Journal of Personality and Social Psychology
1997, Vol. 72, No. 6, 1413-1428

0022-3514/97/$3.00

Approach and Avoidance Motivation in Psychopathic Criminal Offenders During Passive Avoidance

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The authors evaluated competing theories that attribute psychopathic individuals' poor passive avoidance to a strong activating system, a weak inhibitory system, or poor modulation of behavioral activation when inhibitory cues appear. In Study 1, the continuous motor task involved a reward phase to elicit the activating system followed by a passive avoidance phase. Study 2 tested the generality of the theories by using an active avoidance phase to elicit the activating system. Heart rate and response speed results from Study 1 best supported the strong activating system and poor response modulation models in low-anxiety psychopathic offenders. Study 2 results did not clearly support any of the models. Further research is needed to determine if excessive activation by reward and poor response modulation are associated with passive avoidance deficits and other characteristics of low-anxiety psychopathic offenders.

Both clinical observation and experimental research suggest that psychopathic individuals are poor at learning passive avoidance (Cleckley, 1976; Lykken, 1957; Newman & Kosson, 1986; Schachter & Latane, 1964; Schmauk, 1970). They often fail to inhibit behaviors that have, in the past, led to punishment. Because of the importance of passive-avoidance learning in the socialization process (Aronfreed, 1968; Trasler, 1978), it has been studied intensively in psychopathic individuals.

Most attempts to explain psychopathic individuals' poor passive avoidance have focused on their poor fear conditionability (Hare, 1970, 1978; Lykken, 1957; Ogloff & Wong, 1990) or their chronic underarousal (Chesno & Kilmann, 1975; Hare, 1978; Quay, 1965). A variation of the poor fear conditionability hypothesis has been provided by Fowles (1980), who proposed a three-arousal model—an adaptation and extension of Gray's (1975) two-factor learning theory—to account for psychopathic individuals' poor passive avoidance. Fowles's model incorporates the findings that psychopathic individuals show smaller increases in skin conductance (SC) and fewer nonspecific fluctuations in SC in anticipation of aversive unconditioned stimuli and no deficiency in cardiovascular conditioning. The three arousal systems in Fowles's model are (a) the behavioral activation system (BAS), (b) the behavioral inhibition system (BIS), and (c) a nonspecific arousal system (NAS) that receives inputs from both the BAS and the BIS. There are mutually inhibitory inputs between the BAS and BIS. The BAS is activated by and initiates behavior in response to conditioned stimuli for reward or active avoidance and is indexed by heart rate (HR). The BIS is activated by and inhibits behavior in response to conditioned stimuli for punishment (passive avoidance) or frustrative nonreward (extinction) and is indexed by increased electrodermal activity. Consistent with previous investigators (e.g., Gray, 1975; Trasler, 1978), Fowles contended that psychopathic individuals' poor passive avoidance is due to a weak BIS (Figure 1).

Noting a number of inconsistencies in both the poor fear conditionability hypothesis (Hare & Craigen, 1974; Hare, Frazelle, & Cox, 1978) and the low-arousal hypothesis (Blackburn, 1979; Mawson & Mawson, 1977), Gorenstein and Newman (1980) proposed an alternative explanation emphasizing the importance of reward in moderating psychopathic individuals' poor passive avoidance. Since 1980, Newman and his colleagues have reported on findings from a variety of go/no-go discrimination tasks illustrating that psychopathic individuals' difficulty avoiding monetary punishments is relatively specific to tasks involving monetary reward as well as punishments (Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985). On the basis of these and related findings, Newman and his colleagues have proposed that psychopathic individuals' tendency to perseverate a response set for reward makes them less likely to pause after,

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These studies were part of a doctoral dissertation completed by Peter A. Arnett at the University of Wisconsin—Madison under the supervision of Joseph P. Newman. This research was presented in part at the annual meeting of the Society for Psychophysiological Research, Chicago, October 1991 and the annual meeting of the Society for Research in Psychopathology, Palm Springs, November 1992. This research was supported by National Institute of Mental Health Grant MH37711.

Peter A. Arnett wishes to acknowledge his appreciation of the enduring support of Melissa, Marjorie, and Jeffrey Arnett on this article and the dissertation on which it is based. We wish to thank Avshalom Caspi, Richard Davidson, Terrie Moffitt, and Deborah Vandell for serving on the dissertation committee and for their helpful suggestions concerning the study. We also thank Randy Busse for his contribution to this research. We thank the late Arnold Blahnik, Jeff Wydeven, Deb March, Bernice Conner and the staff of the Oakhill Correctional Institution, and the Wisconsin Division of Corrections for their consistent cooperation and support regarding this research project.

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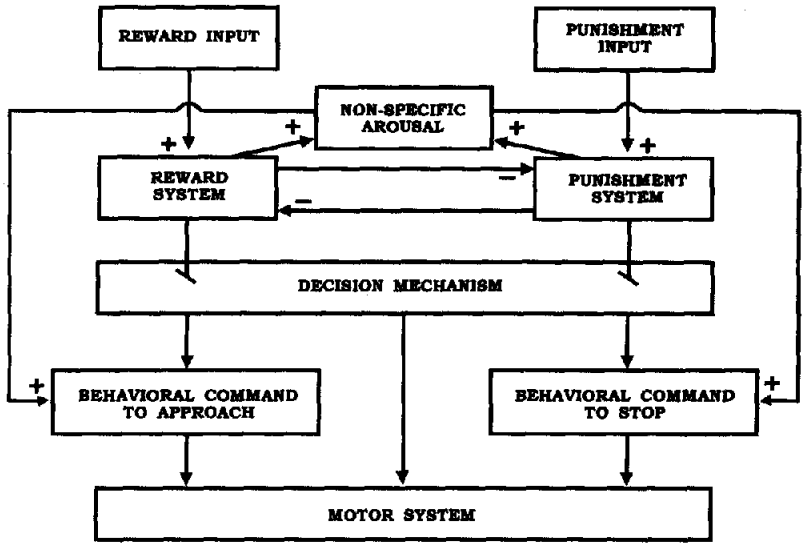

Figure 1. Gray's (1987) neuropsychological model of approach–avoidance learning. The reward system is the behavioral activation system (BAS); the punishment system is the behavioral inhibition system (BIS). From *The Psychology of Fear and Stress* (p. 245) by J. A. Gray, 1987, New York: Cambridge University Press. Copyright 1987 by Cambridge University Press. Adapted and reprinted with the permission of Cambridge University Press.

and therefore less likely to learn from, punishment (Newman, Patterson, & Kosson, 1987; Nichols & Newman, 1986; Patterson & Newman, 1993).

Although research by Newman and his colleagues demonstrates that reward may play an important role in mediating psychopathic individuals' poor passive avoidance, it remains unclear whether psychopathic individuals are truly hypersensitive to reward, in the sense of being physiologically and behaviorally overreactive to it, or whether they simply have difficulty disengaging from their pursuit of reward when environmental cues suggest they should (see Newman, Patterson, Howland, & Nichols, 1990). To use the constructs from Gray's (1975, 1987) model, are psychopathic individuals characterized by an overactive BAS, or do they simply have difficulty modulating BAS activity in response to monetary punishment, perhaps because of an ineffective interrupt or switching mechanism between the BAS and BIS?

To date, no one has assessed sensitivity to reward by comparing the physiological and behavioral responses of psychopathic and nonpsychopathic individuals in a pure BAS context. Although Fowles (1980) asserted that psychopathic individuals show normal BAS activity (i.e., approach and active avoidance), no published study has evaluated their pure BAS activity in response to either reward cues or cues for active avoidance. Scerbo et al. (1990) did find that psychopathic adolescents made more responses than did controls to rewarded stimuli on a go/no-go discrimination task and suggested that these individuals were characterized by greater sensitivity to reward stimuli compared with controls. However, like Arnett, Howland, Smith, and Newman (1993) and Newman et al. (1990), Scerbo et al. collected their data in the context of a task without a pure reward-only phase; instead, the reward/punishment contingencies changed frequently according to participants' responses. Thus there was no way of determining whether the psychopathic individuals' responses to reward really reflected hypersensitivity to reward or reflected some interaction between reward and punishment.

In the present two investigations, we examined the strength of BAS activation in psychopathic individuals and controls in a pure reward-only context. Both studies were designed, in part, to assess peripheral indicators of psychopathic individuals' BAS and BIS activity in the context of a passive-avoidance task. In Study 1, 1 min of BAS activation by reward cues occurred, and its effects on a subsequent passive-avoidance contingency were observed. In Study 2, 1 min of BAS activation by active-avoidance cues occurred before a passive-avoidance contingency were introduced. Study 2 was conducted for three reasons. First, we wished to test the inference from Gray's (1987) model that BAS activation by active-avoidance cues should be similar to and have the same consequences for passive avoidance as BAS activation by reward cues. Second, we wanted to test the alternative hypothesis that psychopathic individuals may react differently to BAS activation from active-avoidance cues than to BAS activation by reward cues, even though Gray's model suggests that the BAS is a unitary construct and should be activated similarly by either type of cue. Third, only two published studies (Chesno & Kilmann, 1975; Newman & Kosson, 1986, Experiment 2) have examined psychopathic individuals' response to active-avoidance cues, whereas several studies have examined passive avoidance. Notably, neither of these studies included a pure active-avoidance phase; the contingencies of the task changed frequently from active to passive avoidance.

From a purely psychometric point of view, given the greater variability and difficulty of learning a passive-avoidance contingency in the context of an active avoidance relative to reward contingency (e.g., Newman & Kosson, 1986), greater passive-

avoidance deficits in psychopathic individuals relative to controls might be expected for the active-avoidance study (Study 2) compared with the reward study (Study 1). However, at least two studies (e.g., Chesno & Kilmann, 1975; Newman & Kosson, 1986) demonstrated passive-avoidance deficits in psychopathic individuals when the passive-avoidance contingency had to be learned in the context of reward, but not in the context of active avoidance. In fact, in conditions in which there was also an ongoing active-avoidance contingency, Chesno and Kilmann (1975, high-stimulation condition) found better passive avoidance in low-anxiety psychopathic individuals than in low-anxiety controls, and Newman and Kosson's (1986, Experiment 2) data were in the direction of better passive avoidance in psychopathic individuals. In addition, theoretical accounts (Gorenstein & Newman, 1980), clinical observations (e.g., McCord & McCord, 1964), and studies of excessive substance use (Smith & Newman, 1990) and reward-seeking behavior (Zuckerman, 1978) suggest a possible reward sensitivity in psychopathic individuals.

We tested hypotheses from three theoretical models using psychophysiological indexes derived from Gray's (1975) model and proposed by Fowles (1980) and additional behavioral indexes suggested by Gray (1975, 1987). It should be noted that two levels of models were considered in the present studies. We used Gray's (1975, 1987) and Fowles's (1980) models as an organizing framework, and we used constructs from these models to derive specific hypotheses for three models of psychopathy. There is the potential for some confusion, because Fowles's model was used both as an organizing framework and to derive specific hypotheses for one of the explanations about processes underlying passive avoidance in psychopathic individuals.

The first explanation of psychopathic individuals' poor passive avoidance, based on Fowles's (1980) model and hereafter referred to as the *weak-BIS model,* is that psychopathic individuals are characterized by a weak BIS, meaning that their behavioral and physiological responses to punishment cues are insufficient to interrupt even normal approach behavior. The second explanation, derived from the work of Gorenstein and Newman (1980) and hereafter referred to as the *strong-BAS model,* is that psychopathic individuals' tendency to perseverate reward-seeking behavior interferes with their ability to process cues for punishment. Despite normal sensitivity to punishment cues, psychopathic individuals' hypersensitivity to reward cues makes the inhibition of ongoing goal-directed behavior unlikely. An extension of this model, considering that both reward and active-avoidance cues are thought to activate the BAS (Gray, 1987), is that hypersensitivity to active-avoidance cues should interfere with the inhibition of goal-directed behavior in psychopathic individuals. The third explanation, the *response modulation model* (Newman et al., 1987; Patterson, Kosson, & Newman, 1987; Patterson & Newman, 1993), is that psychopathic individuals are equally responsive to rewards and punishments in most situations but have difficulty inhibiting a dominant response set when cues for punishment are present, because they are less adept at interrupting or modulating BAS activation. Like the strong-BAS model, this model posits that psychopathic individuals have difficulty assimilating and, ultimately, learning from the punishment cues that appear after reward activation as a result of their failure to pause and process them. Again, an extension of this model in light of Gray's (1987) theory of BAS functioning would suggest that psychopathic individuals should have difficulty learning from punishment cues that appear after the establishment of a dominant response set by active-avoidance cues because of their failure to pause and process them (cf. Newman & Wallace, 1993a; Patterson & Newman, 1993).

All three models predict weak behavioral inhibition to punishment cues (i.e., poor passive avoidance), but the underlying processes are presumed to differ. Using the constructs of Gray's (1975, 1987) general model of learning, the hypothesized processes underlying weak response inhibition for the three models of psychopathy are (a) weak BIS activity, as indexed by weak skin conductance response (SCR) to punishment cues (the weak-BIS model); (b) exaggerated BAS activity, as indexed by increased HR and response speed to reward (Study 1) and active-avoidance cues (Study 2; the strong-BAS model); or (c) poor response modulation, as indicated by failure to suspend goal-directed behavior (e.g., to reward) when punishment cues are encountered. The poor response modulation model predicts difficulty inhibiting responses to punishment cues in the absence of a weak BIS or strong BAS (for a more in-depth treatment of response modulation, see Newman & Wallace, 1993a, 1993b; Patterson & Newman, 1993; see Figure 1).

All three models have received some empirical support, but none has emerged as superior to the others. One reason for this is that, to our knowledge, no study has compared the models in the same paradigm.

Study 1

To evaluate the three models, we used an adaptation of the task reported by Fowles, Fisher, and Tranel (1982). The adaptation involved rewarding participants for responding rapidly to buttons arranged in a semicircle as green lights mounted above the buttons were illuminated. Each reward-only phase was followed by a passive-avoidance phase signaled by the onset of a center red light. In the passive-avoidance phase, participants had to learn to withhold responding to punishment cues (red lights) as they continued to pursue rewards. BAS activation was assessed during the reward-only phase, independent of BIS influence. During the passive-avoidance phase of the task, BIS activation and passive avoidance were assessed.

Heart rate and response rate were used as indicators of BAS activity, and electrodermal activity (EDA) was used as an indicator of BIS activity. Fowles (1980) identified HR as an index of BAS activity, and Gray (1975) proposed that "behavioral vigor," including response speed, can index the NAS, the BAS, or both systems, depending on situational parameters (see also Wallace, Bacharowski, & Newman, 1991). In Study 1, response speed and HR increase were used to index BAS activity during the reward-only phase of the task. In addition, faster response speed after punishment cues was our operationalization of poor response modulation.

A priori hypotheses for the three models targeted participants with low anxiety and negative emotionality for several reasons. First, the absence of neurotic features is considered a cardinal feature of psychopathy by most theorists (e.g., Chesno & Kilmann, 1975; Cleckley, 1976; Fowles, 1980; Hare, 1978; Newman et al., 1990). Therefore, it stands to reason that low-anxiety

Table 1
Hypothesized Differences Between Low-Anxiety Psychopathic Individuals (P) and Controls (C) for Three Models of Psychopathy

Model	Weak behavioral inhibition system	Strong behavioral activation system	Response modulation
	Study 1: Reward versus passive avoidance		
Reward only			
Response speed	P = C	P > C	P = C
Heart rate	P = C	P > C	P = C
SCR (number)	P = C	P = C	P = C
Punishment cue			
Response speed	P > C	P > C	P > C
SCR (amplitude)	P < C	P = C	P = C
Passive avoidance			
SCR (number)	P < C	P = C	P = C
Errors	P > C	P > C	P > C
	Study 2: Active versus passive avoidance		
Active avoidance only			
Response speed	P = C	P > C	P = C
Heart rate	P = C	P > C	P = C
SCR (number)	P = C	P = C	P = C
Punishment cue			
Response speed	P > C	P > C	P > C
SCR (amplitude)	P < C	P = C	P = C
Passive avoidance			
SCR (number)	P < C	P = C	P = C
Errors	P > C	P > C	P > C

Note. SCR = skin conductance response.

psychopathic individuals are closer to what has been referred to as the primary or true psychopathic individual.[1] Second, when groups are equated for anxiety scores, it is possible to state with greater certainty that any differences obtained between low-anxiety psychopathic individuals and low-anxiety controls are due to differences in psychopathy rather than anxiety. This is not the case when all participants are considered, because the anxiety variable is not controlled across groups; high-anxiety control or psychopathic participants could have a disproportionate effect on overall group results. Finally, there is a historical pattern in the literature of finding poorer passive-avoidance learning only in low-anxiety psychopathic individuals (e.g., Chesno & Kilmann, 1975; Newman et al., 1990; Schachter & Latane, 1964; Schmauk, 1970).

Table 1 outlines hypotheses from each model for each dependent variable. On the basis of the weak-BIS model, we expected no group differences in HR or response speed during the reward-only phase unless these differences were mediated by BIS activity, that is, unless there was evidence of weak EDA by psychopathic individuals during this phase. However, we expected that during the passive-avoidance phase, psychopathic individuals would show significantly less BIS activation than controls, as indexed by smaller amplitude SCRs to the punishment cues that initiated the passive-avoidance phase and fewer SCRs during this phase.

On the basis of the strong-BAS model, we expected psychopathic individuals to show greater BAS activation than controls in response to cues for reward. According to this model, psychopathic individuals should show greater increases in HR and response speed from the no-incentive practice period to the reward-only phase of the task than nonpsychopathic individuals should show. To help rule out the possibility that any evidence of strong BAS in psychopathic individuals is secondary to weaker BIS activity during the reward-only phase, we also assessed BIS activity (increase in number of SCRs from the no-incentive practice period) during the reward-only phases.

On the basis of the response modulation model, we hypothesized that low-anxiety psychopathic individuals would show response facilitation, or poor response modulation to punishment cues (i.e., faster response speed after center red light onsets), whereas low-anxiety controls would show greater response inhibition to punishment cues (i.e., slowed response speed after center red light onsets at the beginning of each passive-avoidance phase). Response speed after center red light onsets (cues for possible punishment), rather than response speed change to actual punishment, was used as the measure of response modulation because (a) the onset of the center red light was more salient in that it occurred after 1-min of continuous re-

[1] In this study, we defined low-anxiety participants on the basis of scores on the Welsh Anxiety Scale. Although the term *anxiety* is in the title of this scale, it is more accurate to say that the scale is a measure of anxiety in addition to maladjustment in general and taps into five major content areas: (a) problems in thinking and thought processes, (b) negative emotional tone, (c) permission and lack of energy, (d) personal sensitivity, and (e) deviant thought processes (Greene, 1980). For ease of exposition, however, we refer to participants defined according to Welsh Anxiety Scale scores as having high or low anxiety.

sponding for reward, and (b) some participants made no punished responses and many made only a few. Because no differences in reactivity of BAS and BIS activity are predicted by the response modulation model, we expected no group differences in HR or response speed during the reward-only phases and no differences in EDA during the passive-avoidance phase.

It should be noted that the poor behavioral inhibition to punishment cues in psychopathic individuals predicted by the poor response modulation model could also be predicted from the weak-BIS and strong-BAS models. Table 1 reflects these predictions; however, according to these latter models, poor inhibition to the punishment cues should occur only in the context of weak SCR to the punishment cues (the weak-BIS model) or greater response speed/HR to the reward cues (the strong-BAS model). If response facilitation occurs in the absence of a strong BAS or weak BIS, the results would be most consistent with the poor response modulation model. To use the constructs from Gray's model, the poor response modulation model predicts that psychopathic individuals' faster response speed to the punishment cue (center red light) is caused not by the BAS per se but by the increased NAS arousal from the punishment input, which facilitates the ongoing BAS activity (see Figure 1).

Method

Participants

Participants were 63 Caucasian prison inmates at Oakhill Correctional Institution, a minimum-security prison in southern Wisconsin. After determining each inmate's eligibility for participation by briefly reviewing his institution file, we selected potential participants by identifying every fifth name on the institution roster. Inmates were excluded from participation if they were older than age 40 or younger than age 18, scored below the fourth-grade level on standardized achievement tests, or were identified as actively psychotic or taking psychotropic medication. Approximately 10% of the inmates contacted for interviews and 5% of the inmates called back for the experiment declined to participate. Four participants (2 low-anxiety psychopathic inmates, 1 high-anxiety psychopathic inmate, and 1 low-anxiety control) were excluded from the analyses because their electrocardiograph (ECG) data were insufficient for analysis.

Measures

Psychopathy was assessed by using the revised Psychopathy Checklist (PCL–R; Hare, 1985). Detailed and extensive reliability and validity information on the checklist is available from Hare et al. (1990). Participants who scored 30 or above were designated psychopathic (n = 29), and those who scored 20 or below were designated controls (n = 29). Participants were further divided into high- and low-anxiety subgroups by using a median split on the Welsh Anxiety Scale (Welsh, 1956). Items were embedded in a longer questionnaire assessing impulsivity and sociability. Finally, to obtain estimates of participants' Wechsler Adult Intelligence Scale—Revised (WAIS–R) Full Scale IQs, we administered the Shipley Institute of Living Scale (Zachary, 1986).

Task and Apparatus

The response apparatus was a modification of one used by Fowles et al. (1982). Five response buttons were equally spaced in a 180° semicircle around a center button equidistant from the five outer buttons. Located 2 cm above each of the outer buttons at 45° angles were a small red light and a small green light, each 1 cm in diameter and spaced 5 mm apart. A red light and a green light spaced 5 mm apart and each 2 cm in diameter were set 2 cm above the center button at 45° angles. The semicircle from the center button to the outer edge of the outer lights had a radius of 17.5 cm.

The participant's task, in general, was to press the outer buttons as quickly as possible as the green lights above them were lit. Outer lights were lit if the participant pressed the center button, so the participant continuously responded by pressing either the center button or one of the outer buttons. Because of the rapid nature of the task, feedback was provided only after every five responses. If the participant responded quickly enough, a high (665 Hz) tone sounded for 250 ms. If he responded too slowly, no tone sounded. A small speaker enclosed beneath the response panel provided the auditory feedback.

The entire task comprised a 2-min baseline at the beginning of the task, followed by four 2-min task periods, with 1-min rest periods between each response period. In addition, a 1-min practice period during which no incentives were available followed the baseline. Each 2-min task period consisted of two phases. The first was designed to induce and measure BAS activation and the second, to assess response modulation and BIS activation. During the entire first minute (the reward-only phase), the center green light was lit, signifying that only outer green lights could appear. Participants won 5 cents each time they made five responses in the allotted time. The outer green lights appeared in a quasirandom, unpredictable sequence. The response speed that qualified for reward was adjusted after every five responses to approximate 80% success. At the end of the reward-only phase, the center green light went off for 500 ms, after which both the center green light and red light were lit, signaling the start of the passive-avoidance phase. During this phase, participants could continue to win 5 cents after every five responses, but the red lights next to the green lights could be lit before participants pressed the button. If participants failed to withhold their buttonpress when a red light was lit, a low (94 Hz) tone sounded for 250 ms, and they lost 25 cents. Regardless of whether participants pressed the outer response button when an outer red light appeared, pressing the center button again turned the light off and initiated the next trial.

To ensure that participants did not develop a strategy of pausing before releasing the center button, lights changed to red only after their finger left the center button. Green lights changed to red five times during each passive-avoidance phase. The timing of the red lights during the passive-avoidance phase was varied so that participants would be less likely to anticipate their onset. The time criterion for rewards used in the passive-avoidance phase was set so that participants were able to continue winning at a rate comparable to that of the reward-only phase if they continued responding as quickly, but it also ensured that it was possible for them to lose substantial amounts of money if they failed to inhibit their responding appropriately.

Psychophysiological Recording of Data

Data processing and storage were controlled by a Zenith (Model ZF-158-41) computer. The following physiological signals were processed by using a Beckman R511A dynograph and digitized using a Scientific Solutions 12-bit, 40 kHz analog-to-digital board.

HR. Electrocardiographs were recorded by attaching an electrode to a rib on the right and left side of the torso using adhesive collars and Beckman Standard 1-cm^2 Ag–AgCl electrodes with Spectra 360 electrode gel as the conducting medium. Before placement of the electrodes, the participant's skin was abraded by using gauze moistened with rubbing alcohol. The ECG was recorded by using a Beckman Type 9806A AC coupler with a 0.1-s time constant. The output was directly digitized and recorded by the computer at a rate of 100 Hz.

SC. Skin conductance was recorded from the second and third digits of the nondominant hand by using Beckman Standard 1-cm^2 Ag–AgCl electrodes with a Unibase and saline mixture as the conducting medium (see Fowles et al., 1981, p. 235, for the formula). Skin conductance signals were recorded through a constant-voltage Lykken Skin Conductance Coupler. The output was directly digitized and recorded by the computer at a rate of 20 Hz.

Procedure

All inmates who met the selection criteria were contacted about participating in a study involving an initial 1 ½-hr interview and several behavioral tasks that provided the opportunity to earn money. Participants were paid $3.00 for the interview and were recontacted in 1–4 weeks for the present experiment.

All participants were interviewed prior to the testing day. Details of this interview have been provided by Smith and Newman (1990). Briefly, interview questions were designed to allow an assessment of each participant on the PCL–R items. In addition, questions concerning socioeconomic status, family background, drug and alcohol use, family history of substance abuse and legal difficulties, and head injuries were asked.

All participants were naive to the experimental situation and had not participated in any previous experiments. Each participant was tested on the task by one of two male experimenters unaware of the participant's psychopathy and anxiety status. After the participant signed a consent form on the testing day, his color blindness was assessed by using the Ishihara Tests for Colour Blindness (Ishihara, 1989). If the participant had normal color vision, he was asked to wash his hands as a prelude to attaching SC electrodes. The participant then completed the questionnaire containing the Welsh Anxiety Scale in a separate waiting room. After a minimum of 20 min and a maximum of 30 min had passed, the participant returned to the experimental room, the ECG electrodes were attached to his ribs, and a photo-plethysmograph was attached to his nondominant thumb. After the necessary adjustments on the psychophysiological recording equipment had been made, the inmate was told that a 2-min baseline measurement of his physiology would be taken.

After the baseline measurement, the experimenter began reading the task instructions. On receiving basic instructions for pressing buttons to lights, the participant began the 1-min practice period. At the end of this period, the experimenter told the participant for the first time that he would be performing the task for money. At this point, the experimenter explained the monetary contingencies to the inmate by using a standard set of instructions and laid out poker chips on a table next to where the inmate sat during the task. The participant was reminded again to try to win as much money as possible and was asked if he had any questions prior to starting the task. All participants began the task with $1.00, or 20 poker chips. The experimenter informed the participant of his winnings at the end of each rest period.

At the end of the task, the inmate was told that his winnings would be deposited in his institution account. The inmate then completed the Shipley scale and was thanked for participating.

Data Reduction

HR. The off-line ECG was edited with a computer program that allowed visual inspection of the ECG array to identify and omit invalid heart periods. The remaining heart periods were converted to second-by-second HR (F. K. Graham, 1978).

SC. Skin conductance responses were identified from the digitized data by a Pascal implementation of the WAVE SC scoring program developed by Strayer and Macias (1982). Responses greater than or equal to .05 μS were identified. Three electrodermal measures of BIS activity were used. The first BIS measure consisted of the mean number of SCRs during the passive-avoidance phase. The second BIS measure consisted of the mean amplitude of SCRs (including those with an amplitude of 0) beginning between 1 and 3.0 s after the onset of the center red light (the signal of potential punishment) at the start of each passive-avoidance phase. Examination of SCR traces for the task revealed that the SCRs in response to the center red light onsets were participants' largest SCRs and were clearly delineated from ongoing SC activity. Finally, BIS was also assessed during reward-only phases by using mean number of SCRs.

Analytic Strategy

As described earlier, the a priori hypotheses for all three models involved comparisons of low-anxiety groups. As suggested by Keppel (1991, p. 127), if the homogeneity of variance assumption was met for the overall analysis, the mean square error and degrees of freedom from the overall analysis were used in the planned comparison. Otherwise, the Welch Test was used, with mean square error and degrees of freedom computed on the basis of only the groups being compared. Thus, unless otherwise indicated, any a priori comparison with less than 53 degrees of freedom from Study 1 and less than 60 degrees of freedom from Study 2 involved the use of the Welch Test. Given the number of analyses conducted, alpha was set at .01 for all analyses except a priori comparisons of the low-anxiety groups to reduce the possibility of Type I error.

Many of the hypothesis-testing analyses involved assessing changes in the variables of interest from one part of the task to another. Analysis of covariance (ANCOVA) was used in each case.[2] Although no a priori hypothesis predicted effects across trials, trials was used as a repeated measures factor in all analyses to evaluate (post hoc) the possibility of change over trials. The Tukey honestly significant difference (HSD) test was used for post hoc comparisons, with $p < .05$ as the criterion for statistical significance.

Many analyses involved three or more levels of repeated measures factors. If a significant effect involving repeated measures factors is reported below, the effect remained statistically significant after the p level was adjusted by using the Huynh–Feldt epsilon correction.

Results

Participant Characteristics

Table 2 lists group means and standard deviations for age, PCL–R scores, WAIS–R IQ estimates, and Welsh Anxiety Scale scores. The median Welsh Anxiety Scale score for the sample was 8.0, and this value was used to divide participants into high- and low-anxiety subgroups. A Psychopathy × Anxiety analysis of variance (ANOVA) was used to test for group differences in age and intelligence. No significant main effects or interactions were found. Comparison of the low-anxiety groups on both variables revealed no statistically significant effects.

[2] Difference scores were considered as a strategy, but given the likelihood of multicollinearity introduced by repeated measures on difference scores, an ANCOVA was used instead. In two cases, the ANCOVA assumption of homogeneity of regression slopes was not met, but the use of difference scores did not change the results. In only one case were the results using ANCOVA different from those using difference scores: The a priori comparison of low-anxiety groups for increase in response speed from the practice period to the reward-only phases was statistically nonsignificant when an ANCOVA was used but was statistically significant when difference scores were used, $F(1, 29) = 4.92$, $p < .05$.

Table 2
Participant Characteristics and Passive-Avoidance Errors as a Function of Group in Study 1

	Psychopathic individuals				Controls					
	Low anxiety (n = 13)		High anxiety (n = 16)		Low anxiety (n = 19)		High anxiety (n = 10)		$F(1, 54)$	
Variable	M	SD	M	SD	M	SD	M	SD	Group	Interaction
Age	27.0	3.9	27.4	6.4	28.8	5.1	26.2	5.9	<1.0	1.11
PCL–R	33.0	1.5	33.1	2.5	11.1	5.0	14.6	2.5	471.18***	3.41*
IQ	93.9	11.0	98.4	11.9	99.9	9.4	93.9	8.0	<1.0[a]	3.46*
Welsh Anxiety Scale	4.8	2.4	16.1	6.1	4.4	2.4	18.6	10.0	<1.0	<1.0
Avoidance errors	3.8	1.7	4.8	3.6	4.8	3.6	3.2	2.6	<1.0	2.40

Note. PCL–R = revised Psychopathy Checklist; IQ = Shipley Institute of Living Scale estimate of Wechsler Adult Intelligence Scale—Revised Full Scale IQ. "Group" refers to the psychopathy group main effect; "interaction" refers to the Psychopathy × Anxiety interaction.
[a] For F values for IQ, there are 1 and 52 degrees of freedom.
* $p < .10$. *** $p < .000001$.

Preliminary Analyses

Several preliminary Psychopathy × Anxiety ANOVAs were conducted to assess whether the groups differed on the behavioral and psychophysiological variables prior to the presentation of incentives. Analysis of median response speed during the practice period revealed a significant Psychopathy × Anxiety interaction, $F(1, 54) = 8.89$, $p < .01$. Post hoc analyses indicated that high-anxiety controls responded significantly more slowly than did low-anxiety controls and high-anxiety psychopathic inmates. Separate analyses of median HR during the second minute of the pretask baseline measurement, median HR during the practice period, and number of SCRs during the second minute of the pretask baseline measurement and the practice period revealed no statistically significant effects.[3] For baseline measures, the second minute was chosen for analysis because this was thought to represent a more stable estimate of participants' baseline psychophysiological responding than the first minute or both minutes combined.

Hypothesis-Testing Analyses

Table 3 summarizes the results of the hypothesis-testing analyses and their implications for each of the three models.

Passive-avoidance analyses. Passive-avoidance errors (buttonpresses to red lights during the passive-avoidance phase) were used to assess passive avoidance. There were no statistically significant effects, including the a priori comparison of low-anxiety groups (see Table 2).

BAS analyses. In order to evaluate BAS activity, median response speed during each reward-only phase was used as a repeated measure variable (trials) in the Psychopathy × Anxiety × Trials mixed-model ANCOVA; median response speed during the practice period was used as the covariate. There was a statistically significant Psychopathy × Anxiety × Trials interaction, $F(3, 162) = 5.36$, $p < .005$. Although psychopathic inmates showed faster response speed than did controls during the reward-only phase, $F(1, 53) = 5.09$, $p < .05$, this effect fell short of the alpha level set for non-a-priori analyses. A priori comparison of low-anxiety groups was not statistically significant, $F(1, 53) = 2.32$; however, there was a significant Psychopathy × Trials interaction, $F(3, 90) = 7.00$, $p < .001$. As Figure 2 illustrates, the Psychopathy × Trials effect indicates that response speed to the reward cues increased faster across trials for low-anxiety psychopathic inmates than for low-anxiety controls, $F(1, 30) = 8.39$, $p < .01$, for the linear effect.

It is possible that low-anxiety psychopathic inmates sped up more than low-anxiety controls in general over the four trials of the task and that this acceleration was not specific to their response to reward. To address this possibility, we conducted a Psychopathy × Trials analysis post hoc on the low-anxiety groups, with median response speed during each passive avoidance phase serving as the repeated measures factor. The Psychopathy × Trials effect was not significant, $F(3, 90) = 1.11$, suggesting that low-anxiety psychopathic inmates' speeding up over trials was specific to the reward-only phases.

Heart rate during the reward-only phases provided another means of evaluating BAS activity. An ANCOVA was conducted by using median HR during the practice period as the covariate and median HR during each of the four reward-only phases as the repeated measures factor (trials). There were no statistically significant effects in the overall analysis. A priori comparison of the low-anxiety groups revealed a statistical trend for low-anxiety psychopathic inmates (adjusted mean HR = 94.1) to show greater increase in their HR to the reward cues compared

[3] Because research has shown that psychopathic offenders disproportionately abuse drugs and alcohol in comparison with nonpsychopathic offenders (e.g., Smith & Newman, 1990), preliminary analyses were also conducted comparing psychopathic individuals and controls on the number of lifetime drug and alcohol symptoms, as assessed by the National Institute of Mental Health Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1981). Although psychopathic individuals had significantly more drug and alcohol symptoms than did controls ($p < .05$), there were no significant correlations between number of drug or alcohol abuse symptoms and any of the dependent variables in either study for the low-anxiety groups alone or the high- and low-anxiety groups combined.

Table 3
Summary of Comparisons Between Low-Anxiety Psychopathic Individuals (P) and Controls (C) by Three Models of Psychopathy

Outcome	Result	Model supported
Study 1: Reward versus passive avoidance		
Reward only		
Response speed	P > C	Strong-BAS model
Heart rate	P ~> C	Strong-BAS model
SCR (number)	P = C	Supports all three models
Punishment cue		
Response speed	P > C	Supports all three models
SCR (amplitude)	P = C	Strong-BAS and response modulation models
Passive avoidance		
SCR (number)	P = C	Strong-BAS and response modulation models
Errors	P = C	Contradicts all three models
Study 2: Active versus passive avoidance		
Active avoidance only		
Response speed	P = C	Weak-BIS and response modulation models
Heart rate	P = C	Weak-BIS and response modulation models
SCR (number)	P = C	Supports all three models
Punishment cue		
Response speed	P = C	Contradicts all three models
SCR (amplitude)	P = C	Strong BAS and response modulation models
Passive avoidance		
SCR (number)	P = C	Strong-BAS and response modulation models
Errors	P < C	Contradicts all three models

Note. BAS = behavioral activation system; SCR = skin conductance response; BIS = behavioral inhibition system.

with low-anxiety controls (adjusted mean HR = 90.9), $F(1, 28) = 3.15$, $p < .10$.

BIS analyses. To evaluate BIS activity, we first analyzed mean SCR amplitude to the onset of the four punishment cues (center red lights). A Psychopathy × Anxiety × Trials ANOVA revealed a significant psychopathy main effect, reflecting smaller SCR amplitude in psychopathic inmates than in controls, $F(1, 54) = 8.99$, $p < .005$ (Table 4). A priori comparison of the low-anxiety groups was not statistically significant, $F(1, 24) < 1.0$.

Number of SCRs during the passive-avoidance phases provided another measure of BIS activity. Number of SCRs during each reward-only phase was used as a covariate in the Psychopathy × Anxiety × Trials ANCOVA; number of SCRs during each passive-avoidance phase was the repeated measures factor. No statistically significant effects were found either in the overall analysis or in the a priori comparison of low-anxiety groups. Analysis of the number of SCRs during the reward-only phases revealed no significant differences between the low-anxiety psychopathic and control groups.

Response modulation analyses. Change in response speed in the first 5 s after the onset of the punishment cues (center red lights) that accompanied the four passive-avoidance phases provided the measure of response modulation. An ANCOVA was conducted in which mean response speed in the 5 s after the onset of each punishment cue at the start of the four passive-avoidance phases served as the repeated measures factor and mean response speed in the 10 s before the onset of each of the four punishment cues served as the covariate. A significant Psychopathy × Anxiety effect was found, $F(1, 53) = 9.92$, $p < .005$. Results of the Tukey HSD test revealed significant group differences only between high-anxiety psychopathic inmates and high-anxiety controls; the latter sped up overall, whereas the former slowed down (Figure 3). A priori comparison of the low-anxiety groups revealed a significant psychopathy main effect, $F(1, 28) = 4.57$, $p < .05$, indicating that low-anxiety psychopathic inmates sped up more than did low-anxiety controls after the punishment cue onsets (see Figure 3). Although the Psychopathy × Trials interaction was not statistically significant, $F(3, 89) = 1.67$, $p > .10$, observation of the trial-by-trial data revealed that the group effect for the low-anxiety groups appeared to be due primarily to differences in response to the onset of the first punishment cue: Low-anxiety psychopathic inmates sped up, whereas low-anxiety controls slowed down.

Discussion

The purpose of Study 1 was to assess psychopathic individuals' approach and avoidance motivation in the context of a passive-avoidance task. Contrary to predictions, there were no passive-avoidance differences between low-anxiety psychopathic participants and controls.

Consistent with the strong-BAS model, low-anxiety psychopathic inmates displayed significantly greater increases in response speed across reward-only trials, indicating that the group differences in BAS activation became more pronounced as the task progressed. Post hoc analysis of the low-anxiety groups' median response times during passive-avoidance phases re-

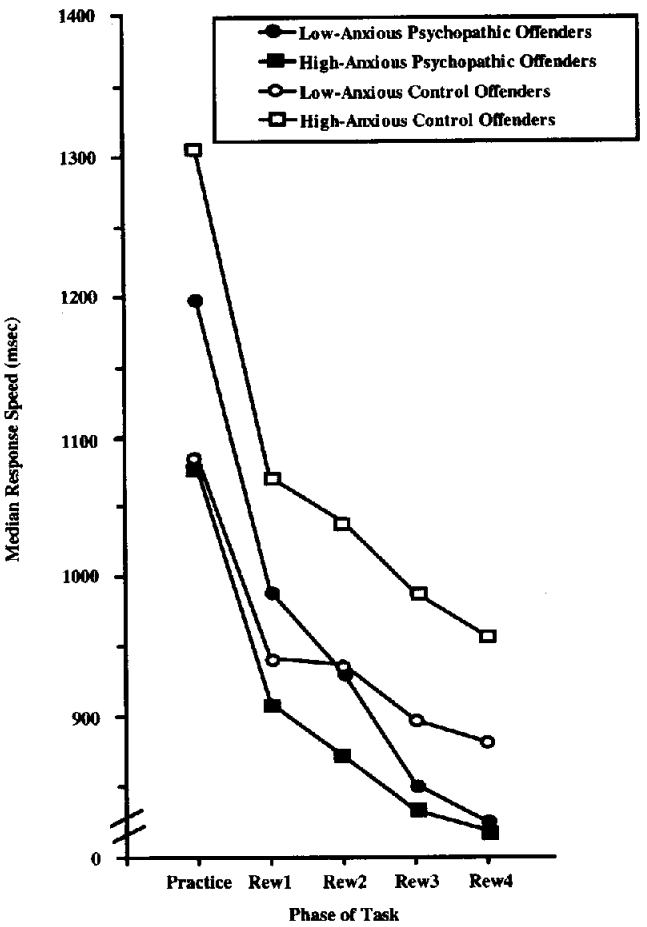

Figure 2. High- and low-anxiety psychopathic offenders and control group unadjusted means for median response speed across reward-only trials in Study 1. Median response speed during the practice phase is included for purposes of comparison with reward-only trials. Rew1 to Rew4 = first to fourth reward-only phases.

vealed no significant Psychopathy × Trials interaction, suggesting that low-anxiety psychopathic inmates' speeding up over trials was specific to the reward-only phases. Also consistent with the strong-BAS model, low-anxiety psychopathic inmates displayed a statistical trend toward greater HR increase to the reward cues. Comparison of the low-anxiety groups revealed significantly greater speeding up after the primary punishment cue in psychopathic inmates than in controls after activation by reward. Although this finding is consistent with the predictions of all three models, the low-anxiety psychopathic inmates' exaggerated response to reward suggests that their weak inhibition may have been due to their exaggerated reward response in the absence of differences in SCR (BIS index) to the punishment cue, thus supporting the strong-BAS model. However, given that evidence for a strong BAS in low-anxiety psychopathic inmates developed over trials and their disinhibition to punishment cues was the most marked on the first trial, it may be that an exaggerated response to reward is not necessary for disinhibition to punishment cues to occur. As the response modulation model predicts, the establishment of a dominant response set for reward may be all that is necessary.

Although there were no differences between the low-anxiety groups in change in number of SCRs from the reward-only to the passive-avoidance phase or in SCR amplitude to the onset of the center red lights, analyses combining low- and high-anxiety participants revealed significantly smaller SCR amplitude to the onset of the center red lights in psychopathic inmates compared with controls.

Study 2

Like Study 1, the second study was designed to assess psychopathic individuals' BAS and BIS activity in the context of a passive-avoidance task. In Study 2, however, BAS activation was generated by active-avoidance cues. That is, instead of consisting of reward-only cues, the first phase consisted of cues for active avoidance, and in the second phase, a passive-avoidance contingency was superimposed on the active-avoidance contingency. According to models presented by Gray (1975, 1987) and Fowles (1980), the BAS is activated by active avoidance as well as by reward cues. So, Gray and Fowles proposed that BAS activation by active-avoidance cues should have the same physiological and behavioral consequences as BAS activation by reward cues. It is important to note, however, that Gorenstein and Newman (1980) suggested that only BAS activation by reward cues produces differences between psychopathic individuals and controls. Similarly, one of the central tenets of the response modulation model is that switching motivational sets (i.e., reward/punishment) is what differentiates the performances of low-anxiety psychopathic individuals and controls. Thus, although neither model makes explicit predictions regarding active-avoidance cues, Study 2 was designed to test the generality of these models to activation by active-avoidance cues. It was also designed to test the generality of the weak-BIS model.

Table 1 outlines the predictions from each model for each dependent variable used in the study. Predictions were identical to those of Study 1. If, as Gray (1987) asserted, the BAS is activated similarly by active avoidance and reward cues, results similar to those found in Study 1 should emerge in Study 2. On the basis of the weak-BIS model, we expected low-anxiety psychopathic individuals to show relatively smaller electrodermal responding after punishment cues at the onset of and during the passive-avoidance phase. If the hypersensitivity to reward model of Gorenstein and Newman (1980) is more generally a strong-BAS model, low-anxiety psychopathic individuals would show greater response speed and HR increase to the active-avoidance cues than would low-anxiety controls. If the response modulation model generalized, low-anxiety psychopathic individuals would show faster response speed after punishment cues at the onset of the passive-avoidance phase than would low-anxiety controls, that is, in the absence of differences in BAS and BIS activity.

As in Study 1, BAS activation was assessed in the first phase of each trial, but this time active-avoidance cues were used to elicit BAS activation. BIS activation and passive avoidance were assessed during the second phase of each trial—the passive-avoidance phase. Heart rate and response speed were again used

Table 4
Skin Conductance Responding Across Task Phases as a Function of Group in Study 1

	Psychopathic individuals				Controls					
	Low anxiety (n = 13)		High anxiety (n = 16)		Low anxiety (n = 19)		High anxiety (n = 10)		$F(1, 54)$	
Variable	M	SD	M	SD	M	SD	M	SD	Group	Interaction
Baseline	3.08	2.29	2.13	2.22	2.42	1.84	3.60	1.51	<1.0	3.86*
Practice	5.85	2.30	6.44	3.01	6.26	2.66	6.90	3.21	<1.0	<1.0
Reward	5.25	1.54	5.75	2.20	5.28	3.25	6.13	2.49	<1.0	<1.0
Passive avoidance	5.90	1.42	5.78	2.03	5.75	2.84	6.88	2.58	<1.0	<1.0
Punishment cue onset	.16	.12	.14	.11	.23	.26	.43	.36	8.99**	3.50*

Note. Values are the number of skin conductance reponses during the phase, with the exception of the punishment cue onset variable. For this variable, the value refers to skin conductance response amplitude. All values reflect means across four task trials. "Group" refers to the psychopathy group main effect; "interaction" refers to the Psychopathy × Anxiety interaction.
* $p < .10$. ** $p < .005$.

as indexes of BAS activity; EDA was used as the index of BIS activity.

Method

Participants

Participants were 71 Caucasian prison inmates at the Oakhill Correctional Institution. The eligibility criteria were the same as in Study 1. Approximately 10% of the inmates contacted for interviews and 9% of the inmates called back for the experiment declined to participate. Five participants (3 low-anxiety psychopathic inmates and 2 low-anxiety controls) with unusable ECG data and 2 participants whose behavioral data were unusable because of equipment malfunction (1 high-anxiety and 1 low-anxiety psychopathic inmate) were excluded from the analyses. Thus, 33 psychopathic inmates and 31 controls remained.

Measures

The measures used in Study 1 were also used in Study 2.

Task and Apparatus

The response apparatus was identical to that used in Study 1. However, in Study 2, if the participant responded *too slowly,* a high (665 Hz) tone sounded for 250 ms. If he responded fast enough, no tone sounded.

The structure of the task was the same as in Study 1. During the active-avoidance phase, the green light near the center response button was lit, again signifying that only outer green lights could appear. However, this time participants lost 5 cents each time they failed to make five responses in the allotted time. The response speed that qualified for avoiding high tones after every five responses was adjusted to approximate 80% success. During the passive-avoidance phase, participants could continue to lose 5 cents after every five responses if they were responding too slowly, but the red lights next to the green lights could also be lit shortly after the green lights were lit. If participants hit buttons associated with the red lights, a low (94 Hz) tone sounded for 250 ms and they lost 25 cents.

Recording of Psychophysiological Data

The procedure for recording psychophysiological data was identical to that used in Study 1.

Procedure

The procedure in Study 2 was identical to that in Study 1, with two exceptions. First, all participants began the task with 140 poker chips worth $7.00. Second, the participants completed the questionnaire that included the Welsh Anxiety Scale and the Shipley Institute of Living Scale on the interview day. None of the inmates from Study 1 participated in Study 2; as in Study 1, all participants were naive to the experimental situation and had not participated in any prior experiments.

Data Reduction

The data reduction methods for all psychophysiological measures were identical to those used in Study 1.

Analytic Strategy

Data were analyzed in the same manner as in Study 1.

Results

Participant Characteristics

Table 5 lists group means and standard deviations for age, PCL–R scores, WAIS–R IQ estimates, and Welsh Anxiety Scale scores. The median Welsh Anxiety Scale score for the sample was 10.0, and this value was used to divide participants into high- and low-anxiety subgroups. This median value differed slightly from the median Welsh Anxiety Scale score used in Study 1 (8.0). However, it seems unlikely that these differences are clinically meaningful, given that the Study 1 median represented a t score of 47 and the Study 2 median, a t score of 50 (J. R. Graham, 1990). A Psychopathy × Anxiety ANOVA was used to test for group differences in age and intelligence. For age, the Psychopathy × Anxiety interaction was statistically significant, $F(1, 60) = 4.52, p < .05$. Planned comparison of the low-anxiety groups indicated that the controls were significantly older than the psychopathic inmates, $F(1, 34) = 5.90, p < .05$. Post hoc analysis of the Psychopathy × Anxiety interaction revealed that the high-anxiety psychopathic inmates were significantly older than the low-anxiety psychopathic inmates.

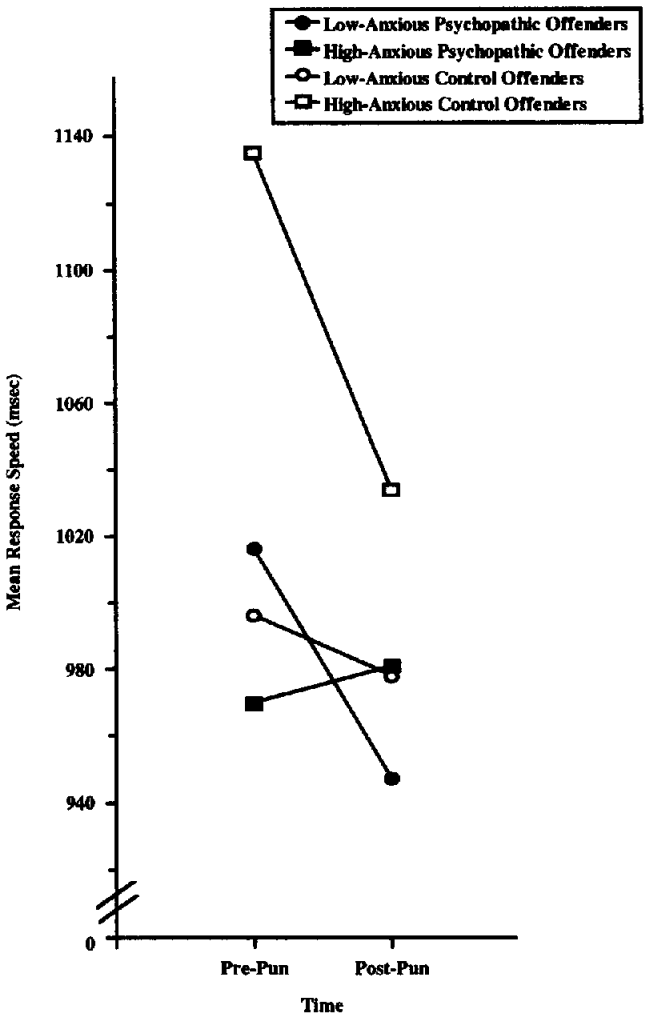

Figure 3. High- and low-anxiety psychopathic offenders and control group unadjusted means for change in response speed to the onset of the center red light (the most salient punishment cue) in Study 1. Pre-Pun = mean response speed in the 10 s before the onset of the center red light across trials; Post-Pun = mean response speed in the 5 s after the onset of the center red light.

Analyses of WAIS–R IQ scores revealed that the controls' scores were significantly higher than the psychopathic inmates' scores, $F(1, 60) = 4.92$, $p < .05$. Planned comparisons of the low-anxiety groups also indicated higher IQ scores for the controls than for the psychopathic inmates, $F(1, 34) = 7.97$, $p < .01$.

In order to explore the possibility that group differences in age and intelligence were associated with the behavioral and psychophysiological variables of interest, correlational analyses were computed. WAIS–R IQ estimate correlated significantly ($p < .05$) with mean SCR amplitude to center red lights ($r = .25$), indicating greater SCR amplitude by higher IQ participants. Thus, WAIS–R IQ was used as a covariate in the SCR amplitude analysis.

Preliminary Analyses

As in Study 1, several preliminary Psychopathy × Anxiety analyses were conducted to assess whether the groups differed on the behavioral and psychophysiological variables prior to the presentation of incentives. No statistically significant effects were found.

Hypothesis-Testing Analyses

Table 3 summarizes the results of the hypothesis-testing analyses and their implications for each of the three models.

Passive-avoidance analyses. As in Study 1, total passive-avoidance errors were used to assess passive avoidance. There was a Psychopathy × Anxiety interaction, $F(1, 60) = 5.62$, $p < .05$, that fell short of the alpha level set for all non-a-priori analyses. A priori comparison of the low-anxiety groups revealed a significant main effect for psychopathy, $F(1, 60) = 4.18$, $p < .05$, with *controls* making more passive-avoidance errors than psychopathic inmates (see Table 5).

BAS analyses. Changes in response speed and HR from the practice period to the active-avoidance phases were used to assess BAS activity in an ANCOVA, as in Study 1. No statistically significant effects involving group were found for any of the analyses, including the a priori comparisons of the low-anxiety groups.

BIS analyses. As in Study 1, BIS activity was first evaluated by examining mean SCR amplitude to the onset of the four punishment cues (center red lights). An ANCOVA was conducted, with estimated WAIS–R IQ as the covariate. No statistically significant effects emerged in the overall analysis or in the a priori comparison of the low-anxiety groups.

Another measure of BIS activity included number of SCRs during each of the passive-avoidance phases as a repeated measures factor, with number of SCRs during each active-avoidance phase used as a covariate. There were no statistically significant findings from the Psychopathy × Anxiety × Trials ANCOVA or the a priori comparison of the low-anxiety groups.

Response modulation analyses. Response modulation was assessed as change in response speed to the punishment cues (center red lights) by using an ANCOVA, as in Study 1. No statistically significant effects were found in either the overall analysis or a priori comparisons.

Cross-Study Analyses

For comparison of groups across studies, post hoc analyses were conducted. Alpha was set at .01 for all analyses to reduce the possibility of Type I error. Psychopathy × Anxiety × Trials × Condition analyses were used. There were no statistically significant Group × Condition effects for any of the dependent variables.

Discussion

The purpose of Study 2 was to assess psychopathic individuals' BAS and BIS activity in the context of a passive-avoidance task. In contrast to Study 1, however, BAS activation was generated by active-avoidance instead of reward cues. Unexpectedly, low-anxiety psychopathic inmates committed significantly fewer

Table 5
Participant Characteristics and Passive-Avoidance Errors as a Function of Group in Study 2

	Psychopathic individuals				Controls					
	Low anxiety (n = 17)		High anxiety (n = 16)		Low anxiety (n = 19)		High anxiety (n = 12)		$F(1, 60)$	
Variable	M	SD	M	SD	M	SD	M	SD	Group	Interaction
Age	24.3	4.3	30.1	6.2	28.9	6.5	28.6	5.6	1.14	4.52*
PCL–R	33.9	2.5	35.3	3.0	16.3	3.9	15.9	3.8	471.39***	1.09
IQ	95.3	12.6	96.8	12.0	105.9	8.1	98.9	12.5	4.92*	2.25
Welsh Anxiety Scale	4.8	2.8	21.0	6.4	6.2	3.5	20.4	5.4	<1.0	<1.0
Avoidance errors	5.2	3.3	7.4	3.8	7.8	4.6	5.4	3.4	<1.0	5.62*

Note. PCL–R = revised Psychopathy Checklist; IQ = Shipley Institute of Living Scale estimate of Wechsler Adult Intelligence Scale—Revised Full Scale IQ. "Group" refers to the psychopathy group main effect; "interaction" refers to the Psychopathy × Anxiety Interaction.
* $p < .05$. *** $p < .000001$.

passive-avoidance errors than did low-anxiety controls; none of the other behavioral or psychophysiological results revealed statistically significant group effects.

General Discussion

The present studies were conducted to evaluate three different explanations of psychopathic individuals' poor passive avoidance. Using constructs from Fowles's (1980) adaptation of Gray's (1975) two-factor learning theory, we hypothesized from three models that psychopathic individuals would show a strong BAS, a weak BIS, or poor response modulation.

Although there was some evidence in support of all three models, the strong-BAS model and the poor response modulation model specific to activation by reward cues (but not active-avoidance cues) appeared to fit the data best. In Study 1, low-anxiety psychopathic inmates showed an exaggerated response to reward cues and weak behavioral inhibition after punishment cues. Specifically, they responded significantly faster than controls to reward cues as the task progressed and showed a statistical trend toward greater HR during the reward-only phases. In addition, they responded faster than the low-anxiety controls after the onset of the most salient punishment cues (center red lights). Given that the low-anxiety psychopathic inmates did not show evidence of attenuated SCR after these punishment cue onsets, weak BIS activity did not appear responsible for their weak behavioral inhibition. Similarly, although low-anxiety psychopathic inmates showed an exaggerated response to reward cues, this did not appear responsible for their response disinhibition to punishment cues. Their exaggerated response to reward only became apparent over trials. In contrast, trial-by-trial changes in response speed to punishment cues revealed that the low-anxiety psychopathic inmates showed the most disinhibition to the punishment cues relative to the low-anxiety controls on the first trial, in which they sped up and low-anxiety controls slowed down. If excessive reward activation was really responsible for low-anxiety psychopathic inmates' response disinhibition to punishment cues, they should have shown this excessive reward activation before the onset of that first punishment cue on the first trial. Consistent with the expectations of the response modulation model, establishment of a dominant response set for reward, but not an *exaggerated* response to reward, appeared to be associated with psychopathic individuals' subsequent response disinhibition.

Given that passive-avoidance phases requiring the avoidance of punishment cues occurred between each successive reward-only phase, low-anxiety psychopathic inmates' increasingly fast response to reward cues across reward phases could reflect a summation of both reward and punishment motivational influences (Newman, Kosson, & Patterson, 1992). Such an interpretation seems less likely, however, when one recalls that low-anxiety psychopathic individuals' speeding up over trials was specific to the reward-only phases; a similar relative speeding up over trials did not occur across the passive-avoidance phases. Therefore, their greater response to reward over trials most likely reflects a buildup of reward activation over time. Nonetheless, a follow-up study involving only reward trials seems warranted to address this issue experimentally.

Because there are no other published reports of psychopathic individuals' being evaluated in a pure reward-only context, there are no data from the literature with which to compare our results. However, the finding of exaggerated reward activation in psychopathic individuals is consistent with Gray's (1987) assertion that antisocial individuals may be characterized by BAS hypersensitivity. Quay's (1988) theory that conduct-disordered children (a group at highest risk for adult antisocial behavior) are characterized by an overactive BAS is also consistent with the current findings. A BAS hypersensitivity specific to reward cues may be associated with personality characteristics of psychopathic individuals as well as their antisocial behavior in certain situations (see Arnett, 1997, for an elaboration of this idea).

The disinhibition to punishment cues we observed in low-anxiety psychopathic individuals is consistent with Newman et al.'s (1990, Experiment 1) finding that when punishment occurs after reward activation, low-anxiety psychopathic individuals slow down less after punishment than do low-anxiety controls. This failure to slow down after an unexpected environmental

event (i.e., punishment cue) may reflect difficulty in switching attention once it has been focused on a motivationally significant goal (in this case, reward).

Low-anxiety psychopathic individuals did not show poor passive avoidance, despite being overactivated by reward and having a disinhibited response to punishment cues. It is possible that these factors interfere with passive avoidance only on tasks in which the demands for information processing are significant. In our studies, there was little to be learned about task performance after passive-avoidance errors. In studies that have demonstrated poor passive avoidance in psychopathic individuals in reward–punishment contexts (e.g., Newman & Kosson, 1986, Experiment 1; Newman et al., 1990, Experiment 1), participants could learn something significant about task performance with each passive-avoidance error, because the error informed them which number on the go/no-go discrimination task would result in punishment. Response disinhibition after punishment in these types of tasks should be more likely to interfere with participants' ability to process the numbers associated with punishment and make them less likely to avoid buttonpresses to those numbers in the future.

Another possible interpretation of our failure to find passive-avoidance deficits in low-anxiety psychopathic inmates relates to the latency between the punishment cues and the need to avoid punishment. If Study 1 had required participants to inhibit responding soon after their disinhibited response to punishment cues, low-anxiety psychopathic inmates may have committed more passive-avoidance errors than low-anxiety controls. In two of the four passive-avoidance phases, the first potential punishment occurred 10 s after the initial punishment cue onset (center red light); in the other two phases, it occurred after 5 s. By the time the first possible punishment occurred, low-anxiety psychopathic inmates may already have recovered from their disinhibition after the initial punishment cue onset and therefore been able to avoid punishment just as effectively as controls. Recent research is consistent with this notion that longer time intervals between punishment or punishment cues and the need to avoid punishment leads to better passive avoidance in psychopathic individuals, and shorter intervals lead to worse passive avoidance (e.g., Arnett et al., 1993; Newman et al., 1990, Experiment 1).

Another factor that may have contributed to our failure to find passive-avoidance deficits in psychopathic inmates, in contrast to reports from several studies in the literature, is the nature of our control group. The control group consisted of criminal offenders who, presumably, were incarcerated because of difficulty inhibiting their behavior appropriately for some reason (poor passive avoidance). It is possible that such offenders show passive-avoidance deficits similar to those of psychopathic individuals, or even greater than those of psychopathic individuals under certain circumstances, but that the situational factors underlying their difficulties are different. The results from Study 2 suggest that low-anxiety incarcerated offenders who are not psychopathic may be especially likely to show passive-avoidance deficits when responding to an active-avoidance contingency. This interpretation is consistent with Chesno and Kilmann's (1975, high-stimulation condition) report of passive-avoidance deficits in low-anxiety *controls* relative to low-anxiety psychopathic individuals in a paradigm that, like our Study 2, included both active- and passive-avoidance contingencies.

Further evidence that the nature of the control group may be an important factor in revealing passive-avoidance deficits in psychopathic individuals comes from studies using Lykken's maze. Lykken (1957) and Schmauk (1970) reported poor passive avoidance in psychopathic individuals, but their comparison groups consisted of nonincarcerated controls. In an attempt to replicate Lykken's study using an incarcerated control group, Schachter and Latane (1964) reported significantly worse passive avoidance in incarcerated psychopathic individuals than in controls. However, this interpretation was based on within-group changes; no statistically significant between-groups differences in passive-avoidance errors were reported. Psychopathic individuals may show passive-avoidance deficits on Lykken's maze only when compared with normal controls and not when compared with incarcerated controls.

The foregoing observations, the data from the present studies, and other studies that have failed to show passive-avoidance deficits in psychopathic individuals (e.g., Arnett et al., 1993; Chesno & Kilmann, 1975, high-stimulation condition) call into serious question the generality of passive-avoidance deficits in psychopathic individuals. If we restrict our definition of passive-avoidance deficits to mean group differences between incarcerated psychopathic individuals and incarcerated nonpsychopathic offenders to control for non-psychopath-specific impulsivity, it seems most likely that psychopathic individuals show passive-avoidance deficits in relatively restricted situations in which (a) a dominant response set for reward has been established before the introduction of the passive-avoidance contingency, (b) relatively rapid avoidance after responses to punishment or punishment cues is required, and (c) significant information processing for improving task performance is necessary after passive-avoidance errors.

Despite the absence of group differences in passive avoidance, our finding of exaggerated response to reward in low-anxiety psychopathic individuals may be related to other characteristics of the psychopathic person. Although psychopathic individuals' impulsivity, sensation seeking, and difficulty delaying gratification have most commonly been explained as manifestations of their insensitivity to punishment (cf. Newman et al., 1992), all could be mediated by hypersensitivity to reward. More thorough measurement of empathy, sensation seeking (especially related to disinhibition and boredom susceptibility), and moral reasoning (see Levenson, 1990) in future research in this area would make it possible to determine whether these core characteristics of psychopathy are associated with behavioral and physiological hypersensitivity to reward. This approach would have the advantage of bridging the more reductionistic behavioral and physiological approaches to psychopathy with the more interpersonal aspects elucidated by Levenson and his colleagues (e.g., Levenson, Kiehl, & Fitzpatrick, 1995).

Data from electroencephalographic research on depression may also be relevant to our finding of hypersensitivity to reward in low-anxiety psychopathic inmates. Phenomenologically, depressed/anxious patients are characterized by excessive social withdrawal and strong avoidance tendencies. This is in marked contrast to psychopathic individuals, who typically show excessive and often inappropriate approach behaviors (e.g., pursuit

of a desired sexual partner to the point of rape and excessive use of stimulating drugs). In contrast to depressed/anxious individuals, psychopathic persons may be disproportionately represented on the approach or reward-seeking end of the motivational continuum. As Davidson (1992) asserted, left frontal activation is associated with heightened sensitivity to reward cues and greater engagement with the environment. If, as Henriques and Davidson (1991) argued, left frontal hypoactivation is a biological substrate of the deficit in reward motivation that is often characteristic of depressed individuals, left frontal hyperactivation could be predicted as a biological substrate of hypersensitivity to reward cues in psychopathic individuals.

An anomalous finding in Study 1 that requires interpretation is that high-anxiety controls sped up significantly more than high-anxiety psychopathic inmates and about the same as low-anxiety psychopathic inmates after punishment cues. Thus, it appears that, at least in our study, weak response inhibition after punishment cues is not specific to low-anxiety psychopathic individuals. However, different processes may mediate the response facilitation of these two groups. First, examination of Figure 2 reveals that compared with high-anxiety controls, low-anxiety psychopathic inmates showed greater response speed increase to reward cues. Second, Table 4 shows that high-anxiety controls had, on average, an SCR amplitude to the punishment cues more than 2 ½ times that of low-anxiety psychopathic inmates. We confirmed this observation statistically by comparing the two groups by using the Tukey HSD post-hoc test. It could be that, whereas BAS activation by reward cues makes it difficult for low-anxiety psychopathic individuals to inhibit their responding when punishment cues appear, excessive BIS activation to the punishment cues makes it difficult for high-anxiety controls to inhibit. Although BIS activation typically causes behavioral inhibition, examination of Gray's (1987) model (see Figure 1) indicates that the opposite effect could result. Given the positive inputs from the BIS to the NAS, excessive BIS activation could activate the NAS, which in turn could accentuate ongoing behavior at the time—reward-seeking behavior in the case of our study. Such an interpretation is consistent with Wallace, Bacharowski, and Newman's (1991) concept of anxious impulsivity. These investigators found that neurotic or high-anxiety introverts displayed disinhibited responding when they were significantly activated by punishment cues. As a final comment, the finding of similarities between low-anxiety psychopathic inmates and high-anxiety incarcerated controls is not unprecedented in the psychopathy literature (e.g., Chesno & Kilmann, 1975, low-stimulation condition). Our understanding of criminal behavior might be broadened by more detailed and systematic examinations of the behavioral and psychophysiological similarities and differences between low-anxiety psychopathic inmates and high-anxiety nonpsychopathic individuals.

Consistent with the weak-BIS model, compared with controls in Study 1 psychopathic inmates showed significantly smaller mean SCR amplitude to the most salient punishment cues (the center red lights). However, this difference was due not so much to abnormally low SCR amplitude among the psychopaths as to aberrant scores by high-anxiety controls, whose mean SCR amplitude was almost double that of any other group, including low-anxiety controls (see Table 4). The absence of significant EDA differences between low-anxiety groups confirms the influence of high-anxiety participants, especially high-anxiety controls, on overall group differences. Consistent with this observation, we found similar aberrant SCR responding to punishment among high-anxiety controls relative to other groups in a previous study (Arnett et al., 1993). These findings highlight the importance of controlling for anxiety to ensure that findings of attenuated EDA to punishment in psychopathic individuals are not due to abnormal responding by high-anxiety controls rather than psychopathic individuals.

An important consideration is the possible relationship between low-anxiety psychopathic individuals' exaggerated response to reward and weak response inhibition to punishment cues in Study 1 and past reports (Hare, 1978) of attenuated SCR in anticipation of punishment in psychopathic individuals. Analysis of SCRs during the reward-only phases and in response to punishment cue onsets in Study 1 revealed no differences between low-anxiety groups. Although it cannot be ruled out definitively that a weak BIS may mediate exaggerated reward responding and disinhibition in some situations, our data suggest that it is not responsible for low-anxiety psychopathic individuals' exaggerated reward seeking or disinhibited response to punishment cues.

One paradoxical result from our research is that we found more group differences in Study 1 than in Study 2, despite using a task that generated fewer errors in performance relative to the task used in Study 2. From a purely psychometric point of view, the more difficult nature of the task used in Study 2 should have produced more group differences. However, even though more group differences were observed in Study 1, the only significant differences in passive avoidance were found in Study 2. Thus, Study 2 did produce a group difference in the area (errors) that should be most affected by the difficulty level of the task, even though the difference was the opposite of the expected one.

A limitation of our research is that only Caucasian male inmates were included in both studies. Thus, our findings clearly cannot be generalized to non-Caucasian or female samples of inmates. Ideally, future research should include different racial groups as well as female offenders.

Another limitation of our study is that, because Study 1 did not result in group differences in passive avoidance between low-anxiety psychopathic inmates and controls, we cannot say that the hypersensitivity to reward and disinhibition to punishment cues we observed are mechanisms underlying psychopathic individuals' poor passive avoidance. Only a future study demonstrating these phenomena in the context of poor passive avoidance in psychopathic individuals can address this issue definitively. Nonetheless, as described earlier, the findings from Study 1 seem relevant for understanding other characteristics of psychopathic persons.

Despite its shortcomings, our research makes six contributions. First, Study 1 is the first controlled experimental demonstration of psychopathic individuals' exaggerated reward responding in a relatively pure reward situation. Furthermore, our results suggest that activation by reward, rather than a weak BIS, interferes with low-anxiety psychopathic individuals' ability to inhibit their responding to cues for punishment. Our findings are consistent with clinical descriptions indicating an exaggerated craving for excitement in psychopathic individuals (McCord & McCord, 1964) and empirical reports characterizing psycho-

pathic persons as engaging in excessive appetitive activities (Smith & Newman, 1990; Zuckerman, 1978). Second, the results from both studies complement recent research showing that anxiety moderates the relationship of psychopathy (e.g., Arnett et al., 1993; Newman et al., 1992; Newman et al., 1990; Newman et al., 1985) and impulsivity (Zinbarg & Revelle, 1989) to performance on tasks using reward and punishment incentives and standardized measures of neuropsychological functioning (Smith, Arnett, & Newman, 1992). Third, the passive-avoidance data from Study 2 are consistent with Chesno and Kilmann's (1975, high-stimulation condition) finding that passive avoidance was better in low-anxiety psychopathic individuals than in low-anxiety controls in a task with active- and passive-avoidance contingencies. Fourth, we found that the significantly smaller amplitude SCRs to punishment cues among psychopathic inmates in Study 1 appeared to be due primarily to the aberrant responding of high-anxiety controls. This suggests that previous reports of weak SCR to punishment in psychopathic individuals may have been due to the unusual response of high-anxiety controls rather than the response of psychopathic individuals. Nonetheless, more research is needed to evaluate whether our finding will generalize to situations in which more aversive punishments (e.g., shock and loud tones) are used. Fifth, our demonstration of hypersensitivity to reward among low-anxiety psychopathic individuals is consistent with results from our earlier study (Arnett et al., 1993) demonstrating greater HR reactivity to reward versus punishment feedback in low-anxiety psychopathic individuals compared with low-anxiety controls. The results also complement our recent report (Arnett, Fischer, & Newby, 1996) suggesting a possible exaggerated reward response in children with attention deficit–hyperactivity disorder (ADHD), a group at high risk for psychopathy (Lilienfeld & Waldman, 1990). Using the same task that we used in Study 1 of the present research, we found that ADHD children who took a placebo showed significantly faster response speed to reward cues than did ADHD children who took Ritalin. Finally, although hypersensitivity to reward, a weak BIS, and poor response modulation are all observed in some psychopathic groups in some situations, none of the three appears sufficient in itself to cause poor passive avoidance in psychopathic individuals. In addition to understanding the significance of these response features for poor passive avoidance, researchers need to clarify the relationship between these features and other characteristics of the psychopathic individual. It will also be important for future behavioral and autonomic psychophysiological research to elucidate the necessary conditions for poor passive avoidance not only for psychopathic inmates but also for other groups of offenders (e.g., low- and high-anxiety nonpsychopathic groups). The amount of information processing required after passive-avoidance errors, the level of prior reward activation, the type of activation (e.g., by reward or by active-avoidance cues), the disinhibited characteristics of control groups, and the immediacy of the need to avoid punishment after punishment or punishment cues are all worthy avenues for research.

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Received March 3, 1995
Revision received June 27, 1996
Accepted July 8, 1996 ■