terms of sensitivity to high magnitude of reward that would be expected to strongly elicit impulsivity, such that inhibitory performance was not improved. As for the L condition, the low

magnitude of reward offered may not have not been sufficient to elicit impulsive behavior in those high psychopathy participants.

A neuropsychological model of the core deficits involved in psychopathy would serve to shed further interpretative light on the results of this study. One such model is the revised reinforcement sensitivity theory (RST) of personality (for a summary see Corr, 2008). The revised RST posits three systems: The behavioral inhibition system (BIS), behavioral approach system (BAS), and the fight-flight-freeze system (FFFS). First, primary psychopathy is associated with significantly lower scores on measures of BIS functioning than non-psychopathic inmates, but normal BAS scores (Newman, MacCoon, Vaughn, & Sadeh, 2005). It is also possible that there is a negative relationship between primary psychopathy and FFFS activity (for a review, see Corr, 2010). On the other hand, in one study BAS scores in secondary psychopaths were significantly higher than those of non-psychopaths (Newman et al., 2005). In the present

study, we controlled for primary psychopathy scores and the SSRTs for high psychopathy participants were significantly faster under the L condition as compared to the other conditions. However, the interaction effect between the groups and conditions disappeared when we controlled for secondary psychopathy. Individuals with high levels of secondary psychopathy did not alter their behavior under the H condition of the present study, presumably because of the higher levels of BAS functioning observed in secondary psychopathy.

Comparing the characteristics of non-clinical and clinical psychopaths has remained a problematic endeavour for a number of reasons. The PCL-R is inappropriate for use in non-clinical settings (Mahmut, Homewood, & Stevenson, 2008). On the other hand, LSRP scores have shown moderate correlations with PCL-R scores among women (Brinkley, Schmitt, Smith, & Newman, 2001). In one study, similar to previous findings with clinical psychopaths, high psychopathy participants performed

significantly worse than those with low psychopathy on the Iowa Gambling Task (Mahmut et al., 2008). Furthermore, previous taxometric studies have indicated that psychopathy is a dimensional construct, whether assessed by self-report (Marcus et al., 2004) or via clinical ratings using the PCL-R (Hare, 1991, 2003; see Edens et al., 2006; Guay et al., 2007). The present study also suggests that psychopathy may be continuously distributed within the general population.

Our study has at least one limitation, in that the motivation of participants during the SSP task was not assessed. Leotti and Wager (2009) found motivational influences on task performance during response inhibition, and noted that when participants tried to accurately inhibit their behavior on the stop trials, SSRTs became short. On the basis of this suggestion, motivation to engage in the present study task might have differed across participants, regardless of their levels of psychopathic traits. Further research needs to investigate the relationships among psychopathy, response disinhibition, and

motivation (as directly measured for the task at hand).

In summary, to our knowledge our study is the first to demonstrate that the presence of reward and punishment has different influences on the inhibitory capabilities of non-clinical psychopathic individuals. A deficit in response inhibition under incentive conditions could be a dimensional feature of both clinical and non-clinical psychopathy.

References

Brinkley, C. A., Schmitt, W. A., Smith, S. S., & Newman, J. P. (2001). Construct validation of a self-report psychopathy checklist-revised. *Personality and Individual Differences*, 31, 1021-1038.

Cleckley, H. (1976). *The mask of sanity*. St. Louis, Mo: Mosby.

Corr, P. J. (2008). Reinforcement sensitivity theory (RST): Introduction. In P. J. Corr (Ed.), *The reinforcement sensitivity theory of personality* (pp. 1-43). Cambridge: Cambridge University Press.

Corr, P. J. (2010). The psychoticism-psychopathy continuum: A neuropsychological model of core deficits. *Personality and Individual Differences*, 48, 695-703.

Edens, J. F., Marcus, D. K., Lilienfeld, S. O., & Poythress, N. G. (2006). Psychopathic, not psychopaths: Taxometic evidence for the dimensional structure of psychopathy. *Journal of Abnormal Psychology*, 115, 131-144.

Gorenstein, E. E., & Newman, J. P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review*, 87, 301-315.

Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & van den Brink, W. (2006). Neurocognitive functions in pathological gambling: A comparison with alcohol dependence, Tourette syndrome and normal controls. *Addiction*, 101, 534-547.

Guay, J. P., Ruscio, J., Knight, R. A., & Hare, R. D. (2007). A taxometric analysis of the latent structure of psychopathy: Evidence for dimensionality. *Journal of Abnormal Psychology*, 116, 701-716.

Gray, J. (1987). *The psychology of fear and stress.* Cambridge: Cambridge University Press.

Hare, R. D. (1991). *The Hare Psychopathy Checklist-Revised*. Toronto, ON: Multi-Health Systems.

Hare, R. D. (1998). Psychopaths and their nature: Implications for the mental health and

criminal justice systems. In T. Millon, E. Simonsen, M. Biket-Smith, & R. D. Davis (Eds.), *Psychopathy: Antisocial, criminal and violent behavior* (pp. 188-212). New York: Guilford Press.

Hare, R. D. (2003). *The Hare Psychopathy Checklist-Revised* (2nd. ed.). Toronto, ON: Multi-Health Systems.

Hare, R. D., & Neumann, C. S. (2008). Psychopathy as a clinical and empirical construct. *Annual Review of Clinical Psychology*, 4, 189-216.

Leotti, L. A., & Wager, T. D. (2009). Motivational influences on inhibition-related brain activity. Poster presented the 16th Annual Meetings of the Cognitive Neuroscience Society, San Francisco, U. S.

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in noninstitutionalized population. *Journal of Personality and Social Psychology*, 68, 151-158.

Logan, G. D. (1994). On the ability to inhibit thought and action: A users' guide to the stop signal paradigm. In D. Dagenbach, & T. H. Carr (Eds.), *Inhibitory processes in attention, memory, and language* (pp. 189-236). San Diego, CA: Academic Press.

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review*, 91, 295-327.

Lynam, D., Whiteside, S., & Jones, S. (1999). Self-reported psychopathy: A validation study. *Journal of Personality Assessment*, 73, 110–132.

Mahmut, M. K., Homewood, J., & Stevenson, R. J. (2008). The characteristics of non-criminals with high psychopathy traits: Are they similar to criminal psychopaths? *Journal of Research in Personality*, 42, 679-692.

Marcus, D. K., John, S. L., & Edens, J. F. (2004). A taxometric analysis of psychopathic personality. *Journal of Abnormal Psychology*, 113, 626-635.

Masui, K., & Nomura M. (in press). A review of impulsivity from structure concept, brain function, and genetic phenotype: The perspectives from stop-signal paradigm. *The Japanese Journal of Research on Emotions*.

Miller, J., Flory, K., Lynam, D., & Leukefeld, C. (2003). A test of the four-factor model of impulsivity-related traits. *Personality and Individual Differences*, 34, 1403-1418.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., & Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry*, 158, 1783-1793.

Monterosso, J. R., Aron, A. R., Cordova, X., Xu, J., & London, E. D. (2005). Deficits in response inhibition associated with chronic methamphetamine abuse. *Drug and Alcohol Dependence*, 79, 273-277.

Newman, J. P., & Kosson, D. S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology*, 95, 257-263.

Newman, J. P., MacCoon, D. G., Vaughn, L. J., & Sadeh, N. (2005). Validating a distinction between primary and secondary psychopathy with measures of Gray's BIS and BAS constructs. *Journal of Abnormal Psychology*, 114, 319-323.

Osumi, T., Kanayama, N., Sugiura, Y., & Ohira, H. (2007). Validation of the Japanese Primary and Secondary Psychopathy Scales. *The Japanese Journal of Personality*, 16, 117-12.

Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review*, 100, 439-446.

Rodrígues-Fornells, A., Lorenzo-Seva, U., & Andrés-Pueyo, A. (2002). Are high-impulsivity and high-risk-taking people more motor disinhibited in the presence of incentive? *Personality and Individual Differences*, 32, 661-683.

Ross, S. R., Moltó, J., Poy, R., Segarra, P., Pastor, M. C., & Montañés, S. (2007). Gray's

model and psychopathy: BIS but not BAS differentiates primary from secondary psychopathy in noninstitutionalized young adults. *Personality and Individual Differences*, 43, 1644-1655.

Solanto, M. V., Abikoff, H., Sonuga-Barke, E., Scharchar, R., Logan, G. D., Wigal, T., et al. (2001). The ecological validity of delay aversion and response inhibition as measures of impulsivity in AD/HD: A supplement to the NIMH multimodal treatment study of AD/HD. *Journal of Abnormal Child Psychology*, 29, 215-228.

Sugiura, Y., & Sato, A. (2005). Validation of the Japanese version of the Primary and Secondary Psychopathy Scale. Poster session presented at the 69th meeting of the Japanese Psychological Association, Tokyo, Japan.

Figure Captions

Figure 1. Mean SSRTs under reward and punishment conditions (*N* = 38). The white bar indicates the mean SSRT for low psychopathy participants, and the black bar indicates the mean SSRT for high psychopathy participants.

Note: N = no reward and punishment control condition, L = low magnitude condition, H = high magnitude condition.

Table 1

Mean reaction times (MRTs) and mean probability of response inhibition under each condition.

	low psychopathy participants (N = 19)	high psychopathy participants (N = 19)
MRT (ms)		
N	630.53 (96.58)	638.92 (103.86)
L	587.06 (84.38)	611.13 (107.56)
H	603.41 (98.33)	603.92 (102.28)
inhibition (%)		
N	59.26 (9.33)	59.84 (9.08)
L	58.29 (9.50)	59.89 (9.70)
H	59.28 (12.37)	57.44 (10.74)

Note: N = no reward and punishment control condition, L = low magnitude condition, H = high magnitude condition.

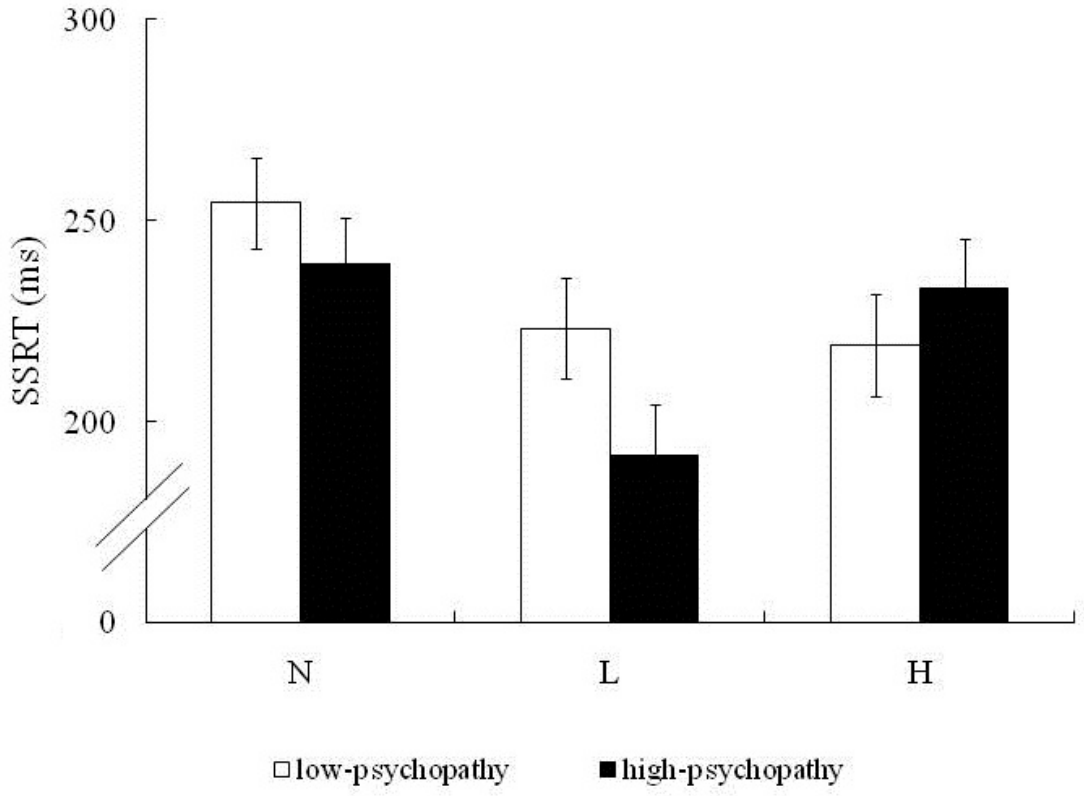

Figure 1