

Deficits in Behavioral Inhibition Predict Treatment Engagement in Prison Inmates

Diana Fishbein · Monica Sheppard ·
Christopher Hyde · Robert Hubal · David Newlin ·
Ralph Serin · George Chrousos · Salvatore Alesci

Published online: 13 January 2009

© American Psychology-Law Society/Division 41 of the American Psychological Association 2009

Abstract Many inmates do not respond favorably to standard treatments routinely offered in prison. Executive cognitive functioning and emotional regulation may play a key role in treatment responsivity. During intake into treatment, inmates ($N = 224$) were evaluated for executive functioning, emotional perception, stress reactivity (salivary cortisol), IQ, psychological and behavioral traits, prior drug use, child and family background, and criminal histories and institutional behavior. Outcome measures included program completion, treatment readiness, responsivity and gain, and the Novaco Reaction to Provocation Questionnaire. Relative deficits in behavioral inhibition significantly predicted treatment outcomes, more so than background, psychological, or behavioral variables, and other neurocognitive and emotional regulatory measures. Future replications of these results have potential to improve assessment and treatment of offenders who are otherwise intractable.

Keywords Correctional treatment · Inmates · Neurocognition · Emotional regulation · Responsivity

Identification of underlying mechanisms in differential responses to treatment for dysregulated behaviors is critical for more effective tailoring of intervention approaches to specific needs of distinctive inmate subgroups (Andrews & Bonta, 2006). Although resources are too scarce to fully match treatments to individuals, at a minimum, the ability to identify characteristics and conditions that compromise treatment responsivity is necessary for the development of interventions that more intensively and effectively target existing deficits. A first step toward a better understanding of mechanisms underlying correctional treatment effects is the elucidation of factors that predict the ability of inmates to engage, persist, and show attitudinal and behavioral improvement in response to existing programs.

Evidence is mounting to suggest that integrity of particular executive cognitive functions (e.g., disinhibition, risky decision making, inattention) and emotional regulation (e.g., facial recognition, emotional responses to threat stimuli, psychophysiological stress responses) may represent neural processes underlying variability in responses to treatment programming for behavioral disorders (e.g., Fishbein et al., 2006; Nixon, Paul, & Phillips, 1998). A few existing studies have identified deficits in these functions that, for example, specifically predict response to treatment for drug abuse (Aharonovich, Nunes, & Hasin, 2003; Bauer, 2001; Forman et al., 2004). Findings provide potentially important clues in the search for conditions that may interfere with intervention efficacy for types of psychopathology that involve high risk behaviors. Intact executive functions and emotional regulation may, in fact, be a prerequisite for processing and

D. Fishbein (✉) · M. Sheppard · R. Hubal · D. Newlin
RTI International, 6801 Eastern Avenue, Suite 203, Baltimore,
MD 21224, USA
e-mail: dfishbein@rti.org

C. Hyde
Bioassessments, Inc., Elkton, MD 21921, USA

R. Serin
Carleton University, 1125 Colonel By Drive, Ottawa, ON K1S
5B6, Canada

G. Chrousos
National Institute of Child Health and Development, NIH, Bldg.
10, Rm. 2D46, 10 Center Dr., Bethesda, MD 20892-1284, USA

S. Alesci
National Institute of Mental Health, Bethesda, MD 20892, USA

executing curriculum materials from any treatment program that invokes higher order cognitive abilities, such as cognitive-behavioral therapies, which are commonly employed in correctional settings. Participants with these skill deficiencies may not be as likely to benefit from programs that do not first address them. Because executive function impairments are malleable (Hermann & Parente, 1996; Manchester, Hodgkinson, & Casey, 1997; Riggs, Greenberg, Kusche, & Pentz, 2006; Rothwell, La Vigna, & Willis, 1999), there is potential for affected individuals to respond favorably to treatment approaches that are specifically targeted to existing deficits (Fals-Stewart, 1993; Grohman & Fals-Stewart, 2004).

NEUROCOGNITIVE AND EMOTIONAL DYSFUNCTION AND DYSREGULATED BEHAVIOR

Impairments in regulation of executive functions and emotional stress responses have been implicated in the development of dysregulated behaviors such as conduct disorder, heightened aggression, rule-breaking, and drug abuse (Deckel, Hesselbrock, & Bauer, 1995; Fishbein, 2000; Giancola, Martin, Tarter, Pelham, & Moss, 1996; Giancola, Mezzich, & Tarter, 1998; Moffitt, Lyman, & Silva, 1994; Seguin, Pihl, Harden, & Tremblay, 1995; Tarter, Mezzich, Hsieh, & Parks, 1995). Aspects of executive functioning that are relevant to an understanding of dysregulated behaviors include behavioral disinhibition, poor decision-making ability, relative insensitivity to negative outcomes, cognitive inflexibility, impaired problem solving and planning, and emotion misattributions (Mirsky & Siegel, 1994). Aberrant physiological responses to stress (e.g., cortisol release) are also implicated given that their regulation is integral to the establishment and integrity of executive function components (Raine, Lencz, Bihle, La-Casse, & Colletti, 2000); a neural network supports a feedback loop in which cognitive demands modify stress responses that, in turn, modify cognitive processing and resultant behaviors (Elliott, Friston, & Dolan, 2000; Liberzon et al., 2000; Steinberg, 2005). Dysfunction of these cognitive and affective domains potentially explains the impulsivity, emotional instability, unresponsiveness to threats of punishment, rules and laws, and negative consequences, as well as an excessive need for stimulation and reward often observed in offender and inmate populations (Dawes et al., 2000; Raine, 1993; Reiss, Miczek, & Roth, 1994; Rogers & Robbins, 2001). The possibility that these behavioral tendencies may further interfere with the ability to respond to treatment makes conceptual sense.

Brain systems that support development and function of these neurocognitive and emotional regulatory functions are

profoundly influenced by experiences of severe and/or chronic stress (Beers & De Bellis, 2002; Bremner, 1999; De Bellis, Keshavan, Spencer, & Hall, 2000; Steckler & Holsboer, 1999). The pattern and efficacy of an individual's responses to stress have been related to the propensity for high risk behaviors (De Bellis, 2002; Giaconia et al., 2000; Kreek, Schlussman, Bart, Laforge, & Butelman, 2004; Nemeroff, 2004). Thus, it is relevant that offender populations have a particularly high prevalence of adversity relative to the general population (Fishbein, 2000; Martell, 1992; Volavka, Martell, & Convit, 1992) and that aberrant stress responses (e.g., low cortisol responsivity) often characterize aggression, psychopathy, and impulsivity (Moss, Vanyukov, Yao, & Kirillova, 1999; Oosterlaan, Geurts, Knol, & Sergeant, 2005; Taylor, Carlson, Iacono, Lykken, & McGue, 1999). Such deficits are considered to reflect an underarousal of emotional systems that regulate abilities to process, interpret, and react to social cues (Walker et al., 1991), potentially impeding the ability to respond favorably to interventions that do not address such impairments.

NEUROCOGNITIVE AND EMOTIONAL DYSFUNCTION AND TREATMENT ENGAGEMENT

Damage to structures that constitute the neural circuitry for cognitive-emotive processes (e.g., by stress or adversity [Davidson, 1994; De Haan, Luciana, Malone, Matheny, & Richards, 1994]), can impair regulatory operations during aversive conditioning, reward-related feedback, and risk-related decision making (see Critchley, Elliot, Mathias, & Dolan, 2000). Theoretically and to some extent empirically, these functional impairments interfere with abilities to integrate new information provided by standard treatment programs and execute the necessary behavioral changes via shifting behavioral strategies based on new information, and/or inhibiting affective responses that promote maladaptive behaviors. This scenario may be especially true for Cognitive Behavioral Therapy-based treatment approaches which form the basis for many standard correctional treatments and reflect Evidence-Based Practice (Serin, 2005). Processing of curriculum materials from Cognitive Behavior Therapy relies on the ability of participants to (a) be cognizant of and responsive to potential negative consequences of their behavior, (b) inhibit inappropriate behavioral responses, and (c) understand and act on the benefits of deliberate and cautious decision making (Blume, Davis, & Schmaling, 1999; Blume, Marlatt, & Schmaling, 2000). Although Cognitive Behavioral Therapy is generally considered to be the most effective approach available to reducing behavioral problems in this population (e.g., Holbrook, 1997;

Nicholaichuk, Gordon, Gu, & Wong, 2000), there is considerable variation in treatment response; a significant subgroup does not respond favorably as indicated by high recidivism and relapse rates, as well as poor attendance, compliance, and retention. Preliminary studies suggest that poor neurocognitive and emotional regulatory function may at least partially explain this variability.

THE CURRENT STUDY

Additional studies are needed to identify neurocognitive and emotional regulatory characteristics that may predict or underlie treatment engagement in prison inmates other than basic background factors which are usually not malleable. To fill this important gap, this study sought to determine whether deficits in executive functions and emotion perception and regulation compromised the ability of inmates to engage, persist, and show attitudinal and behavioral improvement in response to a standard Cognitive Behavioral Therapy-based correctional therapy. We hypothesized that poor treatment engagement, responsiveness, and completion, and related behaviors (e.g., aggression) would be predicted by relative deficits in the ability to effectively process and apply curriculum materials reflected by aberrant processing of neurocognitive and affective stimuli, and attenuated cortisol responses at baseline.

METHOD

Participants

Three medium/maximum custodial level facilities in the State of Maryland's Department of Public Safety and Correctional Services (DPSCS) were selected to participate in this study on the basis of programmatic similarities to ensure continuity and uniformity of treatment duration, type and modality of the program, treatment providers, and other environmental factors. A total of 224 male inmates were recruited over a 2.5-year period from a pool who volunteered for the treatment "Thinking for a Change," as part of the "treatment-as-usual" (TAU) procedure in the state prisons. This protocol was approved by both the RTI Institutional Review Board and the State of Maryland. There was no comparison group given the primary hypothesis that variability in engagement relative to a standard treatment approach would be predicted by neurocognitive and emotional regulatory functioning. Inmates who volunteered were first asked to complete an IQ test (Multidimensional Aptitude Battery); 27 inmates with an IQ below 75 were excluded. Inmates with records indicating that they had mental retardation, dementia, amnesia,

and delirium or who were illiterate were not referred to the study investigators by the facilities due to their inability to understand consent and interference by these conditions on their performance. Inmates who met eligibility criteria for study participation were recruited through identification numbers provided by the facility to include only those between 21 and 49 years old with a minimum of 18 months remaining on their sentence to avoid the stress of pre-release preparations and potential transfers. Participants reflected the ethnic diversity of the offender population in the state system. Those eligible were scheduled for testing and signed the full consent form.

Treatment Program Participation

After baseline assessments, inmates began their participation in the facilities' Cognitive Behavioral Therapy-based program. Cognitive Behavioral Therapy is the most widespread treatment approach used in U.S. correctional institutions to reduce violence, drug abuse, sexual offending, and other behavioral disorders common in inmates (Holbrook, 1997; Nicholaichuk et al., 2000). Cognitive Behavioral Therapy is designed to help inmates develop impulse control, manage anger, and learn new behavioral responses to real-life situations. The underlying assumption is that learning processes play an important role in the development and continuation of antisocial behavior and can be used to help individuals enhance their ability to exert self-control. Cognitive Behavioral Therapy is designed to help patients *recognize* situations in which they are likely to become agitated or aggressive, *avoid* these situations when appropriate, and *cope* more effectively with a range of problems and behaviors associated with aggression.

In the three participating facilities, the DPSCS offers a series of three treatment phases that meet for 90 min twice a week, totaling 50 sessions. The first phase, consisting of 20 sessions delivered by social workers, is called "entry point" and involves curricula on "Thinking, Deciding, Changing." Entry point blends a decision making and cognitive restructuring modality (a self-reflective process to search for triggers of misconduct) into a cognitive-behavioral modality (an external, skill-building process) for self change. Although participation is voluntary, the focus is on inmates who are considered "high risk", including those with multiple incarcerations, history of violence, poor institutional adjustment, and impulsive behavior.

The present study focused on performance and retention during this first session only given routinely high drop-out rates. The sole system requirements for retention in the program are that inmates do nothing to undermine the group process and remain free of serious infractions.

Immediately after inmates completed the first phase, treatment performance was evaluated by staff on the inmate using treatment readiness and gain and responsiveness scales, program completion, and aggressive reactions to provocation.

Procedures

Consenting inmates underwent baseline testing for several complementary dimensions of neurocognition and emotional regulation and conditions that putatively influence their development. Background and psychological instruments were used to characterize inmates with respect to clinically relevant traits (e.g., drug abuse, impulsivity, aggression, and psychopathy). As measures of engagement and responsiveness, social workers provided evaluations and inmates provided self reports. Background, psychological, and neurocognitive and emotional regulation measures were assembled into an assessment battery to determine which variable set best predicted treatment engagement indicators.

Measures

General Intellectual Functioning

The *Multidimensional Aptitude Battery* (MAB; Sigma Assessment Systems, 1999) was self-administered to identify general intellectual deficits that may have resulted from head injury or other causes and to isolate the predictive value of executive functioning to treatment engagement assuming the hierarchical nature of complex functions (Zelazo, Carter, Reznick, & Frye, 1997). When these more basic functions are impaired, integrity of executive functions can be expected to suffer as well. Test-retest reliabilities on separately timed test administrations obtained values for total scores of .95 for Verbal, .96 for Performance, and .97 for the Full Scale.

Behavioral and Psychological Questionnaires

A condensed version of the widely used *Addiction Severity Index* (ASI) (McLellan et al., 1992) was used to assess nature and extent of prior drug use, background factors such as socioeconomic status (Hollingshead rating), religious preference, race/ethnicity, family history of drug use, alcoholism, and mental illness, head injuries, child abuse, medical and psychological status, and other demographic factors. The *Self Report Psychopathy Checklist* (Hare, 1991; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Levenson, Kiehl, & Fitzpatrick, 1995) is a self-report version of the Psychopathy Checklist-Revised (PCL-R) showing high correlation with the original test and good

reliability (Hart et al., 1995). This instrument generates a Primary (e.g., tendency to lie, manipulativeness, and lack of remorse) and Secondary (e.g., impulsivity, quick-temperedness, and lack of long-term goals) psychopathy score. Also, the *Reactive-Proactive Aggression Questionnaire* (Raine et al., 2006) provided an assessment of predatory (proactive) and impulsive (reactive) aggressive inmates. Confirmatory factor analyses indicated that the two-factor structure produced a very good fit and item-total correlations ranged from .41 to .57 for the proactive scale, .45–.58 for the reactive scale, and .41–.60 for the total scale. All the three scales have internal reliabilities in excess of 0.83 (see Raine et al., 2006). The *Early Trauma Inventory*, shown to have acceptable validity and internal consistency (Bremner, Bolus, & Mayer, 2007; Bremner, Vermetten, & Mazure, 2000), was also used as a global indicator of incidences of trauma, witnessing violence, and child abuse and neglect, as well as a subscale measuring symptoms of PTSD.

Executive Cognitive Function Tasks

Knowledge regarding the functional neuroanatomy of cognitive task performance has potential implications for understanding the pathophysiology of behavioral dysregulation and provides a mechanistic account of how interventions mediate their effects (Bremner, 2003; Charney & Deutch, 1996). Thus, the tasks selected for this study have been neuroimaged (either PET or fMRI) to provide information on what areas of the brain are recruited during performance. Importantly, only dimensions of executive functioning that have been associated with behavioral dysregulation and that are conceptually related to treatment responsiveness were used; additional tasks would have been informative, however there were severe time constraints for testing given the prison environment. Neurocognitive and emotional perception tasks were computerized and nonintrusive. Inmates were tested individually at a time that did not interfere with meals, lock-downs, or counts. Questionnaires and neurocognitive tasks (see Table 1) were administered in the mornings (tasks were administered first to avoid the effects of fatigue on cognitive functioning) and the stress task was conducted in the afternoon.

The *Cambridge Decision Making Task* (CDMT; Rogers et al., 1999a, 1999b) was developed to dissect the cognitive components involved in decision making and measures willingness to take risks and relative sensitivity to rewards versus penalties. It has been found to have sensitivity and specificity in high-risk populations (see Fishbein et al., 2005b for a complete description) and reliably activates a portion of the PFC and the anterior cingulate involved in social skills, impulse control, and sensitivity to rewards (Fishbein et al., 2005a). In this task, larger rewards are

Table 1 Test battery

	Measurement instruments
Predictor variables	
Executive cognitive performance	Cambridge Decision Making Task, Logan Stop-Change Task, Stroop Color-Word Interference Task
Emotion perception	Facial Recognition Task
Emotional regulation	Speech Task with Cortisol Sampling SCL-90
Potential moderator variables	
General intellectual functioning	Multidimensional Aptitude Battery
Demographics, drug and alcohol use, family history, etc.	Background Inventory (adapted from the ASI)
Psychopathy traits	Self Report Psychopathy Checklist
Aggression type	The Reactive-Proactive Questionnaire
Past failures/successes	Success Inventory
Events that could alter treatment response	Events Checklist
Lifetime stress exposures	Early Trauma Inventory
Outcome variables	
Change in behavioral control	Novaco: Reactions to Provocation
Institutional behaviors	OBSCIS Data on Good Conduct Points
Treatment response/progress	Responsivity and Gain Scales from Social Workers
Treatment completion	Completion of first session

SCL-90 Symptom Checklist-90, ASI Addiction Severity Index, OBSCIS Offender Based State Correctional Information System

always associated with the least likely outcome, thus capturing the conflict inherent in risk-taking situations. A tendency to take more risks in all conditions in pursuit of a large reward and with a willingness to tolerate an even higher probability of a large loss was expected to predict a lack of positive behavioral change in response to the experimental intervention. Performance measures generated by this task included in our model were percentage of the riskiest decisions and mean reaction time for riskiest decision. Two outliers were discovered for mean reaction time on this task (skewness = 3.5). With their removal, skewness was significantly reduced (skewness = 1.1) and the distribution became more normalized; thus, analyses including this variable excluded these two subjects.

The *Stop-Change Task* (SCT) measures impulsivity and response shifting (Logan & Burkell, 1986 and see Fishbein et al., 2006 for full description) and has been shown to activate the right hemispheric anterior cingulate cortex, supplementary motor area, and inferior prefrontal and parietal cortices, which modulate error monitoring, interference control, and task management (Rubia et al., 2001). This task requires deep concentration, impulse control, timing, and the ability to shift responses in light of newly presented information (a novel stimulus). Measures from this task include percentage correct and reaction time (RT) for all the trials that included presentation of a new stimulus.

The *Stroop Interference Task* uses previously learned information to assess the three attributes of executive

frontal lobe function: complexity, a “nonroutine” nature, and the novel use of old information. Patients with frontal lobe damage are typically influenced by stereotypical thinking, which would interfere with the ability to produce the atypical responses required on the Stroop (Luria, 1980; Mesulam, 1986), and often experience difficulty with mental flexibility (Stuss & Benson, 1986). Studies suggest that the anterior cingulate (believed to be involved in aggression) is primarily recruited for performance on the Stroop (Bench et al., 1993; Pardo, Pardo, Janer, & Raichle, 1990). The primary score generated by the Stroop is the “interference score,” calculated by multiplying the first two conditions ($W = \text{word score}$ and $C = \text{color score}$) and dividing that product by the sum of those conditions to produce a “CW prime” score ($W \times C / W + C = CW'$). CW' is then subtracted from the sum of third condition (CW) score ($CW - CW' = \text{interference}$).

Emotion Perception

Research suggests that emotion-processing deficits lead to a distorted perception of social cues that has been associated with aggressive and disruptive behaviors (Crick & Dodge, 1996; Dodge, 1980). PFC impairment reduces inhibition of emotional behaviors that may be generated from these distorted perceptions; thus, measurement of emotion perception is critical. A *Facial Expressions of Emotion: Stimuli and Test* (FEEST; Ekman & Friesen, 1975) involving a facial recognition technique was used

due to its high level of validity and reliability. Emotion attribution has been directly related to the function of the amygdala (McClure et al., 2004; Whalen et al., 2001; Williams et al., 2004; Yang, Menon, Reid, Gotlib, & Reiss, 2003) and is shown to be impaired in individuals with externalizing disorders, such as ADHD, psychopathy, and drug abuse (Blair, Colledge, Murray, & Mitchell, 2001; Kornreich et al., 2003; Kossen, Suchy, Mayer, & Libby, 2002). This task requires that subjects identify one of six emotions (happiness, sadness, surprise, fear, disgust, and anger) that best describe the faces (10 for each expression). The scores produced included the number of errors in attributions of each emotional expression and total correct responses.

Emotional Regulation and Stress

The significant effect of public speaking on emotional and physiological stress responses has been well demonstrated (see Rohrmann, Hennig, & Netter, 1999). Inmates were instructed to make a 10-min persuasive speech providing justification to a parole board for an early release which was ostensibly judged by the research assistant according to how compelling and effective it was, and in terms of its formal aspects and content. Salivary cortisol levels, which correlate well with free serum cortisol levels, were measured during the treatment baseline to assess inmates' stress response to emotional stimuli (Yao, Moss, & Kirillova, 1998). The task was administered in the afternoon when cortisol is most stable. Two resting state samples of saliva (2 ml) were collected after an acclimation period and immediately before the speech. Two additional samples were taken 20 min after the speech, representing the peak cortisol response. Cortisol tends to rise about 20 min after a stressor and then falls precipitously, thus we were able to evaluate the ascending curve to assess the relationship between treatment engagement and extent of effective elevation in cortisol.¹

In order to evaluate the inmates' present emotional and psychological state which may influence stress responses, the *Symptom Checklist-90* (SCL-90) was administered immediately before this task (Derogatis, Rickels, & Rock, 1976). The Global Severity Index from the SCL-90 was used as a covariate in analyses of cortisol data.

¹ The saliva samples were stored at -20°C until processing. They were then centrifuged to remove particulate matter and extracted with hexane: ethyl acetate. Aliquots of the extract were evaporated to dryness in a rotary evaporator. The dried extract was redissolved in buffer and the cortisol content was determined using a highly sensitive RIA procedure with, ^{125}I -radiolabeled cortisol. Sensitivity of the method is 0.05 mg/dl using 0.2 ml of saliva (Esoterix Inc., Austin, TX).

Treatment Performance Indicators

Behavioral performance measures, questionnaires, official records, and evaluations from social workers were used (Table 1). Treatment readiness and gain and responsivity were evaluated by Social Workers using an instrument designed to assess Cognitive Behavioral Therapy preparedness and performance (Kennedy & Serin, 1997, 1999; Serin, 1998; Serin, Mailloux, & Kennedy, 2007). Each of these three domains is represented by a description and a 4-point rating scale with behavioral examples for each subscale. A Treatment Readiness clinical rating was adapted for use as a self report measure (see Casey, Day, Howells, & Ward, in press; Day et al., 2008; Serin et al., 2007) and has shown acceptable intraclass correlations (Y. Lee, unpublished data; L. Marshall, unpublished data). Subscales of this rating include: problem identification, macro treatment benefits, micro treatment benefits, treatment distress, treatment goals, treatment behaviors, behavioral congruency, and treatment support. Subscales were combined to produce a total readiness score for this performance domain (Cronbach's $\alpha = .73$ in the present study, and .83 in Serin et al., 2007). This instrument was administered at baseline, prior to neurocognitive assessments and treatment participation. Although this measure is technically not an outcome measure, it was included in a separate regression model to determine whether neurocognitive, emotional regulation, and background measures predicted the extent to which inmates perceived themselves to be ready for treatment.

Treatment indicators were further evaluated by social workers using a combination of the *Treatment Responsivity and Gain Scales* immediately after the first treatment phase. The Responsivity subscales reflect personality dimensions putatively related to treatment performance and include Callousness, Denial, Procrastination, Intimidation, Power and Control, Rigidity, Victim Stance and Procriminal Views (Cronbach's $\alpha = .85$). The Gain subscales reflect program participation and performance, such as Evidence of Increased Skills from Program, Disclosure in Program, Application of Knowledge, Application of Skills, Depth of Emotional Understanding of Program Content, Appropriateness of Behavior in Group, Participation, and Therapeutic Alliance (Cronbach's $\alpha = .90$). Items within each scale, in a 4-point Likert scale format (i.e., 0–3), were completed by social workers based on their observations of inmates participating in their treatment groups and in the research protocol. Social workers were instructed to consider *change* in behavior, skills, attitude from commencement to present evaluation in their responses. Descriptions were provided of each subscale; e.g., Evidence of Increased Skills From Program subscale: "This item considers the development of specific skills the

treatment program is intended to impart. A higher score indicates the inmate has exhibited the increased ability to do more than simply repeat in group that which has been demonstrated in previous group sessions.”

The *Novaco Reaction to Provocation* inventory (Novaco, 1994) was given at baseline and again after completion of each treatment phase. There are two parts, the first set of subscales assessing cognitive, arousal, and behavioral domains and the second assessing impulsive reaction, verbal aggression, physical confrontation, and indirect expression. This instrument is sensitive to behavioral change particularly in response to an intervention.

Noncompletion of the program due to disinterest, commission of an infraction and/or time in segregation, disruption to the group, lack of cooperation, or a related ‘negative’ reason was considered a poor treatment outcome. Noncompletion due to a ‘neutral’ cause such as obtaining a job, mandatory transfer, or other reason unrelated to noncompliance was excluded in analyses; their reasons for noncompletion were considered “neutral” and not indicative of a good or bad outcome. Inmates who dropped out of treatment were retained in the study and in all possible cases were tested according to the same schedule as those who remained in treatment. Also, only those who remained in treatment for at least two weeks were included in the analyses; treatment engagement and responsivity cannot be measured in inmates who discontinued early as exposure would be insufficient to obtain self evaluations and social workers would not have sufficient familiarity with the inmates to provide an evaluation.

RESULTS

Statistical Techniques

Primary analyses were restricted to performance during and immediately after the first treatment phase due to the large numbers who did not progress beyond that stage in treatment (see Table 2). Extraordinarily high attrition rates are common in prison treatment programs (Hiller, Knight, & Simpson, 1999); thus, one focus of this study was to determine whether neurocognitive and emotional regulation factors predicted attrition. Also note that sample sizes differ for various analyses given missing data. In all the analyses, $p < 0.05$ was considered significant.

A score ($t_2 - t_1$) reflecting change from baseline to the time period after the first treatment phase (whether or not participants completed) was calculated for the total score on the Reaction to Provocation measure as another outcome potentially related to treatment participation. A linear regression analysis was conducted with change scores as the dependent variable and background and cognitive measures as predictor variables.

Two salivary cortisol samples were obtained prior and subsequent to stress task administration during the treatment baseline; each subset of two samples was averaged to generate a baseline and posttest value. In cases where only one sample was viable, no averaging was conducted. Standardized values (z -scores) were calculated for baseline and posttest cortisol levels and relative change from baseline was computed by subtracting baseline from posttest z -scores. Analyses including cortisol measures were adjusted for Body Mass Index (BMI) given the inverse

Table 2 Sample descriptives

	<i>N</i>	Minimum	Maximum	Mean	Std. Deviation
Age of inmate	224	21	49	31.08	5.76
Verbal IQ	224	70	117	88.04	11.49
Performance IQ	224	70	136	90.46	15.11
Full scale IQ	224	70	122	88.50	12.37
Months in prison	221	1	251	38.67	40.53
Years of education	224	4	21	11.39	1.69
Weight (lbs)	220	135	360	202.37	36.94
Height (inches)	220	60	83	67.26	3.08
Treatment for alcohol abuse	225	0	20	.77	2.11
Treatment for drug abuse	225	0	24	1.36	3.14
PCL-primary psychopathy	222	12	54	32.29	8.60
PCL-secondary psychopathy	222	9	37	23.17	5.58
Proactive aggression	225	0	24	6.29	4.76
Reactive aggression	225	0	24	11.16	4.58
SCL-GSI	195	34	81	59.99	12.56

PCL Psychopathy CheckList, SCL-GSI Symptom Checklist-90 Global Severity Index

relationship between abdominal obesity (as measured by BMI) and HPA Axis function (Rosmond, Dallman, & Björntorp, 1998). BMI was calculated by dividing weight (kg) by height (meters squared). Also, since correlations between SCL-90 scores and cortisol levels were nonsignificant, cortisol change scores were used in regression models that only included adjustments for BMI.

A hierarchical multiple regression (“sequential regression” statistical model) was conducted to attempt to delineate which of the neurocognitive and emotional regulation variables best predicted each treatment outcome (dependent variable). This hierarchical approach to multiple regression allows determination of whether the final predictor—the neurocognitive or emotional regulation measure—entered into the regression equation can explain a significant proportion of the variance in the dependent variable over and above that of which is attributable to earlier data sets. It thus represents a conservative test of the effect of the variable(s) of interest in that variables first entered are maximized. For all models, background covariates included age, IQ, and education. Additional covariates were included in the secondary set of predictors based on a significant correlation with either treatment outcomes or neurocognitive and emotional regulation measures; thus, for all models, covariates included the primary psychopathy measure, reactive aggression, the global severity score from the SCL-90, family history of alcoholism, and average amount of previous drug usage. For Treatment Gain and Treatment Responsivity, and Reaction to Provocation outcomes, a history of physical abuse was added. Also for Reaction to Provocation, a history of emotional abuse was added based on their correlation, and the reactive and proactive aggression measures were excluded given their conceptual and empirical overlap with this outcome. An R^2 change, an F change, degrees of freedom (df), and the t statistic are reported to explain how much each individual predictor (i.e., neurocognitive or emotional regulation measure) contributed to the variation in the dependent variable controlling for both the first (background) and second (psychological and behavioral) sets of variables. And covariates that were significantly related to each outcome in the final models are included.

Treatment completion was coded as 0 or 1, reflecting completion or discontinuation of the first phase. Each treatment outcome was modeled as a linear function of each neurocognitive and emotional regulation measure and covariate factors using logistic regression methods. Covariates included in models were those that were significantly related to either neurocognitive/emotional regulation measures or outcomes and were entered in a stepwise fashion. Age, education, and IQ were entered into the model first, and history of physical abuse and

psychological problems in the past 30 days were entered second. Omnibus tests were conducted of the net effect of each neurocognitive and emotional regulation variable separately on treatment outcomes after adjustment for covariates. Thus, χ^2 , degrees of freedom, Cox & Snell R^2 (which provides a test of strength of the association and not percentage of variance explained in a logistic regression model) are reported for the third block of each model. Also, those covariates that were significantly related to each outcome in each model are shown.

Preliminary Analyses

Table 2 presents a description of the combined population including inmates from the three prison facilities combined. In total, 17.7% were Caucasian, 73.7% were African American, and 5.6% fell into other ethnic/racial categories. Approximately 44% reported having had depression, 38% anxiety, 44% violent behavior, and 13% had suicide attempts in their lifetimes. Data gleaned from official records (OBSCIS) indicated that 3% committed primarily property offenses, 32% were primarily drug offenders, and 65% had violent offense records. The following percentages reflect a history of psychopathology among immediate family members: alcoholism = 46.4%; drug abuse = 52.2%; and mental illness = 22.8%. A total of 28.3% ($n = 71$) reported severe head injury (>3 min unconsciousness). A preliminary analysis was conducted to determine whether history of head injury predicted treatment response; those with head injury had less treatment gain ($p < 0.05$) than those without head injury. The only neurocognitive or emotional regulatory dimension related to head injury was inhibition/response shifting and for all central neurocognitive and emotional regulation variables, their contributions to treatment gain appeared to be independent; interaction effects between head injury and each neurocognitive and emotional regulation domain on treatment outcomes were insignificant. On the other hand, those with head injuries had more psychological problems in the last 30 days, a greater history of physical, emotional, and sexual abuse, and higher levels of both proactive and reaction aggression.

Correlation analyses were conducted between predictor variables. Results suggest a relatively low level of multicollinearity: the Stroop Interference Score and cortisol levels ($R = .211$, $p < 0.05$), reaction time on the CDMT ($R = -.22$, $p < 0.01$), and percentage correct on the SCT ($R = .26$, $p < 0.005$). The percentage correct on the SCT was also related to the total score on the FEEST ($R = .19$, $p < 0.05$) and the CDMT reaction time ($R = -.16$, $p < 0.05$). Reaction time on the SCT and percentage of safe choices on the CDMT were not significantly related to any other independent measure.

Correlations were also computed to assess relationships among treatment outcome variables (with the exception of the dichotomous variable treatment completion). For most measures, outcomes were unrelated to one another, suggesting that they are likely measuring different treatment dimensions (results not shown) and providing justification for including them as separate constructs. Thus, these instruments are likely measuring different treatment outcome dimensions. Due to strong convergence, however, between scores on the Treatment Responsivity and Gain subscales ($R = .965, p < 0.001$), responses were combined to produce a total responsivity and gain score for these performance domains. Cronbach's α for this combined construct was .92, suggesting high internal validity.

A final preliminary analysis assessed correlations between each treatment outcome and background, behavioral, and psychological variables. Unexpectedly, very few significant relationships emerged between background, behavioral and psychological characteristics, and treatment outcomes. There were no variables significantly related to Treatment Readiness. Those inmates with greater Treatment Responsivity and Treatment Gain reported less physical abuse: $R = -.17, p < 0.05$ and $R = -.14, p < 0.05$, respectively. Reaction to Provocation Change scores were related to history of physical abuse ($R = -.30, p < .0001$), history of emotional abuse ($R = -.31, p < .0001$), proactive aggression ($R = -.35, p < 0.0001$), and reactive aggression ($R = -.35, p < 0.0001$). And in an ANOVA analysis, program completers reported a lesser history of physical abuse ($F = 8.89, p = 0.003$) and experiencing psychological problems a fewer number of days in the last month ($F = 6.19, p = 0.01$). As stated above, background, psychological, and behavioral variables that were significantly related to each outcome or neurocognitive/emotional regulation measure were

included as covariates in the models described below; e.g., childhood physical abuse was included as a covariate in models with Reaction to Provocation Scores.

Neurocognitive and Emotional Regulatory Predictors of Treatment Responses/Outcomes

Treatment Readiness

Higher levels of self-reported treatment readiness were associated with fewer correct ($t = -2.64, p < 0.05$) and shorter reaction times ($t = -2.02, p < 0.05$) in response to delay tone trials on the impulsivity task. Also, reaction times in response to risky decisions on the CDMT were shorter in those who perceived greater treatment readiness ($t = -2.58; p < 0.01$). Change in cortisol during the stress task was more pronounced in those who viewed themselves as more treatment ready relative to those who reported less readiness ($t = 2.03; p < 0.05$) (see Table 3).

Treatment Gain and Responsivity

Treatment Gain and Responsivity scores were strongly and positively related to percent correct ($t = 4.44, p = 0.0001$) and reaction times ($t = 3.59, p < 0.0001$) on the SCT. RT during risky decision making on the CDMT was also predictive of Responsivity ($t = 1.83, p < 0.05$) (Table 4).

Treatment Completion

Inmates who completed the first treatment phase, and excluding those who began treatment but dropped out for reasons unrelated to treatment engagement or progress (e.g., transfer, work, etc.), exhibited less impulsivity with a greater percentage of correct responses on the SCT

Table 3 Hierarchical regression analyses demonstrating contribution of neurocognitive measures to variance in Treatment Readiness

Neurocognitive measure	R ² change	F change	N (df)	t	Sig. Covariates
SCT: total % correct	.04	6.96**	166 (1,156)	-2.64	n/a
SCT: average RT	.02	4.68*	166 (1,156)	-2.02	n/a
CDMT: percent safe trials	.01	0.91	177 (1,167)	.95	n/a
CDMT: RT to risky trials	.04	6.64**	175 (1,165)	-2.58	n/a
Stroop: interference score	.00	0.04	139 (1,129)	-.20	n/a
FEEST: total correct	.01	1.16	177 (1,167)	1.08	n/a
Cortisol: change score	.04	4.12*	107 (1,97)	2.03	Education

SCT Stop Change Task, CDMT Cambridge Decision Making Task, FEEST Facial Expressions of Emotion: Stimuli and Test, RT Reaction Time

* $p < 0.05$, ** $p < 0.01$

Model 1: IQ, age of inmate, years of education

Model 2: IQ, age of inmate, years of education, PCL-prim, SCL-GSI, reactive aggression, average amount of use of all drugs, family history of alcoholism

Model 3: IQ, age of inmate, years of education, PCL-prim, SCL-GSI, reactive aggression, average amount of use of all drugs, family history of alcoholism, one central measure from each neurocognitive task entered in separate models

Table 4 Hierarchical regression analyses demonstrating contribution of neurocognitive measures to variance in Treatment Gain and Responsivity

Neurocognitive measure	R^2 change	F change	N (df)	t	Sig. Covariates
SCT: total % correct	.11	19.75**	164 (1,153)	4.44	famhx alc
SCT: average RT	.07	12.91**	164 (1,153)	3.94	n/a
CDMT: percent safe trials	.00	0.19	175 (1,164)	-0.43	phys abuse
CDMT: RT to risky trials	.02	3.33*	173 (1,162)	1.83	famhx alc, phys abuse
Stroop: interference score	.01	2.02	140 (1,129)	1.42	reactive agg, famhx alc
FEEST: total correct	.00	1.01	175 (1,164)	-1.01	famhx alc, phys abuse, age
Cortisol: change score	.00	0.23	106 (1,95)	0.48	age, psychopathy

SCT Stop Change Task, CDMT Cambridge Decision Making Task, FEEST Facial Expressions of Emotion: Stimuli and Test, RT Reaction Time
 * $p < 0.05$, ** $p < 0.001$

Model 1: IQ, age of inmate, years of education

Model 2: IQ, age of inmate, years of education, history of physical abuse, PCL-prim, SCL-GSI, reactive aggression, average amount of use of all drugs, family history of alcoholism

Model 3: IQ, age of inmate, years of education, history of physical abuse, PCL-prim, SCL-GSI, reactive aggression, average amount of use of all drugs, family history of alcoholism, one central measure from each neurocognitive task entered in separate models

($\chi^2 = 4.61$, $p < 0.05$) and longer RTs ($\chi^2 = 3.43$, $p < 0.05$) than noncompleters. They also showed lengthier reaction times in response to risky trials on the CDMT ($F = 14.98$, $p < 0.05$) (see Table 5).

Change in Reaction to Provocation Score

As seen in Table 6, less improvement in terms of reactions to provocation before and after the first treatment group was associated with fewer safe choices in the CDMT ($t = 2.03$, $p < 0.05$).

DISCUSSION

The most prominent finding from this research was that inmates exhibiting a relative lack of behavioral inhibition

and inability to shift responses based on new information were less likely to progress favorably in a standard correctional treatment program, more likely to drop out early, and less likely to report improvement in aggressive reactions to provocation. Indicators of impulsivity included both the inability to respond correctly in the presence of a distracter stimulus and reaction time, which was significantly shorter in poor responders. This behavioral orientation has implications for social competency skills that may influence amenability to treatment and ultimate behavioral outcomes.

Impulsivity has been consistently found to act as an important risk factor in a wide range of high risk behaviors, including aggression and drug abuse (Dawe & Loxton, 2004; de Wit & Richards, 2004). Individuals with impulse dyscontrol disorders have difficulties in generating socially adaptive behaviors and executing skills to avoid aggressive

Table 5 Logistic regression model showing group differences between drop out and completion groups

Neurocognitive measure	Drop outs N	Completers N	χ^2	Cox & Snell R^2	Sig. Covariates
SCT: total % correct	57	81	4.61*	0.10	psyc problems
SCT: average RT	57	81	3.43*	0.09	psyc problems
CDMT: % safe trials	65	85	0.31	0.08	psyc problems, phys ab
CDMT: RT to risky trials	65	83	14.98*	0.10	psyc problems, phys ab
Stroop: interference score	44	73	0.88	0.08	psyc problems, phys ab
FEEST: total correct	66	85	1.12	0.10	phys ab
Cortisol change score	33	58	1.37	0.06	n/a

SCT Stop Change Task, CDMT Cambridge Decision Making Task, FEEST Facial Expressions of Emotion: Stimuli and Test, RT Reaction Time
 * $p < 0.05$

Statistics represent a test of the predictive power of the cognitive variable entered into the third block, after adjusting for block 1 (IQ, age, education) and block 2 (history of physical abuse, psychological problems in last 30 days)

Table 6 Hierarchical regression analyses demonstrating contribution of neurocognitive measures to variance in Change in Reaction to Provocation

Neurocognitive measure	R^2 change	F change	N (df)	t	Sig. Covariates
SCT: total % correct	.011	1.51	123 (1,112)	−1.23	hx emo abuse
SCT: average RT	.00	0.01	123 (1,112)	0.02	hx emo abuse
CDMT: percent safe trials	.028	4.11*	127 (1,116)	2.03	hx emo abuse, hx phys abuse
CDMT: RT to risky trials	.02	2.67	125 (1,114)	1.63	hx emo abuse
Stroop: interference score	.00	0.21	105 (1,94)	−.46	hx emo abuse
FEEST: total correct	.001	0.077	127 (1,116)	0.28	hx emo abuse
Cortisol: change score	.01	1.12	84 (1,73)	−1.06	hx phys abuse

SCT Stop Change Task, CDMT Cambridge Decision Making Task, FEEST Facial Expressions of Emotion: Stimuli and Test, RT Reaction Time
* $p < 0.05$

Model 1: IQ, age of inmate, years of education

Model 2: IQ, age of inmate, years of education, history of physical abuse, history of emotional abuse, PCL-prim, SCL-GSI, average amount of use of all drugs, family history of alcoholism

Model 3: IQ, age of inmate, years of education, history of physical abuse, history of emotional abuse, PCL-prim, SCL-GSI, average amount of use of all drugs, family history of alcoholism, one central measure from each neurocognitive task entered in separate models

responses or stressful interactions. Also, compromised impulse control over behavior may permit hostility, negative affective states, and other maladaptive responses to dominate behavior (Giancola, 1995). These dimensions of impulsivity may operate to interfere with the ability to engage and persist in treatment programs as well. Additional support for this finding would suggest that behavioral inhibition may be a prerequisite for program participation and retention, particularly when interventions do not first reinforce the building blocks for effective behavioral inhibition. Thus, impulsivity may play a critical role in treatment responses which rely heavily upon the ability to resist impulses to engage in behavior that has yielded immediate intrinsic rewards (e.g., the control from aggressive behavior or euphoria from illicit drugs) despite longer term negative consequences. Recent data such as those presented in this study suggest the need to match interventions to relevant personality traits such as impulsivity to improve intervention outcomes in general (Moeller et al., 2001; Staiger, Kambouropoulos, & Dawe, 2007).

Impulsive responding on neurocognitive tasks similar to that used in this study has been consistently shown to recruit the brain's prefrontal cortex (PFC) and its neural circuitry (King, Tenney, Rossi, Colamussi, & Burdick, 2003; Martin & Potts, 2004; Watanabe et al., 2001) which regulates planning skills, sensitivity to consequences, impulse control, and other complex social behaviors. Development of the brain circuitry underlying impulse control is in transition throughout adolescence and into early adulthood; the PFC and its connections remain underdeveloped relative to other brain regions until about age 25 (Giedd, 2004). As a result, impulsive behaviors are developmentally expected in adolescence. Enhancing risk for developmental delays that may lead to enduring impulsive behavior is the heightened

sensitivity to severe psychosocial and physical stressors (e.g., head injury, drug or alcohol use, deprivation, child abuse) during this critical developmental period (Chambers, Taylor, & Potenza, 2003). Evidence for this possibility is seen in adults with a history of trauma who exhibit high levels of impulsivity and other risk taking behaviors; several studies have reported PFC developmental delays or deficits in this population (e.g., Berlin, Rolls, & Iversen, 2005; Rodriguez-Jimenez et al., 2006; Spinella, 2004). Perhaps not coincidentally, therefore, physical and psychological trauma which can compromise PFC development and latter functioning is more prevalent in criminal offenders than in the general population (Blake, Pincus, & Buckner, 1995; Neller, Denney, Pietz, & Thomlinson, 2006). These findings provide one potential explanation for their high rates of impulsivity and, possibly for inmates' particular intractability to treatment.

Interventions may be more beneficial to the inmate population if they first address the level of proficiency of an inmate's behavioral inhibition skills and their use of techniques for weighing consequences. Training in the prediction of outcomes and development of a future orientation among other related skills may, then, reinforce the ability to withhold impulsive responses and act on an assessment of consequences (Trad, 1993). Treatment program strategies to help inmates "slow down and think" and control impulsive reactions may be indicated given that the subgroup most likely to be unresponsive to treatment may be impulsive, resulting in a counterproductive decisions. Cognitive rehabilitation approaches may be particularly effective in addressing efficiency of behavioral responding. Remediation targets learning, attention, problem-solving, and visual-spatial skills using two approaches: repeated exposure to a task and/or the deconstruction of complex

tasks into their simpler component parts. For example, components of a complex task such as scanning or psychomotor speed are trained separately and then integrated into performance on the complex target task. The training also focuses on memory and problem-solving, stressing the use of strategies such as forming images or drawing diagrams.

Another significant finding in this study is that stress reactivity, as measured by cortisol levels, predicted treatment readiness, consistent with van de Wiel, van Goozen, Matthys, Snoek, and van Engeland (2004) and Zimmermann, Blomeyer, Laucht, and Mann (2007); lower cortisol reactivity was associated with self evaluations of lower readiness. Abnormally high levels of cortisol release are related to acute stress and anxiety, however, relative cortisol insensitivity may be reflective of either a genetic propensity to lower stress reactivity or a dampening of stress responses that can occur after chronic exposure to stressful situations. It is also possible that a stress response or some level of negative affect is required to engage in treatment (McKay, Alterman, & Mulvaney, 1999). This study cannot determine the origins of low stress responses in those who performed less well in treatment. However, given that chronic or severe stressful experiences are known to (a) produce cognitive impairment and stress response dampening, (b) dysregulate emotional responses, (c) increase the likelihood of recidivism and relapse, and (d) interfere with treatment benefits, approaches to minimize the impact of stress may eventually prove to be a critical component of treatment for affected inmates. Cortisol dysregulation as one index of physiological stress reactivity has been found to be malleable in response to interventions. For example, Fisher, Gunnar, Chamberlain, and Reid (2000) and other investigators provide evidence for the impact of a targeted intervention on cortisol (Bruce, Kroupina, Parker, & Gunnar, 2006; Gunnar, Morison, Chisholm, & Schuder, 2001; Hammerfald et al., 2006; Mommersteeg, Keijsers, Heijnen, Verbreeak, & van Doornen, 2006). There are also potential implications for altering autonomic reactivity in general with appropriate interventions (e.g., McCraty, Atkinson, Tomasino, Goelitz, & Mayrovitz, 1999; Raine, 1996), suggesting a possible mediating role for stress responsivity. Nevertheless, the relationship between stress and perceived treatment readiness is still a poorly understood phenomenon.

Other Related Findings

IQ did not relate to treatment outcomes and most of the models remained significant with its inclusion, suggesting that higher order cognitive functions, and not subservient cognitive functions, played a more direct role in treatment engagement outcomes. Also, younger inmates fared better

in treatment, but total months in prison were not predictive of outcomes. This finding may be interpreted to suggest that younger inmates may be more tractable irrespective of how long they have been in prison. Additional background variables were similarly unrelated directly to treatment outcomes, including education, duration of total prison time, family history of criminality and mental illness, drug abuse history, and psychopathy. On the other hand, there was a general pattern for physical abuse and psychological problems in the last 30 days to contribute independently to poor treatment response. In light of the extant research on this subject, it is critical that treatment providers consider childhood trauma and its related psychological manifestations as possible concomitant conditions that can potentially disengage inmates from treatment and result in diminished amenability to program demands. Nevertheless, when these factors were included in models with neurocognitive and emotional regulation measures, they did not retain their significance. And importantly, although various measures from each of the neurocognitive and emotional regulation tasks significantly predicted particular treatment outcomes and low cortisol responses also predicted treatment completion (data not shown), these factors also did not retain their significance with the inclusion of relevant covariates in the models. These results suggest the potentially influential role of disinhibition in treatment engagement over and above other inmate characteristics.

Interestingly, self evaluations of treatment readiness differed from social worker evaluations and were not predictive of actual treatment performance indicators. We speculate that those inmates who have an inflated sense of how well they may fare in treatment may be at particular risk for negative outcomes. It may also be relevant that a robust cortisol response to a stressor typified those who evaluated themselves as being highly treatment ready. Based strictly on years of observations of prisoners, this finding may suggest that inmates who report being treatment ready and who have lowered stress responsivity may be less able to predict their performance in treatment, and less enthusiastic and compliant. Inmates with a higher cortisol response and who report less treatment readiness may simply be more committed to change and anxious about their ability to perform well; i.e., they may have a higher level of responsivity to social stimulation. The implications of this finding for treatment will be explored in future research.

FINAL REMARKS

There are several limitations in this study. High levels of noncompletion in prison treatment programs make it difficult to assess the 'effects' of treatment on cognitive

functioning or any other measure. On the other hand, this study intended to predict individual differences in level of engagement, persistence, and improvement in a correctional treatment program, which may be more reflective of the inmates' baseline willingness and ability to respond favorably and not the effects of any given treatment approach. Similarly, the lack of a placebo control group limits conclusions to some extent; however, the purpose of this study was to compare inmates with varying levels of participation rather than evaluate the treatment program. Another shortcoming is that there is no method by which we could assess selection bias. Inmates were referred to the study team based on preexisting criteria, thus, a comparison of inmates referred with those who were not referred was not possible. The response rate was exceedingly high, however, in that all of those recruited and considered eligible participated in the research. Also, use of self reports to evaluate behaviors, including nature and extent of drug use, and psychological traits is not ideal without corroboration or more extensive testing. Primary measures, however, were performance tasks and not prone to errors due to recall, selective memory, bias, and other common issues in surveys.

In short, our findings tentatively suggest that behavioral disinhibition may be informative in identifying treatment readiness preparations that are necessary for inmates to be able to respond to existing intervention strategies. Replications of these preliminary findings would indicate that new approaches to correctional treatment may include a cognitive neurorehabilitation component designed to enhance connectivity between neural systems and strengthen these malleable cognitive and emotional regulatory functions to more effectively modify behavior in inmates. Incorporation of this knowledge into criminal justice policies and practices may eventually alter their course substantially, dramatically improving agencies' ability to assess, detect, and treat offenders who are currently considered intractable. (Note that if this represented only 10% of inmates in custody in the US, based on Bureau of Justice Statistics December 2005 statistics, this would affect about 219,000 individuals).

Acknowledgments This research was funded by the National Institute of Justice, Grant No. 2002-MU-BX-0013. We thank the Maryland Department of Correction, their Social Work and Correctional Office staff for their full cooperation and assistance in conducting this study.

REFERENCES

- Aharonovich, E., Nunes, E., & Hasin, D. (2003). Cognitive impairment, retention and abstinence among cocaine abusers in cognitive-behavioral treatment. *Drug and Alcohol Dependence*, *71*, 207–211. doi:10.1016/S0376-8716(03)00092-9.
- Andrews, D. A., & Bonta, J. (2006). *The psychology of criminal conduct* (4th ed.). Cincinnati, OH: Anderson.
- Bauer, L. O. (2001). Predicting relapse to alcohol and drug abuse via quantitative electroencephalography. *Neuropsychopharmacology*, *25*, 332–340. doi:10.1016/S0893-133X(01)00236-6.
- Beers, S. R., & De Bellis, M. D. (2002). Neuropsychological function in children with maltreatment-related posttraumatic stress disorder. *The American Journal of Psychiatry*, *159*, 483–486. doi:10.1176/appi.ajp.159.3.483.
- Bench, C. J., Frith, C. D., Grasby, P. M., Friston, K. J., Paulesu, E., Frackowiak, R. S. J., et al. (1993). Investigations of the functional anatomy of attention using the Stroop test. *Neuropsychologia*, *31*, 907–922. doi:10.1016/0028-3932(93)90147-R.
- Berlin, H. A., Rolls, E. T., & Iversen, S. D. (2005). Borderline personality disorder, impulsivity, and the orbitofrontal cortex. *The American Journal of Psychiatry*, *162*(12), 2360–2373. doi:10.1176/appi.ajp.162.12.2360.
- Blair, R. J., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of said and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, *29*, 491–498. doi:10.1023/A:1012225108281.
- Blake, P. Y., Pincus, J. H., & Buckner, C. (1995). Neurologic abnormalities in murderers. *Neurology*, *45*, 1641–1647.
- Blume, A. W., Davis, J. M., & Schmalting, K. B. (1999). Neurocognitive dysfunction in dually-diagnosed patients: A potential roadblock to motivating behavior change. *Journal of Psychoactive Drugs*, *31*, 111–115.
- Blume, A. W., Marlatt, G. A., & Schmalting, K. B. (2000). Executive cognitive function and heavy drinking behavior among college students. *Psychology of Addictive Behaviors*, *14*, 299–302. doi:10.1037/0893-164X.14.3.299.
- Bremner, J. D. (1999). Alterations in brain structure and function associated with post-traumatic stress disorder. *Seminars in Clinical Neuropsychiatry*, *4*, 249–255.
- Bremner, J. D. (2003). Long-term effects of childhood abuse on brain and neurobiology. *Child and Adolescent Psychiatric Clinics of North America*, *12*, 271–292. doi:10.1016/S1056-4993(02)00098-6.
- Bremner, J. D., Bolus, R., & Mayer, E. (2007). Psychometric properties of the Early Trauma Inventory-Self Report. *The Journal of Nervous and Mental Disease*, *195*, 211–218. doi:10.1097/01.nmd.0000243824.84651.6c.
- Bremner, J. D., Vermetten, E., & Mazure, C. M. (2000). Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: The Early Trauma Inventory. *Depression and Anxiety*, *12*, 1–12. doi:10.1002/1520-6394(2000)12:1<1::AID-DA1>3.0.CO;2-W.
- Bruce, J., Kroupina, M., Parker, S., & Gunnar, M. (2006). *The relationships between cortisol patterns, growth retardation, and developmental delays in postinstitutionalized children*. Paper presented at the International Conference on Infant Studies, Brighton, UK.
- Casey, S., Day, A., Howells, K., & Ward, T. Assessing suitability for offender rehabilitation: Development and validation of the Treatment Readiness Questionnaire. *Criminal Justice and Behavior*, in press.
- Chambers, R. A., Taylor, J. R., & Potenza, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *The American Journal of Psychiatry*, *160*, 1041–1052. doi:10.1176/appi.ajp.160.6.1041.
- Charney, D. S., & Deutch, A. (1996). A functional neuroanatomy of anxiety and fear: Implications for the pathophysiology and treatment of anxiety disorders. *Critical Reviews in Neurobiology*, *10*, 419–446.

- Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms in reactive and proactive aggression. *Child Development, 67*, 993–1002. doi:10.2307/1131875.
- Critchley, H. D., Elliot, R., Mathias, C. J., & Dolan, R. J. (2000). Neural activity relating to generation and representation of galvanic skin conductance responses: A functional magnetic resonance imaging study. *The Journal of Neuroscience, 20*, 3033–3040.
- Davidson, R. (1994). Asymmetric brain function, affective style and psychopathology: The role of early experience and plasticity. *Development and Psychopathology, 58*, 741–758. doi:10.1017/S0954579400004764.
- Dawe, S., & Loxton, N. J. (2004). The role of impulsivity in the development of substance use and eating disorders. *Neuroscience and Biobehavioral Reviews, 28*, 343–351. doi:10.1016/j.neubiorev.2004.03.007.
- Dawes, M. A., Antelman, S. M., Vanyukoy, M. M., Giancola, P., Tarter, R. E., Susman, E. J., et al. (2000). Developmental sources of variation in liability to adolescent substance use disorders. *Drug and Alcohol Dependence, 61*, 3–14. doi:10.1016/S0376-8716(00)00120-4.
- Day, A., Howells, K., Casey, S., Ward, T., Chambers, J. C., & Birgden, A. (2008). Assessing treatment readiness in violent offenders. *Journal of Interpersonal Violence*. Epub ahead of Print.
- De Bellis, M. D. (2002). Developmental traumatology: A contributory mechanisms for alcohol and substance use disorders. *Psychoneuroendocrinology, 27*, 155–170. doi:10.1016/S0306-4530(01)00042-7.
- De Bellis, M. D., Keshavan, M. S., Spencer, S., & Hall, J. H. (2000). N-Acetylaspartate concentration in the anterior cingulate of maltreated children and adolescents with PTSD. *The American Journal of Psychiatry, 157*, 1175–1177. doi:10.1176/appi.ajp.157.7.1175.
- De Haan, M., Luciana, M., Malone, S., Matheny, L., & Richards, L. (1994). Development, plasticity, and risk: Commentary on Huttenlocher, Pollitt and Gorman and Gottesman and Goldman. In C. A. Nelson (Ed.), *Threats to optimal development: Integrating biological, psychological and social risk factors*. Hillsdale, NJ: Lawrence Erlbaum.
- de Wit, H., & Richards, J. B. (2004). Dual determinants of drug use in humans: Reward and impulsivity. *Nebraska Symposium on Motivation, 50*, 19–55.
- Deckel, A. W., Hesselbrock, V., & Bauer, L. (1995). Relationship between alcohol-related expectancies and anterior brain functioning in young men at risk for developing alcoholism. *Alcoholism, Clinical and Experimental Research, 19*, 476–481. doi:10.1111/j.1530-0277.1995.tb01534.x.
- Derogatis, L. R., Rickels, K., & Rock, A. F. (1976). The SCL-90 and the MMPI: A step in the validation of a new self-report scale. *The British Journal of Psychiatry, 128*, 280–289. doi:10.1192/bjp.128.3.280.
- Dodge, K. A. (1980). Social cognition and children's aggressive behavior. *Child Development, 51*, 162–170. doi:10.2307/1129603.
- Ekman, P., & Friesen, W. V. (1975). *Pictures of facial affect*. Palo Alto, CA: Consulting Psychologists Press.
- Elliott, R., Friston, K. J., & Dolan, R. J. (2000). Dissociable neural responses in human reward systems. *The Journal of Neuroscience, 20*, 6159–6166.
- Fals-Stewart, W. (1993). Neurocognitive deficits and their impact on substance abuse treatment. *Journal of Addictions & Offender Counseling, 13*, 46–57.
- Fishbein, D. H. (2000). The import of neurobiological research to the prevention of antisocial behavior and drug abuse. *Prevention Science, 1*, 89–106. doi:10.1023/A:1010090114858.
- Fishbein, D. H., Eldreth, D., Hyde, C., Matochik, J., London, E., Contoreggi, C., et al. (2005a). Risky decision making and the anterior cingulate in abstinent drug addicts and nondrug users. *Brain Research. Cognitive Brain Research, 23*, 119–136. doi:10.1016/j.cogbrainres.2004.12.010.
- Fishbein, D. H., Hyde, C., Eldreth, D., Paschall, M. J., Tarter, R., Das, A., et al. (2006). Neurocognitive skills moderate urban male adolescents' responses to preventive intervention materials. *Drug and Alcohol Dependence, 82*, 47–60. doi:10.1016/j.drugalcdep.2005.08.008.
- Fishbein, D. H., Hyde, C., London, E. D., Matochik, J., Ernst, M., Isenberg, N., et al. (2005b). Cognitive and physiological differences between abstinent drug abusers and controls. *Experimental and Clinical Psychopharmacology, 13*, 25–40. doi:10.1037/1064-1297.13.1.25.
- Fisher, P. A., Gunnar, M. R., Chamberlain, P., & Reid, J. B. (2000). Preventive intervention for maltreated preschool children: Impact on children's behavior, neuroendocrine activity, and foster parent functioning. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*, 1356–1364. doi:10.1097/00004583-200011000-00009.
- Forman, S. D., Dougherty, G. G., Kelley, M. E., Stenger, V. A., Pizarov, L. A., & Wick-Hull, C. (2004, December). *Brain activity of opiate addicts predicts subsequent treatment retention*. Panel session presentation at the annual meeting of the American College of Neuropsychopharmacology. San Juan, Puerto Rico.
- Giaconia, R. M., Reinherz, H. Z., Hauf, A. C., Paradis, A. D., Wasserman, M. S., Langhammer, D. M., et al. (2000). Comorbidity of substance use and post-traumatic stress disorders in a community sample of adolescents. *The American Journal of Orthopsychiatry, 70*, 253–262. doi:10.1037/h0087634.
- Giancola, P. R. (1995). Evidence for dorsolateral and orbital prefrontal cortical involvement in the expression of aggressive behavior. *Aggressive Behavior, 21*, 431–450. doi:10.1002/1098-2337(1995)21:6<431::AID-AB2480210604>3.0.CO;2-Q.
- Giancola, P. R., Martin, C. S., Tarter, R. E., Pelham, W. E., & Moss, H. B. (1996). Executive cognitive functioning and aggressive behavior in preadolescent boys at high risk for substance abuse/dependence. *Journal of Studies on Alcohol, 57*, 352–359.
- Giancola, P. R., Mezzich, A. C., & Tarter, R. E. (1998). Disruptive, delinquent and aggressive behavior in female adolescents with a psychoactive substance use disorder: Relation to executive cognitive functioning. *Journal of Studies on Alcohol, 59*, 560–567.
- Giedd, J. N. (2004). Structural magnetic resonance imaging of the adolescent brain. *Annals of the New York Academy of Sciences, 1021*, 77–85. doi:10.1196/annals.1308.009.
- Grohman, K., & Fals-Stewart, W. (2004). *Cognitive rehabilitation in substance abuse treatment: Effects on length of stay and posttreatment abstinence*. Poster presented at the Annual Meeting of the Research Society on Alcoholism, Vancouver, Canada.
- Gunnar, M., Morison, S. J., Chisholm, K., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romania orphanages. *Development and Psychopathology, 13*, 611–627. doi:10.1017/S095457940100311X.
- Hammerfeld, K., Aberle, C., Grau, M., Kinsperger, A., Zimmermann, A., Ehlert, U., et al. (2006). Persistent effects of cognitive-behavioral stress management on cortisol responses to acute stress in healthy subjects—A randomized controlled trial. *Psychoneuroendocrinology, 31*, 333–339. doi:10.1016/j.psyneuen.2005.08.007.
- Hare, R. D. (1991). *The Hare psychopathy checklist-revised*. Toronto: Multi-Health Systems.
- Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity

- disorder in boys: A four-year longitudinal study. *Journal of Abnormal Child Psychology*, 23, 729–749. doi:10.1007/BF01447474.
- Hermann, D., & Parente, R. (1996). *Retraining cognition: Techniques and application*. Baltimore, MD: Aspen Publishers.
- Hiller, M., Knight, K., & Simpson, D. D. (1999). Risk factors that predict dropout from corrections-based treatment for drug abuse. *Prison Journal*, 79, 411–430.
- Holbrook, M. I. (1997). Anger management training in prison inmates. *Psychological Reports*, 81, 623–626.
- Kennedy, S., & Serin, R. C. (1997). Treatment responsivity: Contributing to effective correctional programming. *International Community Corrections Association Journal*, 7, 46–52.
- Kennedy, S., & Serin, R. C. (1999). Examining offender readiness to change and the impact on treatment outcome. In P. M. Harris (Ed.), *Research to results: Effective community corrections* (pp. 215–232). Lanham, MD: American Correctional Association.
- King, J. A., Tenney, J., Rossi, V., Colamussi, L., & Burdick, S. (2003). Neural substrates underlying impulsivity. *Annals of the New York Academy of Sciences*, 1008, 160–169. doi:10.1196/annals.1301.017.
- Kornreich, C., Foisy, M. L., Philippot, P., Dan, B., Tecco, J., Noel, X., et al. (2003). Impaired emotional facial expression recognition in alcoholics, opiate dependence subjects, methadone maintained subjects, and mixed alcohol-opiate antecedents subjects compared with normal controls. *Psychiatry Research*, 199, 251–260. doi:10.1016/S0165-1781(03)00130-6.
- Kossen, D. S., Suchy, Y., Mayer, A. R., & Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion*, 2, 398–411. doi:10.1037/1528-3542.2.4.398.
- Kreek, M. J., Schlussman, S. D., Bart, G., Laforge, K. S., & Butelman, E. R. (2004). Evolving perspectives on neurobiological research on the addictions: Celebration of the 30th anniversary of NIDA. *Neuropharmacology*, 47, 324–344. doi:10.1016/j.neuropharm.2004.07.024.
- Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing psychopathic attributes in a noninstitutionalized population. *Journal of Personality and Social Psychology*, 68, 151–158. doi:10.1037/0022-3514.68.1.151.
- Liberzon, I., Taylor, S. F., Fig, L. M., Decker, L. R., Koeppe, R. A., & Minoshima, S. (2000). Limbic activation and psychophysiological responses to aversive visual stimuli. Interaction with cognitive task. *Neuropsychopharmacology*, 23, 508–516. doi:10.1016/S0893-133X(00)00157-3.
- Logan, G. D., & Burkell, J. (1986). Dependence and independence in responding to double stimulation: A comparison of stop, change and dual-task paradigms. *Journal of Experimental Psychology. Human Perception and Performance*, 12, 549–563. doi:10.1037/0096-1523.12.4.549.
- Luria, A. R. (1980). *Higher cortical functions in man*. New York: Basic Books.
- Manchester, D., Hodgkinson, A., & Casey, T. (1997). Prolonged, severe behavioral disturbance following traumatic brain injury: What can be done? *Brain Injury*, 11, 605–617. doi:10.1080/026990597123296.
- Martell, D. A. (1992). Estimating the prevalence of organic brain dysfunction in maximum-security psychiatric patients. *Journal of Forensic Sciences*, 37, 878–893.
- Martin, L. E., & Potts, G. F. (2004). Reward sensitivity in impulsivity. *Neuroreport*, 15(9), 1519–1522. doi:10.1097/01.wnr.0000132920.12990.b9.
- McClure, E. B., Monk, C. S., Nelson, E. E., Zarahn, E., Leibenluft, E., Bilder, R. M., et al. (2004). A developmental examination of gender differences in brain engagement during evaluation of threat. *Biological Psychiatry*, 55, 1047–1055. doi:10.1016/j.biopsych.2004.02.013.
- McCarty, R., Atkinson, M., Tomasino, D., Goelitz, J., & Mayrovitz, H. N. (1999). The impact of an emotional self-management skills course on psychosocial functioning and autonomic recovery to stress in middle school children. *Integrative Physiological and Behavioral Science*, 34, 246–268. doi:10.1007/BF02688693.
- McKay, J. R., Alterman, A. I., & Mulvaney, F. D. (1999). Predicting proximal factors in cocaine relapse and near miss episodes: Clinical and theoretical implications. *Drug and Alcohol Dependence*, 56, 67–78. doi:10.1016/S0376-8716(99)00013-7.
- McLellan, A. T., Kushner, H., Metzger, D., Peters, R., Smith, I., Grissom, G., et al. (1992). The fifth edition of the addiction severity index: Historical critique and normative data. *Journal of Substance Abuse Treatment*, 9, 199–213. doi:10.1016/0740-5472(92)90062-S.
- Mesulam, M. M. (1986). Frontal cortex and behavior. *Annals of Neurology*, 19, 319–323.
- Mirsky, A. F., & Siegel, A. (1994). The neurobiology of violence and aggression. In A. J. Reiss Jr., K. A. Miczek & J. A. Roth (Eds.), *Violence: Biobehavioral influences* (Vol. 2, pp. 59–172). Washington, DC: National Academy Press.
- Moeller, F. G., Dougherty, D. M., Barratt, E. S., Schmitz, J. M., Swann, A. C., & Grabowski, J. (2001). The impact of impulsivity on cocaine use and retention in treatment. *Journal of Substance Abuse Treatment*, 21, 193–198. doi:10.1016/S0740-5472(01)00202-1.
- Moffitt, T. E., Lyman, D. R., & Silva, P. A. (1994). Neuropsychological tests predicting persistent male delinquency. *Criminology*, 2, 277–300. doi:10.1111/j.1745-9125.1994.tb01155.x.
- Mommersteeg, P. M. C., Keijsers, G. P. J., Heijnen, C. J., Verbreaak, M. J. P. M., & van Doornen, L. J. P. (2006). Cortisol deviations in people with burnout before and after psychotherapy: A pilot study. *Health Psychology*, 25, 243–248. doi:10.1037/0278-6133.25.2.243.
- Moss, H. B., Vanyukov, M., Yao, J. K., & Kirillova, G. P. (1999). Salivary cortisol responses in prepubertal boys: The effects of parental substance abuse and association with drug use behavior during adolescence. *Biological Psychiatry*, 45, 1293–1299. doi:10.1016/S0006-3223(98)00216-9.
- Neller, D. J., Denney, R. L., Pietz, C. A., & Thomlinson, R. P. (2006). The relationship between trauma and violence in a jail inmate sample. *Journal of Interpersonal Violence*, 21, 1234–1241. doi:10.1177/0886260506290663.
- Nemeroff, C. B. (2004). Neurobiological consequences of childhood trauma. *The Journal of Clinical Psychiatry*, 65, 18–28.
- Nicholaichuk, T., Gordon, A., Gu, D., & Wong, S. (2000). Outcome of an institutional sexual offender treatment program: A comparison between treated and matched untreated offenders. *Sexual Abuse*, 12, 139–153. doi:10.1177/107906320001200205.
- Nixon, S. J., Paul, R., & Phillips, M. (1998). Cognitive efficiency in alcoholics and polysubstance abusers. *Alcoholism, Clinical and Experimental Research*, 22, 1414–1420. doi:10.1111/j.1530-0277.1998.tb03929.x.
- Novaco, R. W. (1994). Anger as a risk factor for violence among the mentally disordered. In J. Monahan & H. J. Steadman (Eds.), *Violence, mental disorder: Developments in risk assessment*. Chicago: University of Chicago Press.
- Oosterlaan, J., Geurts, H. M., Knol, D. L., & Sergeant, J. A. (2005). Low basal salivary cortisol is associated with teacher-reported symptoms of conduct disorder. *Psychiatry Research*, 134, 1–10. doi:10.1016/j.psychres.2004.12.005.
- Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences of the United States of America*, 87, 256–259. doi:10.1073/pnas.87.1.256.

- Raine, A. (1993). *The psychopathology of crime: Criminal behavior as a clinical disorder*. New York: Academic Press.
- Raine, A. (1996). Autonomic nervous system factors underlying disinhibited, antisocial, and violent behavior. Biosocial perspectives and treatment implications. *Annals of the New York Academy of Sciences*, 794, 46–59. doi:10.1111/j.1749-6632.1996.tb32508.x.
- Raine, A., Dodge, K., Loeber, R., Gatzke-Kopp, L., Lynam, D., Reynolds, C., et al. (2006). The Reactive-Proactive Aggression Questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys. *Aggressive Behavior*, 32, 159–171. doi:10.1002/ab.20115.
- Raine, A., Lencz, T., Bihle, S., LaCasse, L., & Colletti, P. (2000). Reduced prefrontal gray matter volume and reduced autonomic activity in antisocial personality disorder. *Archives of General Psychiatry*, 57, 119–127. doi:10.1001/archpsyc.57.2.119.
- Reiss, A. J., Jr., Miczek, K. A., & Roth, J. A. (1994). *Understanding and preventing violence: Volume 2. Biobehavioral influences*. Washington, DC: National Academy Press.
- Riggs, N. R., Greenberg, M. T., Kusche, C. A., & Pentz, M. A. (2006). The mediational role of neurocognition in the behavioral outcomes of a social-emotional prevention program in elementary school students: Effects of the PATHS Curriculum. *Prevention Science*, 7, 91–102. doi:10.1007/s11121-005-0022-1.
- Rodriguez-Jimenez, R., Avila, C., Jimenez-Arriero, M. A., Ponce, G., Monasor, R., Jimenez, M., et al. (2006). Impulsivity and sustained attention in pathological gamblers: Influence of childhood ADHD history. *Journal of Gambling Studies*, 22(4), 451–461. doi:10.1007/s10899-006-9028-2.
- Rogers, R. D., Blackshaw, A. J., Middleton, H. C., Matthews, K., Hawtin, K., Crowley, C., et al. (1999a). Tryptophan depletion impairs stimulus-reward learning while methylphenidate disrupts attentional control in healthy young adults: Implications for the monoaminergic basis of impulsive behaviour. *Psychopharmacology*, 146, 482–491. doi:10.1007/PL00005494.
- Rogers, R. D., Owen, A. M., Middleton, H. C., Williams, E. J., Pickard, J. D., Sahakian, B. J., et al. (1999b). Choosing between small, likely rewards and large, unlikely rewards activates inferior and orbital PFC. *The Journal of Neuroscience*, 20, 9029–9038.
- Rogers, R. D., & Robbins, T. W. (2001). Investigating the neurocognitive deficits associated with chronic drug misuse. *Current Opinion in Neurobiology*, 11(2), 250–257. doi:10.1016/S0959-4388(00)00204-X.
- Rohrmann, S., Hennig, J., & Netter, P. (1999). Changing psychobiological stress reactions by manipulating cognitive processes. *International Journal of Psychophysiology*, 33, 149–161. doi:10.1016/S0167-8760(99)00036-7.
- Rosmond, R., Dallman, M. F., & Björntorp, P. (1998). Stress-related cortisol secretion in men: Relationships with abdominal obesity and endocrine, metabolic and hemodynamic abnormalities. *The Journal of Clinical Endocrinology and Metabolism*, 83, 1853–1859. doi:10.1210/jc.83.6.1853.
- Rothwell, N. A., La Vigna, G. W., & Willis, T. J. (1999). A non-aversive rehabilitation approach for people with severe behavioral problems resulting from brain injury. *Brain Injury*, 13, 521–533. doi:10.1080/026990599121421.
- Rubia, K., Taylor, E., Smith, A. B., Oksanen, H., Overmeyer, S., Newman, S., et al. (2001). Neuropsychological analyses of impulsiveness in childhood hyperactivity. *The British Journal of Psychiatry*, 179, 138–143. doi:10.1192/bjp.179.2.138.
- Seguin, J. R., Pihl, R. O., Harden, P. W., & Tremblay, R. E. (1995). Cognitive and neuropsychological characteristics of physically aggressive boys. *Journal of Abnormal Psychology*, 104, 614–624. doi:10.1037/0021-843X.104.4.614.
- Serin, R. C. (1998). Treatment responsivity, intervention, and reintegration: A conceptual model. *Forum on Corrections Research*, 10, 29–32.
- Serin, R. (2005). *Evidence-based practice: Principles for enhancing correctional results in prisons*. Washington, DC: National Institute of Corrections.
- Serin, R. C., Mailloux, D. L., & Kennedy, S. M. (2007). Development of a clinical rating scale for offender readiness: Implications for assessment and offender change. *Issues in Forensic Psychology*, 7, 70–80.
- Spinella, M. (2004). Neurobehavioral correlates of impulsivity: Evidence of prefrontal involvement. *The International Journal of Neuroscience*, 114, 95–104. doi:10.1080/00207450490249347.
- Staiger, P. K., Kambouropoulos, N., & Dawe, S. (2007). Should personality traits be considered when refining substance misuse treatment programs? *Drug and Alcohol Review*, 26(1), 17–23. doi:10.1080/09595230601036952.
- Steckler, T., & Holsboer, F. (1999). Corticotropin-releasing hormone receptor subtypes and emotion. *Biological Psychiatry*, 46, 1480–1508. doi:10.1016/S0006-3223(99)00170-5.
- Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences*, 9, 69–74. doi:10.1016/j.tics.2004.12.005.
- Stuss, D. T., & Benson, D. F. (1986). *The frontal lobes*. New York: Raven.
- Sigma Assessment Systems, Inc. (1999). *Multidimensional Aptitude Battery*. P.O. Box 610984, Port Huron, MI 48061-0984, USA.
- Tarter, R. E., Mezzich, A. C., Hsieh, Y. C., & Parks, S. M. (1995). Cognitive capacity in female adolescent substance abusers. *Drug and Alcohol Dependence*, 39, 15–21. doi:10.1016/0376-8716(95)01129-M.
- Taylor, J., Carlson, S. R., Iacono, W. G., Lykken, D. T., & McGue, M. (1999). Individual differences in electrodermal responsivity to predictable aversive stimuli and substance dependence. *Psychophysiology*, 36(2), 193–198.
- Trad, P. V. (1993). The ability of adolescents to predict future outcome. Part I: Assessing predictive abilities. *Adolescence*, 28, 533–555.
- van de Wiel, N. M., van Goozen, S. H., Matthys, W., Snoek, H., & van Engeland, H. (2004). Cortisol and treatment effect in children with disruptive behavior disorders: A preliminary study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 1011–1018. doi:10.1097/01.chi.0000126976.56955.43.
- Volavka, J., Martell, D., & Convit, A. (1992). Psychobiology of the violent offender. *Journal of Forensic Sciences*, 37, 237–251.
- Walker, J. L., Lahey, B. B., Russo, M., Frick, P. J., Christ, M. A. G., McBurnett, K., et al. (1991). Anxiety, inhibition, and conduct disorder in children, I: Relations to social impairment and sensation seeking. *Journal of the American Academy of Child and Adolescent Psychiatry*, 30, 187–191. doi:10.1097/00004583-199103000-00004.
- Watanabe, J., Sugiura, M., Sato, K., Sato, Y., Maeda, Y., Matsue, Y., et al. (2001). The human prefrontal and parietal association cortices are involved in NO-GO performances: An event-related fMRI study. *NeuroImage*, 17(3), 1207–1216. doi:10.1006/nimg.2002.1198.
- Whalen, P. J., Shin, L. M., McInerney, S. C., Fischer, H., Wright, C. I., & Rauch, S. L. (2001). A functional MRI study of human amygdala responses to facial expressions of fear vs anger. *Emotion*, 1, 70–83. doi:10.1037/1528-3542.1.1.70.
- Williams, L. M., Brown, K. J., Das, P., Boucsein, W., Sokolov, E. N., Brammer, M. J., et al. (2004). The dynamics of cortico-amygdala and autonomic activity over the experimental time course of fear perception. *Brain Research. Cognitive Brain Research*, 21, 114–123. doi:10.1016/j.cogbrainres.2004.06.005.

- Yang, T. T., Menon, V., Reid, A. J., Gotlib, I. H., & Reiss, A. L. (2003). Amygdalar activation associated with happy facial expressions in adolescents: A 3-T functional MRI study. *Journal of the American Academy of Child and Adolescent Psychiatry*, *42*, 979–985. doi:[10.1097/01.CHI.0000046886.27264.BA](https://doi.org/10.1097/01.CHI.0000046886.27264.BA).
- Yao, J. K., Moss, H. B., & Kirillova, G. P. (1998). Determination of salivary cortisol by nonisotonic immunoassay. *Clinical Biochemistry*, *31*, 190–198. doi:[10.1016/S0009-9120\(98\)00004-6](https://doi.org/10.1016/S0009-9120(98)00004-6).
- Zelazo, P. D., Carter, A., Reznick, J. S., & Frye, D. (1997). Early development of executive function: A problem solving framework. *Review of General Psychology*, *1*, 198–226. doi:[10.1037/1089-2680.1.2.198](https://doi.org/10.1037/1089-2680.1.2.198).
- Zimmermann, U. S., Blomeyer, D., Laucht, M., & Mann, K. F. (2007). How gene–stress–behavior interactions can promote adolescent alcohol use: The roles of predrinking allostatic load and childhood behavior disorders. *Pharmacology, Biochemistry, and Behavior*, *86*, 246–262. doi:[10.1016/j.pbb.2006.09.024](https://doi.org/10.1016/j.pbb.2006.09.024).