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Etiology And Treatment Of Neuropsychological And Cognitive Deficits In Relation To Antisocial Behavior

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Etiology And Treatment Of Neuropsychological And Cognitive Deficits In Relation To Antisocial Behavior

Abstract

Impaired neuropsychological and cognitive functioning are well-understood to be risk factors for antisocial behavior. There are, however, gaps in our knowledge of the etiology and effective treatment of neuropsychological and cognitive deficits. My dissertation examines these questions in a series of three papers. The first paper proposes a serial mediation model wherein neighborhood disadvantage and subsequent impaired neuropsychological functioning represent a partial explanation of the race-antisocial behavior relationship. In a community sample of male and female young adolescents, the hypothesized sequential path accounted for 10.8% of the relationship between race and antisocial behavior. The second paper examines the relationship between sleep and antisocial behavior, which has primarily been examined via correlational or extreme sleep deprivation studies. Using National Incidence-Based Reporting System and city-reported data, this paper exploits the natural experiment of daylight saving time (DST) to examine the effects of a very mild change in sleep on assault rates. The Monday directly following the advent of DST was associated with 3% fewer assaults as compared to the Monday a week later, which we hypothesize may be the result of fatigue. In contrast, we saw 3% more assaults following the return to standard time. The final paper examines a sample of incarcerated male adolescents longitudinally to test whether incarceration results in impaired cognition, and if so, whether a Cognitive Behavioral Therapy/Mindfulness intervention can protect against such impairments. Performance on three measures derived from an emotional go/no-go task significantly worsened from baseline to follow-up, however, two marginally significant time x group interactions suggest mindfulness may be potentially effective in buffering the adverse effects of imprisonment. While many scholars have postulated about adverse psychological effects of incarceration, this is one of the first papers to empirically document such effects. In totality, the proposed dissertation is intended to improve our understanding of the association between cognition and antisocial behavior through examining external and environmental influences on the brain. From a theoretical perspective, findings highlight the need to explore environmental correlates of neuropsychological and cognitive deficits. From an applied and policy perspective, findings indicate potential avenues for individual-level treatment that may positively impact behavior.

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Adrian Raine

ETIOLOGY AND TREATMENT OF NEUROPSYCHOLOGICAL AND COGNITIVE

DEFICITS IN RELATION TO ANTISOCIAL BEHAVIOR

Rebecca Umbach

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in

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2018

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ETIOLOGY AND TREATMENT OF NEUROPSYCHOLOGICAL AND COGNITIVE

DEFICITS IN RELATION TO ANTISOCIAL BEHAVIOR

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ABSTRACT

ETIOLOGY AND TREATMENT OF NEUROPSYCHOLOGICAL AND COGNITIVE

DEFICITS IN RELATION TO ANTISOCIAL BEHAVIOR

Rebecca Umbach, M.S.

Adrian Raine, D. Phil.

Impaired neuropsychological and cognitive functioning are well-understood to be risk factors for antisocial behavior. There are, however, gaps in our knowledge of the etiology and effective treatment of neuropsychological and cognitive deficits. My dissertation examines these questions in a series of three papers. The first paper proposes a serial mediation model wherein neighborhood disadvantage and subsequent impaired neuropsychological functioning represent a partial explanation of the race-antisocial behavior relationship. In a community sample of male and female young adolescents, the hypothesized sequential path accounted for 10.8% of the relationship between race and antisocial behavior. The second paper examines the relationship between sleep and antisocial behavior, which has primarily been examined via correlational or extreme sleep deprivation studies. Using National Incidence-Based Reporting System and city-reported data, this paper exploits the natural experiment of daylight saving time (DST) to examine the effects of a very mild change in sleep on assault rates. The Monday directly following the advent of DST was associated with 3% fewer assaults as compared to the Monday a week later, which we hypothesize may be the result of fatigue. In contrast, we saw 3%

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GENERAL INTRODUCTION

It is generally agreed that research incorporating individual and environmental risk factors can help us better understand the etiology of antisocial behavior. Advances in technology and multidisciplinary approaches from a wide range of fields support the idea that biological, psychological, and environmental factors, alone and in conjunction, can influence both pro- and antisocial behavior. Across many disciplines, one of the best-supported individual-level variables at the intersection of genetics and environment is cognition—the unconscious and conscious processing of information (Beaver et al., 2009; Hughes et al., 2005; Wright, Beaver, Delisi, & Vaughn, 2008).

The areas of cognition particularly implicated in antisocial behavior include executive functioning and emotion processing, domains that have been linked to the prefrontal cortex (Miyake et al., 2000) and the amygdala (E. A. Phelps & LeDoux, 2005), respectively. Impairment of executive functioning may lead to poor self-control, an inability to plan for the future and adjust to changes, and weakened attention abilities (Morgan & Lilienfeld, 2000; Ogilvie, Stewart, Chan, & Shum, 2011), among other negative effects. Deficient emotion regulation may result in over- or under-regulation of emotions, in turn leading to internalizing or externalizing behavior (Roberton, Daffern, & Bucks, 2012). The important role of cognition in antisocial behavior is supported by a robust body of literature using various methodologies.

Cognition is not fixed from birth, and the plasticity of the human brain has been well-established (Duffau, 2006; Taupin, 2006). This plasticity can be a positive or a negative. On the one hand, studies have found various types of cognition can be

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improved through techniques of mindfulness (Zeidan, Johnson, Diamond, David, & Goolkasian, 2010), fitness programs (Colcombe & Kramer, 2003; Donnelly & Lambourne, 2011) and noninvasive brain stimulation (Brunoni & Vanderhasselt, 2014). On the other hand, sustained exposure to chemical neurotoxins (Bellinger, Stiles, & Needleman, 1992) and long-term exposure to trauma and sustained stress (Jaffee & Maikovich-Fong, 2011; Sampson, Sharkey, & Raudenbush, 2008; Sharkey, Tirado-Strayer, Papachristos, & Raver, 2012) are significant risk factors for long-term and deleterious effects on the brain and cognitive functioning. Furthermore, these neurotoxic risk factors are not independent—many of them share common etiologies such as living in a disadvantaged neighborhood (Noble, Houston, Kan, & Sowell, 2012; Noble, McCandliss, & Farah, 2007) and/or being incarcerated (Glenn & Raine, 2014).

While continued stress and negative environmental exposure has a well-documented and more permanent effect on the brain, it is also true that short-term and situational effects can produce notable consequences. Small things like mild loss of sleep (e.g., the time shift associated with daylight saving time [Gaski & Sagarin, 2011]) and experiences that produce stereotype threat (Davies, Spencer, Quinn, & Gerhardstein, 2002; Steele & Aronson, 1995) have been shown to affect cognitive performance. Cognition is not a stable trait, but rather a set of processes that can vary within as well as between individuals.

Given general agreement that environment and context play a role in helping shape human behavior and cognition, it follows that better understanding these

relationships can promote prosocial policy. On the front end, better understanding of environmental variables that can affect cognition can support policy to reduce exposure to negative environments in a number of realms, including criminal justice and city planning. On the back end, this research provides evidence for the importance of treatment programs and research that can help us harness the plasticity of the brain in an effort to interrupt antisocial trajectories.

The goal of this dissertation is to demonstrate the important influence of the environment on cognition, and identify the pathways through which impaired cognition can be linked to increased likelihood of antisocial behavior. This dissertation consists of three papers that examine cognition in three very different populations, showing that environmental effects are pervasive. By using differing methodologies to operationalize antisocial behavior and cognition, these papers provide convergent evidence for the importance of linking environmental and cognitive risk factors serially to better understand the etiology of antisocial behavior.

Paper 1 Summary

The first paper in this dissertation approaches the topic of the relationship between race and antisocial behavior by examining intermediate factors that may help explain the relationship. Namely, that blacks are more likely to reside in a disadvantaged neighborhood due to a number of historical and contemporary factors (e.g., legalized discrimination in housing). Because disadvantaged neighborhoods are associated with a variety of cognitive risk factors, including increased exposure to sustained stress and

neurotoxins such as lead, we propose a serial pathway from race to increased antisocial behavior through increased neighborhood disadvantage followed by impaired executive functioning.

This paper uses data from 341 11-12 year old males and females recruited from Philadelphia and surrounding suburbs. Based on their primary home address, participants were assigned a level of neighborhood disadvantage according to a composite score of variables that have been used in previous studies on neighborhood disadvantage. Executive functioning was measured using three measures derived from two neuropsychological tasks, which were then z-scored and combined for a composite score. Antisocial behavior was reported by both the parents and the children using a variety of measures including the Antisocial Screening Processes Device, the Reactive Proactive Questionnaire, and the Child Behavior Checklist. A serial mediation model (PROCESS Model 6) was employed to test whether the indirect pathway from race to neighborhood disadvantage to executive functioning to antisocial behavior was a significant mediator of the relationship between race and antisocial behavior. This significant pathway accounted for approximately 10.8% of the relationship. This result highlights two potential areas to address with interventions: improving neighborhood conditions, which is a macro-level and expensive proposition; and individual-level executive functioning, around which there is a body of research.

Paper 2 Summary

This paper examined whether short-term changes in sleep could affect behavior. A robust body of literature has connected poor sleep to increased aggression, negative mood, and antisocial behavior (Kamphuis, Meerlo, Koolhaas, & Lancel, 2012), while a couple of experimental studies have failed to find effects or found effects in the opposite direction (Cote, McCormick, Geniole, Renn, & MacAulay, 2013; Vohs, Glass, Maddox, & Markman, 2010). Much of the correlational literature depends on self-report measures of both sleep and antisocial behavior. The remaining literature uses experimental methods to test the effects of large amounts of sleep deprivation on performance on laboratory aggression paradigms. There is no existing literature on the effects of a small amount (i.e., one hour) of sleep change on antisocial behavior. This study exploits the advent and conclusion of daylight saving time to test the effects of sleep change on aggression.

Assault data from the National Incidence-Based Reporting System was combined with city-reported data from Philadelphia, Chicago, Los Angeles, and New York. Reported number of assaults the Monday following daylight saving time were compared with assaults the Monday one week later, to best control for weather and lighting. The same analysis was applied to the Monday following the return to standard time in the fall and the Monday one week later. Contrary to much of the existing literature, daylight saving time and the loss of an hour of sleep was associated with a 3% increase in assaults the day following, as compared to one week later. The return to standard time was associated with a 3% decrease in assaults, as compared to one week later. Multiple

robustness analyses supported the strength of the spring daylight saving time finding, but were less conclusive on the fall effects.

These findings indicate that the relationship between sleep and antisocial behavior is more complicated than it would appear from the existing literature. It may be that while poor/less sleep is associated with negative affect and intentions to act more aggressively, the behavioral result of a short-term loss of sleep (and perhaps, fatigue) is reduced aggression.

Paper 3 Summary

There is a long-standing belief that incarceration can have deleterious effects on cognition and psychological well-being (Haney, 2003, 2012). These hypothesized effects have ranged from subtle psychological effects to clinical levels of mental illness. Despite conjecture, however, no study to date has empirically and longitudinally looked at the effects of incarceration on cognitive functioning (with one notable exception using these data, Leonard et al., 2013). Accordingly, this paper examined whether incarceration is associated with a decline in cognitive performance, specifically in cognitive control, emotion regulation, and emotion recognition (all measured by a single neuropsychological task). In the event that findings provided an affirmative answer to the first research question, this paper also tested whether a Cognitive Behavioral Therapy/Mindfulness Training (CBT/MT) intervention can help buffer negative effects.

CBT/MT has been studied in healthy and clinical populations, as well as some forensic populations. It is thought to improve self-regulation through its focus on self-

awareness, attentional control, and emotion regulation (Tang, Hölzel, & Posner, 2015). Studies on the efficacy of mindfulness in treating antisocial behavior and associated criminogenic constructs have focused predominantly on outcomes of substance abuse and recidivism in incarcerated adult populations (Shonin, Van Gordon, Slade, & Griffiths, 2013).

This study recruited a sample of 197 incarcerated adolescent males from Rikers correctional facility to participate in a group-randomized longitudinal study. The experimental group received a combined mindfulness/cognitive behavioral therapy (Casarjian & Casarjian, 2003) for approximately 750 minutes over 3-5 weeks, while the active control received a sexual health and drug use reduction program during the same period. A computerized emotional go/no-go task (Tottenham, Hare, & Casey, 2011) was administered pre-treatment and three months after the conclusion of the treatment. Outcomes of emotion regulation, emotion recognition, and cognitive control were derived from the task.

Although both groups declined in performance over four months, the decline in emotion regulation and cognitive control was only significant for the control group. The decline in emotion recognition was significant for both groups. This study provides empirical and important support for a long-standing hypothesis about the negative cognitive effects of incarceration. Moreover, it demonstrates tentative support for the potential use of a mindfulness/CBT treatment in mitigating those negative effects.

PAPER 1. NEIGHBORHOOD DISADVANTAGE AND NEUROPSYCHOLOGICAL FUNCTIONING AS PART MEDIATORS OF THE RACE-ANTISOCIAL RELATIONSHIP: A SERIAL MEDIATION MODEL[1]

Abstract

We test a serial multiple mediation model in which the relationship between ethnicity and antisocial behavior is sequentially mediated by disadvantaged neighborhoods and impaired neuropsychological functioning. Parental and self-report measures of antisocial behavior were assessed in a community sample of 341 adolescent male and female children. Neighborhood disadvantage was assessed from census data. Neuropsychological functioning was evaluated using a computerized battery. Separate serial multiple mediation models were tested using nonexecutive functioning and executive functioning. The serial mediation model for executive functioning was supported, with the pathway from race to antisocial behavior through neighborhood disadvantage and executive functioning in serial accounting for 10.8 percent of the total effect of race on antisocial behavior. Findings support social neurocriminology theory by integrating neighborhood disadvantage and executive functioning as sequential mediators of the race – antisocial relationship. To our knowledge, these are the first findings to explain the race – antisocial relationship in terms of connected social and neuropsychological processes. While this pathway is significant, the effect is still

relatively small and thus should be understood as one of many mechanisms through which race may affect antisocial behavior. From a translational science standpoint, the identification of neurocognitive mechanisms by which neighborhood disadvantage predisposes to antisocial behavior suggests the potential benefits of cognitive enhancement techniques to remediate the negative effects of adverse neighborhoods on brain functioning in at-risk minority groups.

Background

There are well-documented differences in rates of antisocial behavior and violent offending among some ethnic and racial groups, particularly between blacks and whites. Blacks are more likely to engage in violent behavior than their white counterparts (Farrington, Loeber, & Stouthamer-Loeber, 2003; Felson & Kreager, 2015; Hawkins, Laub, Lauritsen, & Cothern, 2000; LaFree, 1995). The race differences in antisocial behavior are arguably spurious, confounded by the relationship between race and socioeconomic status (SES), and subsequent criminogenic risk factors to which blacks are disproportionately exposed (Kawachi, Kennedy, & Wilkinson, 1999; Piquero, Moffitt, & Lawton, 2005; Sampson, Morenoff, & Raudenbush, 2005; Shaw & McKay, 2014; Wilson, 2009; Zimmerman & Messner, 2013). Sociological explanations of the mechanisms of the race-antisocial behavior relationship often focus on neighborhood-level concepts such as collective efficacy and social disorganization (Bellair & McNulty, 2005; Lauritsen & White, 2001; McNulty, Bellair, & Watts, 2012; Sampson & Wilson, 1995). As mechanisms, these "place-based" macrosocial theories are strongly supported

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by the literature, which finds that controlling for neighborhood and family disadvantage greatly attenuates or eliminates the relationship between race and antisocial behavior (Bellair & McNulty, 2005; Brody et al., 2001; Lauritsen & White, 2001; Sampson et al., 2005).

Perhaps because of the strength of the support for traditional sociological explanations for the race-antisocial behavior relationship, there are few studies proposing additional mechanisms for how neighborhood disadvantage influences antisocial behavior, representing a gap in the literature that needs addressing. Exceptions include Kaufman et al.'s (2008) examination of the racial differences in criminal offending using General Strain Theory, Barnes et al.'s (2016) finding that pre- and perinatal risk factors help explain the mean differences in self-regulation between races, and studies by McNulty, Bellair, and Watts (2012) and Choy et al. (2015) that integrate neighborhood-level and individual-level theories into "social neurocriminology" models. These latter studies show that environmental risk factors can affect biological risk factors such as cortisol, heart rate, and neuropsychological functioning in a way to predispose to antisocial behavior (Choy et al., 2015; Kishiyama, Boyce, Jimenez, Perry, & Knight, 2009; McGrath, Matthews, & Brady, 2006; McNulty et al., 2012).

This research is designed to fit within the social neurocriminology framework by testing for the presence of an additional mechanism in the race to neighborhood disadvantage to antisocial behavior relationship. We utilize a mediation model (Hayes, 2013), which uses OLS regression to partition the total effect of one variable (X) on

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another (Y) into direct and indirect effects. Consistent with the prior literature, we expect to account for a significant amount of the relationship between race and antisocial behavior through mediating variables. Specifically, we aim to test a serial multiple mediation model process in which being a racial minority (X) is associated with an increased degree of neighborhood disadvantage (M_1), which then leads to impaired neuropsychological functioning (M_2), predisposing to antisocial behavior (Y). This framework is similar to a structural equation modeling framework, but utilizes OLS regression instead of maximum-likelihood methods (see Hayes, 2013, p. 159 for a full discussion on the merits of each).

Race and Neighborhood Disadvantage

Racial minority individuals in the US, specifically blacks, are significantly more likely than whites to live in disadvantaged neighborhoods and communities for a number of reasons rooted in economic and social discrimination (Adelman, Tsao, Tolnay, & Crowder, 2001; Chauhan & Reppucci, 2009; Krivo, Peterson, Rizzo, & Reynolds, 1998; Piquero et al., 2005; Sampson & Wilson, 1995). The deindustrialization of the inner city, declining need for low-skill manufacturing jobs, and the rise of jobs requiring education and advanced skills in the 1960s led to an increase in the concentration of poverty and disorder in inner-city neighborhoods, which prompted middle-class, predominantly white, families to migrate to the suburbs (Rankin & Quane, 2000; Sampson & Wilson, 1995; Wilson, 2012). Blacks faced strong barriers to residential mobility, including insufficient resources and formalized discriminatory housing market practices (Peterson

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& Krivo, 1999; Sampson & Wilson, 1995). While the formalized practices have been discontinued, the ghettoization of blacks is ongoing. One cause is neighborhood appreciation inequality, wherein homes in disadvantaged neighborhoods and neighborhoods with high proportions of blacks appreciate more slowly than comparable housing in white communities, resulting in a racial wealth gap that would make it difficult for blacks to sell a home and move to a better neighborhood (Flippen, 2004). In summary, as described by Sampson (1995), macrostructural factors including racial segregation, housing discrimination, structural economic transformation, and class-linked out-migration from the inner city have combined to concentrate urban black poverty, family disruption, and disorganized communities in the inner city.

Low social capital, poor collective efficacy, and social isolation can prevent residents of disadvantaged neighborhoods from developing informal social control networks and enacting meaningful and comprehensive change (Bursik & Grasmick, 1993; Sampson, Raudenbush, & Earls, 1997). Resulting effects of this residential segregation and ghettoization of blacks include advantages for whites and costly criminogenic outcomes for blacks, such as increased rates of unemployment, teenage pregnancy, increased risk of chronic maltreatment, and academic failure (Jaffee & Maikovich-Fong, 2011; Massey, Condran, & Denton, 1987; Peterson & Krivo, 1999; Ross & Mirowsky, 2001). The intergenerational transmission of poverty furthermore suggests that many of the individuals born in disadvantaged neighborhoods and subjected to detrimental outcomes remain there as adults, maintaining the past and current

residential segregation by race (Sampson et al., 2008). Given these facts, it is expected (not surprisingly) that blacks will report higher levels of neighborhood disadvantage as compared to whites.

Neighborhood and Neuropsychological Functioning

As noted by Moffitt (1994), even early in life, blacks are disproportionately affected by risk factors for impaired neuropsychological functioning (e.g., insufficient prenatal care, poor infant nutrition). These individual risk factors are often compounded by differential exposure to neighborhood disadvantage and associated constructs (e.g., poverty, sustained stress). Well-replicated risk factors for reduced cognitive functioning include family dysfunction, exposure to environmental toxins, lack of access to material resources and sufficient schooling, sustained poverty, and higher rates of stress (Bradley & Corwyn, 2002; Kohen, Leventhal, Dahinten, & McIntosh, 2008; Lanphear, Byrd, Auinger, & Schaffer, 1998). The relationship between neighborhood disadvantage and neuropsychological deficits is supported by a robust literature employing a variety of measures such as traditional academic-based testing methods (Leventhal & Brooks-Gunn, 2000; Sharkey, 2010; Sharkey, Schwartz, Ellen, & Lacoe, 2014; Waber, Gerber, Turcios, Wagner, & Forbes, 2006) and computerized or pen-and-paper neurocognitive batteries (Farah et al., 2006; Raver, Blair, & Willoughby, 2013). Neighborhood disadvantage has been associated with impaired cognitive performance in general, and more specifically, with verbal and language ability, and executive functioning processes including memory and emotion regulation (Farah et al., 2006; Pechtel & Pizzagalli, 2011; Sampson et al.,

2008). The notion that neighborhood disadvantage can affect neuropsychological functioning in individuals is in line with social neurocriminology perspectives that have begun to integrate environmental and biological risk factors into models (Choy et al., 2015; McNulty et al., 2012; Moore et al., 2016).

Although neighborhood disadvantage and associated constructs are related to numerous types of specific neuropsychological functioning, one of the strongest correlates of such disadvantage is executive functioning, which include complex cognitive processes such as mental flexibility, selective attention, and inhibitory control (Farah et al., 2006; Hackman & Farah, 2009; Hackman, Gallop, Evans, & Farah, 2015; Moore et al., 2016; Noble et al., 2007; Noble, Norman, & Farah, 2005; Pechtel & Pizzagalli, 2011; Raver et al., 2013; Waber et al., 2006).

This study aims to add to the social neurocriminology literature by focusing on executive functioning as a mediator of the neighborhood disadvantage-antisocial behavior relationship. Executive control has been linked to frontal lobe functioning, and is considered necessary for goal-oriented and prosocial behavior (Morgan & Lilienfeld, 2000). Executive functioning may be particularly susceptible to sustained stress for two specific reasons: first, the prefrontal cortex has a high density of hormone transmitters and receptors that are stress-susceptible, and second, the prefrontal cortex is a higher-order structure, and thus develops later in life (Giedd & Rapoport, 2010; Pechtel & Pizzagalli, 2011) when individuals tend to experience more stress (Hammen & Rudolph, 2003). Accounting for differential exposure to neighborhood disadvantage, blacks would

be particularly at risk for the effects of chronic stress on executive functioning. Accordingly, we anticipate that higher levels of neighborhood disadvantage will be negatively associated with scores on a computerized neuropsychological battery. Additionally, it is expected that the negative association between neighborhood disadvantage and neuropsychological functioning will be strongest for measures of executive functioning, as compared to measures of non-executive functioning, which includes processes such as short-term memory and visual-spatial perception (Arffa, 2007).

Executive Functioning and Antisocial Behavior

While Moffitt (1990) and others (Marsh & Blair, 2008; McNulty et al., 2012) have found links between antisocial behavior and non-executive functioning processes, executive functioning is considered the best-replicated cognitive correlate of antisocial behavior (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Executive dysfunction can interfere with an individual's ability to control their own behavior and thus may result in maladaptive behaviors (Moffitt, 1993, 1994) and inappropriate emotional responses, such as aggression, reward seeking, and inappropriate sexual behavior (Williams, Suchy, & Rau, 2009). The relationship between neuropsychological functioning and antisocial behavior is not moderated by age, sex, or ethnicity (Bellair, McNulty, & Piquero, 2016; Morgan & Lilienfeld, 2000), however, one finding (Bellair and McNulty, 2010) suggests that the relationship may be moderated by neighborhood. Furthermore, Moffitt (1993) proposes that neurocognitive deficits may characterize persistently antisocial individuals,

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as opposed to adolescent-limited ones, suggesting the relationship is developmentally specific. Significantly, while the antisocial literature has often focused on the executive function of self-control (Cauffman, Steinberg, & Piquero, 2005), this paper aims to test executive functioning more broadly due to developmental and age-related differences in separate components of executive functioning (Huizinga, Dolan, & van der Molen, 2006).

Support for the importance of executive functioning in relation to antisocial behavior is derived primarily from three types of studies: (1) neurological case studies of subjects with frontal lobe damage (S. W. Anderson, Bechara, Damasio, Tranel, & Damasio, 1999; Bechara, Damasio, Damasio, & Anderson, 1994); (2) brain imaging studies examining the structure and function of the frontal cortex (Y. Yang & Raine, 2009); and (3) studies utilizing computer-based measurements of neurocognitive ability (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Patients with frontal lobe damage can exhibit acquired psychopathy, demonstrating many of the same traits found in antisocial personality disorder (Damasio, Tranel, & Damasio, 1990). Brain imaging studies of antisocial populations are fairly consistent in showing significantly reduced prefrontal structure and function in antisocial populations (Kiehl, 2006; Motzkin, Newman, Kiehl, & Koenigs, 2011; Y. Yang & Raine, 2009). Meta-analyses by Morgan and Lilienfeld, (2000) and Ogilvie et al., (2011) of executive functioning in antisocial populations found mean Cohen's *d* effect sizes of 0.62 and 0.47, respectively. Relatively consistent findings from different but related studies provide support for another hypothesis of the current

study, which is that executive functioning will be negatively associated with antisocial behavior.

We consider testing a non-executive functioning (specifically incorporating episodic memory and emotion recognition) model as a type of sensitivity analysis. There is a lacuna in the neurocognitive literature regarding the relationship between most types of non-executive functioning and antisocial behavior. This may be a result of the "file drawer problem" (Rosenthal, 1979). Indeed, findings from this study, null or otherwise, will help to fill this gap regarding the role of non-executive functioning in antisocial behavior.

Conceptual Framework for the Current Study

There are at least two frameworks spawned by Moffitt's taxonomy that guide the current study. The work of Piquero et al. (2005) and McNulty et al. (2012) provide two examples of frameworks that look directly at the relationships between race, antisocial behavior, neuropsychological risk factors, and neighborhood disadvantage. Piquero et al. (2005) used race-specific models to see whether contextual-, familial- and individual-level risk factors predict antisocial behavior differently for blacks and whites and across different neighborhood contexts. We note that while they also examine neighborhood disadvantage and neuropsychological functioning (through a birth weight proxy) as risk factors for antisocial behavior, their theoretical pathway differs significantly from ours. Moffitt's work hypothesized that the neurocognitive impairments associated with life-course persistent offending would be present at birth, due to pre and peri-natal risk

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factors. Following Moffitt's hypothesis, the framework of Piquero et al. (2005) assumes that neighborhood disadvantage would compound the negative effects of these early impairments, not necessarily cause them. In contrast, as laid out explicitly below, we posit that neighborhood disadvantage alone can have a deleterious effect on neuropsychological functioning.

Alternatively, McNulty et al. (2012) tested a model pathway more closely resembling ours. Using five waves of the 1997 National Longitudinal Survey of Youth, they found that neighborhood disadvantage and resulting verbal deficits, as measured by word knowledge and paragraph comprehension, serially mediated the race-violence relationship. Their longitudinal data allows for a stronger claim to a causal integrated model, which in turn provides support for our model as our cross-sectional data precludes a definitive temporal ordering. We build on their study by making use of a computerized neurocognitive battery designed to test several domains, including executive functioning, arguably the best-replicated domain of neuropsychological functioning associated with antisocial behavior (Morgan and Lilienfeld, 2000; Ogilvie et al., 2011). Furthermore, McNulty et al. (2012) demonstrated the importance of this pathway for predicting violence (whether the respondent had attacked someone with the intention of hurting them in the past, and the frequency of such acts), a relatively severe measure of antisocial behavior. The richness of our data allows for a less severe and more nuanced measure of antisocial behavior, created by combining a number of parental and self-report questionnaires.

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Summary of Study Hypotheses: A Serial Mediation Model

This paper tests several inter-related initial hypotheses that lay the foundation for our primary serial mediation hypothesis. First, it is hypothesized that race and neighborhood disadvantage will be related, such that being black will be associated with higher levels of neighborhood disadvantage. Second, we hypothesize that higher levels of neighborhood disadvantage will be associated with poorer neuropsychological functioning, particularly in the domain of executive functioning. Finally, we hypothesize that impaired executive functioning will be associated with increased levels of antisocial behavior.

Based on these initial hypotheses, for our primary hypothesis four models were tested in total as there were two distinct measures of neuropsychological functioning (executive and non-executive functioning), and two reporters of antisocial behavior (the child and the parent). The expectation is that the serial mediation path in the executive functioning models, broadly illustrated in Fig. 1.1, will be significant. Thus, we hypothesize that adverse environment and frontal lobe dysfunction sequentially mediate the relationship between race and antisocial behavior. On the other hand, the relatively weak literature supporting the role of non-executive functioning in antisocial behavior leads to a prediction of null findings in models including these cognitive processes.

Methods

Participants

This study draws on data from the Healthy Brains and Behavior study. Full details of recruitment and exclusionary criteria can be found in Liu et al. (2013) and Richmond et al. (2013). Briefly, the sample consisted of 11 and 12 year old male and female children residing in the city of Philadelphia, PA, or the surrounding suburbs. Within the study area, subjects were recruited through advertisements, personal referrals, targeted mailings, and fliers placed in recreation centers, daycares, schools, and other community centers. Youths diagnosed with psychosis, mental retardation, or a pervasive developmental disorder were excluded.

The original sample consisted of 454 subjects. Of this original group, eight subjects were later deemed ineligible or withdrew. Complete data were collected for 370 subjects. 29 subjects who identified as Hispanic/Latino, Asian/Pacific Islander, Native American, "Other" and "Multiracial" were eliminated from analyses, leaving a total of 341 subjects. The resulting sample was 50.1 percent male and had a mean age of 11.9 years (SD = 0.58). No significant differences were observed in age or sex between individuals who were included in the analyses and those who were not (ps > 0.05). The caregiver of each participant served as an informant for the child's behavior, demographics, and living circumstances. Caregiver participation primarily involved the biological mothers (89.7 percent). Written informed consent and assent were obtained from both parents and children, and the study protocols were approved by both the Institutional Review Board of the University of Pennsylvania and the Philadelphia Department of Health.

Race

Of the 370 participants with complete data, 13.0 percent ($n = 48$) were white, 79.2 percent ($n = 293$) were black, and 7.8 percent ($n = 29$) identified as Hispanic, Asian, Native American, Other, or Multiracial. Subjects who did not self-identify as white or black were dropped from analyses, leaving 341 subjects in the analyses. Race was coded as 1 if black, and 0 if white.

Antisocial Measures

The child and parent separately reported on the child's antisocial behavior using several questionnaires, described in detail below. The subscale totals of each of the questionnaires for each informant were then subjected to a principal factor analysis, providing two overall measures of child antisocial behavior (parent-report and child self-report).

Parent-report externalizing behavior

Subject caregivers completed the Child Behavior Checklist (CBCL), a well-standardized and widely used psychometric instrument (Achenbach, McConaughy, & Howell, 1987). The externalizing behavior subscale consists of the 17-item aggressive behavior (e.g. "destroys his/her things") and 15-item rule-breaking behavior (e.g. "steals at home") scales. These two syndromes have been used in previous research on childhood antisocial behavior (Baker, Jacobson, Raine, Lozano, & Bezdjian, 2007; Choy et al., 2015). The internal reliabilities (Cronbach's alpha) of the 15-item rule-breaking behavior scale and 17-item aggressive behavior scale were 0.96 and 0.95, respectively.

Parent-report disruptive behaviors disorders

Caregivers completed the 24-item Conduct Disorder and Oppositional Defiance Disorder Questionnaire (COD; Raine, unpublished). The instrument was designed to assess conduct disorder and oppositional defiant disorder in terms of the Diagnostic and Statistical Manual of Mental Disorders-IV-TR symptoms. The questionnaire asks about the occurrence of behaviors in the past year based on a 3-point ordinal scale: 0 (never), 1 (sometimes), or 2 (often). Items used to assess conduct disorder and oppositional defiant disorder, respectively, include how often a subject has "bullied or threatened someone," and how often a subject has "argued with adults" (see Appendix A). The internal reliabilities (Cronbach's alpha) of the 15-item conduct disorder scale and 8-item oppositional defiant disorder scale were 0.78 and 0.90, respectively.

Parent-report child psychopathy

The 20-item Antisocial Processes Screening Device (Frick, Bodin, & Barry, 2000) was completed by parents. The scale assesses psychopathic traits in children by asking parents to report how well items such as "engages in illegal activities" describe their child on a three-point scale. The overall scale consists of three subscales to assess narcissism (Cronbach's alpha = 0.79), impulsivity (Cronbach's alpha = 0.68), and callous-unemotional traits (Cronbach's alpha = 0.65).

Overall parent-reported antisocial score

To create an overall antisocial measure for mediation analyses, the above 7 subscales were factor analyzed using principal component analysis with Kaiser's criterion used to

select number of factors. Only the first factor had an eigenvalue > 1, accounting for 64 percent of the total variance. With the exception of APSD callous-unemotional scale, which loaded 0.50, all scales loaded between 0.80 and 0.89 on this first principal component, with standardized factor scores saved using the regression method (see Appendix B for details of factor loadings). Higher scores on this scale reflected worse behavior. These scores were used for the main analyses. As noted below, another factor analysis was conducted excluding the poorest loading item (the APSD callous-unemotional scale). Again, only the first factor had an eigenvalue > 1, which accounted for 56 percent of the variance. All scales loaded between 0.64 and 0.85 on this first principal component. As outlined in the supplemental analyses section, analyses were re-run using the resulting factor scores.

Self-report aggression

Subjects completed the 15-item short version of the Aggression Questionnaire (Buss & Warren, 2000). The scale consists of five subscales composed of three items each: physical aggression (Cronbach's alpha = 0.73), verbal aggression (Cronbach's alpha = 0.68), indirect aggression (Cronbach's alpha = 0.64), anger (Cronbach's alpha = 0.61), and hostility (Cronbach's alpha = 0.70). We note that the reliability of each of the scales was low to moderate, mostly likely due to the few items per subscale (Gliem & Gliem, 2003). The internal reliability of the total scale, however, was high (Cronbach's alpha = 0.88).

Self-report reactive and proactive aggression

Specific types of aggression were measured using the reactive and proactive subscales of the Reactive-Proactive Aggression Questionnaire (Raine et al., 2006). The reactive subscale consists of 11 items (e.g. "hit others to defend yourself") and the proactive subscale consists of 12 items (e.g. "had fights to show who was on top"). The RPQ has been validated in multiple adolescent samples with documented cross-cultural validity (Baker et al., 2007; Raine et al., 2006; Raine, Fung, & Lam, 2011). Subjects responded on a three-point scale (0 = never, 1 = sometimes, 2 = often). The scores for each subscale were summed. The internal reliability of each of the reactive and proactive subscales were high (Cronbach's alpha = 0.81 and 0.82, respectively).

Self-report disruptive behavior disorders

In addition to caregivers, subjects also completed the 24-item self-report version of the Conduct and Oppositional Defiance Disorder Questionnaire (COD; Raine, unpublished). The internal reliabilities (Cronbach's alpha) of the 15-item conduct disorder scale and 8-item oppositional defiant disorder scale were 0.83 and 0.83, respectively.

Self-report child psychopathy

The 20-item Antisocial Process Screening Device (APSD; Frick et al., 2000) was also completed by the subjects, edited minimally to reflect self-reporting (e.g. "[your child] engages in illegal activities" became "you engage in illegal activities"). Reliability in general was poorer in self-report than in parent-report. The Cronbach's alpha for the narcissism, impulsivity, and callous-unemotional traits scales was 0.35, 0.68, and 0.59,

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respectively. An acknowledged weakness of this scale is low reliability within subscales for children, even with slightly older samples (Achenbach et al., 1987; Munoz & Frick, 2007), although we do note that the internal reliability (Cronbach's alpha) for the total scale was 0.71.

Self-report externalizing behavior

Subjects completed the Youth Self-Report (YSR), the self-report complement to the Child Behavior Checklist (Achenbach et al., 1987). Like the CBCL, the scales of interest were the 17-item aggressive behavior (e.g. "I get in many fights") and 15-item rule-breaking behavior (e.g. "I disobey my parents"). The internal reliabilities (Cronbach's alpha) of the 15-item rule-breaking behavior scale and 17-item aggressive behavior scale were 0.83 and 0.69, respectively.

Overall child self-report score

To create an overall self-report antisocial measure for mediation analyses, the above 14 self-report subscales were factor analyzed using principal component analysis with Kaiser's criterion used to select number of factors. Only the first factor had an eigenvalue > 1, accounting for 52 percent of the total variance. All scales loaded between 0.64 and 0.86 on this first principal component, except for the APSD callous-unemotional subscale, which loaded at 0.20 (see Appendix B for details of the factor loadings). Standardized factor scores were saved using the regression method. Higher scores reflect more antisocial behavior, and these scores were used for the main analyses. As in the parent self-report factor, we note the low loading of the APSD callous-unemotional item,

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and ran additional analyses (as outlined in the supplemental analyses section) using scores derived from a principal component analysis excluding this scale. Only the first factor had an eigenvalue > 1, which accounted for 56 percent of the variance. All scales loaded between 0.66 and 0.86 on this first principal component. As outlined in the supplemental analyses section, analyses were re-run using the resulting factor scores.

Neighborhood Disadvantage

The degree of neighborhood disadvantage experienced by the subject was based on the census block-group in which the subject resided. Of the 341 subjects in the analysis, subjects lived in a total of 258 block groups, with an average of 1.32 subjects living in each group. Items comparable to those used in previous research (Fagan & Wright, 2012; Piquero et al., 2005; Sampson et al., 1997; Simons, Simons, Chen, Brody, & Lin, 2007) to evaluate structural neighborhood disadvantage were included in a principal components analysis. Data regarding these items were derived from the most temporally proximate available American Community Survey 5-year estimate (2005-2009; U.S Census Bureau). Based on their correlation with the first principal component, 7 items were selected: percent of female headed family households with children under age 18 years, percent of population that is 25 years or over that has less than a high school education, percent of population that is less than 18 years old, percent of households receiving public assistance income, percent of occupied housing units that are renter occupied, percent vacant housing units, and percent of population living below the poverty level. The standardized Cronbach's alpha of the scale was 0.77. Standardized

scores were summed to create a neighborhood disadvantage index, with higher scores indicating higher levels of neighborhood disadvantage.

In addition to this comprehensive variable, two other neighborhood disadvantage variables were computed. The first is like the one above, but excludes the percent of occupied housing units that are renter occupied to best isolate neighborhood disadvantage as opposed to residential turnover. The second is a simple summed measure of the standardized scores of percent of households receiving public assistance income and percent of female headed family households with children under age 18 years. This more closely resembles the scale used in the prior literature (e.g., Bellair & McNulty, 2005; McNulty, Bellair, & Watts, 2012). Analyses were also rerun using these variables.

Neuropsychological Functioning

Neuropsychological functioning was assessed from five subtests of the Penn Computerized Neurocognitive Battery (Gur et al., 2001, 2010), a neuropsychological test battery that has been validated in healthy children (Gur et al., 2012). In this study, individual tests were selected based on prior findings on cognitive correlates of antisocial behavior and an aim to be comprehensive across the domains. The resulting 30-minute battery included: two tests of executive function, the Penn Conditional Exclusion Test (PCET), and the Penn Continuous Performance Test (PCPT); two tests of episodic memory, the Penn Word Memory Test (PWMT) and the Visual Object Learning Test (VOLT); and one measure of social cognition, the Penn Emotion Identification Test

(PEIT). In all cases, scores were corrected (multiplied by -1) as necessary such that higher scores indicate better performance.

The CNB was administered by assessors trained in a standard protocol. The didactic and hands-on training sessions included exercises in proctoring instruction, assignment of validity codes to indicate data quality, and noting protocol deviation as well as technical and security issues. Prior to administering the CNB to research participants, assessors observed administrations and conducted mock administrations (Gur et al., 2010). Additionally, their first CNB administrations were observed to ensure professionalism and adherence to protocol.

Penn conditional exclusion test (PCET)

The PCET is a measure of abstraction and concept formation, and is considered a test of executive functioning. Participants select the object that does not belong with the other three based on one of three latent principles (line thickness, shape, and size). The participant is guided by feedback, and sorting principles change after the participant completes 10 successful trials. Subject's score was calculated as the number of categories solved multiplied by the proportion of correct choices, with one added to the number of categories to avoid a floor effect of zero for individuals who did not solve any category.

Penn continuous performance test (PCPT)

The PCPT is an executive functioning task that measures visual attention and vigilance. 7-segment displays are presented at 1 hertz. Participants are asked to press the keyboard space bar when they form a digit (for the first half of the test) or a letter (for the

second half of the test). The test is a total of 6 minutes long. The number of true

positives, true negatives, false positives, and false negatives were calculated. This test

contributed separate measures of inattention and impulsivity through number of omission

(false negative) and commission (false positive) errors, respectively (e.g. Yakir et al.,

2007). Error measures were multiplied by -1 for consistency of interpretation across test

scores, so that higher scores reflect better performance.

Penn word memory test (PWMT)

This task measures episodic memory using verbal stimuli. The PWMT presents 20

target words mixed with 20 distractors equated for frequency, length, concreteness and

low imageability. A 20-minute delayed recognition test follows in which subjects are

asked whether a word presented was included on the target list on a 1 to 4 scale

(definitely yes; probably yes; probably not; definitely not). Subject's score was calculated

as number of correct responses.

Visual object learning test (VOLT)

This task measures episodic memory for spatial stimuli. The VOLT uses the same

procedure as the PWMT, employing Euclidean shapes instead of words as stimuli.

Subject's score was calculated as number of correct responses.

Penn emotion identification test (PEIT)

The PEIT is designed as a measure of social cognition. 40 photographs of faces

depicting various emotions are displayed one at a time, and the participant's task is to

identify which one of five emotions (i.e., happy, sad, angry, fearful, and neutral) best

describes the emotion depicted. The faces are balanced for sex, age, and ethnicity. Subject's score was calculated as number of correct responses.

Overall scores

The raw scores for each test were z-transformed. The a priori interest in the specificity of neuropsychological domains dictated the need for separate executive functioning and non-executive functioning scores. The three scores for the two executive functioning tests (i.e. PCPT false positives, PCPT false negatives, and PCET) were summed to create an index of executive functioning, with higher scores indicating greater executive functioning. The PCET is specifically designed to measure executive functioning (Kurtz, Wexler, & Bell, 2004), while the PCPT has been assigned to the executive functioning domain in prior literature (Gur et al., 2001, 2010, 2012). Executive functioning deficits have been robustly associated with antisocial behavior (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011), and composite measures of executive functioning have been used in prior research on childhood antisocial behavior (Fatima & Sheikh, 2014). The non-executive functioning composite score was computed by summing the standardized scores of the remaining tests: the PWMT, the VOLT, and the PEIT. Mirroring the executive functioning composite score, a higher score reflects better performance. While these tests target neuropsychological domains that have been related to antisocial behavior in some studies (Marsh & Blair, 2008; Raine et al., 2005), they have not been as robustly supported as executive functioning (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011).

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Potential Confounds

Age, gender, and parental education and offending were examined as possible confounders of this mediation model. Gender was coded as 0 for female or 1 for male. A proxy for parental IQ was based on parental education, calculated as the mean years of education of the biological mother and father. Caregivers reported whether either biological parent had ever been imprisoned. If either had been imprisoned, the child was given a score of 1. Accounting for these parental factors was part of an effort to control for the genetic and environmental influences of an antisocial parent as much as possible within the limited methodology of this study, which does not employ a twin sample (J. C. Barnes et al., 2014).

Statistical Analyses

Bivariate correlations among the observed study variables were performed. Independent sample t-tests were conducted to test for differences between blacks and whites on the study variables. Tests were conducted using SPSS statistical software (IBM SPSS Statistics Version 22.0). A serial multiple mediation model was tested in the PROCESS macro (Hayes, 2013) to test neighborhood disadvantage and neuropsychological functioning as serial mediators of the race-antisocial behavior relationship. It was hypothesized that race would indirectly exacerbate risk for antisocial behavior through these mediators. Specifically, in serial fashion, it is hypothesized that being a minority subject (X) would be associated with higher levels of neighborhood disadvantage (M_1), and in turn, poorer neuropsychological functioning (M_2), and thus

higher levels of antisocial behavior (Y). A multiple serial mediator model with two mediators (neighborhood disadvantage and neuropsychological functioning) provides three specific indirect effects that sum to a total indirect effect. The specific indirect effects in this model were (1) through neighborhood disadvantage (a_1b_1); (2) through neuropsychological functioning (a_2b_2); and (3) through neighborhood disadvantage and neuropsychological functioning in sequence ($a_1a_3b_2$). This final indirect effect is the specific indirect effect that, if significant, supports serial multiple mediation (Hayes, 2013). Additionally, the percentage of the total effect explained by the specific indirect pathways are calculated through division.

Models were run using the executive functioning and non-executive functioning scores, and each type was tested separately using child self-report and parental report antisocial behavior as outcomes. By testing multiple models, this study aims to distinguish between executive and non-executive functioning, as the former is particularly implicated in the antisocial behavior literature (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011).

Analyses require (1) point estimates to be calculated for the direct and indirect effects linking race and antisocial behavior and (2) inferential tests to be conducted to determine whether these effects are different from zero (Preacher & Hayes, 2008). To conduct the inference tests for the indirect effects, bootstrapping was used taking 10,000 samples from the original data set ($N = 341$ for the parent-report models, and $N = 307$ for the child self-report models) to construct 95 percent bias-corrected confidence intervals

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(Hayes & Scharkow, 2013). Bootstrapping is considered superior to the Sobel test because it makes no assumptions about normality in the sampling distribution, and reduces likelihood of Type I error (MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008). Following convention, an indirect effect is significant if the 95 percent confidence interval does not include 0.

Results

Bivariate Associations

Bivariate correlations among the observed variables are shown in Table 1.1. Correlations were consistent with expectations. Higher levels of antisocial behavior and neighborhood disadvantage were observed in black subjects ($p < 0.05$). Neighborhood disadvantage was positively associated with antisocial behavior ($p < 0.01$). Lower scores on the executive functioning composite score are associated with higher levels of parent-reported antisocial behavior and neighborhood disadvantage, and being a minority participant (ps < 0.01). In contrast, lower scores on the non-executive functioning score are not significantly associated with race, antisocial behavior, or neighborhood disadvantage. Notably, although child-report measures trend in the same direction, effects for the parent-reported antisocial behavior measures are stronger and more in line with expectations based on the established literature. Specifically, Fisher r to z transformations reveal the child self-report antisocial factor to be significantly less correlated with executive functioning ($r = -0.03$), as compared to the parent-report factor ($r = -0.24$, $p < 0.01$).

Independent Sample T-tests

Independent sample t-tests revealed significant differences in means for neighborhood disadvantage, executive functioning, and antisocial behavior (both child self-report and parent report) between blacks and whites. There were no significant differences in non-executive functioning for blacks as compared to whites (see Table 1.2).

Mediating Mechanisms

In order to test the final (and primary) hypothesis, a serial multiple mediator model was specified (Andrew F Hayes, 2013; PROCESS Model 6). This model examined whether the effect of race on antisocial behavior is mediated serially, with the hypothesized causal flow moving from race (X) to neighborhood disadvantage (M_1) to poorer neuropsychological functioning (M_2) to antisocial behavior (Y). Because our cross-sectional data restricts our ability to infer the temporal order of our variables, we base these pathways on theory and prior literature (McNulty et al., 2012). To assess specificity, we compared the two neuropsychological domains (non-executive functioning and executive functioning) against parent-report and child-report antisocial measures as outcomes in separate serial multiple mediation models.

Parent-report data: executive functioning

The first mediation model tested the executive functioning composite score. All path coefficients for the full PROCESS model are shown in Fig. 1.2 and the first column of Table 1.3. Results of this analysis revealed that the total effect of race on antisocial

behavior was significant ($c = 0.37$, $p < 0.05$, 95% CI = 0.08 to 0.66); this total effect remained unchanged in both parent-report data models (using executive and non-executive functioning scores as mediators), as X and Y remained constant. The results of this model supported our hypothesis that neighborhood disadvantage and executive functioning together would mediate the relationship between race and antisocial behavior. Accounting for the mediators (total indirect effect = 0.36, 95% CI = 0.21 to 0.52) eliminated the relationship between race and antisocial behavior, and the total direct effect was non-significant ($c' = 0.05$, $p = 0.75$, 95% CI = -0.27 to 0.38).

The predicted serial mediation model was supported, as the specific indirect effect of race on antisocial behavior through neighborhood disadvantage and executive functioning was significant ($a_1a_3b_2 = 0.04$, CI = 0.01 to 0.09) and accounted for 10.8 percent of the total effect. Additionally, the remaining specific indirect effects were significant ($a_1b_1 = 0.21$, CI = 0.04 to 0.39; $a_2b_2 = 0.07$, CI = 0.01 to 0.15), accounting for 56.8 percent and 17.8 percent, respectively, of the total effect of race on antisocial behavior. In all, the indirect effects accounted for 85.5 percent of the total effect.

Parent-report data: non-executive functioning

Non-executive functioning was also tested. Coefficients can be found in Fig. 1.3 and the second column of Table 1.3. The total indirect effect was significant (indirect effect = 0.27, CI = 0.09 to 0.44), and accounting for the mediators rendered the total direct effect non-significant ($c' = 0.11$, $p = 0.52$). However, results of this analysis revealed insufficient support for the hypothesized serial mediation. The specific indirect

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effect of race on antisocial behavior through both neighborhood disadvantage and non-executive functioning was statistically non-significant ($a_1a_3b_2 = -0.00$, CI = -0.02 to 0.01). Examination of each specific indirect effect (see Table 1.3) indicated that the only statistically significant indirect effect was through neighborhood disadvantage in isolation ($a_1b_1 = 0.25$; CI = 0.08 to 0.43), accounting for 67.8 percent of the total effect of race on antisocial behavior.

Child self-report data

The same analyses were run using the child self-report antisocial factor (see columns three and four of Table 1.3). Neither of the models were supported using this outcome measure. In both the executive functioning model and the non-executive functioning model, the confidence intervals for the specific indirect effects of interest contained zero ($a_1a_3b_2 = -0.00$; CI = -0.02 to 0.02; $a_1a_3b_2 = -0.00$; CI = -0.02 to 0.01; respectively) as can be seen the third and fourth columns of Table 1.3.

Potential Confounds

After controlling for age, gender, years of parental education and parental offending, results remained substantively unchanged. As one example, in the model with executive functioning and parent-reported ASB, the total indirect effect was significant (indirect effect = 0.20, CI = 0.03 to 0.37), and accounting for the mediators rendered the total direct effect non-significant ($c' = 0.01$, $p = 0.94$). The specific indirect effect of race on antisocial behavior through both neighborhood disadvantage and non-executive functioning remained statistically significant ($a_1a_3b_2 = 0.03$, CI = 0.00 to 0.08).

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Supplemental Analyses

Analyses were rerun using parent and self-report factors that excluded the callous-unemotional scale, which had a relatively modest loading on the factor. As seen in Table 1.4, significant results remained unchanged for every analysis. For example, using the parent-report measure of antisocial behavior, the relevant indirect effect through executive functioning remained significant, and the others were very similar ($a_1b_1 = 0.22$, CI $= 0.05$ to 0.41; $a_2b_2 = 0.06$, CI $= 0.01$ to 0.14, $a_1a_3b_2 = 0.04$, CI $= 0.01$ to 0.08).

Additionally, analyses were rerun using the two modified neighborhood measures. With regard to the simple two item measure, as seen in Table 1.5, the indirect effect of interest was no longer significant ($a_1a_3b_2 = 0.01$, CI $= -0.01$ to 0.04).

Using the measure that simply excluded percent of occupied housing units that are renter occupied resulted in findings comparable to our main results ($a_1a_3b_2 = 0.07$, CI $= 0.01$ to 0.15). See Table 1.6 for all full details of the model.

Finally, analyses using all of the subjects were re-ran after imputing missing data using expectation maximization. These results again were unchanged in all models. For example, using parent-reports for the executive functioning model, the specific indirect effect of interest ($a_1a_3b_2$) was 0.07 with a confidence interval of 0.03 to 0.12.

Discussion

The main aim of this study was to test neighborhood disadvantage and neuropsychological functioning as serial mediators of the established race-antisocial behavior relationship. Based on parent-report data, this study found that being black in

the US was associated with higher levels of antisocial behavior, a relationship that is fully mediated by neighborhood disadvantage and executive functioning. The model specifying executive functioning (but not non-executive functioning) was supported, with the pathway through neighborhood disadvantage and executive functioning in series accounting for 10.8 percent of the total effect explained by race. Together, the three indirect effects accounted for 85.5 percent of the total effect of race on antisocial behavior.

The findings of the parent-report data are in line with the only prior study integrating neighborhood-level effects and individual cognitive differences sequentially to explain race differentials in antisocial behavior (McNulty et al., 2012). However, our results expand on that research in several ways. In addition to using more comprehensive measures of neighborhood disadvantage and antisocial behavior, this study used a neuropsychological battery rather than a single measure of verbal ability verbal scores, as used by McNulty and colleagues (2012). Neuropsychological tests measuring executive functioning are a more direct method of measuring executive functioning than traditional intelligence quotient tests (Ardila, Pineda, & Rosselli, 2000; Arffa, 2007; Friedman et al., 2006).

The importance of neurocognitive functioning as a risk factor for antisocial behavior has been laid out explicitly by Moffitt (1993, 1997) in her seminal taxonomy and later work. Notably, Moffitt identifies many individual and family-level risk factors for neuropsychological functioning (e.g. fetal maldevelopment, genetics, infant nutrition,

child abuse). Here, we highlight the potential influence of macrosocial variables (neighborhood) on neurobiological variables (neuropsychological functioning). The current findings both support and expand Moffitt's work by looking outside the household towards less proximal causes of neuropsychological deficits.

An additional finding of note is that the relationship between race and neurocognitive functioning was entirely mediated by neighborhood disadvantage. As mentioned earlier, blacks in the US are more likely to live in disadvantaged neighborhoods, and are thus more likely to be subjected to risk factors for neuropsychological deficits (Sampson and Winter, 2016; Wilson, 2012). Accordingly, neighborhood disadvantage has been associated with performance deficits in a variety of areas, including memory, emotion regulation, verbal and language ability, and executive functioning (Farah et al., 2006; Pechtel & Pizzagalli, 2011; Sampson et al., 2008). While the association between race and executive functioning was indeed significant ($p < 0.001$), this relationship was eliminated after accounting for neighborhood disadvantage in linear regression. This finding is in keeping with the sociological literature implicating environmental disadvantage in neuropsychological deficits (Sharkey et al., 2012; Waber et al., 2006) and speaks to a possible mechanism for race differences in academic performance (Jencks & Phillips, 2011).

Executive vs. Non-Executive Functioning

Neither of the serial mediator models using the non-executive functioning measure were supported. This finding is in line with expectations, as there is a dearth of

literature supporting a relationship between episodic memory (the majority of tasks comprising the non-executive functioning measure) and antisocial behavior. Indeed, there is evidence supporting intact performance on episodic memory tasks in at least one antisocial population (Hart, Forth, & Hare, 1990). In contrast, the significant findings of the executive functioning model (using parent-report data) highlight the importance of cognitive processes that include attention, working memory, and impulse control. Executive functioning is considered developmentally sensitive, and if impaired may lead to antisocial behavior through decreased behavioral inhibition, poor understanding of behavioral consequences, and inappropriate evaluation of punishment and rewards (Carlson, 2005; Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Concerning covariates, the model on executive functioning remained significant after controlling for age, gender, parental antisocial behavior, and parental years of education. These findings are consistent with previous meta-analyses regarding executive functioning and antisocial behavior, which also find no moderating effect of age and gender (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011).

Extensions of the Current Model

This study focused specifically on neuropsychological functioning as the neurobiological variable of interest. More broadly, this serial multiple mediator model can nevertheless be extended beyond executive functioning to examine other biological mechanisms that may account for the neighborhood-antisocial behavior component of the race-antisocial relationship. At least three other biological areas of interest are candidates

as mediators in future work extending the current serial mediation model: lead exposure, structural/functional brain differences, and potentially epigenetics. First, in the context of the current serial mediation results, the relationship between high lead levels and the three components of the serial mediation model (race, neighborhood, and antisocial behavior) makes lead exposure an attractive candidate mediator in a future test of this model (Lanphear & Roghmann, 1997; Reyes, 2007, 2011; Surkan et al., 2007). Second, given documented relationships between brain integrity and both macrosocial constructs and antisocial behavior (Henry & Moffitt, 1997; Raine, 1993; Y. Yang & Raine, 2009), and given also that poor executive functions are a proxy for poor prefrontal functioning (Roberts, Robbins, & Weiskrantz, 1998; A. D. Wagner, Maril, Bjork, & Schacter, 2001), brain imaging measures of prefrontal structure and function may be substituted into this model in place of executive functioning in future studies. Third, future studies could substitute epigenetic processes (the effect of the environment on gene expression) for executive functioning in the current study to test whether the race-antisocial relationship is mediated serially by neighborhood disadvantage and consequent epigenetic processes (Beach, Brody, Todorov, Gunter, & Philibert, 2010, 2011).

Limitations

The results from this study should be interpreted in the context of some limitations. First, all measures were cross-sectional and thus causal direction can only be hypothesized and not ascertained. Nevertheless, in addition to the precedence set by at least one previous study proposing a similar causal model (McNulty et al., 2012), there

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are two reasons for cautious confidence in the causal direction in this particular study. At one level, the young age of the subjects provides some confidence in the hypothesized causal flow. Given that children would be expected to have little control over their residential circumstances, the first mediator (neighborhood disadvantage) is unlikely to be caused by child neuropsychological functioning or child antisocial behavior, which we hypothesize to follow later in development. With regard to the brain-antisocial behavior relationship, precedence in the literature supports a causal flow from impaired brain dysfunction to antisocial behavior (Raine & Yang, 2006), especially given neurological studies on the effects of prefrontal cortical damage resulting in later antisocial behavior (S. W. Anderson et al., 1999; Bechara et al., 1994). Future studies can use these findings as a basis for a longitudinal design in which the direction of the associations can give more assurance to the hypothesized causal model.

A second limitation is the mixed findings depending on type of informant. The parent-report, but not self-report, data supported our executive functioning model. Inconsistent findings dependent on informant-type are not wholly unexpected, given the relatively low cross-informant correlations found in childhood psychopathology (Achenbach et al., 1987; Jensen et al., 1999). It has been suggested that parents may be more reliable in reporting externalizing behavior problems that go unacknowledged by the child, especially with younger children (Edelbrock, Costello, Dulcan, Conover, & Kala, 1986). Nevertheless the use of multiple informants needs to be examined in future studies that aim to replicate and extend the current findings.

A third limitation is that the specific indirect effect of race on antisocial behavior through neighborhood disadvantage and executive functioning, although statistically significant, was relatively small. The specific indirect effect of race on antisocial behavior through neighborhood disadvantage alone was larger than the specific indirect effect of race on antisocial behavior through neighborhood disadvantage and neuropsychological functioning in series in every model. This is consistent with the literature (McNulty et al., 2012), and indicates that the role of neuropsychological functioning, while significant, is merely one of several mechanisms through which neighborhood disadvantage can affect antisocial behavior. Sociological explanations that invoke multiple social mechanisms likely contribute to this association alongside other neurobiological variables. At the same time, the specific indirect effect of neuropsychological functioning alone was significant, documenting a path from race to executive functioning to antisocial behavior. This could potentially speak to another pathway described by Moffitt (1993), in which blacks are disproportionately less likely to have access to prenatal care (Piquero et al., 2005) and more likely to experience pre- and peri-natal complications (James, Jamison, Brancazio, & Myers, 2006; Taveras, Gillman, Kleinman, Rich-Edwards, & Rifas-Shiman, 2010). Infants born after labor complications may experience oxygen deprivation and resulting brain damage. This theory that neurocognitive impairments are present at birth is in keeping with the framework of Piquero et al. (2005). While the effect of the pathway of interest was small, we believe the significance of the other pathways calls for future studies to examine additional

intervening mechanisms for how these two mediators predispose to antisocial behavior in the context of the race – antisocial behavior relationship.

Finally, the indirect effect of interest derived from using the simpler measure of neighborhood disadvantage was nonsignificant. This could be due to the significant positive skew of this new measure (skewness = 1.04, *SE* = 0.12), and the loss of sampling validity associated with fewer variables composing the measure. The skewness of both the original variable and the measure excluding houses occupied by renters was less than 1 (skewness = 0.48, *SE* = 0.12 and skewness = 0.45, *SE* = 0.12, respectively).

Conclusions and Future Directions

In conclusion, results of this study are consistent with a growing body of literature advocating the integration of neighborhood-level effects with biological risk factors to explain antisocial behavior (Choy et al., 2015; Dodge & Pettit, 2003; McNulty et al., 2012). Findings have both theoretical and potential policy implications. With respect to theory, this study is indicative of the value of integrating classic criminology and sociology theories with biology-based theories (J. C. Barnes, 2012; J. C. Barnes & Jacobs, 2013; Beaver et al., 2009; Beaver, Vaughn, DeLisi, Barnes, & Boutwell, 2012; DeLisi, Beaver, Wright, & Vaughn, 2008; Tuvblad, Grann, & Lichtenstein, 2006), in particular, the social neurocriminology perspective advocated by Choy et al. (2015), which argues for the importance of the social environment in shaping the biological factors that increase the risk for crime and delinquency. Classic explanations of the race-antisocial behavior relationship have traditionally examined criminogenic environmental

risk factors associated with being of a minority status (particularly black) (Brody et al., 2001; Conger et al., 2002; Lambert, Brown, Phillips, & Ialongo, 2004; Morrison Gutman, McLoyd, & Tokoyawa, 2005). Indeed, neighborhood-level constructs such as social disorganization and collective efficacy likely have a significant impact on the predisposition to antisocial behavior (Morenoff, Sampson, & Raudenbush, 2001; Sampson & Groves, 1989; Sampson et al., 1997). This study supplements that literature by showing that neighborhood disadvantage can also influence antisocial behavior at the individual-level by impairing neuropsychological functioning.

One potentially important implication of identifying a biological mechanism in the race-antisocial behavior relationship is the recognition of individual-level treatment opportunities to break the pathway of relationships documented in this study. While neighborhood disadvantage and the race differential in exposure to neighborhood disadvantage need to be eradicated, neighborhood disadvantage is unlikely to change quickly (Sampson, 2012). That is not to say that eliminating poverty should be abandoned as a goal, but rather that an alternative approach suggested by our findings is to intervene on neurocognitive functioning at the individual level, given that poor executive functioning lies on the putative causal chain from neighborhood to antisocial behavior. Methylphenidate, a stimulant typically prescribed for ADHD, has been shown to improve executive functioning (Elliott et al., 1997; Mehta et al., 2000; Turner et al., 2003), although widespread and indiscriminate medication of whole populations is certainly not appropriate. Less controversially, behavioral therapies have also been

identified, and interventions with positive findings in a variety of populations (e.g. older adults, schizophrenics, young adults) have included computer-based training (Dahlin, Nyberg, Bäckman, & Neely, 2008), piano lessons (Bugos, Perlstein, McCrae, Brophy, & Bedenbaugh, 2007), neurocognitive enhancement therapy paired with work therapy (Bell, Bryson, Greig, Corcoran, & Wexler, 2001), and neurofeedback treatment (Kouijzer, de Moor, Gerrits, Congedo, & van Schie, 2009). Another recent intervention is mindfulness training, which is hypothesized to improve executive functioning (Y. Tang, Yang, Leve, & Harold, 2012). Indeed, at least one study treated antisocial children with mindfulness training and found improvements in attention and behavior (Bögels, Hoogstad, van Dun, de Schutter, & Restifo, 2008). Thus, there appears to be a number of potential individual-level interventions that may help improve neuropsychological functioning in children exposed to neighborhood disadvantage.

The findings of this study demonstrate that executive functioning, in particular, in sequence with neighborhood disadvantage, explains approximately 10.8 percent of the race-antisocial behavior relationship. In light of these findings, a few propositions may be considered. First, future studies can bolster and clarify this relationship through longitudinal designs (Murray & Farrington, 2010), which may help demonstrate whether these effects are lasting. That is, does early neighborhood disadvantage and later executive dysfunction in childhood predispose towards antisocial behavior in adulthood? Longitudinal data would also allow validation of our temporal ordering, which could only be hypothesized given the cross-sectional nature of the data. Second, as discussed above,

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additional studies examining biological variables such as lead exposure and brain structure and function may use this study's model as a basis within which to further test social neurocriminology theory (Choy et al., 2015). Finally, policy suggestions to close the race-antisocial behavior gap could consider incorporating forms of intervention to address neuropsychological functioning deficits in disadvantaged children (Bierman, Nix, Greenberg, Blair, & Domitrovich, 2008).

Paper 1 Tables and Figures

Table 1.1 Summary Statistics and Bivariate Correlations among Observed Study Variables

Variable	1	2	3	4	5	6	7	8	9	10	11	12
1. Race	1.00											
2. Parent-Report Antisocial Behavior	0.14*	1.00										
3. Child-Report Antisocial Behavior	0.12*	0.34**	1.00									
Potential Mediators												
4. EF Total Score	-0.19**	-0.24**	-0.03	1.00								
5. PCET	-0.20**	-0.15**	0.06	0.70**	1.00							
6. PCPT FN	-0.09	-0.10	0.01	0.56**	0.12*	1.00						
7. PCPT FP	-0.05	-0.20**	-0.13*	0.57**	0.13*	-0.10	1.00					
8. Non-EF Total Score	-0.06	-0.09	-0.03	0.25**	0.14**	0.14*	0.18**	1.00				
9. PWMT	-0.05	-0.06	-0.06	0.11*	0.06	0.12*	0.02	0.66**	1.00			
10. VOLT	-0.08	-0.04	0.02	0.23**	0.17**	0.08	0.16**	0.74**	0.29**	1.00		
11. PEIT	0.01	-0.07	-0.02	0.16**	0.05	0.07	0.17**	0.59**	0.04	0.16**	1.00	
12. Neighborhood Disadvantage	0.48**	0.21**	0.17**	-0.21**	-0.10	-0.19**	-0.09*	-0.03	0.03	-0.08	-0.01	1.00
Mean	0.86	-0.05	-0.05	0.05	0.02	0.03	0.00	0.07	0.04	-0.00	0.03	0.10
SD	0.35	0.95	0.99	1.75	0.98	0.95	0.96	1.90	0.93	1.01	0.92	4.54

Note. Sample sizes: whites *n* = 48; blacks = 293; for Child-Report Antisocial Behavior, whites *n* = 47; blacks *n* = 260
Abbreviations: PCET = Penn Conditional Exclusion Test; PCPT FN = Penn Continuous Performance Test False Negatives; PCPT FP = Penn Continuous Performance Test False Positives; PEIT = Penn Emotion Identification Test; PWMT = Penn Word Memory Test; VOLT = Visual Object Learning Test
*$p < 0.05$; ** $p < 0.01$.

Table 1.2 Difference of Means T-Tests for Study Variables for Whites and Blacks

Variable	Whites		Blacks		df	t-value	95% CI
	M	SD	M	SD			
Neighborhood Disadvantage	-5.31	2.34	0.99	4.18	104.67	-15.13***	-7.13 to -5.47
Executive Functioning	0.87	1.42	-0.08	1.76	339	3.57***	0.04 to 1.48
Non-Executive Functioning	0.52	1.89	0.02	1.97	80.45	1.45	-0.13 to 0.81
Parent-Report Antisocial Behavior	-0.38	0.80	-0.00	0.96	339	-2.55*	-0.06 to -0.09
Child Self-Report Antisocial Behavior[a]	-0.33	0.73	-0.00	1.02	82.76	-2.65*	-0.57 to -0.08

Note. Sample sizes: whites $n = 48$; blacks $n = 293$

[a] Sample sizes reduced for this particular variable. whites $n = 47$; blacks $n = 260$

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

Table 1.3 Path Coefficients and Standard Errors from Serial Mediation Models Estimated using PROCESS

	Parent Report ASB		Child Self-Report ASB	
Path	EF Model (Figure 2)	Non-EF Model (Figure 3)	EF Model	Non-EF Model
Total effect (c)	0.37*(0.15)	0.37*(0.15)	0.33*(0.16)	0.33*(0.16)
95% CI	0.09 to 0.66	0.09 to 0.66	0.02 to 0.64	0.02 to 0.64
Direct effect (c')	0.05(0.16)	0.11(0.17)	0.13(0.18)	.12(.18)
95% CI	-0.27 to 0.38	-0.22 to 0.43	-0.23 to 0.48	-0.23 to 0.47
a_1	6.30*** (0.62)	6.30*** (0.62)	6.22***(0.63)	6.22***(0.63)
95% CI	5.08 to 7.52	5.08 to 7.52	4.98 to 7.46	4.98 to 7.46
a_2	-0.60(0.30)	-0.35(0.34)	-0.53(0.31)	-0.37(0.35)
95% CI	-1.19 to 0.00	-1.02 to 0.32	-1.14 to 0.08	-1.06 to 0.32
a_3	-0.06*(0.02)	0.00(0.03)	-0.05(0.02)	0.00(.03)
95% CI	-0.10 to -0.01	-.05 to 0.05	-0.10 to 0.00	-0.05 to 0.06
b_1	0.03**(0.01)	0.04**(0.01)	0.03*(0.01)	0.03*(0.01)
95% CI	0.01 to 0.06	0.02 to 0.07	0.00 to 0.06	0.00 to 0.06
b_2	-0.11*** (0.03)	-0.04(0.03)	0.00(0.03)	-0.01(0.03)
95% CI	-0.17 to -0.05	-0.09 to 0.01	-0.06 to 0.07	-0.07 to 0.04
Indirect Effects				
a_1b_1	0.21*(0.09)	0.25*(0.09)	0.20*(0.10)	0.21*(0.10)
95% CI	0.04 to 0.39	0.08 to 0.43	0.01 to 0.40	0.01 to 0.41
a_2b_2	0.07*(0.03)	0.01(0.02)	-0.01 (0.02)	0.00(0.02)
95% CI	0.01 to 0.15	-0.01 to 0.07	-0.05 to 0.04	-0.01 to 0.06
$a_1a_3b_2$	0.04*(0.02)	-0.00(.01)	-0.00 (0.01)	-0.00(0.01)
95% CI	0.01 to 0.09	-0.02 to 0.01	-0.02 to 0.02	-0.02 to 0.01
Total indirect effect	0.32*(0.09)	0.27*(0.09)	0.20*(0.10)	0.21(0.10)
95% CI	0.15 to 0.49	0.09 to 0.44	0.01 to 0.40	0.01 to 0.41
	$R^2 = 0.09$ F(3, 337) = 10.75, $p < 0.001$	$R^2 = 0.05$ F(3, 337) = 6.31, $p < 0.001$	$R^2 = 0.03$ F(3, 303) = 3.25, $p < 0.05$	$R^2 = 0.03$ F(3, 303) = 3.31, $p < 0.05$

Note. Sample sizes: for parent report ASB, whites $n = 48$, blacks = 293; for child self-report ASB, whites $n = 47$, blacks $n = 260$

*$p < 0.05$, ** $p < 0.01$, ***$p < 0.001$

Figure 1.1 Proposed Serial Mediation Model Linking Race to Antisocial Behavior Through Neighborhood Disadvantage and

Executive Functioning as Serial Mediators
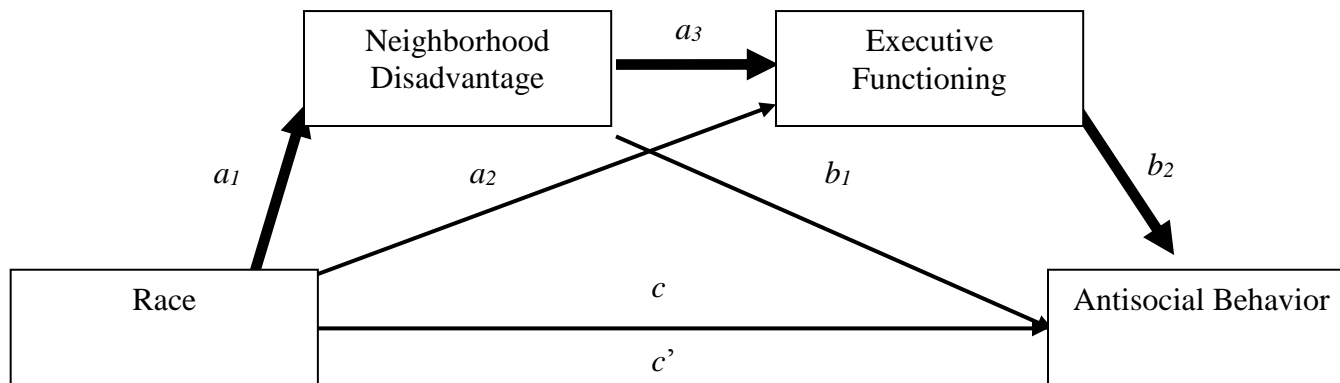
Figure 1.2 Serial Mediation Model Showing the Direct Effect and Path Coefficients Linking Race to Antisocial Behavior Through

Neighborhood Disadvantage and Executive Functioning as Serial Mediators.
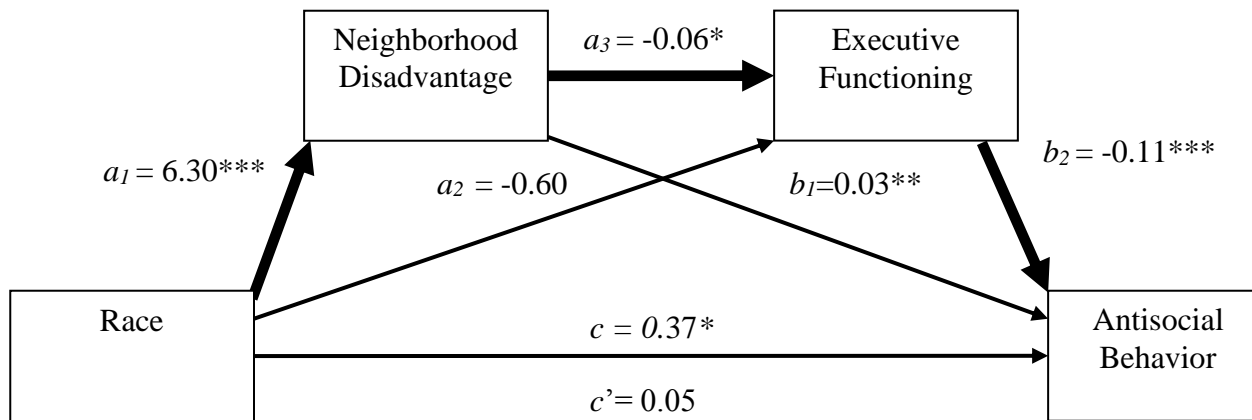
Note: N = 341
*p < 0.05; **p < 0.01, ***p < 0.001, two-tailed

Figure 1.3 Serial Mediation Model Showing the Direct Effect and Path Coefficients Linking Race to Parent-Reported Antisocial Behavior Through Neighborhood Disadvantage and Non-Executive Functioning as Serial Mediators.
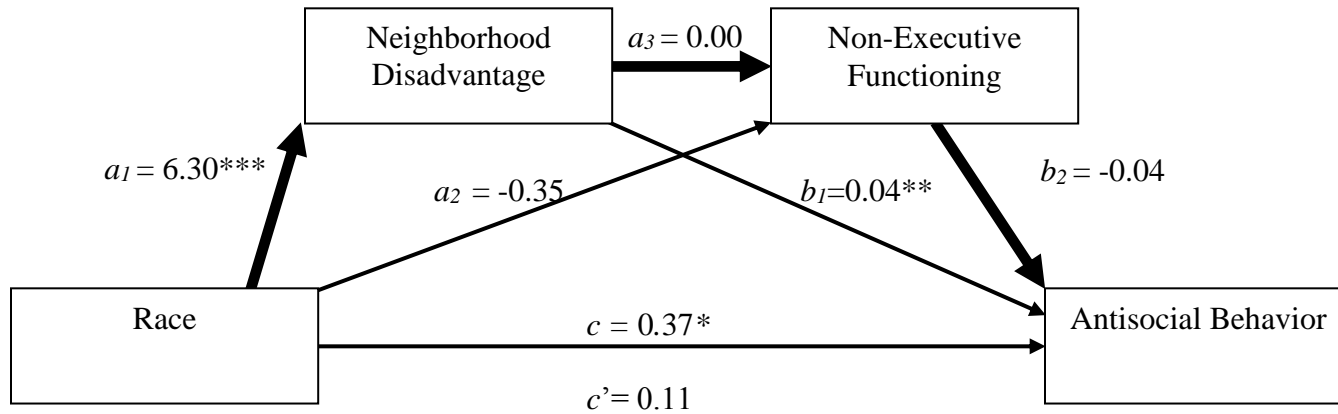
Note.: $N = 341$
*p < 0.05; **p < 0.01, ***p < 0.001.*

Table 1.4 Path Coefficients and Standard Errors from Serial Mediation Models with no CU ASB measure

Path	Parent Report ASB		Child Self-Report ASB	
	EF Model	Non-EF Model	EF Model	Non-EF Model
Total effect (c)	0.31*(0.15)	0.31*(0.15)	0.31*(0.16)	0.31*(0.16)
95% CI	0.02 to 0.60	0.02 to 0.60	0.00 to 0.61	0.00 to 0.61
Direct effect (c')	-0.1(0.16)	0.04(0.17)	0.11(0.18)	.10(.18)
95% CI	-0.34 to 0.31	-0.29 to 0.36	-0.25 to 0.46	-0.25 to 0.45
a_1	6.30*** (0.62)	6.30*** (0.62)	6.21***(0.63)	6.21***(0.63)
95% CI	5.08 to 7.52	5.08 to 7.52	4.98 to 7.44	4.98 to 7.44
a_2	-0.60(0.30)	-0.35(0.34)	-0.53(0.31)	-0.35(0.35)
95% CI	-1.19 to 0.00	-1.02 to 0.32	-1.13 to 0.08	-1.04 to 0.34
a_3	-0.06*(0.02)	0.00(0.03)	-0.05*(0.02)	0.00(.03)
95% CI	-0.10 to -0.01	-0.05 to 0.05	-0.10 to -0.00	-0.05 to 0.05
b_1	0.03**(0.01)	0.04**(0.01)	0.03*(0.01)	0.03*(0.01)
95% CI	0.01 to 0.06	0.02 to 0.07	0.01 to 0.06	0.01 to 0.06
b_2	-0.11*** (0.03)	-0.03(0.03)	0.00(0.03)	-0.01(0.03)
95% CI	-0.16 to -0.05	-0.08 to 0.02	-0.06 to 0.07	-0.07 to 0.05
Indirect Effects				
a_1b_1	0.22*(0.09)	0.26*(0.09)	0.21*(0.10)	0.21*(0.10)
95% CI	0.06 to 0.41	0.09 to 0.44	0.02 to 0.40	0.01 to 0.40
a_2b_2	0.06*(0.03)	0.01(0.02)	-0.01 (0.02)	0.00(0.02)
95% CI	0.01 to 0.14	-0.01 to 0.07	-0.05 to 0.04	-0.01 to 0.06
$a_1a_3b_2$	0.04*(0.02)	-0.00(.01)	-0.00 (0.01)	0.00(0.01)
95% CI	0.01 to 0.08	-0.02 to 0.01	-0.02 to 0.02	-0.01 to 0.01
Total indirect	0.32*(0.09)	0.27*(0.09)	0.20*(0.10)	0.21(0.10)
effect	0.15 to 0.50	0.10 to 0.46	0.01 to 0.40	0.01 to 0.41
95% CI				
	$R^2 = 0.08$	$R^2 = 0.05$	$R^2 = 0.03$	$R^2 = 0.03$
	$F_{(3, 337)} = 9.42, p < 0.001$	$F_{(3, 337)} = 5.43, p < 0.01$	$F_{(3, 307)} = 3.13, p < 0.05$	$F_{(3, 307)} = 3.18, p < 0.05$

Note. Sample sizes: for parent report ASB, whites $n = 48$, blacks = 293; for child self-report ASB, whites $n = 47$, blacks $n = 264$

*$p < 0.05$, ** $p < 0.01$, ***$p < 0.001$

Table 1.5 Path Coefficients and Standard Errors from Serial Mediation Models with Two Variable Neighborhood Measures

Path	Parent Report ASB		Child Self-Report ASB	
	EF Model	Non-EF Model	EF Model	Non-EF Model
Total effect (c)	0.37*(0.15)	0.37*(0.15)	0.33*(0.16)	0.33*(0.16)
95% CI	0.09 to 0.66	0.09 to 0.66	0.02 to 0.64	0.02 to 0.64
Direct effect	0.14(0.16)	0.22(0.16)	0.27(0.17)	.27(.17)
(c')	-0.17 to 0.44	-0.01 to 0.53	-0.07 to 0.61	-0.07 to 0.61
95% CI				
a_1	1.96*** (0.26)	1.96*** (0.26)	1.95***(0.26)	1.95***(0.26)
95% CI	1.45 to 2.47	1.45 to 2.47	1.44 to 2.47	1.44 to 2.47
a_2	-0.87**(0.29)	-0.48(0.32)	-0.77**(0.29)	-0.35(0.34)
95% CI	-1.44 to -0.30	-1.11 to 0.15	-1.35 to -0.19	-1.02 to 0.32
a_3	-0.04(0.06)	0.07(0.06)	-0.03(0.06)	0.00(0.03)
95% CI	-0.15 to 0.07	-0.05 to 0.19	-0.15 to 0.09	-0.05 to 0.05
b_1	0.06*(0.03)	0.07*(0.03)	0.03(0.03)	0.03(0.03)
95% CI	.00 to 0.12	0.01 to 0.13	-0.04 to 0.09	-0.04 to 0.09
b_2	-0.12*** (0.03)	-0.04(0.03)	-.01(0.03)	-0.01(0.03)
95% CI	-0.18 to -0.06	-0.10 to 0.01	-0.07 to 0.61	-0.07 to 0.04
Indirect Effects				
a_1b_1	0.12*(0.06)	0.14*(0.06)	0.05(0.07)	0.05(0.07)
95% CI	-0.01 to 0.25	0.02 to 0.27	-0.10 to 0.21	-0.09 to 0.20
a_2b_2	0.10*(0.04)	0.02(0.02)	0.01 (0.03)	0.01(0.02)
95% CI	0.04 to 0.19	-0.00 to 0.08	-0.05 to 0.07	-0.02 to 0.06
$a_1a_3b_2$	0.01(0.01)	-0.01(.01)	0.00 (0.00)	-0.00(0.01)
95% CI	-0.01 to 0.04	-0.03 to 0.00	-0.01 to 0.01	-0.02 to 0.00
Total indirect	0.24*(0.07)	0.15*(0.07)	0.06(0.08)	0.06(0.08)
effect	0.11 to 0.38	0.03 to 0.29	-0.10 to 0.21	-0.10 to 0.21
95% CI				
	$R^2 = 0.08$	$R^2 = 0.04$	$R^2 = 0.02$	$R^2 = 0.02$
	$F_{(3, 337)} = 9.76, p < 0.001$	$F_{(3, 337)} = 4.73, p < 0.01$	$F_{(3, 303)} = 3.13, p = 0.17$	$F_{(3, 303)} = 1.73, p = 0.16$

Note. Sample sizes: for parent report ASB, whites $n = 48$, blacks = 293; for child self-report ASB, whites $n = 47$, blacks $n = 264$

$*p < 0.05$, $** p < 0.01$, $***p < 0.001$

Table 1.6 Path Coefficients and Standard Errors from Serial Mediation Models with Neighborhood Measure excluding Percent Renters Variable

Path	Parent Report ASB		Child Self-Report ASB	
	EF Model	Non-EF Model	EF Model	Non-EF Model
Total effect (c)	0.37*(0.15)	0.37*(0.15)	0.33*(0.16)	0.33*(0.16)
95% CI	0.09 to 0.66	0.09 to 0.66	0.02 to 0.64	0.02 to 0.64
Direct effect (c')	0.07(0.16)	0.12(0.16)	0.15(0.18)	0.15(0.18)
95% CI	-0.25 to 0.38	-0.20 to 0.44	-0.20 to 0.50	-0.20 to 0.50
a_1	5.29***(0.55)	5.29***(0.55)	5.24***(0.55)	5.24***(0.55)
95% CI	4.21 to 6.36	4.21 to 6.36	4.15 to 6.32	4.15 to 6.32
a_2	-0.63*(0.30)	-0.38(.34)	-0.56(0.31)	-0.38(0.35)
95% CI	-1.22 to -0.03	-1.04 to 0.28	-1.16 to 0.04	-1.07 to 0.31
a_3	-0.06*(0.27)	0.01(.03)	-0.05(0.03)	0.00(0.03)
95% CI	-0.11 to -0.01	-0.05 to 0.07	-0.11 to 0.00	-0.06 to 0.07
b_1	0.04**(0.01)	0.05**(0.01)	0.03*(0.02)	0.03*(0.02)
95% CI	0.01 to 0.07	0.02 to 0.07	0.00 to 0.07	.00 to 0.07
b_2	-0.11***(0.03)	-0.04(0.03)	0.00(0.03)	-0.01(0.03)
95% CI	-0.17 to -0.06	-0.09 to 0.01	-0.07 to 0.06	-0.07 to 0.04
Indirect Effects				
a_1b_1	0.20*(0.08)	0.24*(0.09)	0.18(0.10)	0.18(0.10)
95% CI	0.04 to 0.37	0.08 to 0.42	-0.02 to 0.37	-0.01 to 0.37
a_2b_2	0.07*(.03)	0.02(0.02)	0.00(0.02)	0.00(0.02)
95% CI	0.02 to 0.15	-0.00 to 0.07	-0.04 to 0.04	-0.01 to 0.06
$a_1a_3b_2$	0.04*(0.02)	-0.00(0.01)	0.00(0.01)	-0.00(0.01)
95% CI	0.01 to 0.08	-0.00 to 0.09	-0.02 to 0.02	-0.01 to 0.01
Total indirect effect	0.31*(0.08)	0.25*(0.09)	0.18(0.10)	0.18(0.10)
95% CI	0.14 to 0.47	0.09 to 0.43	-0.12 to -0.38	-0.01 to 0.38
	$R^2 = 0.09$	$R^2 = 0.05$	$R^2 = 0.03$	$R^2 = 0.03$
	$F(3, 337) = 10.70, p < 0.001$	$F(3, 337) = 6.25, p < 0.001$	$F(3, 303) = 2.92, p < 0.05$	$F(3, 303) = 2.99, p < 0.05$

Note. Sample sizes: for parent report ASB, whites $n = 48$, blacks $n = 293$; for child self-report ASB, whites $n = 47$, blacks $n = 264$

*$p < 0.05$, ** $p < 0.01$, ***$p < 0.001$

Table 1.7 Path Coefficients and Standard Errors from Serial Mediation Models with Full Imputed Data

	Parent Report ASB		Child Self-Report ASB	
Path	EF Model	Non-EF Model	EF Model	Non-EF Model
Total effect (c)	0.19**(0.07)	0.19*(0.07)	0.16*(0.07)	0.16*(0.07)
95% CI	0.05 to 0.33	0.05 to 0.33	0.02 to 0.30	0.02 to 0.30
Direct effect (c')	0.04(0.08)	0.06(0.08)	0.09(0.08)	0.09(0.08)
95% CI	-0.12 to 0.19	-0.09 to 0.22	-0.06 to 0.25	-0.07 to 0.25
a_1	3.13***(0.30)	3.13***(0.30)	3.13***(0.30)	3.13***(0.30)
95% CI	2.55 to 3.72	2.55 to 3.72	2.55 to 3.72	2.55 to 3.72
a_2	-0.28*(0.14)	-0.20(0.16)	-0.28*(0.14)	-0.20(0.16)
95% CI	-0.55 to -0.00	-0.52 to 0.11	-0.55 to -0.00	-0.52 to 0.11
a_3	-0.06**(0.02)	-0.01(0.02)	-0.06**(0.02)	-0.01(0.02)
95% CI	-0.11 to -0.02	-0.06 to 0.04	-0.11 to -0.02	-0.06 to 0.04
b_1	0.04**(0.01)	0.04**(0.01)	0.02(0.01)	0.02(0.01)
95% CI	0.01 to 0.06	0.01 to 0.06	-0.00 to 0.05	-0.00 to 0.05
b_2	-0.14***(0.03)	-0.05(0.02)	-0.00(0.03)	-0.01(0.02)
95% CI	-0.19 to -0.08	-0.10 to 0.00	-0.06 to 0.05	-0.06 to 0.04
Indirect Effects				
a_1b_1	0.09*(0.04)	0.11*(0.04)	0.07(0.04)	0.07(0.04)
95% CI	0.01 to 0.17	0.04 to 0.19	-0.01 to 0.15	-0.01 to 0.15
a_2b_2	0.04*(0.02)	0.01(0.01)	0.00(0.01)	0.00(0.01)
95% CI	0.01 to 0.08	-0.00 to 0.04	-0.02 to 0.02	-0.01 to 0.02
$a_1a_3b_2$	0.03*(0.01)	0.00(0.00)	0.00(0.01	0.00(0.00)
95% CI	0.01 to 0.05	-0.01 to 0.01	-0.01 to 0.02	-0.00 to 0.01
Total indirect effect	0.15*(0.04)	0.12*(0.04)	0.07(0.04)	0.07(0.04)
95% CI	0.08 to 0.23	0.05 to 0.21	-0.01 to 0.15	-0.01 to 0.16
	$R^2 = 0.09$	$R^2 = 0.05$	$R^2 = 0.02$	$R^2 = 0.02$
	$F(3, 405) = 14.14, p < 0.001$	$F(3, 405) = 6.84, p < 0.001$	$F(3, 405) = 2.97, p < 0.05$	$F(3, 405) = 3.05, p < 0.05$

Note. Sample sizes: whites $n = 52$, blacks $n = 357$

*$p < 0.05$, ** $p < 0.01$, ***$p < 0.001$

PAPER 2. AGGRESSION AND SLEEP: A DAYLIGHT SAVING TIME NATURAL EXPERIMENT ON THE EFFECT OF MILD SLEEP LOSS AND GAIN ON ASSAULTS[2]

Abstract

The purpose of this study was to test the effect of a mild, short-term sleep loss/gain on assault rates. Using National Incidence Based Reporting System data and city-reported data from Chicago, New York, Philadelphia, and Los Angeles, we calculated the difference in assault rates on the Monday immediately following daylight saving time (DST) as compared to the Monday a week later using a Poisson quasi-maximum likelihood estimator model. The same analyses were performed to examine effects of the return to standard time in the fall. We employed several falsification checks. There were 2.9% fewer (95% CI: -4.2%, -1.6%, $p < 0.0001$) assaults immediately following DST, when we lose an hour, as compared to a week later. In contrast, there was a 2.8% rise in assaults immediately following the return to standard time, when an hour is gained, as compared to a week later (95% CI: 1.5%, 4.2%, $p < 0.0001$). Multiple falsification analyses suggest the spring findings to be robust, while the evidence to support the fall findings is weaker. This study suggests that mild and short-

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term changes in sleep do significantly affect rates of assault. Specifically, there is support for the theory that mild sleepiness possibly associated with an hour loss of sleep results in reduced assaults. This contradicts the simple inverse relationship currently suggested by most of the correlational literature. This study and the mixed findings presented by experimental studies indicate that measurement variability of both sleep and aggression may result in conflicting findings.

Background

Low quantity and quality of sleep is increasingly thought to be a causal factor in antisocial behavior and aggression (Kamphuis et al., 2012). The evidence surrounding the relationship between sleep and antisocial behavior has been derived primarily from small samples in experimental settings involving significant sleep deprivation (Cote et al., 2013; Cote, Mondloch, Sergeeva, Taylor, & Semplonius, 2014), and via self-reports of sleep, affect, and antisocial behavior (e.g., Adrian Raine & Venables, 2017), although there are exceptions, including studies on children using teacher and parent reporters (Chervin, Dillon, Archbold, & Ruzicka, 2003; Fallone, Acebo, Seifer, & Carskadon, 2005). This quasi-experimental study uses an exogenous shock to sleep to measure the effects of mild sleep reductions and increases on quantifiable measures of antisocial behavior (i.e., assaults), using a larger and more representative sample than previously studied. We exploit the switch to daylight saving time (DST) in the spring and the return to standard time in the fall as a mild exogenous shock to sleep to test whether even one night of a mild change in sleep affects rates of assault on the Monday after a switch. As noted in more detail below, although the switch occurs early Sunday morning, the literature generally agrees that effects are most acutely felt on Monday, when inflexible business and school hours enforce a specific wake time. As a secondary issue, cross-disciplinary research has called into question the energy-saving premise of the DST policy and revealed significant unintended effects of DST, both negative and positive. Consequently, there is ongoing legislation in several states surrounding the policy (Victor

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2016). This study aims to provide more knowledge about a costly outcome (assault), which would be relevant to policy-makers, researchers involved in policing and corrections, and the general public.

Prior research indicates that DST affects quantity and quality of sleep and that changes in sleep can alter physiological pathways that, in turn, affect aggression.

Sleep and Antisocial Behavior

A positive relationship has been proposed between poor sleep quality and anger, short- temperedness, delinquency, and impulsive aggression (Catrett & Gaultney, 2009; Kamphuis et al., 2012; Lindberg et al., 2003). The research thus far has focused largely on antisocial or psychiatric populations (Ireland & Culpin, 2006; Lindberg et al., 2003) and healthy individuals with consistent sleep disturbances (Coulombe, Reid, Boyle, & Racine, 2011; Granö, Vahtera, Virtanen, Keltikangas-Järvinen, & Kivimäki, 2008; Gregory & O'Connor, 2002) or short-term extreme sleep deprivation (multiple hours) (Christian & Ellis, 2011; Cote et al., 2013; Vohs et al., 2010). Despite the varied methodology, subject pools, and operationalization of both sleep and antisocial behavior, a positive relationship between poor sleep and antisocial behavior has been found in cross-sectional (Kamphuis et al., 2012) and longitudinal studies (Gregory & O'Connor, 2002; Raine & Venables, 2017), studies of adults (Granö et al., 2008; Shin et al., 2005; Taub, 1977; Vaughn, Salas-Wright, White, & Kremer, 2015), and children and adolescents (Backman et al., 2015; Becker, Langberg, & Evans, 2015; Catrett & Gaultney, 2009; Chervin et al., 2003; Clinkinbeard, Simi, Evans, & Anderson, 2011;

Kamphuis et al., 2012; Meijer, Reitz, Deković, Van Den Wittenboer, & Stoel, 2010). It is important to note that many of these studies considered the consequences of long-term poor sleep and were reliant on self-report measures.

Of particular relevance to this study, and as noted by a narrative review (Krizan & Herlache, 2016), direct tests of the results of short-term significant sleep deprivation on a number of antisocial outcomes using experimental methods have reported mixed findings. One study (Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006) deprived subjects of sleep for 55 hours and then measured aggression and hostility using fill-in-the-response vignettes. They found that subjects were more likely than controls to direct blame and/or hostility towards others, and to be unwilling to alleviate a conflict by accepting the blame (Kahn-Greene et al., 2006). A contradictory study (Cote et al., 2013) observed reduced reactive aggression (as measured by a Point Subtraction Aggression Paradigm task) in sleep-deprived men as compared to controls, and no relationship between sleep deprivation and aggression in women after 33 hours of sleep deprivation. A study by Vohs et al. (2010) examined the effects of sleep deprivation on reactive aggression as measured by volume of noise chosen by the participant to be blasted at their opponent in a game. In reporting null results, they were unable to show support for effects of sleep deprivation on aggression in either direction. Finally, Haack and Mullington (2005) assessed affect every day for 12 days in an experimentally sleep-deprived group and a control group, and found that sleep restriction did result in higher self-reported anger/aggression, but effects were not seen until a few days of consistent

sleep restriction. Moreover, effects were reversed upon a single day of sleep recovery. These experimental tests suggest that the consequences of short-term sleep deprivation are still poorly understood, and results may vary significantly by methodology. Because of these experimental findings, we propose competing hypotheses as to the effects of DST and standard time switches (and by extension, a single hour of sleep loss or gain) on rates of assault. That is, for each switch, assault rates on the following Monday could plausibly increase or decrease.

Possible Pathways Between Sleep and Aggression

Less sleep, more aggression?

One plausible pathway through which poor sleep may lead to increased antisocial behavior could be through impaired neurocognitive functioning or sleepiness and subsequent low self-control. There is supporting evidence for each of these relationships and for some of the relationships in sequence, even if the entire sequential pathway has not been explicitly tested.

First, studies involving both sleep disordered (Fulda & Schulz, 2001) and healthy populations (e.g., Kronholm et al., 2009; Nilsson et al., 2005; Sadeh, Gruber, & Raviv, 2002; Touchette et al., 2007) have linked poor sleep quality to impairment in a number of cognitive functions. Some studies identified regions of interest a priori, and found sleep quality to be associated with cognitive functions that draw specifically from emotion-processing (e.g., Baum et al., 2014; van der Helm, Gujar, & Walker, 2010) and executive

functioning parts of the brain (Cote et al., 2014; Rossa, Smith, Allan, & Sullivan, 2014; Williamson & Feyer, 2000).

Second, employing self-control (for example, to inhibit emotional responses stemming from the limbic system) draws on a limited pool of mental resources (namely executive control functions centered in the prefrontal cortex), and when that pool is lessened, for example by sleep, self-control may be impaired until the pool can be replenished (Hagger, Wood, Stiff, & Chatzisarantis, 2010).

Finally, the last pathway, between low self-control and increased antisocial behavior, has been formalized in the well-supported, seminal Self-Control Theory (Evans, Cullen, Burton, Dunaway, & Benson, 1997; Gottfredson & Hirschi, 1990). An inability to regulate one's impulses and immediate desires has been associated with antisocial behavior and delinquency (Hay, 2001), with convergent findings across a number of samples and methodologies (Cheung & Cheung, 2008; Gibbs & Giever, 1995; Vazsonyi, Pickering, Junger, & Hessing, 2001). In short, both psychological and criminological literature has provided supporting evidence for a causal chain that could link poor sleep quantity/quality and antisocial behavior.

Less sleep, less aggression?

In addition to correlational studies suggesting a simple inverse relationship between sleep and aggression, there is some experimental research documenting the opposite relationship—more sleep deprivation resulting in reduced reactive aggression (Cote et al., 2013). Cote et al. (2013) used a sample of 49 undergraduate students and the Point

Subtraction Aggression Paradigm to look at the effect of 33 hours of sleep deprivation on aggression. Predictably, the sleep deprivation subjects self-reported more negative mood than the controls. However, when it came to actual behavioral aggression, sleep- deprived women stole at the same rate as the controls, and sleep-deprived men stole less than the controls. Cote et al. (2013) proposed that lessened sleep may result in increased physiological and cognitive responsiveness, but decreased behavioral aggression. Additionally, as sleep deprivation disrupted the relationship between testosterone and reactive aggression in male subjects, Cote et al. (2013) hypothesized that the reduced testosterone in the sleep-deprived subjects may play a role in the observed reduced reactive aggression.

Vohs et al. (2010) also used undergraduate students to examine the effects of 24 hours of sleep deprivation on reactive aggression (operationalized as level of volume blasted at an imaginary opponent as punishment). In explaining unexpected null findings, Vohs et al. (2010) concluded that rather than sleep deprivation causing aggression, it might be methodological issues and the oft-associated self-regulation depletion driving the general consensus in the correlational literature. They also noted the benefits of measuring behavior as opposed to intention, as was seen in earlier experimental studies (Kahn-Greene et al., 2006). It could be that when presented with hypothetical situations, individuals will rely on lay beliefs as to how they, a sleep-deprived person, should and would respond. Indeed, when looking at how healthy individuals deal with a mild decrease in sleep quantity/quality, it may be that, despite an increase in irritability and

negative affect, sleepiness and lethargy reduces the likelihood of their acting on their aggressive impulses.

Daylight saving time, sleep, and cognitive functioning

Anticipating significant differences in assault counts as a result of the reduced sleep caused by DST requires first that the shift to DST results in decreased sleep. Previous research conducted on DST supports this core assumption (C. M. Barnes & Wagner, 2009). Using a large sample from the American Time Use Survey conducted by the Bureau of Labor Statistics, Barnes and Wagner (2009) found DST to be associated with a self-reported decrease of 40 min of sleep from Sunday to Monday. Proposing DST effects on assault also assumes that the amount of sleep lost is enough to affect cognitive functioning and energy. Research surrounding DST does indicate that even the loss of one hour can result in sluggish cognitive functioning as measured by performance on the SAT (Gaski & Sagarin, 2011). Employing methods so as to focus on the loss of sleep, specific connections have been drawn between DST and a resulting rise in car accidents (Harrison, 2013), stock market losses (Kamstra, Kramer, & Levi, 2000; Pinegar, 2002), work-place injuries (C. M. Barnes & Wagner, 2009), the workplace use of the internet for personal reasons, or cyberloafing (D. T. Wagner, Barnes, Lim, & Ferris, 2012), reduced test scores (Gaski & Sagarin, 2011), and even suicide rates (Berk et al., 2008). These studies have demonstrated that the sleep effects of DST are sufficiently strong for these researchers to have statistical power to detect relationships between sleep and a variety of outcome measures. We note that many of these studies have focused on the transition to

DST without examining the switch back to standard time. As a result, while the literature

suggests that DST (and a loss of one hour of sleep) has an acute negative effect on

cognitive functioning, there is less evidence to support the inverse.

Fall shift to standard time and sleep

The popularly held belief is that we lose an hour of sleep in the switch to DST, but

gain an hour in the return to standard time in the fall. The former part of the hypothesis is

better supported than the latter. Using a nationally representative sample; Barnes and

Wagner (2009) found the advent of DST to be associated with a loss of 40 minutes of

sleep, but no change in sleep duration after the fall shift. Furthermore, there is evidence

that the adjustment back to standard time is easier and completed faster than its DST

counterpart (Kantermann, Juda, Merrow, & Roenneberg, 2007). In accordance with a

general lack of consensus and literature, we allow for competing hypotheses as to the

effect of the switch back to standard time on assaults.

This Study

This study uses the exogenous mild shocks of DST to assess the effect of a short-

term, and mild, reduction in sleep time on crime. The advantages of this methodology as

compared to earlier sleep/antisocial research is that it uses a representative and large

sample of the United States, thus looking directly at a real-world outcome of interest,

assaults, as opposed to self-reported aggression or simulated antisocial behavior in an

experimental setting. Considering that most adults have adjusted their bed and rise times

within one week post-switch (Harrison, 2013) we expect to see significant effects of the

policy on crime isolated on the Monday immediately following switches (as compared to one week post-switch). We note that by focusing on a one-time loss or gain of a single hour, our study diverges from the existing sleep literature. Additionally, our outcome (assaults severe enough to warrant police involvement) is a highly valid measure of aggression, as opposed to laboratory tests employed in previous studies. Thus, while we do look at a similar cause, sleep, it is difficult to ascertain how our findings will compare to previous findings.

Broadly, our research question is: Does a small change in sleep duration result in increased aggression? Specifically, we will assess whether (1) the rate of assaults is affected by a potential one hour loss of sleep on the Monday following the transition to DST, as compared to the Monday one week after, and whether (2) the rate of assaults is affected by a potential gain of one hour of sleep on the Monday following the return to standard time, as compared to the Monday one week later.

Methods

Data

Due to a need to examine data at a daily level, we used publicly available data from the National Incident-Based Reporting System (NIBRS) from 2001 to 2014, the only national crime database with a daily level of detail. NIBRS is an incidence-based reporting system used by local, state, and federal law enforcement agencies. Small jurisdictions are disproportionately represented in NIBRS. To provide a more representative view of the effects of DST, we combined these data with city-reported

publicly available data from the cities of Chicago, Los Angeles, New York, and Philadelphia. These cities provide data on an incident-level, with the same requisite variables (date, incident type) as the NIBRS data; however, they do not participate in NIBRS. The NIBRS database provides detailed crime data in the form of incident counts for 22 Group "A" offenses, including aggravated assault and simple assault. For each analysis, we summed both types of assault to provide an overall measure of assault (using all assaults that occurred during the 24 hours). Using the combined NIBRS and city-reported data, the number of observed assaults used in the main analyses in the spring and fall is $n = 60,333$ and $n = 62,546$, respectively.

While the switches to and from DST actually go into effect at 2:00 a.m. Sunday morning, the aforementioned literature around DST suggests that the effects are not felt until Monday, when traditional business/school hours preclude sleeping in to compensate (Varughese & Allen, 2001). Accordingly, we compared the Monday after the transitions to and from DST with the Monday a week later (rather than the Monday before) because it best controls for (a) daylight, (b) day effects, and (c) any possible seasonal effects. We essentially expected those two Mondays to be, on average, very similar in terms of weather and lighting (as opposed to the Monday prior to the switch, when there is an additional hour of sunlight or darkness during typical activity hours). This methodology has been used in prior daylight saving time literature (D. T. Wagner et al., 2012). Like Doleac and Sanders (2015), who used DST to look at the effects of ambient light on crime, we discarded data for years when holidays fell on one of the days of interest. For

example, in the fall of 2005, Halloween, which may artificially inflate crime rates,

coincided with the Monday directly following DST. Likewise, we discarded the data for

the spring of 2008 and 2014, when St. Patrick's Day fell on the comparison Monday (the

Monday one week following). Because NIBRS includes information on the age and

gender of the offender, we also looked at potential moderating effects of age and gender

with interaction terms. Counts of assaults per year are presented in Table 2.1.

Statistical model

We estimated the effect of DST using a Poisson quasi-maximum likelihood

estimator (QMLE) regression model, fit separately to the spring data and the fall data. We

preferred a Poisson QMLE model over a negative binomial model because it is, on

balance, more efficient and robust (Wooldridge 2010).

$$y_{itd} \sim Poisson(\lambda_{itd}) \text{ and } \log{(\lambda_{itd})} = \beta d + \alpha_i + \xi_t$$

where y_{itd} is the number of assaults reported in city i, in year t, on Monday d (where $d = 1$

is the Monday immediately following the switch and $d = 0$ is the Monday one week after

that). α_i represents a city fixed effect and ξ_t represents a year fixed effect. Of primary

interest is β, where $e^{\beta} - 1$ is the estimated fraction increase in crime immediately

following the change to/from DST relative to the Monday one week later. This method of

comparing the Mondays is consistent with previous literature on the effects of DST

(Coren, 1996; D. T. Wagner et al., 2012), although we note that we did not make any

comparisons using the Monday prior to DST, so as to best isolate the effects of sleep as

opposed to changes in daylight hours and ambient lighting.

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To look at gender and age of the offender as potential moderators, we calculated interaction terms using dichotomous and categorical variables, respectively. Age was discretized into three categories, 14–25, 26–40, and 41 and older.

Robustness checks

We tested the robustness of our findings using four falsification checks. First, we took advantage of the 2007 switch in the advent and end of DST legislated in the federal Energy Policy Act of 2005. From 2007 on, DST was extended by one month, by shifting the start from the first Sunday in April up to the second Sunday of March, and shifting the end from the last Sunday of October to the first Sunday of November. We reversed the definitions, coding days before 2006 using the post-2007 definition and days after 2007 using the pre-2007 DST definition. We expect no effect since no DST changes occurred under this flipped coding definition. If the Monday coefficient is nonsignificant, this robustness check supports the theory that sleep, as opposed to weather or another non-DST factor, is driving the effects. Second, we compared the Monday one week post-switch to the Monday two weeks post switch, expecting to see no significant difference between the two, as people seem to adjust their sleep patterns by that point (Harrison, 2013). Our final two robustness checks compared other weekdays (i.e., Wednesday and Thursday) immediately following the switch to their corresponding day the following week. Wednesday and Thursday best balance our interest of looking at weekdays and looking at days in which DST would have lessened effects as compared to Monday. We expected to see at least weakened, if not null effects, as it seems plausible that people will

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not have adjusted to the switch entirely by those days, but will be less affected than on Monday. We wished to include in our falsification checks an analysis of Arizona, which does not observe DST, but we could not compile a sufficient number of assaults from NIBRS or localities.

Results

Spring Analyses: Potential One Hour Loss of Sleep

We examined whether we find an effect of the potential loss of an hour of sleep in the spring transition to daylight saving time. Table 2.2 shows that, on average, the potential one hour loss of sleep was associated with a decrease in assaults. There were 3% (95% CI: -4.3%, -1.6%, $p < 0.001$) fewer assaults on the Monday immediately following DST as compared to the Monday one week later. There was no moderating effect of gender ($p = 0.48$) or age ($p = 0.56$ and $p = 0.34$).

The spring robustness checks (also shown in Table 2.2) generally resulted in null findings. The reverse coding check, reversing the changes stated in the Energy Policy Act of 2005, was nonsignificant ($p = 0.39$). Comparing the Mondays one week and two weeks after DST when any effect of DST should wear off was also not significant ($p = 0.66$). We also found no effect when analyzing Wednesdays ($p = 0.62$), though we found a marginal effect when looking at Thursdays ($p = 0.05$); however this finding is not significant if adjusted for the number of falsification tests. These nonsignificant falsification tests are in striking contrast to the strong finding from the analysis of the correct labeling of Mondays.

Fall Analyses: Potential One Hour Gain Of Sleep

   The return to standard time in the fall and potential gain of an hour of sleep was associated with an increase in assaults as shown in Table 2.3. Indeed, we found what amounted to almost a mirror image of our spring findings, as there were 2.8% (95% CI: 1.5%, 4.2%, $p < 0.001$) more assaults immediately following the switch as compared to the following week. There was no moderating effect of gender ($p = 0.33$) or age ($p = 0.19$ and $p = 0.63$).

   Reversing the Energy Policy Act of 2005 coding indicated no effect ($p = 0.28$). Unlike our analysis in spring, analyses of other days following the return to standard time showed effects on the same scale as the primary analysis. The comparison of the Mondays one week and two weeks after the return to standard time, the comparison of the two Thursdays following the return to standard time, and the comparison of the two Wednesdays following the return to standard time showed significant increases in assaults (in all cases $p < 0.005$).

Discussion

   This study leveraged the exogenous short-term shock to sleep provided by the daylight saving time policy to test whether a potential loss or gain of an hour of sleep results in increased aggression. The springtime results were strong and robust and showed that a mild and short-term reduction in sleep quantity resulted in fewer assaults (as compared to a week later). This finding supports lack of motivation/energy due to sleepiness rather than reduced self-control as the effect of mild short-term sleep loss.

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Collectively, these tests indicate there is something particularly special about the Monday following DST that causes the observed decrease in assaults, suggesting that the 3% decline in assaults is likely due to DST and its effect on sleep.

On the other hand, while the fall finding showed a corresponding finding—a mild and short-term sleep gain results in increased assaults—findings are not robust to the falsification analyses as demonstrated by significant findings in almost all of those tests. This weaker finding is consistent with the lack of evidence that the fall return to standard time results in any discernable sleep gain (Harrison, 2013), which may explain the previous studies that have examined solely the springtime shift, and ignored the fall switch (Kotchen & Grant, 2011; D. T. Wagner et al., 2012). It is possible that the results are simply more long-lasting in the fall, but it seems unlikely considering most of the DST literature suggests less of a significant change in sleep in the fall than in the spring (it is easier for people to phase delay than phase advance) (Kantermann et al., 2007). Indeed, it could also be that people are getting more sleep, but increased motivation/energy alone is not a risk factor for increased aggression.

Sleep loss has wide-ranging effects on the body including physiological (Van Cauter, Spiegel, Tasali, & Leproult, 2008), cognitive (e.g., Sadeh, Gruber, & Raviv, 2002) and mood (Cote et al., 2013) alterations. This study suggests it also has distinct behavioral consequences. Unfortunately, our methodology restricts our ability to provide a causal explanation for the relationship between mild sleep loss and assaults; thus, we cannot provide insight into the biological pathways through which one hour of sleep

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might cause the observed reduction in assaults. It could be that the lethargy induced by DST results in a reduction in motivated behavior, particularly that which requires significant physical outlay (e.g., assaults). Nevertheless, recent longitudinal research indicates that daytime drowsiness at age 15 is associated with increased crime at age 23 (Raine & Venables, 2017). This contrary finding further suggests the need to differentiate the effects of limited sleep loss on short-term violence from the effect of more systemic sleep disruption and drowsiness on longer-term offending.

These findings and similar mixed findings in the sleep deprivation experimental studies coalesce to suggest that there is more nuance to the sleep-antisocial literature than those suggested by correlational studies. Neither the correlational nor the few experimental studies in the sleep literature are easily comparable to a one-time loss or gain of a single hour, which we aimed to operationalize here. Our findings suggest that a mild loss of sleep may result in lethargy or reduced motivation and thus reduced assaults. The methodology of this study contributes in a novel way to the sleep- antisocial behavior literature.

These findings have potentially important implications. This study demonstrates that the relationship between quantity/quality sleep and antisocial behavior may be more nuanced than suggested by the current literature (Kamphuis et al., 2012). Indeed, the mixed findings of the experimental literature suggest the importance of future research carefully considering how to measure sleep and aggression (Cote et al., 2013; Haack & Mullington, 2005). It seems plausible that a one hour loss as opposed to 24 hours, or 33

hours, or multiple hours over multiple days, will have differential effects on behavior. Moreover, Vohs et al. (2010) note that differential findings in the experimental literature may be attributable to the variable methods that have been used to measure aggression, for example, attributing blame in a vignette vs. a computerized "game." By using a significant and criminal level of aggression that requires physical exertion and some degree of motivation, this study supports the suggestion put forth by Cote et al. (2013) regarding the possibility that sleep loss may influence behavioral responsiveness very differently than it does cognitive or physiological responsiveness. The advent of DST could result in a reduction in motivation levels, accounting for a decrease in assaults despite an increase in negative mood.

With regard to criminal justice policy, our findings suggest that the crime-reducing effects of DST go further than the ambient lighting effects suggested by the findings of Doleac and Sanders (2015) and Calandrillo and Buehler (2008). Their findings focus on the fact that DST results in more ambient lighting at a high-crime time. We found decreased assaults immediately following DST, without robust findings suggesting a corresponding increase in the switch to standard time. Both of these findings are in agreement with regard to positive unintended effects of DST on crime. Because of the ongoing and competing bills surrounding DST in various state legislatures, it is important that legislators have as much information as possible about the unintended consequences of DST, particularly those related to crime. However, a 3% reduction in assaults one day out of the year has a negligible impact on the volume of assaults. A 3%

decrease in assaults on one day per year reduces the nation's assaults by about 330, a small fraction of the 800,000 aggravated assaults and 3.2 million simple assaults reported in the 2015 National Crime Victimization Survey. Therefore, crime effects as a result of the hour of lost sleep should not factor into policy-making on DST, although we refrain from concluding the crime effects of DST in general are insubstantial (e.g., Doleac & Sanders, 2015).

There are some limitations to this study. First, in four of the 12 years, the Monday immediately after the start of DST had a higher number of assaults, but these counts are consistent with our estimated year to year extra-Poisson variation. Nevertheless, unaccounted-for events on just the right dates could explain the results, though these events would need to have broad impact since NIBRS includes data on assaults from a wide geographical area. Second, while the spring findings were generally strong and robust to falsification checks, the evidence surrounding the effects of the fall switch are far weaker. It seems possible that weather effects may be more significant in the late fall, when early snow storms and falling temperatures can be expected, particularly in three of the four cities included in the analyses (i.e., New York City, Chicago, and Philadelphia). This makes some sense given the generally significant declines from week one to week two, regardless of the day, in the fall falsification analyses. Routine activities theory (L. E. Cohen & Felson, 1979) would suggest that winter weather could impact assaults during both work hours (if a storm causes businesses to close) and after-work hours (if individuals choose to stay at home) by keeping possible victims and assailants from

interacting. Nevertheless, the reverse coding robustness check, which is arguably the best robustness check as it uses days that could plausibly be the relevant days, found no effect.

In taking advantage of city-reported and NIBRS data, advantages of this sample include its size and broad coverage of the United States. The disadvantages of these data include our inability to examine and control for various factors, such as criminal records and sleep durations. Our assumptions around the sleep effects of the switches were therefore based on prior literature (i.e., C. M. Barnes & Wagner, 2009), but could not be confirmed in this sample.

Though the evidence suggests that the loss of sleep is the likely cause of the increase in assaults, it is possible that people, unaffected by sleep quantity or quality, simply alter their activities immediately following DST in such a way that could change the likelihood of both assaulting and being assaulted (e.g., staying home instead of going to a bar), and return to their habits a week later.

The mild exogenous shock to sleep provided by the DST policy allowed us to look at a large and representative population to examine the effects of a short-term loss of sleep. The advent of DST specifically is thought to be responsible for 40 min of lost sleep, if not more (C. M. Barnes & Wagner, 2009). DST already has shown itself to generate useful natural experiments for researchers to examine car accidents (Harrison, 2013), stock market losses (Kamstra et al., 2000; Pinegar, 2002), work-place injuries (C. M. Barnes & Wagner, 2009), cyberloafing (D. T. Wagner et al., 2012), reduced test scores (Gaski & Sagarin, 2011), suicide rates (Berk et al., 2008), crime due to changes in

ambient lighting (Doleac & Sanders, 2015), and racial profiling (Grogger & Ridgeway, 2006). In this paper we have harnessed this natural experiment generator again to learn about sleep and assault on a large national dataset. While the bulk of the literature has suggested a simple inverse relationship between sleep quantity/sleep quality and aggression, our findings are more in line with the mixed findings presented by sleep deprivation experimental studies. Indeed, the measurement of sleep and aggression appear to provide the differences. It seems possible that while mild loss of sleep may indeed induce aggressive feelings or negative affect, the associated sleepiness and lethargy impedes physically acting on those feelings.

Paper 2 Tables

Table 2.1 Number of assaults on Mondays immediately after and one week after transitions to and from DST

Year	Spring		Fall	
	Monday immediately following start of DST	Second Monday after start of DST	Monday immediately following end of DST	Second Monday after end of DST
2001	1797	1827	1673	1593
2002	1652	2015	1671	1625
2003	1695	2032	1732	1951
2004	3597	3512	4019	3458
2005	2396	2355	Not Included Due to Halloween	
2006	2624	2766	2645	2430
2007	2681	2556	2397	2432
2008	Not Included Due to St. Patrick's Day		2639	2215
2009	2780	2740	2525	2642
2010	2618	2623	2480	2481
2011	2574	2836	2551	2750
2012	2799	2882	2473	2420
2013	2406	2570	2442	2259
2014	Not Included Due to St. Patrick's Day		2501	2542
Total	60,333		62,546	

Table 2.2 Estimates of the Effects of Spring Changes to Daylight Saving Time on Assault, including Falsification Tests

Primary Hypotheses	
% increase in assault (95% CI)	-2.9%(-4.2%, -1.6%)
p-values	< 0.001
n	60,333
Falsification Analyses	
Reverse Coding	
% increase in assault (95% CI)	0.6% (-0.7%, 2.0%)
p-values	0.37
n	58,547
Monday one week and two weeks after DST	
% increase in assault (95% CI)	0.3% (-1.2%, 1.7%)
p-values	0.71
n	59,443
Wednesday immediately following and one week after DST	
% increase in assault (95% CI)	-0.3% (-1.5%, 1.0%)
p-values	0.62
n	67,997
Thursday immediately following and one week after DST	
% increase in assault (95% CI)	-1.2% (-2.4%, 0.0%)
p-values	0.05
n	68,658

Table 2.3 Estimates of the Effects of Fall Changes to Standard Time on Assault, including Falsification Tests

Primary Hypotheses	
% increase in assault	2.8% (1.5%, 4.2%)
p-values	< 0.001
n	62,546
Falsification Analyses	
Reverse Coding	
% increase in assault (95% CI)	0.8% (-0.6%, 2.2%)
p-values	0.28
n	58,966
Monday one week and two weeks after DST end	
% increase in assault (95% CI)	2.1% (0.7%, 3.4%)
p-values	< 0.005
n	60,740
Wednesday immediately following and one week after DST end	
% increase in assault (95% CI)	4.5% (3.1%, 5.8%)
p-values	< 0.001
n	62,875
Thursday immediately following and one week after DST end	
% increase in assault (95% CI)	2.1% (0.8%, 3.5%)
p-values	< 0.005
n	59,051

PAPER 3. COGNITIVE DECLINE AS A RESULT OF INCARCERATION AND THE EFFECTS OF A CBT/MINDFULNESS TRAINING INTERVENTION: A CLUSTER-RANDOMIZED CONTROLLED TRIAL[3]

Abstract

This study primarily tests whether incarceration negatively affects cognitive functioning; namely, emotion regulation, cognitive control, and emotion recognition. As a secondary interest, we test protective effects of a cognitive behavioral therapy/ mindfulness training (CBT/MT) intervention. Dormitories containing 197 incarcerated males aged 16 to 18 years were randomly assigned to either a CBT/MT program or an active control condition. A cognitive task was administered pretreatment and again 4 months later, upon treatment completion. Performance on all outcome variables was significantly worse at follow-up compared with baseline. There were marginally significant group by time interactions. While the control group performance significantly declined in both cognitive control and emotion regulation, the CBT/MT group showed no significant decline in either outcome. This is the first study to probe the effects of incarceration on these three cognitive processes. Findings suggest that incarceration worsens a known risk factor for crime (cognitive functioning), and that a CBT/MT intervention may help buffer against declines.

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[3] A version of this paper was published as Umbach, R., Raine, A., & Leonard, N. R. (2018). Cognitive Decline as a Result of Incarceration and the Effects of a CBT/MT Intervention: A Cluster-Randomized Controlled Trial. *Criminal Justice and Behavior*, *45*(1), 31-55.

Background

The negative outcomes associated with incarceration are argued to be well-documented and wide-ranging, including increased antisocial behavior of offspring (Mears & Siennick, 2016; Murray & Farrington, 2008; Rose, 1998) and impaired health of those previously incarcerated (Schnittker & John, 2007). Most studies have focused on psychological and social effects, resulting in a gap in the literature regarding the effects of incarceration on cognitive functioning. Compromised cognitive functioning, particularly executive functioning, is a well-replicated risk factor for antisocial behavior (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Accordingly, this study uses a longitudinal design to look at cognitive functioning in young incarcerated males, providing a new social neurocriminological perspective on the criminogenic effects of incarceration (Choy et al., 2015). Evidence that incarceration negatively alters cognitive functioning of prisoners in a way that may promote future recidivism would also speak to the possible benefits of alternative forms of justice, including drug courts and restorative justice approaches.

As a secondary and exploratory aim, this study additionally experimentally investigates the effects of a group-based cognitive behavioral therapy/mindfulness training (CBT/MT) intervention. While programming directed at improving prisoner outcomes is commonplace in correctional settings, mindfulness training as an intervention has become popular only recently. Most studies looking at the effects of mindfulness in offender populations have focused on recidivism as the outcome of

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interest (Alexander & Orme-Johnson, 2003; Bleick & Abrams, 1987; Himelstein, 2011).

while effects on cognitive functioning remain a gap in the literature, despite the

possibility that improved cognitive functioning may be a mechanism for these reductions

in recidivism. Leonard et al. (2013), using these data, provided a notable exception by

looking at the effects of incarceration and CBT/MT on a task of attention. Because

incarceration is hypothesized to lead to impaired cognitive functioning, and because

executive functioning expands beyond attention to include a wide range of processes

including emotion regulation and cognitive control, this study tests as a secondary aim

whether CBT/MT can help mitigate negative effects on these processes specifically.

Finally, this study discusses potential policy and criminal justice implications that follow

from our findings.

Executive Functioning: Cognitive Control, Emotion Regulation, And Emotion Recognition

Executive Functioning

Executive functioning is an overarching term used to refer to higher order

cognitive processes, which include dynamic decision making, attending, cognitive

control, and emotion regulation, all of which are considered necessary for prosocial

behavior (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Although there are a number

of executive functions that have been associated with antisocial behavior, the emotional

go/no-go task used in this study measures three related but distinct processes: cognitive

control, emotion regulation, and emotion recognition.

Cognitive Control

Poor inhibition and low self-control are executive functions well acknowledged by the literature to be associated with antisocial behavior (Gottfredson & Hirschi, 1990; Ogilvie et al., 2011). Gottfredson and Hirschi's general theory of crime argues that low self-control is the single most important predictor of crime, although many argue the need to incorporate situational characteristics such as opportunity (Grasmick, Tittle, Bursik, & Arneklev, 1993; Osgood, Wilson, O'Malley, Bachman, & Johnston, 1996). An ability to inhibit inappropriate responses is argued to be necessary in the achievement of future-oriented goals and prosocial behavior generally.

Emotion Regulation

Impaired emotion regulation has been associated with antisocial behavior (Lewis, Granic, & Lamm, 2006; Long, Felton, Lilienfeld, & Lejuez, 2014; Roberton et al., 2012; Roberton, Daffern, & Bucks, 2014). Under- and overregulation of emotion are both considered pathways to aggressive behavior (Roberton et al., 2012). Those who underregulate may act out to try to repair, end, or avoid uncomfortable emotional states, while those who overregulate may have increased negative affect and physiological arousal, and reduced inhibitions against aggression (Roberton et al., 2012). Being unable to manage and modify one's reactions appropriately is maladaptive and therefore likely to result in negative immediate, and long-term, outcomes.

Emotion Recognition

There is a strong body of evidence supporting a relationship between facial

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emotion recognition ability and antisocial behavior (Marsh & Blair, 2008).The prevailing

hypothesis behind this relationship is that poor recognition of negative affect (particularly

fear) is associated with impaired empathic development and, thus, a greater

predisposition to antisocial behavior. This cognitive process is thought to derive from

some of the same areas of the brain as emotion regulation and inhibition (Streit et al.,

2003), although to date there is no existing support for the hypothesis that mindfulness

may impact performance in this area.

Incarceration

After peaking in 2009, the incarceration rate in the United States has gradually

declined, standing at 458 prisoners sentenced to more than one year per 100,000 U.S.

residents of all ages in 2015 (Carson & Anderson, 2016; Travis, Western, & Redburn,

2014). Despite this declining trend, the incarceration rate of the United States continues

to be the highest in the world. A variety of factors will influence an inmate's incarcerated

experience, including the physical and cultural characteristics of a facility, academic

resources and life skills training classes, recreational time, and correctional officers. In

addition, characteristics of the prisoner (e.g., type of offense for which they were

convicted) will likely influence the stressors experienced.

Prison Programming

The availability of prison programming reflects not only different mandates at the

federal and state levels but also shifting societal attitudes toward the purpose of

incarceration and increased awareness regarding the effectiveness of programming.

Today, there has been a shift away from academic programming toward practical, targeted interventions (e.g., budgeting and parenting classes) designed to help prisoners succeed upon reentry (M. S. Phelps, 2011).

There is a dearth of reports evaluating the recent history of program offerings; however, at least one paper suggests that while more prisons are offering more programs, overall rates of inmate participation are anemic or decreasing. This is because, despite increases in facilities offering programming, the burgeoning numbers of inmates and the logistical restrictions (e.g., class sizes cannot just expand to meet demand, given security risks) prevent a commensurate increase in the rate of inmate participation in educational, vocational, or prison industry programming (Travis et al., 2014).

Negative Effects of Incarceration

Despite programming intended to help with rehabilitation and reentry, incarceration remains an overwhelmingly negative experience for the majority of offenders. Considering the number of individuals affected, it is important to examine the lived realities of incarceration. The literature surrounding "prisonization," or the process of socialization in a prison setting, suggests that prisoners develop coping mechanisms to adapt to the informal "code" practiced in prison. Studies suggest that the incarcerated experience is characterized by bullying, substance use, emotional flattening, psychological distress, strain on social bonds, self-isolation, and violence (Ashkar & Kenny, 2008; Haney, 2012; Schnittker & John, 2007; S. Yang, Kadouri, Révah-Lévy, Mulvey, & Falissard, 2009).

Moreover, negative effects of incarceration appear to be enduring and widespread, extending outside of prison. In addition to mental and physical health issues, the formerly incarcerated experience reduced status in the labor market (Schnittker & John, 2007), increased rates of drug-related death and homicide (Lim et al., 2012), and greater incidence of delinquency in their offspring (Murray & Farrington, 2008).

Importantly, incarceration experiences likely vary significantly conditional on the specific characteristics of a facility (Travis et al., 2014). Naturally, jails and prisons will operate differently, as will correctional facilities at different levels of security, and state prisons as opposed to federal prisons. Even within facilities, the experience of inmates will vary widely due to factors including physical layout (Wolff, Blitz, Shi, Siegel, & Bachman, 2007), resources (Duwe & Clark, 2014; Gallant, Sherry, & Nicholson, 2015), and quality of correctional staff (Reisig & Mesko, 2009). Moreover, the aforementioned factors likely feed into each other. For example, lesser resources may result in more prisoner misconduct, leading to frustrated and fearful staff and thus, more prisoner misconduct, which could result in additional removal of resources as punishment, and so on.

Inmate characteristics, such as criminal record, age, mental health, gender, and race, will also impact threats to personal safety and stress levels (Ashkar & Kenny, 2008). Young inmates, inmates with mental health disorders, and new offenders may be perceived as particularly vulnerable, and thus easy targets for victimization (Wolff, Blitz, & Shi, 2007; Wolff, Shi, Blitz, & Siegel, 2007). Certain types of offenders, such as

domestic or child abusers and sex offenders, may suffer significantly more prison victimization (Wolff, Shi, et al., 2007).

Psychological and Cognitive Effects of Incarceration

Despite a large body of literature exploring the effects of incarceration, the impact of incarceration on cognitive functioning is largely understudied. Some have speculated that incarceration has negative psychological effects (Haney, 2003, 2012) and that such effects may range from subtle psychological deficits to clinical levels of mental illness. For example, in generating hypotheses on the effects of incarceration in a supermax prison, Haney (2003) noted that the rigid structure, lack of stimuli, and loss of autonomy may result in the loss of the prisoners' " . . . ability to initiate or to control their own behavior, or to organize their own lives" and may cause them to " . . . find it difficult to focus their attention, to concentrate, or to organize activity" (p. 139). Haney does not explicitly identify these symptoms as cognitive issues. Nevertheless, both of these purported consequences are arguably indicators of impaired executive functioning, on the one hand, impaired attention and, on the other, loss of self-control ((Morgan & Lilienfeld, 2000; Ogilvie et al., 2011). Although supermax is the most extreme form of incarceration, others have suggested cognitive deficits could result from the loss of personal control associated with any type of incarceration (Goodstein, MacKenzie, & Shotland, 1984).

Beyond these high level potential risk factors, many other well-supported risk factors for executive function deficits are likely present in correctional facilities. Briefly,

sustained stress or trauma and lack of enrichment activities, both physical and material (Colcombe & Kramer, 2003; Hackman & Farah, 2009; Kramer et al., 1999; Noble et al., 2012, 2007, 2005; Öhman, Nordin, Bergdahl, Birgander, & Neely, 2007; Sarsour et al., 2011); sleep deprivation (Durmer & Dinges, 2005; Goel, Rao, Durmer, & Dinges, 2009); and institutional violence exposure (Glenn & Raine, 2014; Schretlen & Shapiro, 2003) all present as potential threats to cognitive integrity in incarcerated individuals. In this study in particular, exposure to institutional violence, specifically violence inflicted by correctional staff, is of serious concern (U.S. Department of Justice, Office of the United States Attorney Southern District of New York, 2014).

Despite these obvious risk factors, and the significant hypothesizing by Haney and others, there is little to no empirical data on the effects of incarceration on cognitive functioning. An exception includes a cluster-randomized controlled trial conducted by Leonard et al. (2013) using these participants but a different cognitive task, the Attention Network Task (Fan, McCandliss, Sommer, Raz, & Posner, 2002). In the task, various cueing conditions were followed by a central arrow pointing left or right, either alone or sandwiched between arrows pointing in the same direction or the opposite direction. Participants were asked to press the arrow key corresponding to the direction of that arrow. Leonard et al. (2013) examined three separate attentional networks (alerting, orienting, and conflict monitoring). They found that, although CBT/MT somewhat mitigated the deleterious effects of incarceration, performance on the task significantly decreased from baseline to follow-up (approximately 4 months later) across participants.

The observed decline due to incarceration is consistent with Haney's (2003) and Goodstein and colleagues' (1984) hypotheses that incarceration may have harmful psychological effects. As Leonard et al. (2013) focused on the effects of CBT/MT in buffering declines in attention, the current study aims to expand on these findings by probing the effects of incarceration on different types of executive function (i.e., inhibition and emotion regulation).

Mindfulness

Mindfulness, mindfulness training, and mindfulness meditation practices fall under the umbrella of general meditation practices. A general consensus definition of mindfulness involves two components: (a) the self-regulation of attention and (b) detached self-observation of the present moment in a nonjudgmental and accepting way (Bishop et al., 2004; Kabat-Zinn, 1982). Mindfulness is often incorporated into clinically oriented, group-based meditation programs such as Mindfulness-Based Cognitive Therapy (Teasdale et al., 2000) and Mindfulness-Based Stress Reduction (Kabat-Zinn, 1982). Although specific programs may incorporate various complementary therapeutic approaches, mindfulness is associated with improved self-regulation through its focus on self-awareness, attentional control, and emotion regulation (Y.-Y. Tang, Hölzel, & Posner, 2015).

With regard to cognition, mindfulness has been associated with improved executive functioning, particularly in the areas of attention (Chiesa, Calati, & Serretti, 2011; Eberth & Sedlmeier, 2012; Jha, Krompinger, & Baime, 2007), emotion regulation,

and cognitive control (Eberth & Sedlmeier, 2012; Holzel et al., 2011; Y. Tang et al., 2012; Wupperman, Neumann, & Axelrod, 2008). It has been hypothesized that one mechanism underlying this relationship is the upregulation of brain areas associated with executive functioning that results in improved neurocognition (Hölzel et al., 2011; Y.-Y. Tang & Posner, 2009). This is supported by brain imaging studies that find increased activation of the prefrontal cortex following mindfulness (Chiesa & Serretti, 2010). It is also conceivable that stress reduction may play a large part in improving cognition, as stress is associated with impaired neurocognition (Öhman et al., 2007), and mindfulness is often specifically targeted at reducing stress (Goyal et al., 2014).

Studies on the efficacy of mindfulness in treating antisocial behavior and associated criminogenic constructs have focused predominantly on outcomes of substance abuse and recidivism in incarcerated adult populations (Shonin, Van Gordon, Slade, & Griffiths, 2013). Within offender populations, mindfulness is argued to reduce recidivism (Alexander & Orme-Johnson, 2003; Bleick & Abrams, 1987; Himelstein, 2011), decrease hostility and depression, and increase self-esteem (Shonin et al., 2013) and self-reported self-regulation, including suppression of aggression (Evans-Chase, 2013). Despite the increasing interest in the viability of mindfulness as an effective intervention in incarcerated populations, methodological issues are widespread in the current literature, as noted by Shonin et al. (2013) in their review. Among the various studies included in their review, they note unreported attrition rates, small sample sizes,

and possible selection bias due to self-selection into the intervention group, among other issues.

Mindfulness has been shown to be effective in improving executive functions in community and clinical samples, but the current study will test whether it is successful in buffering deficits associated with the sustained stress of incarceration. Because chronic stress is associated with impaired cognitive functioning (Öhman et al., 2007), it seems plausible that stress reduction, perhaps the best-supported benefit of mindfulness (Barrett, 2016; Chiesa & Serretti, 2009), may provide a protective effect in a forensic sample. In addition, mindfulness encourages the meditator to acknowledge and accept his current emotions without acting on them, which may promote more effective and adaptive emotion regulation (Barrett, 2016; Eberth & Sedlmeier, 2012).

This Study

This study has primary and secondary research questions which aim to bring together several disparate literatures on the effects of incarceration and CBT/MT on cognitive functioning and the effectiveness of CBT/MT in correctional settings. Our primary research question is as follows:

Research Question 1: Does time spent incarcerated result in deficits in emotion recognition, cognitive control, and emotion regulation as measured by an emotional go/no-go task?

Assuming the answer to the first question is yes, our secondary research question is as follows:

Research Question 2: Does CBT/MT protect against incarceration-related cognitive deficits?

Although Leonard et al. (2013) used this sample, they used a task of attention and focused particularly on the protective effects of CBT/MT. By expanding these findings to different tasks measuring other executive functioning processes, this study aims to build upon those findings and to test the extent to which incarceration impairs cognitive functioning more generally—a theory that has been put forth (Goodstein et al., 1984; Haney, 2003), but has so far remained untested empirically.

Methods

Participants

As part of a larger study, 268 sentenced or detained male youths (M_{age} = 17.4 years, SD = 0.71, range = 16-18) were recruited from a large correctional facility in New York City between August 2009 and December 2010. Youth were invited to participate if they (a) had at least 6 weeks remaining on their sentence or estimated length of stay, (b) could complete an interview in English, and (c) were between the ages of 16 to 18 years. Youth at Rikers are assigned to one of two buildings depending on their status (sentenced

vs. awaiting trial), which consist of multiple dormitories. Dormitories from both buildings and the participating participants within were assigned randomly to receive either a CBT/MT intervention or an active control intervention. This cluster randomization was necessitated by concerns of contamination of the treatment effect.

Only a subset of participants completed both waves of data collection ($N = 197$) for the following reasons. As per the study protocol, participants ($n = 24$) who were transferred or released after the T1 assessment, but before the intervention began, were not contacted for follow-up assessment. Some participants ($n = 28$) completed the intervention, but were later transferred to a facility where study activities were prohibited by correction officials, and thus were unable to complete the follow-up evaluation. In addition, nine computer files were entirely corrupted, four computer files were missing data specifically for the follow-up emotional go/no-go task, four participants refused to complete the T2 assessment entirely, one participant was deported out of the country, and one participant turned 19 before the intervention began. Participants with complete data did not differ in age, race, or days incarcerated at baseline from those excluded from the study.

Of the participants with complete data, 88 participants were enrolled into the control group and 109 participants were enrolled into the experimental CBT/MT group. The groups did not differ in race, percent reporting violent or nonviolent crime, number of days in Rikers at baseline, or self-reported age of onset of offending. The experimental group was older than the control group by approximately 1 month (17.52 years vs. 17.40

years, $p = .005$). In all, 97% of the participants were Black or Latino, and mean length of time already spent in the correctional facility at baseline was nonnormally distributed ($M = 103.93$ days, $Mdn = 73$ days, interquartile range [IQR] = 111 days), with skewness of 3.43 ($SE = 0.17$) and kurtosis of 16.89 ($SE = 0.35$). Table 3.1 presents descriptive statistics for included participants. The participants also self-reported on types of offending, full details of which can be found in Table 3.2.

All youth incarcerated at Rikers are required to attend high school programming for five hours a day, unless in court or in solitary for rule infractions. All youth participating in this experiment continued to attend the General Educational Development program. There is no other educational or mental health programming offered to juvenile inmates. Youth who were 18 years old or legally emancipated signed informed consent. Youth less than 18 years of age signed informed assent, and parental consent was obtained for participation. All procedures were approved by the New York University Institutional Review Board and the New York City Department of Corrections.

Interventions

CBT/MT

Power Source (PS) is a group-based CBT/MT intervention for at-risk youth (Casarjian & Casarjian, 2003). Full details of the intervention and control condition can be found in Leonard et al. (2013). The theoretical underpinning of PS is the Process Model of Emotion Regulation (Gross, 1998), which outlines five major points of focus during emotion regulation: situation selection, situation modification, attentional

deployment, cognitive change, and response modification. PS combines traditional CBT practices with mindfulness training, which aims to assist in modulating physiological responses to stressful and risky situations to encourage prosocial behavioral responses. PS is designed to blend the social-cognitive change components of CBT with the attentional and response modification (including inhibition) elements of mindfulness. Specifically, with regard to the latter elements, PS trains youth to attend to situational characteristics, identify personal triggers for antisocial behavior, and direct attention away from those triggers and toward elements of the situation that encourage prosocial behavior. While CBT itself is a stand-alone intervention for antisocial behavior (Lipsey, Chapman, & Landenberger, 2001), it has been suggested that mindfulness may complement traditional CBT by increasing individuals' ability to be open to and acquire CBT skills and concepts (Teasdale, Segal, & Williams, 2003).

PS trains youth to choose prosocial peers and self-select into low-risk situations to reduce the likelihood of offending behavior. Youth are taught to identify high-risk situations and personal triggers for antisocial behavior, and direct their attention toward elements of situations that encourage prosocial behavior. Youth are trained to reappraise the meaning of situations to alter their emotional impact, reducing hostile attributional biases that may be present in an incarcerated sample (Dodge, Price, Bachorowski, & Newman, 1990).

The intervention consisted of weekly or biweekly group sessions with two clinicians trained in mindfulness meditation, and an accompanying book with role model

stories and mindfulness meditation exercises that were practiced in the group sessions. Group sessions consisted of cognitive behavioral exercises, videos for meditation instruction, and formal meditation practice, including body scans, sitting meditation, and walking meditation. To maintain internal validity, adherence to the PS protocol (in cognitive behavioral exercises, types of meditations, and reading assignments) was accomplished through the use of a manual and the videos. In addition to the group sessions, participants were also encouraged to engage in independent mindfulness meditation practice.

Cognitive-Perception Control intervention

The control group consisted of weekly or biweekly group sessions in which participants received portions of two evidence-based interventions: a cognitive-perception intervention focused on attitudes and beliefs about substance use and violence (Sussman, Rohrbach, & Mihalic, 2004) and a sexual-risk reduction intervention (Rotheram-Borus et al., 2003). The curriculum of each intervention was modified to exclude any skills or concepts that were under investigation in the PS intervention, thus controlling for the effects of common therapeutic factors such as therapeutic alliance, empathic counselors, attention, and group cohesion (Del Boca & Darkes, 2007; Safer & Hugo, 2006).

Treatment Procedure

The CBT/MT and control groups met separately for a total of approximately 750 minutes over three to five weeks. Timing varied slightly based on the security demands

of the separate housing areas. Each session was administered by two of four possible

clinicians (dependent on the clinicians' schedules), each session lasted approximately 75

minutes, and each group contained between eight and 12 participants. All four trained

clinicians received weekly clinical supervision to ensure fidelity to the respective

manuals. In addition, sessions were audio recorded, and approximately 10% of session

recordings were subject to quality assurance ratings for fidelity to both the control and PS

interventions. Fidelity was high across both conditions. Make-up sessions were offered in

small groups or individually for participants who missed sessions due to disciplinary

infractions or court appearances as possible. Baseline interviews were conducted prior to

onset of the intervention. Follow-up interviews occurred approximately 21 weeks after

baseline (range = 11-79 weeks), and there was no significant difference in time between

interviews for the treatment group ($M = 21.3$, $SD = 8.6$) and the control group ($M = 20.7$,

$SD = 11.0$; $t = -0.45$, $p = .66$). Participants in both groups received \$5.00 for every

session they attended, and \$25.00 in their commissary accounts for participation in each

interview.

Measures

Emotional Go/No-Go Task

Participants underwent two administrations of a computerized emotional go/no-go

task. The emotional go/no-go task is a variant on the classic go/no-go test, which allows

for the measurement of the respondent's ability to inhibit responses to emotional stimuli.

Because the traditional go/no-go task is considered a measure of behavioral inhibition

and cognitive control, often the stimuli are neutral, such as objects (Rubia et al., 2001) and shapes (Schulz et al., 2007); however, affective tests have become more common (e.g., Elliott, Rubinsztein, Sahakian, & Dolan, 2000).

Cognitive underpinnings of the emotional go/no-go

In this study, cognitive control, emotion regulation, and emotion recognition are measured by the emotional go/no-go task (Tottenham et al., 2011). The classic go/no-go task is commonly understood to be a test of executive functioning, a domain thought to be instantiated in the prefrontal cortex (Casey et al., 1997, 2011; Rubia et al., 2001). The emotional version of the task engages the amygdala (Hare, Tottenham, Davidson, Glover, & Casey, 2005) in addition to the prefrontal cortex (Wessa et al., 2007), allowing for a measure of emotion regulation, defined here as the ability to inhibit behavioral responding when presented with an emotionally stimulating situation (Tottenham, 2015).

Task procedure. As part of the interview process, participants completed two waves (one baseline and one approximately 4 months post-baseline) of a computerized emotional go/ no-go paradigm. The task required participants to press a button when a given facial expression target (e.g., anger) was displayed, and to refrain from pressing if they saw any other expression (the "no-go" or distracter expression). The target trials occurred more frequently (70% of trials were "go" trials) to create a tendency to respond. In total, there were eight conditions, each consisting of a neutral expression paired with one of four possible emotional expressions (happiness, sadness, fear, and anger).

Depending on the task, either the emotional expression or the neutral expression

served as the target. For example, there were two sad/neutral types of tasks: one in which

the sad face was the target, and one in which the neutral face was the target.

The facial images were color photographs of 10 adult male and female faces drawn from

the NimStim set (available at www.macbrain.org), representing a variety of

races/ethnicities. Faces were pseudorandomized across the block to control for order of

presentation, and the order of the eight blocks was randomized across participants.

Stimulus duration was 500 ms with 1,500 ms interstimulus intervals to ensure the

participants had sufficient time to respond. Ten practice trials were administered to

ensure that participants understood the task and could execute the responses. To fully

capture the treatment-related changes in behavior, composites previously associated with

this type of task were utilized (Casey, 2007; Schulz et al., 2007; Tottenham et al., 2010,

2011). The emotional go/no-go task has been validated for use with adults (Hare et al.,

2008), as well as community and clinical samples of children and adolescents

(Grunewald et al., 2015; Hare et al., 2008; Ladouceur et al., 2006; Tottenham et al., 2010,

2011). It has not been previously used in a forensic population. Measures of three main

constructs are derived from the emotional go/ no-go task: emotion recognition, cognitive

control, and emotion regulation.

Emotion recognition. D-prime provides an index of accuracy accounting for

response bias and is considered a measure of emotion recognition. It is calculated by

subtracting the *z*-transformed false alarm (FA) rate from the *z*-transformed hit rate. Higher scores reflect better performance.

Cognitive control. Overall FA rate was our index of cognitive control. In each trial, there are 10 possible FAs wherein a participant "hits" on a distractor emotion. The FA rate is the average proportion of incorrect responses and was calculated for all eight conditions, both when emotions were "go" and "no-go" stimuli. Higher scores indicate poorer performance.

Emotion regulation. FA rate to emotional "no-go" stimuli was an index of emotion regulation, with higher scores indicating poorer performance.

Covariates

Wide Range Achievement Test-4 (WRAT-4) reading subscale scores, self-reported mental health problems, and duration of time between baseline and follow-up were considered as possible confounders. The mean WRAT-4 reading raw score was 38.88 (equivalent to a seventh-grade reading level) and did not differ between groups ($p = .205$).

In addition to basic demographic information, participants completed a shortened version of the Youth Self-Report (YSR) questionnaire as a measure of mental health (Achenbach, 1991). The YSR is a well-used scale that has demonstrated significant generalizability (Ivanova et al., 2007). The six scales derived were Withdrawn, Somatic Complaints, Anxious/Depressed, Delinquent Behavior, Attention Problems, and

Aggressive Behavior. The two groups did not differ on any of the subscales (all $ps >$ .109).

Despite precautions taken to ensure even treatment application across the CBT/MT group, it seems possible that the clinicians could have improved over time, resulting in unintentional differences in treatment. This possibility was also examined through the use of repeated-measures ANOVAs.

Finally, the length between baseline interview and follow-up interview was examined as a possible covariate. The groups did not differ on length of time between interviews ($p = .678$). Detailed descriptive statistics of the potential covariates can be found in Table 3.1.

Data Analyses

The initial analyses were conducted using SPSS statistical software (IBM SPSS Statistics Version 22.0). Fully factorial repeated-measures ANOVAs were run on each of the three measures with a between-participants factor of treatment group (treatment, control group) and a within-participants factor of time (baseline, post-treatment). Within- and between- group changes in cognitive performance over time were used to assess whether incarceration caused declines in cognitive functioning, and whether CBT/MT affected those declines. We calculated the effect size f for the ANOVA main effects and interactions by using η^2, which is the ratio between the between-groups variance and the total variance. Effect size f is commonly understood such that $f = 0.10$ is a small effect, $f = 0.25$ is a medium effect, and $f = 0.40$ is a large effect (J. Cohen, 1969). Post hoc paired-

samples *t* tests were used to examine whether follow-up differed from baseline within groups. We calculated effect sizes for the paired-sample *t* tests using Cohen's *d* (J. Cohen, 1969). We corrected for dependence among the means using Morris and DeShon's (2002) Equation 8.

A secondary concern was the potential effects of covariates of interest. We conducted repeated-measures fully factorial ANCOVAs with additional within-subjecct factors of WRAT-4 score, the six subscales of the YSR Questionnaire (Achenbach, 1991), and the duration of time between baseline and follow-up to determine whether any of the covariates had significant main or interaction effects (Thomas et al., 2009).

Finally, we supplemented our main repeated-measures ANOVA analyses with a Bayesian approach, run using JASP statistical software (Version 0.8.0.1). We estimate a Bayes factor using Bayesian Information Criteria (Wagenmakers, 2007), comparing the fit of the data under the null hypothesis and the various alternative hypotheses. Bayesian analyses work to overcome some of the limitations of pure null-hypothesis significance testing by providing more information about both the null and alternative hypotheses and reducing dependence on sample size (Jarosz & Wiley, 2014). In short, the Bayesian approach is a model selection procedure that provides information to prefer one model over the others. Although there are a number of equivalent statistics that can be derived from Bayesian analyses, here we prefer BF_{10}, which frame the results in the context of the alternate hypothesis as opposed to the null hypothesis. For example, a BF_{10} of two

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means the data are two times more likely under the alternate hypothesis than the null hypothesis.

Results

Primary Research Question

The primary research question of this article was whether incarceration is associated with cognitive decline in cognitive control, emotion recognition, and emotion regulation. There was support for this hypothesis.

Cognitive Control

There was a main effect of time, $F(1, 195) = 11.84$, $p = .001$, $\eta^2 = 0.06$, $f = 0.25$, demonstrating significant decline from baseline to follow-up.

Emotion Regulation

There was a main effect of time, $F(1, 195) = 5.66$, $p = .018$, $\eta^2 = 0.03$, $f = 0.18$, again indicating significant decline from baseline to follow-up.

Emotion Recognition

There was a main effect of time, $F(1, 195) = 65.55$, $p < .001$, $\eta^2 = 0.25$, $f = 0.58$, such that performance significantly declined from baseline to follow-up.

Secondary Research Question

Our secondary research question was, given a rejection of the null hypothesis in the primary research question, whether CBT/MT could buffer deleterious effects of incarceration on cognition. This question was exploratory due to the gap in the literature regarding the effects of CBT/MT on cognitive functioning in an incarcerated population.

Cognitive Control

The two-way interaction of time by treatment group, $F(1, 195) = 3.47$, $p = .064$, $\eta^2 = 0.02$, $f = 0.14$, did not reach the traditional significance threshold of $p < .05$. Paired-sample t tests were used to probe the treatment effects. As shown in Figure 3.1, the control group significantly declined from baseline, $M = 0.24$, $SD = 0.14$, to follow-up, $M = 0.32$, $SD = 0.19$; $t(87) = -3.71$, $p < .001$, Cohen's $d = 0.41$. In contrast, the treatment group demonstrated no significant difference in the scores for baseline, $M = 0.26$, $SD = 0.17$, and follow-up, $M = 0.29$, $SD = 0.19$, assessments; $t(108) = -1.14$, $p = .255$, Cohen's $d = 0.13$.

Emotion Regulation

While the two-way interaction of time by treatment group, $F(1, 195) = 3.21$, $p = .075$, $\eta^2 = 0.02$, $f = 0.14$, also did not achieve traditional significance, the interaction term was probed by paired-sample t tests. As visually depicted in Figure 3.2, we observed significant decline in the control group performance from baseline, $M = 0.29$, $SD = 0.15$, to follow-up, $M = 0.37$, $SD = 0.33$, waves; $t(87) = -2.91$, $p = .005$, Cohen's $d = 0.32$. However, in the treatment group, there was no significant decline from baseline, $M = 0.32$, $SD = 0.19$, to follow-up, $M = 0.33$, $SD = 0.22$; $t(108) = -0.43$, $p = .670$, Cohen's $d = 0.05$.

Emotion Recognition

There was no significant two-way interaction, $F(1, 195) = 0.68$, $p = .410$, $\eta^2 = 0.00$, $f = 0.00$. As shown in Figure 3.3, paired-sample t tests showed significant decline in

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performance in both the control group—baseline: $M = 2.21$, $SD = 0.83$; follow-up: $M = 1.54$, $SD = 0.87$; $t(87) = 6.49$, $p < .001$, Cohen's $d = 0.69$—and the treatment group—baseline: $M = 2.14$, $SD = 0.87$; follow-up: $M = 1.55$, $SD = 0.95$; $t(108) = 5.14$, $p < .001$, Cohen's $d = 0.49$.

Potential Confounds

The data were reanalyzed using a repeated-measures fully factorial ANCOVA, with additional within-participant factors of WRAT-4 score, the six subscales of the YSR Questionnaire (Achenbach, 1991), and the duration of time between baseline and follow-up. This second part of the analysis allows us to examine the main effect of each of the covariates and the interaction of the covariates with our variables of interest (Thomas et al., 2009). None of the covariates had significant main or interaction effects (all $ps > .30$).

Despite adherence to a manual and the use of videos, it seemed possible that treatment may have differed over time (i.e., the trainers may have improved over time resulting in inconsistent treatment application). The treatment group was divided equally into "early" ($n = 54$) and "late" ($n = 55$) groups, which were then submitted to a repeated-measures ANOVAs using group type as the between-groups factor. Results demonstrated no significant differences between the two groups (all $ps > .182$).

Bayesian Supplemental Analyses

In addition to the standard repeated-measures ANOVAs, Bayesian repeated-measures ANOVAs with default prior scales were run. With regard to our primary research question, all models preferred the model with solely the within-participants

measure of time to the null (BF_{10} = 18.35, 19.10, and 1.25×10^{11}, for the cognitive control, emotion regulation, and emotion recognition models, respectively).

With regard to our more exploratory secondary research question, all models preferred the model with solely the within-participants measure of time to the model incorporating the interaction term (BF_{10} = 9.17, 8.96, and 28.20, for the cognitive control, emotion regulation, and emotion recognition models, respectively).

Possible Cluster Effects

As randomization was by dormitory to avoid treatment contamination and due to administrative reasons, we tested for possible effects due to individual dormitories using intracluster correlation (ICC) coefficients for performance on the three measures at baseline to investigate the variability within dormitories versus between dormitories. Because treatment and dormitory effects cannot be separated at follow-up, analyses were restricted to baseline data. We used the following formula to account for variable cluster sizes (Shrout & Fleiss, 1979)(Shrout & Fleiss, 1979): $MS_{between}$ - $MS_{within}/MS_{between}$ - $MS_{within(m0)}$ where m_0 = [1/k − 1] [n - $\Sigma m^2_i /n$], k is the total number of clusters, and m_i is the number of participants in each cluster. At baseline, the ICCs for cognitive control, emotion regulation, and emotion recognition were 0.00, 0.02, and 0.03, which are all small in size ($\leq$ 0.05) and can be interpreted as the proportion of overall response variation in individual responses that can be accounted for by within-dorm variation.

Effects of Out-of-Session Practice

We conducted additional analyses to assess the effects of out-of-session practice on cognitive performance by looking at participants in the CBT/MT arm who self-reported practicing ($n = 89$), and those in the CBT/MT arm who self-reported not practicing ($n = 20$). The groups did not differ in baseline performance (all ps > .39). Repeated-measures ANOVAs revealed no differences between groups with regard to emotion recognition ($p = .159$). Repeated-measures ANOVAs did find that the PS participants that independently practiced did better than the PS participants that did not independently meditate in emotion regulation ($p = .008$) and cognitive control ($p = .002$).

Discussion

This study set out to address a number of important gaps in the literature surrounding the cognitive effects of incarceration. We primarily hypothesized that incarceration negatively affects the executive functions of emotion regulation, emotion recognition, and cognitive control. We found that incarceration was associated with significant declines in specific aspects of executive functioning. The current study is the first to use a cognitive battery to longitudinally and empirically demonstrate the negative impact of incarceration on emotion regulation, cognitive control, and emotion recognition, which are key processes implicated in antisocial behavior (Marsh & Blair, 2008; Ogilvie et al., 2011; Roberton et al., 2012).

These findings provide empirical support for long-standing, but predominantly untested, hypotheses on the negative neuropsychological effects of incarceration (Goodstein et al., 1984; Haney, 2003, 2012). A secondary, exploratory question was

whether CBT/MT would have protective effects against any cognitive decline. Although the Time × Group interaction coefficients for cognitive control and emotion regulation did not reach the traditional significance threshold of $p < .05$, they are suggestive of the potential of CBT/MT interventions in buffering against some of these effects. Bayes factor repeated-measures ANOVAs, which are model based, preferred the "only time" model to both the null and interaction models in all three analyses.

Additional analyses incorporating potentially confounding covariates demonstrated no significant main or interaction effects, suggesting that cognitive decline due to incarceration can be expected regardless of initial reading ability, self-reported mental health, or length of time between baseline and follow-up. Furthermore, analyses revealed no significant facilitator improvement over time, supporting overall consistent treatment application.

We have documented cognitive declines in offenders over four months, but what may account for this decline? The methodology of this study restricted our ability to identify direct mechanisms for the relationship between incarceration and the decline in executive functioning. Aforementioned characteristics of prison, both designed and unintentional, may exert significant influence on individual psychological and cognitive functioning. Briefly, deprivation of self-determination and autonomy, sustained psychological and physical stress, lack of stimulation, victimization (physical and/or psychological), and sleep deprivation are potential mediators on a causal pathway from incarceration to impaired cognition (Blevins, Listwan, Cullen, & Jonson, 2010;

Colcombe & Kramer, 2003; Durmer & Dinges, 2005; Goel et al., 2009; Hackman & Farah, 2009; Ireland & Culpin, 2006; Kamphuis et al., 2012; Kramer et al., 1999; Maski & Kothare, 2013; Noble et al., 2012, 2007, 2005; Öhman et al., 2007; Sarsour et al., 2011; Shao et al., 2014; Vogler, Perkinson-Gloor, Brand, Grob, & Lemola, 2014).

One finding of note is the highly significant decline over time for both groups in emotion recognition, and the lack of even marginal mindfulness buffering effects for this task specifically. There are several plausible reasons for this finding. The decline in emotion recognition could be the result of short-term extreme sleep deprivation, which has been shown to impair emotion recognition in healthy adults (van der Helm et al., 2010). In addition, qualitative studies have suggested inmates self-isolate (S. Yang et al., 2009) or become emotionally numb to avoid displaying weakness or vulnerability to other inmates (Liem & Kunst, 2013). If offenders are exposed to a limited range of emotions for extended periods of time, their ability to identify emotions may be diminished. Finally, as noted earlier, of the three cognitive functions measured here, mindfulness has been shown to specifically affect emotion regulation and cognitive control (Eberth & Sedlmeier, 2012; Holzel et al., 2011; Y. Tang et al., 2012; Wupperman et al., 2008). In contrast, to date, there has been no support for a positive effect of mindfulness on emotion recognition. Furthermore, the strong decline in emotion recognition may have been too great for CBT/MT to remediate this cognitive deterioration, suggesting that any buffering attempts against the effects of incarceration on cognitive loss may need to be implemented relatively quickly.

Limitations

It is conceivable that this study may be underestimating the true effects of incarceration on cognition and the buffering effect of CBT/MT. With regard to the former, under normal conditions, performance at follow-up would be expected to be the same or even slightly better than at baseline, due to learning effects from repeating the same task (Morris & DeShon, 2002). Learning, or retest, effects refer to the improvement often observed in participants when they are repeatedly administered the same or similar tests (Bachoud-Levi et al., 2001; Salthouse, Schroeder, & Ferrer, 2004). Such practice effects may well attenuate any effect of imprisonment on cognitive decline.

With regard to the effects of CBT/MT, institutional constraints dictated an active control group. Thus, it is plausible that a true control group, and one more representative of the lack of actual programming available in this facility (e.g., a waitlist comparison), would have deteriorated even more significantly, providing a clearer measurement of both the effects of incarceration on cognition and the potentially protective effects of CBT/MT. We further acknowledge the limitation that the intervention did not improve cognitive functioning, as has been seen in community samples (Diamond & Lee, 2011), but merely limited the decline.

Although we derived three measures of cognitive functioning, this study focuses on a single cognitive domain (executive functioning) using a single cognitive task. There are many other unexplored cognitive and brain processes that may be affected by

incarceration and involved in recidivism, such as episodic memory and language processing, and at this point, findings cannot be generalized to other cognitive functions. Our secondary question only received mixed support. The results of the repeated-measures ANOVAs and the group-by-time interactions for cognitive control and emotion regulation did not reach the traditional significance threshold of $p < .05$ ($p = .064$ and $p = .075$, respectively). Regardless, evolving notions of the role of the p value (Goodman, 1999; Kyriacou, 2016) suggest that blind observance of p values restricts the balanced judgment of experimental findings. Type II error may be of particular importance in the early phase of a field of study, where false rejection of a true effect may foreclose the development of a new field of enquiry. Accordingly, we also ran a Bayes factor repeated-measures ANOVA, which preferred the model with a main effect of time alone, as opposed to the model with the interaction term. Together, these results do not provide enough evidence to unilaterally support protective effects of mindfulness.

Despite this, in recognizing the potential protective effects of CBT/MT, we note both the marginal interactions from the repeated-measures ANOVAs and the traditionally significant results of the paired-sample t tests, which suggest some degree of buffering against cognitive decline in the CBT/MT group compared with the significant decline in the control group. Moreover, we submit that the clear deleterious effects of incarceration on cognition necessitate an ongoing pursuit of potential cognitive interventions. Replication and extension of this study with a larger sample would provide clarity as to the clinical significance of these marginal interactions.

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This study intentionally incorporated elements of both CBT and mindfulness training, both of which have strong bodies of literature suggesting positive effects on various out- comes in incarcerated populations (e.g., recidivism, self-esteem; Landenberger & Lipsey, 2005; Shonin et al., 2013). While the blending of the two interventions was intentional due to a belief in their complementary nature, it does restrict our ability to attribute the results solely to either CBT or mindfulness training.

We demonstrated the deleterious effects of incarceration on cognitive functioning and the positive effects of CBT/MT; however, this study focused on a very specific population—16- to 18-year-old males in one facility. Moreover, it has been documented in this particular facility that a disproportionate proportion of the youth inmates were subjected to serious physical harm by correctional staff at the time this study was being conducted (U.S. Department of Justice, Office of the United States Attorney Southern District of New York, 2014). Therefore, we cannot generalize findings to other populations, but we nevertheless do provide the basis for future studies on older males, women, and prisoners in other facilities.

Contributions and Future Directions

The above limitations should be viewed in the context of several strengths of the current study. While there is a significant body of literature identifying lasting psychological and social effects of incarceration, including negative outcomes both for the former convicts and their families (e.g., marital instability, offspring antisocial problems, physical and mental health problems; Murray & Farrington, 2008; Schnittker

& John, 2007), this article identifies another negative outcome that has largely been ignored—cognitive impairments, particularly in executive functions associated with antisocial behavior (Ogilvie et al., 2011; Roberton et al., 2012, 2014). In addition to being a risk factor for antisocial behavior (Morgan & Lilienfeld, 2000; Ogilvie et al., 2011), impaired executive functioning is also associated with a variety of other negative life states, including substance use (Giancola, Mezzich, & Tarter, 1998; Giancola, Shoal, & Mezzich, 2001), impaired social integration (Hanks, Rapport, Millis, & Deshpande, 1999), and other psychopathology (Donohoe & Robertson, 2003; Moritz et al., 2002). Perhaps most fundamentally, our findings with regard to our primary research question speak to the benefits of alternatives to traditional incarceration, such as drug courts and restorative justice practices, especially for low-level offenders.

This randomized controlled trial also makes a second contribution by recognizing the possible role of CBT/MT in buffering against these impairments. Although analysis of the effects of CBT/MT on recidivism in this particular sample was beyond the scope of this study, this study provides a context to previous findings regarding the efficacy of CBT/MT in reducing recidivism likelihood (Alexander & Orme-Johnson, 2003; Bleick & Abrams, 1987; Himelstein, 2011). While the literature suggests positive effects of CBT/MT on recidivism likelihood, there is a lacuna as to the actual mechanisms driving that relationship. The experimental nature of this study goes beyond previous literature by allowing for a more rigorous exploration of the potential buffering effects of CBT/MT in protecting against cognitive decline. It seems possible that protective effects of

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CBT/MT on executive functioning may help account for some reduction in recidivism likelihood. Implementing an intervention such as CBT/MT or other cognitive interventions may be costly in the short term, but could be cost-effective in the long term by helping former prisoners transition successfully back into society (Dafoe & Stermac, 2013). While the lack of significance of the interaction precludes any definitive rejection of the null hypothesis, we hope that these initial results will encourage future work in this field.

This preliminary work can provide a basis for expansion and replication. We were restricted in our methodology to cognitive tasks, but future studies can expand on these cognitive findings by incorporating other measures of brain functioning, including electro- encephalogram (EEG), event-related potentials, and brain imaging (Raine, 2013). In addition, future interventions may use four groups to separate mindfulness from cognitive behavioral therapy to assess whether in isolation or in combination these treatments may protect against cognitive decline. Future studies may also work to identify the mediating variable between incarceration and cognitive decline, looking at variables such as victimization (by staff and/or other inmates) and stress, among others. It would also be informative to examine recidivism as an outcome to test whether cognition predicts recidivism likelihood. Finally, in addition to replication and extension, understanding the mechanism of action of how CBT/MT appears to buffer against cognitive decline remains an important future challenge.

Conclusion

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There are already numerous causes of concern regarding the effects of incarcerating youth. For example, aggregating troubled youth with other antisocial youth could lead to a peer contagion effect (Dishion & Dodge, 2005). In addition, more recent analyses of labeling theory have affirmed the notion that official interventions in adolescence may result in exclusionary circumstances, reducing opportunities for conventional success and contributing toward increased risk for adult offending (Bernburg & Krohn, 2003). Finally, the aforementioned well-supported experiences of being incarcerated could be exacerbated in youth, who may well be being separated from their family and friends for the first time and are characterized by many of the risk factors for victimization within prison (Wolff, Shi, et al., 2007). This study provides another compelling reason to try to keep adolescents with still- developing cognitive functioning out of correctional facilities.

Treatment of youth by the criminal justice system in recent years has reflected competing interests—on one hand, state and federal courts have retained the ability, and in some cases, the mandate, to waive particularly violent youth to the adult court system. On the other hand, Supreme Court decisions (e.g, *Roper v. Simmons,* 2005; *Graham v. Florida,* 2010; *Miller v. Alabama,* 2012) in the past two decades have recognized that youth offenders are fundamentally different from their adult counterparts, and present more potential for maturation into prosocial members of society. Such decisions suggest that a reversal from harsh, punitive treatment of youth offenders helped by growing research into characteristics of adolescence. Furthermore, from a purely economic, cost-

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saving perspective, there is evidence that treatment oriented policies (e.g., multisystemic therapy, aggression-oriented programs) are far more cost-effective than retributive incarceration (Travis et al., 2014).

This study provides another cause of concern, which is that cognitive functioning may decline as a result of incarceration, particularly in domains and processes already tied to antisocial behavior. Given the fiduciary and ethical concerns surrounding the enormous cost of mass incarceration, this study provides even more support for the use of alternative methods of punishment, such as drug courts and restorative justice courts. Keeping youth out of the "system" and protecting them when they are most emotionally and cognitively susceptible to the negative effects of incarceration may well be the best policy in terms of both efficacy and cost-effectiveness. Barring such a substantial shift in criminal justice policy, however, it is important for researchers and policymakers alike to continue to seek out and evaluate potential interventions to mitigate the negative effects of incarceration on inmates.

Paper 3 Tables and Figures

Table 3.1 Descriptive Statistics

Variable	Full Sample, $N = 197$		Control, $n = 88$		Powersource, $n = 109$		
	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	t
Age	17.40	0.71	17.24	0.71	17.52	0.69	-2.83*
WRAT (reading subscore)	38.88	8.65	38.00	10.03	39.61	7.32	-1.32
Last Grade Completed	9.93	1.38	10.00	0.94	9.87	1.66	0.69
Black	0.51	0.50	0.55	0.50	0.51	0.50	0.70
Hispanic	0.30	0.46	0.28	0.45	0.31	0.47	-0.42
White	0.01	0.07	0.00	0.00	0.01	0.10	-0.90
Multiracial/Other	0.18	0.38	0.17	0.38	0.18	0.39	-0.24
Any violent crimes	0.57	0.50	0.56	0.50	0.58	0.50	-0.30
Nonviolent crimes	0.78	0.41	0.81	0.40	0.76	0.43	0.76
Number of Days in Rikers at Baseline	103.93	120.44	94.06	84.63	111.90	142.88	-1.09
Log Number of Days in Rikers at Baseline	4.15	1.02	4.15	0.94	4.16	1.07	-0.09
Days Between Baseline and Follow-Up Interviews	146.89	68.26	144.64	76.92	148.72	60.68	-0.42
Age at Onset of Offending	10.50	4.53	9.94	4.60	10.98	4.42	-1.50
Score on YSR subscales							
Anxious/Depressed	4.33	3.88	4.03	3.54	4.57	4.14	-0.96
Withdrawn	5.16	3.19	5.07	2.83	5.24	3.46	-0.38
Somatic Complaints	1.65	2.13	1.41	2.03	1.84	2.20	-1.43
Delinquent Behavior	10.45	4.57	10.90	4.12	10.09	4.88	1.23
Aggressive Behavior	7.76	4.99	8.40	5.02	7.25	4.93	1.61
Attention Problems	5.38	3.32	5.56	3.57	5.23	3.12	0.69
Other Problems	4.21	2.53	4.39	2.86	4.06	2.23	0.89

Note. *$p < 0.01$

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Table 3.2 Self-Reported Crime by Group

Type of Self-Reported Crime	Of Reporters, % and Number Reporting Yes		
	Total	Power Source	Control
Carried a hidden weapon	72%	72.9%	69.3%
	$N = 193$	$n = 107$	$n = 86$
Purposely set fire to a house, building, car, or vacant lot	6.3%	3.8%	9.3%
	$N = 192$	$n = 106$	$n = 86$
Entered or broke into a building to steal something	31.3%	30.2%	32.6%*
	$N = 192$	$n = 106$	$n = 86$
Stolen something from a store	66.0%	61.9%	70.9%
	$N = 191$	$n = 105$	$n = 86$
Snatched someone's purse or wallet or picked someone's pocket?	22.5%	21.9%	23.3%
	$N = 191$	$n = 105$	$n = 86$
Taken something that did not belong to you from a car?	29.8%	33.3%	25.6%
	$N = 191$	$n = 105$	$n = 86$
Stolen a car or motorcycle to keep or sell?	13.1%	12.4%	14.0%
	$N = 191$	$n = 105$	$n = 86$
Sold drugs such as marijuana, cocaine, crack, or heroin?	63.4%	61.9%	65.1%
	$N = 191$	$n = 105$	$n = 86$
Attacked someone with a weapon?	41.9%	42.9%	40.7%
	$N = 191$	$n = 105$	$n = 86$
Had or tried to have sexual relations with someone against their will?	1.0%	1.9%	
	$N = 191$	$n = 105$	0.0%*
			$n = 86$
Used a weapon or force to get money or things from people?	36.9%	35.7%	38.3%
	$N = 179$	$n = 98$	$n = 81$
Threatened someone with a gun or another weapon?	47.8%	49.5%	45.7%
	$N = 178$	$n = 97$	$n = 81$
Been loud, rowdy, or unruly in a public place?	52.2%	50.5%	54.3%
	$N = 178$	$n = 97$	$n = 81$
Avoided paying for things such as movies, trains, or bus rides	64.0%	60.8%	67.9%
	$N = 178$	$n = 97$	$n = 181$
Attempted to kill or seriously injure someone?	25.7%	23.5%	28.4%
	$N = 179$	$n = 98$	$n = 81$

Note. *$p < 0.05$

Figure 3.1 Cognitive Control at Baseline and Follow-Up as a Function of Treatment
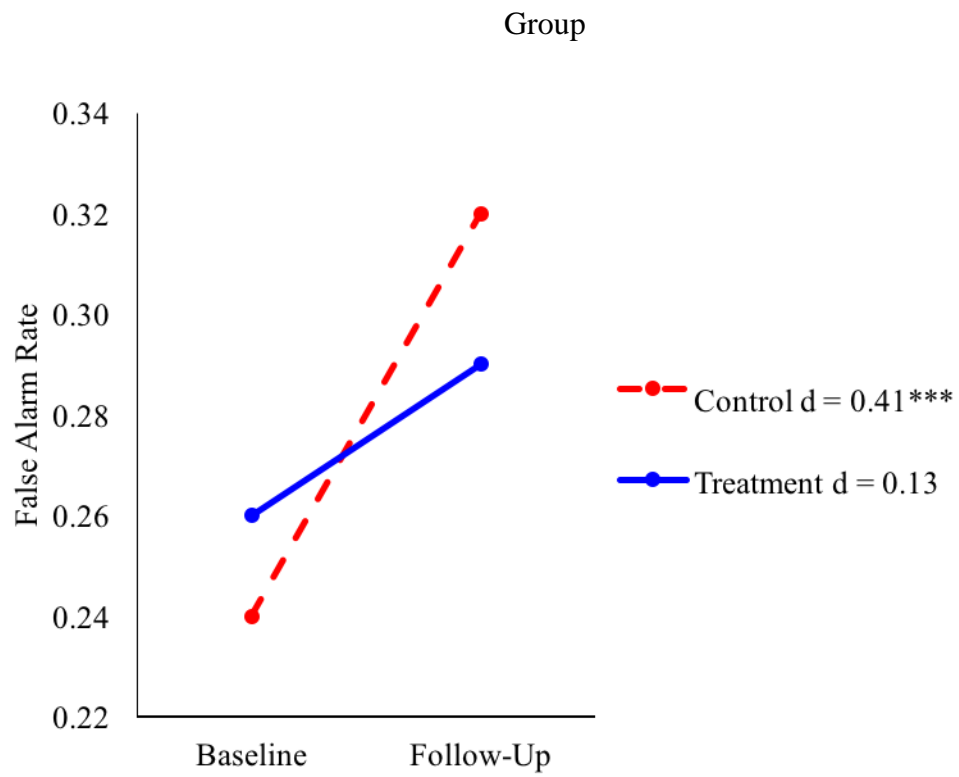
Group

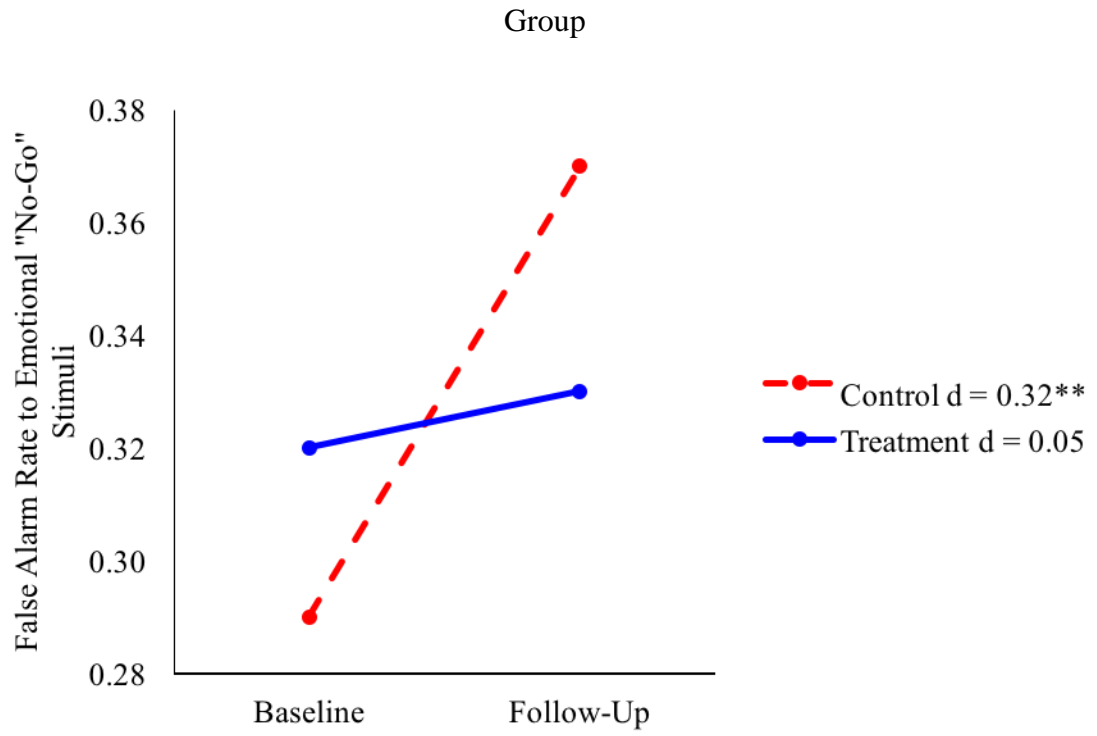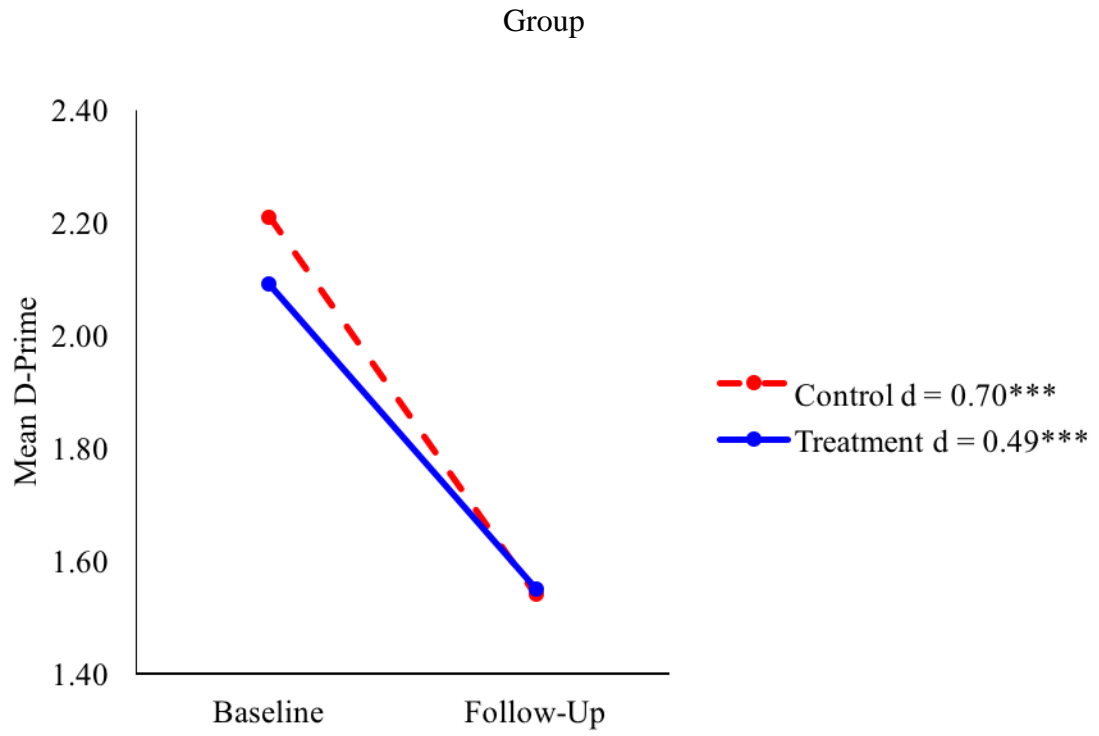Figure 3.2 Emotion Regulation at Baseline and Follow-Up as a Function of Treatment

Group

Figure 3.3 Emotion Recognition at Baseline and Follow-Up as a Function of Treatment

Group

GENERAL DISCUSSION

This dissertation set out to examine environmental and situational influences on cognition and corresponding behavior. Findings suggest A) that individual cognition can be affected by negative environments, B) cognition can then be directly linked to antisocial behavior, and C) short-term situational changes can affect behavior. The first paper in this dissertation demonstrated that the link between race and antisocial behavior can be at least partially explained by a pathway from race to neighborhood disadvantage to impaired executive functioning to antisocial behavior. The second paper in this dissertation found that the hour loss of sleep associated with daylight saving time corresponded with a 3% decrease in assaults, as compared to the Monday following. The return to standard time in the fall resulted in a 3% increase in assaults, as compared to the Monday following, however the finding was not as robust to the falsification analyses conducted. The third paper in this dissertation examined the effects of incarceration on cognitive functioning. An emotional go/no-go task was given to adolescent male offenders, and then repeated four months later. Findings showed that performance on the three outcomes derived from the task (cognitive control, emotion regulation, and emotion recognition) all declined significantly. A secondary research question investigated whether a Cognitive Behavioral Therapy/Mindfulness Training intervention could help mitigate these effects. Marginally significant time x group interactions for the outcomes of cognitive control and emotion regulation suggest that it may be a useful avenue to pursue to ameliorate the negative cognitive consequences of incarceration.

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In criminology research, environment has long been understood to have an important effect on behavior, but has often been examined more on the macro level (e.g., built environment, informal social control). There is increasing evidence and support for the importance of exploring the relationship between environmental, psychosocial, and biological factors in predicting antisocial behavior (Beaver, Gibson, DeLisi, Vaughn, & Wright, 2012; Choy et al., 2015). These three papers contribute to that evidence by demonstrating the significant ways in which environment can influence individual cognitive functioning. Additionally, they highlight the fact that small situational shifts can affect behavior in a tangible way. Broadly, impaired executive functioning, and more specifically, cognitive functions such as sustained attention, emotion regulation, and cognitive control, have long been implicated in delinquency and antisocial behavior.

On a theoretical level, findings help provide evidence for a biopsychosocial approach to criminology, in which environment can affect individual functioning (Choy et al., 2015). From an applied viewpoint, these findings help support the importance of ameliorating toxic environments, and for examining potential unintended consequences of seemingly harmless legislation. For example, findings from paper 3 would suggest that redirecting juvenile offenders from traditional incarceration towards alternatives such as drug courts or restorative justice may protect them from cognitive harm. Barring significant investment in improving disadvantaged neighborhoods and making prisons more stimulating, findings from paper 1 help identify an alternative pathway—cognition—to target in an effort to mitigate the potential negative effects of adverse

environments. Finally, while the findings from paper 2 suggest a net neutral effect of daylight saving time on assaults (based solely on the sleep change, disregarding any effects on light), nevertheless, the findings do demonstrate the importance of probing for potential unintended consequences of existing and/or proposed legislation.

APPENDIX

Appendix A

Items for Conduct and Oppositional Defiance Disorder Questionnaire (COD)

Conduct Disorder

      bullied or threatened someone
      started a physical fight
      used a weapon to harm someone
      been physically cruel to someone
      been physically cruel to an animal
      stolen or grabbed things from someone
      forced someone into a sexual activity
      started a fire to damage things
      destroyed people's things
      broken into a house, building, or car
      lied to get things or favors, or to avoid doing something
      stolen things or shoplifted
      stayed out at night without permission
      ran away from home for a while
      stayed off school without permission.

Oppositional Defiance Disorder

      lost their temper
      argued with adults
      refused to follow requests or rules
      deliberately annoyed people
      blamed others for their own mistakes or bad behavior
      been touchy or easily annoyed
      been angry or resentful
      been spiteful or mean.

Appendix B

Table B.1 Factor loadings based on a principal components analysis for parent-report and child self-report measures of antisocial behavior

	Antisocial Behavior	
	Child Self-Report	Parent Report
COD		
Oppositional Defiance Disorder	.85	.81
Conduct Disorder	.78	.83
APSD		
Callous Unemotional	.20	.50
Narcissistic	.76	.83
Impulsivity	.71	.80
CBC/YSR		
Rule Breaking	.69	.86
Aggressive	.86	.89
AQ		
Physical Aggression	.74	-
Verbal Aggression	.72	-
Anger	.72	-
Hostility	.64	-
Indirect Aggression	.69	-
RPQ		
Proactive Aggression	.78	-
Reactive Aggression	.79	-

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