The Role of Top-Down Attentional Control and ADHD Symptoms in Predicting Future Motor Vehicle Crash Risk

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Abstract

Objective: ADHD confers elevated risk for automobile crashes, both as a clinical syndrome and continuously when examining risk as a function of symptom severity. However, the neurocognitive mechanisms and processes underlying this risk remain poorly understood. The current longitudinal study examined whether attention network components reflect neurocognitive pathways linking ADHD symptoms with adverse driving outcomes.

Method: Prospective monitoring of objectively-identified crashes, near-crashes, and crash fault in 3,226 drivers from six U.S. sites participating in the Strategic Highway Research Program (SHRP-2) Naturalistic Driving Study. At study entry, drivers were assessed for ADHD symptoms, completed the Conners' CPT-II, and were then followed continuously for 1-2 years of routine, on-road driving using technology-enhanced in-car monitoring. Bias-corrected, bootstrapped mediation models examined the extent to which attention network components mediated the association between ADHD symptoms and future driving risk, controlling for known risk factors.

Results: As expected, self-reported ADHD symptoms predicted all markers of future driving risk. Higher ADHD symptoms were associated with reduced inhibitory control, lower levels of top-down attentional control (endogenous orienting), and greater arousal decrements (phasic alertness). Controlling for ADHD symptoms, top-down attentional control uniquely predicted future crashes, near-crashes, and culpability for future crashes/near-crashes; only arousal decrements portended future near-crashes. Only top-down attentional control significantly mediated the association between baseline ADHD symptoms and future driving risk.

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Conclusions: The driving risks associated with ADHD appear to be conveyed in part by impairments in the top-down, voluntary control of attention, rather than to difficulties sustaining attention over time or inhibiting impulses as is often assumed.

The National Highway Traffic Safety Administration (2014) estimates that approximately 34,000 people die in motor vehicle crashes per year, making it one of the leading causes of death in the United States. Drivers with high incidence psychiatric disabilities such as ADHD contribute disproportionately to these rates (Aduen et al., 2018; Barkley & Cox, 2007; Vaa, 2014). Replicated evidence indicates that drivers with ADHD experience more frequent crashes, near-crashes, and traffic violations (Barkley et al., 1993; Chang et al., 2014; Fischer et al., 2007; Vaa, 2014), with evidence suggesting that these rates do not decline in adulthood like they do for neurotypical drivers (Kay et al., 2009). Further, drivers with ADHD are more likely to be involved in more severe crashes and be adjudicated 'at fault' for driving errors that lead to traffic collisions (Aduen et al., 2015; Jerome et al., 2006; Vaa, 2014). In the context of these findings, driving safety has emerged as a significant functional impairment in adolescent and adult ADHD (Barkley, 2004; Fabiano et al., 2016). However, less is known about the underlying mechanisms and processes that link ADHD behavioral symptoms with this increased crash risk. The current study used a prospective, longitudinal design with a large, nationwide sample of drivers with and without ADHD to examine the link between ADHD symptoms, objectively-assessed components of the cognitive attention network (Posner & Peterson, 1990), and future risk for crashes, near-crashes, and crash/near-crash fault during routine driving.

ADHD and Driving Risk: Cognitive and Behavioral Pathways

Studies examining the link between ADHD symptoms and adverse driving outcomes have consistently implicated inattention as a primary contributor to motor vehicle accidents and violations (Dingus et al., 2016; Fuermaier et al., 2015). For example, drivers with ADHD have a greater susceptibility for distraction, such that they are more likely than drivers without ADHD to allocate their attention to in-vehicle distractors (Pope et al., 2017; Reimer et al., 2010), which is problematic given that attentional lapses as short as two seconds have been shown to result in severe or fatal driving outcomes (Horrey & Wickens, 2006). Drivers with ADHD are also less likely to maintain attention to driving-relevant cues over time, as evidenced by less monitoring of changing road conditions and traffic demands, and extended glances away from the roadway (Biederman et al., 2007; Fuermaier et al., 2015; Kingery et al., 2015). However, no study to date has differentiated among cognitive subcomponents of attention to assess the extent to which these observable behaviors are attributable to difficulties with vigilance/sustained attention, arousal/alertness, impairments in top-down (voluntary) attentional control, and/or difficulties with inhibitory control. Differentiating among these interrelated but separable components of the attention network (Posner & Peterson, 1990) is critical given evidence that some of these components may not be impaired in individuals with ADHD (e.g., Huang-Pollock et al., 2003). Moreover, there is mixed evidence linking disinhibition with crashes, traffic violations, and risky driving behaviors in simulated high stress driving scenarios (Barkley & Cox, 2007; Bioulac et al., 2016; Groom et al., 2015; cf. Biederman et al., 2007; Reimer et al., 2006).

Linking Neurocognitive Mechanisms with Driving Safety

Barkley (2004) proposed a testable model regarding the behavioral and cognitive mechanisms through which ADHD symptoms interfere with driving performance. Briefly, driving requires three hierarchical levels of competency – *operational* (immediate vehicle control), *tactical* (traffic and in-vehicle demands), and *strategic* (higher-order planning, goal-directed behavior) – that increase in complexity, cognitive load, and susceptibility to disruption. A key component of this model is its emphasis on the permeating impact of inattention and disinhibition on each level of driving performance (Barkley, 2004; Barkley & Cox, 2007). As such, neurocognitive processes of inattention and disinhibition are positioned as likely candidate mechanisms underlying driving risk in ADHD, given established associations with ADHD behavioral symptoms (Biederman et al., 2007; Hervey et al., 2004; Woods et al., 2002) and adverse driving outcomes (Barkley, 2004; Fuermaier et al., 2015; Groom et al., 2015; Reimer et al., 2010). Building on this model, the current study adopts the Posner & Peterson (1990) attention network framework to examine the extent to which cognitive attention components, including disinhibition, account for the adverse driving risk associated with ADHD symptoms.

Arousal and vigilance decrements (phasic and tonic alertness).

Attention is a complex, multi-component construct that can be parsed into separate but interrelated neural subsystems and functions (Riccio et al., 2002). Three attention networks frequently emerge: the *alerting* network, the *orienting* network, and the *executive* attention network, each associated with distinct neural correlates and functionally separate attentional processes (Petersen & Posner, 2012). *Alerting* refers to bottom-up (involuntary) processes responsible for enhancing physiological activation to maintain a state of high awareness to incoming stimuli (Posner & Petersen, 1990; Sturm et al., 1999), supported by lateralized right-hemisphere frontal and posterior parietal cortices, the locus coeruleus, and brainstem, and is primarily reliant on noradrenergic systems (Petersen & Posner, 2012; Posner, 2008; Samuels & Szabadi, 2008; Sturm & Willmes, 2001). The alerting system can be further subdivided into the vigilance and arousal systems. The vigilance system (*tonic alertness*) maintains an individual's trait level of psychophysiological arousal over time. The arousal system (*phasic alertness*) is responsible for increasing alertness following an external cue or warning, characterized by the basic orienting response to changes in the environment (Oken et al., 2006). Interestingly, despite 'difficulties sustaining attention' being included in the diagnostic criteria for ADHD, meta-analytic evidence indicates that ADHD is likely not characterized by dysfunction in tonic alertness (Huang-Pollock & Nigg, 2003). However, adults with ADHD have been shown to have slower response times with longer interstimulus intervals, providing evidence for phasic arousal decrements as a function of increased task duration (e.g., Wiersema et al., 2006).

Top-down attentional control (endogenous orienting).

Endogenous orienting refers to top-down processes involved in voluntarily directing attention toward an object in the environment (Berger et al., 2005; Jonides, 1981; Posner, 1980), supported by anterior cingulate gyrus, supplementary motor cortex, and mid-

prefrontal cortex (Berger & Posner, 2000; Posner & Raichle, 1994). Studies examining attention network components provide the most evidence for dysfunction in endogenous orienting in samples of children and adults with ADHD (d=0.30-1.32) (Nigg et al., 1997; Novak et al., 1995), albeit with inconsistent results across studies (Huang-Pollock & Nigg, 2003). In contrast, the evidence more consistently indicates that exogenous orienting (bottom-up, automatic orienting) is likely intact in ADHD (Aman et al., 1998; Epstein et al., 1997; Huang-Pollock & Nigg, 2003; Perchet et al., 2001).

Disinhibition.

The *executive attention* network encompasses higher-order attentional mechanisms responsible for monitoring, selecting, and resolving conflict between opposing responses (Posner & Petersen, 1990) supported by prefrontal and lateral ventral cortices, anterior cingulate, supplementary motor cortex, and basal ganglia (Botvinick et al., 2001; Fan et al., 2005). Several processes commonly attributed to broader executive functions (EF), such as inhibitory control, are subsumed within this subcomponent of attention (Posner & DiGirolamo, 1998). Of interest in the current study due to its prominence in modern theoretical accounts of ADHD (e.g., Barkley, 1997; Sonuga-Barke et al., 2010), *inhibitory control* refers to a set of interrelated cognitive processes that underlie the ability to withhold (action restraint) or stop (action cancellation) an on-going behavioral response (Alderson et al., 2007; Schachar et al., 2000). Adults with ADHD have demonstrated poorer performance on response inhibition tasks compared to neurotypical adults and adults with anxiety (Epstein et al., 2001). Nevertheless, studies examining differences in inhibition between ADHD, other clinical groups, and healthy control groups have failed to consistently replicate these findings (Lijffijt et al., 2005; Nigg, 2001), with recent meta-analyses concluding that inhibition processes may be generally intact in ADHD (Alderson et al., 2007).

Current Study

The current study is the first longitudinal examination of the extent to which impairments in inhibitory control (disinhibition), top-down attentional processing (endogenous orienting), arousal (phasic alertness), and vigilance (tonic alertness) reflect neurocognitive mechanisms through which ADHD confers risk for crashes, near-crashes, and culpability for crashes/near-crashes during routine driving. We expected higher self-reported ADHD symptoms at study entry to portend increased risk for future adverse driving outcomes. We hypothesized that higher ADHD symptoms would be associated with deficits in top-down attentional control (Nigg et al., 1997) and arousal (i.e., phasic alertness; Wiersema et al., 2006), but not disinhibition (Alderson et al., 2007) or vigilance (i.e., tonic alertness; Huang-Pollock & Nigg, 2003) as measured by the CPT-II (Conners et al., 2000). Finally, we predicted that reduced top-down attentional control and decrements in arousal would reflect neurocognitive mechanisms underlying the link between ADHD symptoms and adverse driving outcomes (i.e., would mediate the link between baseline ADHD symptoms and future crash/near-crash risk; Dingus et al., 2016; Fuermaier et al., 2015) given prior cross-sectional work linking neuropsychological test performance and driving (Barkley, 2004; Lincoln & Radford, 2013).

Method

SHRP-2 Design and Overview

The Strategic Highway Research Program (SHRP-2) Naturalistic Driving Study included 3,600 drivers from six U.S. sites (Bloomington, IN; Central PA; Tampa, FL; Buffalo, NY; Durham, NC; Seattle, WA). Technical reporting of study design and recruitment, probability-based sampling, and methodological plans are summarized by Antin (2011). Sample characteristics and comparisons with the U.S. population of licensed drivers are provided in Antin (2015). Comparisons indicate close approximation to U.S. drivers for the sample. Sample demographics are shown in Table 1, and were controlled in all analyses. The sample was clinically heterogeneous, including 275 drivers with ADHD (n=46 with comorbid anxiety) and 251 drivers with Depression (n=81 comorbid with anxiety) as identified by Aduen et al. (2015). Detailed data reduction methods, software-based trigger algorithms and validation, vehicle sensor calibration, data reductionist training, and reliability are provided in Hankey et al. (2016). Each driver's car was outfitted with five high-speed video cameras, speed/brake monitors, accelerometers, and GPS to continuously capture routine driving from engine-on to engine-off for 1-2 consecutive years (Antin et al., 2011). Participants were protected by a national Certificate of Confidentiality and not required to report crashes or surrender crash-relevant evidence to authorities (Hankey et al., 2016). Upon study entry, drivers completed a battery that included driving history and demographic questionnaires, the Barkley ADHD Quick Screen (BAQS; Barkley et al., 2010), and the Continuous Performance Test (CPT-II; Conners et al., 2000).

Continuous Monitoring and Event Triggers

SHRP-2 collected approximately 2,000,000 gigabytes of data spanning 5,512,900 individual trips that occurred between 2010 and 2013. Software-based trigger algorithms and 100% double-coded manual validation identified 4,254 safety-critical events (SCEs; 1,549 crashes, 2,705 near-crashes). These algorithms used kinematic and behavioral signatures previously identified as present with high probability during crashes (e.g., longitudinal deceleration < −0.65g, lateral acceleration >0.75g) (Hankey et al., 2016). All SCEs were verified by comparing event videos with pre-recorded index images to ensure the consented participant was driving. Manual video review then verified if an SCE occurred. Verified events were coded by extensively trained data reductionists (100% SCEs coded by ≥2 coders; reliability=91%) (Antin et al., 2011; Hankey et al., 2016). Coders completed ~10 days of training, demonstrated 90% proficiency prior to coding, were retested frequently using the same 90% criterion, and completed ≤4 hour shifts (mandatory breaks/hour) to minimize fatigue-related errors. Coders were blind to driver ADHD symptoms and CPT-II performance (Hankey et al., 2016).

Prospective Driving Outcomes

Primary outcomes included number of *crashes* (SCEs involving any contact between participant vehicle and fixed or moving object, at any speed where kinetic energy is measurably transferred or dissipated), *near-crashes* (SCEs requiring rapid, evasive maneuver by participant vehicle to avoid imminent crash), and crash and near-crash *fault* (observable

evidence that the participant driver committed the error leading to the crash/near-crash) recorded during prospective monitoring.

Barkley Adult ADHD Quick Screen (BAQS)

The BAQS is a 6-item self-report questionnaire that assesses ADHD symptom quantity/frequency in adults on a 4-point Likert scale (0=never/rarely, 1=sometimes, 2=often, 3=very often; range=0-18). Psychometric support includes high internal consistency (α=.90) and concurrent validity for self-reported (r=.97) and other-reported (r=.68) ADHD symptoms compared to full, 18-item DSM-based checklists. The BAQS has also demonstrated predictive validity for self-reported (r=.87) and other-reported impairment (r=.67), and high sensitivity (.93) and specificity (.97) for differentiating adults with ADHD from neurotypical adults (cut-off score=7; Barkley et al., 2010). Internal consistency in the current sample was adequate (α =.70).

Demographic Questionnaire and Driving History

Upon enrollment in SHRP-2, participants completed the driving demographic questionnaire, which assessed participant age, sex, educational level, income, and marital status. The driving history questionnaire also assessed vehicle year and estimated annual miles driven.

Conners' Continuous Performance Test, Second Edition (CPT-II)

The CPT-II (Conners et al., 2000) is a 14-minute computerized visual-motor task that requires participants to rapidly and accurately respond to letters that continuously appear on the screen (250 ms presentation rate; 1, 2, or 4 second ISI). Participants respond by pressing the spacebar for every letter ('go' targets) except 'X' ('no-go' targets). When the letter 'X' appears, participants are instructed to interrupt their continuous motor response and inhibit responding. Despite being labeled a continuous performance test, the CPT-II uses a go/no-go paradigm with a high 'go' to 'no-go' (90:10) ratio and manipulations of interstimulus intervals to assess inhibitory control as well as multiple subcomponents of attention (Bytoft et al., 2017; Egeland & Kovalik-Gran, 2010a; Egeland & Kovalik-Gran, 2010b).

The CPT-II has been deemed to possess ecological validity with motor vehicle driving due to the shared monotonous nature of each task (Barkley et al., 2002). Individuals with ADHD have been documented to have poorer CPT-II performance (Barkley, 1991; Biederman et al., 2006; Epstein et al., 2001, 2003). The CPT-II provides 12 outcome measures that were used to derive the study's theoretically-informed attention subcomponents (Table 2). Raw scores are converted to T-scores based on age and sex (national standardization sample N= 2,521; Conners et al., 2000).

CPT-II factor structure.—The current study used a factor analytic approach to derive our primary neurocognitive indices. This method is considered advantageous to approaches that use single markers of performance (e.g., commission errors) because it provides more accurate estimates of higher-order neurocognitive abilities by isolating reliable variance among related variables (e.g., Miyake et al., 2000). The CPT-II's psychometric structure varies across published reports, with evidence supporting three (Conners et al., 2000), four (Conners, 2014), and five factor solutions based on exploratory models (Bytoft et al., 2017;

Egeland & Kovalik-Gran, 2010a). Interestingly, to our knowledge no study to date has successfully replicated a previously published CPT-II factor structure using confirmatory methods despite multiple attempts (e.g., Bytoft et al., 2017; Vertinski et al., 2014). Thus, our data reduction approach began by testing each of the previously reported 3-, 4-, and 5-factor models via confirmatory factor analysis, as well as an *a priori* specified 1-factor model using *MPlus* v7.4 (Muthén & Muthén, 2015). Unfortunately, the 3-, 4, and 5-factor models were all misspecified (i.e., large negative residual variances across factors) and the single-factor model indicated poor fit (e.g., RMSEA=.39, CFI=.301, TLI=.146). Thus, none of the previously published CPT-II factor structures were considered appropriate for use in the current study.

Because none of the previously published confirmatory models demonstrated adequate fit, our next step was to test the factorability of the 12 CPT-II outcome variables. Results provided significant evidence for the factorability of the CPT-II variables (i.e., KMO = 0.64; Bartlett's test of sphericity $\chi^2[66] = 17119.67$, $p < .0001$). We therefore used principal component analysis (PCA) to identify the latent structure across its 12 outcome variables as a prerequisite to using these factors for the current study's primary mediation analyses. This approach was considered advantageous because the current study reflects the largest sample to date to examine the CPT-II's factor structure.

Both theoretical and empirical evidence were considered when deciding on the number of factors to retain: (a) unrotated factors satisfying Kaiser's (1958) criterion of eigenvalues greater than 1.00; (b) accepted configurations accounting for an appreciable percentage of total score variance (i.e., ≥ 50%); (c) solutions meeting Cattell's (1966) scree test; (d) rotated factors each including at least two appreciable factor loadings (i.e., ≥ 0.40; Stevens, 2002); and (e) compatibility of the final solution with theoretical models of the mental processes involved in go/no-go tasks that vary stimulus presentation rates over an extended duration (Huang-Pollock et al., 2012; Soreni et al., 2009).

Results of the varimax-rotated PCA are shown in Table 2 and indicated 4 distinct and theoretically-linked neurocognitive constructs (disinhibition, endogenous orienting deficits, arousal decrements, vigilance decrements) that were saved using the Bartlett method. Bartlett scores use maximum likelihood methods that yield unbiased estimates of true factor scores (DiStefano et al., 2009). The final factor structure was broadly consistent with the updated normative sample factor structure of the CPT-3 (Conners, 2014), with minor exceptions noted in Table 2. However, the factors were labeled to be consistent with terminology implemented in the ADHD and cognitive literatures rather than those used by the Conners CPT.[a]

[a]The first factor was labeled *Disinhibition* rather than Impulsivity to better capture the cognitive construct of response inhibition assessed through commission errors (Congdon et al., 2012; Epstein et al., 2001; van der Meere, 2002). The second factor, characterized as Inattention by the CPT-3 (Conners, 2014), was labeled *Endogenous Orienting Deficits* to specify the subcomponent of attention measured (Berger, Henik, & Rafal, 2005; Posner & Petersen, 1990) and distinguish it from the Vigilance Decrement factor that indexes changes in alertness over time (Huang-Pollock & Nigg, 2003; Woods, Lovejoy, & Ball, 2002). Given that both omission errors and anticipatory responses (Perseverations) were among the variables that loaded onto the Endogenous Orienting Deficits factor, this construct was determined to most accurately describe endogenous as opposed to exogenous orienting (Carter et al., 1995; Huang-Pollock & Nigg, 2003; Swanson et al., 1991). Lastly, the CPT-3 (Conners, 2014) factors of Sustained Attention and Vigilance were re-labeled *Arousal Decrements* and *Vigilance Decrements* in the current study given that sustained attention and vigilance are often used interchangeably in the literature (Oken, Salinsky, & Elsas, 2006). The *Arousal Decrements* (impaired phasic alertness) factor is

Data Analysis Overview

Bias-corrected, bootstrapped mediation (Hayes, 2013; Williams & MacKinnon, 2008) with 10,000 bootstrapped resamples was implemented using PROCESS (Hayes, 2013) to examine the extent to which the risk for future adverse driving outcomes associated with ADHD symptoms is conveyed via neurocognitive impairments in one or more subcomponents of the attention network. Each model included one predictor (self-reported ADHD symptoms: BAQS Score), four neurocognitive parallel mediators (Disinhibition, Endogenous Orienting, Arousal Decrements, Vigilance Decrements) and one outcome variable. Separate models were analyzed for each adverse driving outcome (Crashes, Near-Crashes, Crash/Near-Crash Fault; Figure 1). We then tested exploratory models, substituting the BAQS total scores with separate estimates of ADHD attention problems and then hyperactivity/impulsivity symptoms.

Results

Preliminary Analyses

Missing data ranged from 0.1-0.9% for sex, age, education, and marital status. Education served as a proxy for socioeconomic status; income was not controlled due to high missing data (16.2%). Sample retention was excellent: 3,226 of 3,600 enrolled cases (89.6%) were followed prospectively and included in analyses. Retrospective self-reported and prospectively monitored crashes, near-crashes, and crash fault are reported in Aduen et al., 2015 and 2018, respectively. CPT-II data has not been previously reported for this sample. All CPT-II variables were screened for univariate/multivariate outliers and tested against $p<.001$, resulting in four identified outlier scores that were subsequently recoded as missing. Intercorrelations among study variables are reported in Table 3.

Primary Analyses: Neurocognitive Processes Mediating the Link Between ADHD Symptoms and Future Crash/Near-Crash Risk

All models controlled for age, sex, education, marital status, time in study, and miles driven. Results are organized by pathway (Figure 1). As expected (Aduen et al., 2018), greater ADHD symptoms at study entry portended increased risk for future crashes (c pathways; $\beta=.03$, $p=.001$), near-crashes ($\beta=.04$, $p=.003$), and at-fault crashes/near-crashes ($\beta=.05$; $p=.0001$). Of interest in the current study, greater ADHD symptoms predicted higher levels of Disinhibition (a pathways; $\beta=.03$, $p=.0001$), Endogenous Orienting Deficits ($\beta=.04$, $p<.001$), and Vigilance Decrements ($\beta=.02$, $p=.02$), but not Arousal Decrements ($p=.44$). After accounting for ADHD symptoms, Endogenous Orienting Deficits at study entry predicted future crashes (b pathways; $\beta=.04$, $p=.04$), near-crashes ($\beta= .05$, $p=.04$), and at-fault crashes/near-crashes ($\beta= .07$, $p=.03$), whereas Arousal Decrements predicted number of near-crashes ($\beta= -.06$, $p=.03$). None of the other neurocognitive/driving outcome relations reached significance (all $p>.11$).

consistent with literature investigating arousal deficits in ADHD through manipulation of ISIs (Oken, Salinsky, & Elsas, 2006; Raymaekers et al., 2007; van der Meere, 2005), while the *Vigilance Decrements* (impaired tonic alertness) factor describes fluctuations of attention as a function of time (Cohen, 2014).

Importantly, there was significant evidence for mediation via the Endogenous Orienting Deficits pathway, such that ADHD symptoms at study entry exerted indirect effects on future crash risk (ab pathways; β=.001, Effect Ratio [ER]=.06; 95%CI= .0002-.003), future near-crash risk (β=.002, ER=.05; 95%CI=.0001-.005), and future at-fault crash/near-crash risk (β=.002, ER= .05; 95%CI=.0004-.005) via its association with Endogenous Orienting. No additional indirect effects were detected (all 95%CI include 0.0). After accounting for these effects, self-reported ADHD symptoms continued to predict risk for future crashes (c' pathways; β=.02, p=.002), near-crashes (β=.04, p=.005), and at-fault crashes/near-crashes (β=.05, p=.0001).

Exploratory Analyses

ADHD symptom clusters.—Next, we repeated the primary analyses, this time substituting the overall ADHD symptoms predictor with separate estimates of ADHD attention problems (3 items; α = .64) and then hyperactivity/impulsivity (3 items, α = .60), which were moderately correlated (r=.41, p<.001). Hyperactive/impulsive symptoms were controlled in the attention problems model, and vice versa. Results were generally consistent with the primary analyses, with a more nuanced pattern suggesting that (a) the main effects on driving outcomes were conveyed via attention problems and the indirect effects on driving outcomes were conveyed via hyperactivity/impulsivity, and (b) the relations between ADHD symptoms and Disinhibition were conveyed via the attention problems symptoms, whereas the ADHD/Endogenous Orienting relations were conveyed via hyperactivity/impulsivity symptoms. Neither symptom cluster uniquely predicted Arousal Decrements, suggesting that this finding in the primary analyses was likely conveyed via ADHD symptoms generally rather than a specific symptom subcluster.

Specifically, attention problems exerted direct effects on crashes, near-crashes, and crash/near-crash fault both before and after controlling for the neurocognitive factors (β=.04-.06, p<.004), but did not exert indirect effects via any of the neurocognitive pathways (all 95%CIs include 0.0). In contrast, hyperactivity/impulsivity did not directly predict any of the adverse driving outcomes (β=.004-.03, p>.09), but exerted indirect effects on crashes, near-crashes, and crash-near-crash fault via the Endogenous Orienting pathway (β=.002-.004, 95%CIs exclude 0.0). In terms of the neurocognitive factors, attention problems predicted Disinhibition only (β=.04, p=.009) whereas hyperactivity/impulsivity predicted Endogenous Orienting only (β=.06, p<.0005). Neither ADHD symptom subcluster predicted Arousal Decrements or Vigilance Decrements (β=-.01 to .02, all p>.08). After accounting for each ADHD symptom cluster, Endogenous Orienting Deficits at study entry predicted future crashes (both β=.04, p<.006), near-crashes (both β=.05, p=.04) and at-fault crashes/near-crashes (both β= .07, p=.004), whereas Arousal Decrements predicted number of near-crashes (both β= −.04, p=.04).

Discussion

The present study examined the extent to which vulnerabilities in subcomponents of the attention network reflect neurocognitive pathways through which ADHD symptoms impact future crash risk. Through a dimension reduction approach using performance variables

yielded by the CPT-II (Conners et al., 2000), the role of baseline impairments in specific subcomponents of attention – including top-down attentional control (endogenous orienting deficits), inhibitory control (disinhibition), arousal (phasic alertness) and vigilance (tonic alertness) – were examined in relation to objectively-assessed motor vehicle driving outcomes up to two years later (Riccio et al., 2002; Petersen & Posner, 2012). These attention network subprocesses have been proposed as foundational cognitive mechanisms for both basic and complex aspects of motor vehicle driving (Barkley & Cox, 2007; Posner & Petersen, 1990; Ranney, 1994), but until now had not been linked prospectively with adverse outcomes as they occur during routine, day-to-day driving. Additional strengths of the study include the longitudinal, prospective design; large, nationwide sample; and objective assessment of on-road crashes, near-crashes, and fault for crashes/near-crashes.

Consistent with previous research, ADHD symptoms at study entry prospectively predicted future crashes, near-crashes, and at-fault crashes up to 2 years later, even when controlling for known risk factors including exposure (miles driven, days monitored), age, sex, education, and marital status (Aduen et al., 2018; Cox et al., 2011; Vaa, 2014). These findings continue to position ADHD as an important risk factor for adverse driving outcomes. Interestingly, self-reported ADHD symptoms were significantly related to neurocognitive attentional processes involving less developed inhibitory control, top-down attentional control, and phasic arousal. These findings are generally consistent with prior evidence linking the behavioral sequelae of ADHD with poorer neurocognitive performance (Barkley, 1991; Epstein et al., 2003; Weaver et al., 2009), and extend these findings by providing additional evidence that ADHD symptoms do not appear to be related to deficits in vigilance/sustained attention processes. While these findings are generally consistent with meta-analytic evidence (Huang-Pollock & Nigg, 2003), they contradict clinical observations and DSM-5 criteria that describe sustained attention difficulties as a core symptom of ADHD (APA, 2013).

Of primary interest was the extent to which ADHD behavioral symptoms predicted future driving risk through their association with neurocognitive components of the attention network system. Interestingly, top-down attentional control (endogenous orienting) portended future crashes, near-crashes, and culpability for driving errors that led to crashes/near-crashes, both independently when controlling for ADHD symptoms (b pathways) and via its association with ADHD behavioral symptoms (ab mediation pathways). The consistency of this finding in predicting not only future crashes, but also near-crashes and crash/near-crash fault, suggests that deficits in top-down attentional processing may be a primary contributor to adverse driving outcomes. These findings are consistent with simulator studies showing that attention lapses and off-road glances often precede adverse driving outcomes, predict more driving errors, and interfere with successful avoidance of crashes (Barkley & Cox, 2007; Cox et al., 2004; Jerome et al., 2006). Implications for deficits in top-down attentional control are vast given that it is considered integral to operational aspects of driving performance and may be exacerbated in more complex driving scenarios involving increased passenger, road, and traffic demands (Barkley, 2004). Vulnerabilities in top-down attentional control also increase the potential for increased distractibility to secondary tasks (Reimer et al., 2006, 2007, 2010), which becomes particularly problematic given increased use of cellular telephones and in-vehicle technology

while driving (El Farouki et al., 2014; Kingery et al., 2015; Narad et al., 2013; Reimer et al., 2010). Thus, the current findings indicate that higher levels of voluntary attentional control are critical for actively scanning roadways as a prerequisite to efficiently detecting immediate changes in the environment that require adjustments to vehicle speed/position and/or more rapid evasive maneuvering (Brouwer, 2002).

Conversely, decrements in arousal (phasic alertness) were associated with near-crash risk, even after controlling for self-reported ADHD symptoms. Phasic alertness refers to processes involved in increasing alertness following an external cue or warning (Posner & Peterson, 1990). Managing imminent driving situations is heavily dependent on one's physiological state of alertness (Collet et al., 2005; Mehler et al., 2009), and as such the current findings indicate that individuals with better-developed phasic alertness are more likely to be able to respond to a sudden road hazard/changing conditions and avoid situations in which an imminent crash is likely. Phasic alertness's association with *near-crashes* rather than crashes suggests that higher physiological responsivity to changing driving conditions reduces the risk for an imminent crash (i.e., drivers are able to mitigate the risk earlier and are therefore less likely to engage in the sudden evasive maneuvering that characterizes a near-crash). In this context, it appears that phasic alertness processes provide compensatory functions. For example, visual cues associated with a soon-to-be-imminent crash would be expected to recruit phasic alertness processes to temporarily actuate the underdeveloped alertness system associated with higher ADHD symptoms to reorient and respond quickly enough to avoid a crash becoming imminent. This hypothesis is consistent with evidence that critical driving situations are associated with a physiological response that increases arousal (Collet et al., 2005); thus, this elicited (phasic) change in physiological arousal may provide a window for drivers to successfully maneuver early enough to prevent an imminent crash. A practical implication of these findings, in the context of reduced vigilance and top-down attention control (endogenous orienting) among individuals with elevated ADHD symptoms, is that vehicle-enhanced technologies such as driving alert monitoring systems (a) may be particularly important for reducing risk in these drivers by alerting them to impending danger, and (b) may need to be tailored to be more sensitive to provide earlier warnings given evidence for reduced phasic alerting in ADHD (Wiesema et al., 2006).

Limitations

The present study sought to better understand the cognitive mechanisms that underlie increased risk for adverse driving outcomes in ADHD. In doing so, multiple components of neurocognition were examined as mechanisms through which ADHD symptoms predict increased risk for future criterion-based, objectively-measured crashes, near-crashes, and crash/near-crash culpability. Despite the study's methodological refinements, the following caveats must be considered when interpreting the results. Given the large-scale nature of the study, only one neurocognitive instrument was available to characterize inhibition and attention subprocesses. Although our dimension reduction approach provided superior control for construct-irrelevant variance within the task, the use of multiple tests spaced across multiple testing days would have provided increased control for task- and session-specific error. Thus, although this process provided estimates of theoretically-linked components of the attention network model, several important neurocognitive processes with

links to ADHD symptoms could not be measured (e.g., working memory; Kasper et al., 2012).

In addition, despite demonstrating that subcomponents of attention derived from a 14-minute test predict adverse driving outcomes up to 2 years later, we were unable to assess for more proximal risk factors for crashes, such as *in situ* driver behaviors, cell phones, substance use, or whether medication was metabolically active at the time of a crash. As such our models explained only a minority of the variance in crash/near-crash risk, and the magnitude of the associations between neurocognitive predictors and driving outcomes were fairly small. While these findings suggest that relatively large changes in attentional control would be needed to significantly reduce an individual's crash risk, it is important to contextualize these findings in the context of the longitudinal design. In that context, it is striking that performance on a 14-minute test can predict crash risk two years later, even when controlling for ADHD symptoms that may themselves reflect the behavioral outcome of neurocognitive impairments (Kofler et al., 2018). In other words, associations of this magnitude suggest limited prediction at the individual patient level (i.e., impaired CPT test performance would not be sufficient to revoke an individual's driving privileges) despite having clinically meaningful implications when considered at the public health level. The naturalistic study design precluded multi-informant assessment or clinical interviewing regarding perceived medication efficacy, emergent effects, or timing in relation to crashes, and thus informs relations with self-reported symptoms and medication treatment-as-usual rather than optimal dosing and gold standard psychiatric classification. Finally, although we replicated prior failures to replicate the CPT-II factor structure (Bytoft et al., 2017; Vertinski et al., 2014) using the largest sample to date, the exploratory methods may have dampened associations among neurocognitive predictors and driving outcomes.

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Clinical and Research Implications

Overall, the present results provide evidence that deficits in top-down attentional control (endogenous orienting) mediate the risk for future crashes, near-crashes, and crash/near-crash culpability conveyed by ADHD. The consistency of this finding across driving outcomes highlights the importance of this mechanism for understanding why drivers with higher ADHD symptoms are at increased risk for adverse driving outcomes. This finding creates an important opportunity for clinicians to assess, intervene, and make recommendations based on the specific findings of this task as it relates to driving (e.g., discussing available driver assist technologies in the context of the individual's specific attention network profile). Future studies are warranted to examine *in-situ* driving behaviors to better characterize the inattentive errors that lead to increased crashes (e.g., eyes off road, looking at objects outside/inside the car, adjusting in-vehicle devices, using technology). Determining whether similar behaviors precede crashes across ADHD severity levels will be helpful for developing transdiagnostic and disorder-specific interventions to reduce adverse driving outcomes. Identifying inattention as a key mechanism through which risk is transmitted has significant public health implications for designing effective preventative intervention methods (e.g., driver training, technology-enhanced accommodations, vehicle adaptations). Implementation of these interventions has the potential to reduce the social, financial, health, and legal outcomes associated with motor vehicle collisions for drivers with high incidence disabilities such as ADHD.

Key Points

Question:

Why do drivers with ADHD experience more motor vehicle crashes and near-crashes than their peers?

Findings:

The driving risks associated with ADHD appear to be conveyed in part by impairments in the top-down, voluntary control of attention, rather than to difficulties sustaining attention over time or inhibiting impulses as is often assumed.

Importance:

These findings provide the first longitudinal evidence linking neurocognitive impairments in specific components of the attention network with adverse motor vehicle driving outcomes during routine, on-road driving.

Next Steps:

Investigating additional neuropsychological functions (e.g., working memory) and linking these abilities with the in-car behaviors that immediately proceed crashes/near-crashes will be important for developing harm reduction strategies that improve driving safety for individuals with neurocognitive vulnerabilities.

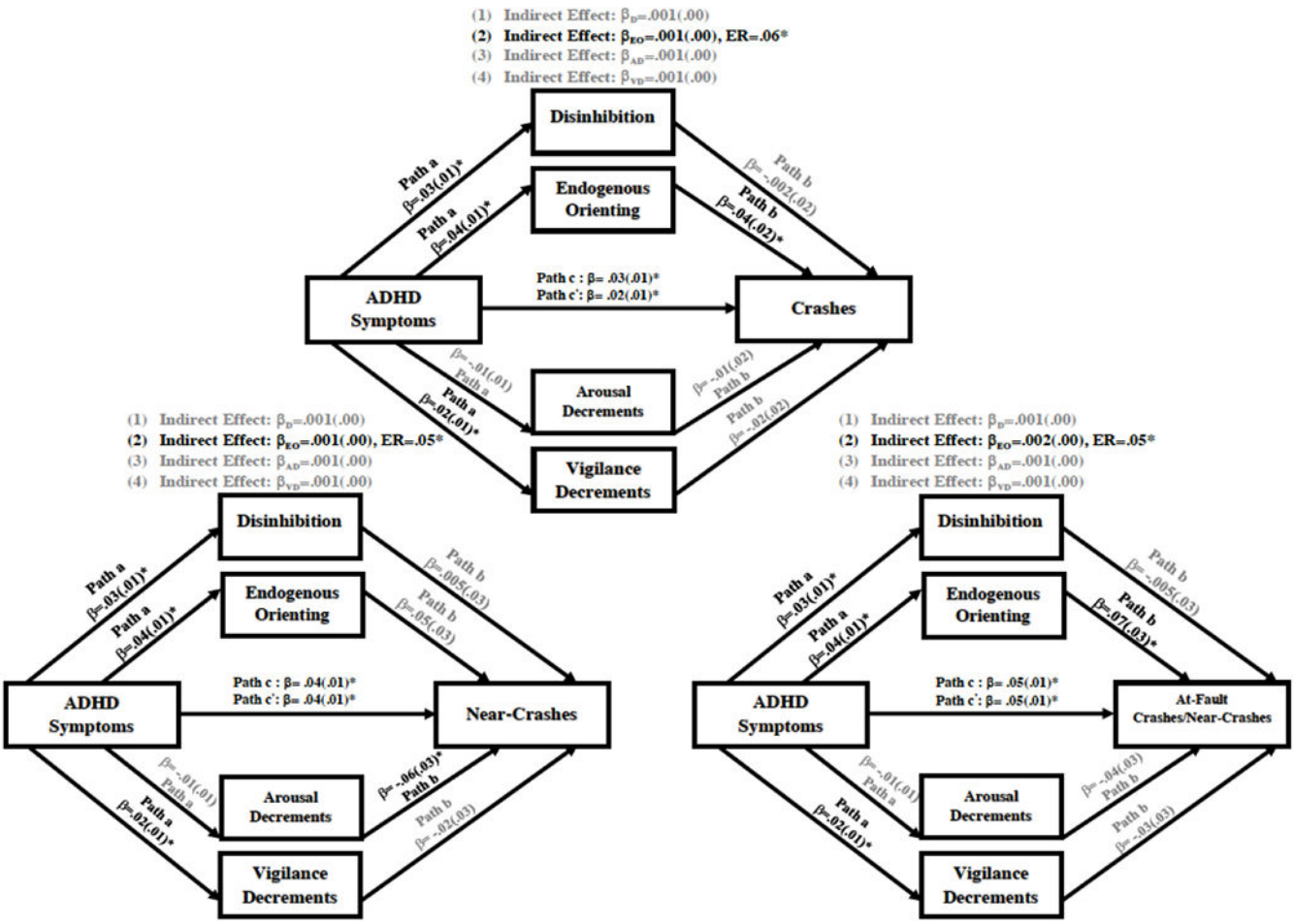

Figure 1.

ADHD Symptoms (BAQS Score) predictor and neurocognitive mediators of crashes, near-crashes, and at-fault crashes/near-crashes. Four neurocognitive mediators, (1) Disinhibition, (2) Endogenous Orienting Deficits, (3) Arousal Decrements, (4) Vigilance Decrements, were uncorrelated by design and tested simultaneously. All models controlled for age, sex, education, marital status, time in study, and miles driven. Significant pathways (*) shown in black font; non-significant *(ns)* pathways shown in grey font. Results are reported as β (SE). ER = Effect Ratio (shown only for significant indirect effects).

Table 1.

Driver demographic data (adapted from Aduen et al., 2018)

	Overall Sample (N=3,226)	
	M	SD
Miles Driven	9527.9	7315.8
Days Monitored	440.49	210.65
BAQS ADHD Score	3.20	2.18
Percent involved in		
No crashes	70.3%	
Single crash	19.9%	
Multiple crashes	9.8%	
No near-crashes	61.7%	
Single near-crash	21.7%	
Multi. near-crashes	16.6%	
Age Group	N	%
16-17	258	8.0
18-20	520	16.1
21-25	597	18.5
26-35	327	10.1
36-50	349	10.8
51-65	383	11.9
66-75	345	10.7
75+	442	13.7
Not reported	5	0.2
Sex		
Male	1537	47.6
Female	1661	51.5
Missing	28	0.9
Education		
Some high school	271	8.4
H.S. graduate	1241	38.5
College degree +	1692	52.4
Not reported	22	0.7
Marital Status		
Not Married	1989	61.7
Married	1207	37.4
Not reported	30	0.9
Annual Income		
Under $29K	556	17.2
$30K to $39K	378	11.7
$50K to $69K	537	16.6
$70K to $99K	551	17.1

	Overall Sample (N=3,226)	
$100K to $149K	462	14.3
$150K or higher	219	6.8
Not reported	523	16.2

Note. BAQS = Barkley Adult ADHD Quick Screen.

Table 2.

Principal component analysis (PCA) results for CPT-II, 4-factor solution, varimax rotation (*N*=3,138)

Factor Loadings					
Variable	**Description**	**Disinhibition**	**Endogenous Orienting Deficits**	**Arousal Decrements**	**Vigilance Decrements**
Commission Errors	Incorrect responses to non-targets (i.e., letter X)	**.92**	.17	−.04	.09
Detectability (D')	Discrimination between non-targets and targets	**.90**	.17	−.04	.06
HRT	Response speed for non-perseverative responses (ms)	**−.77**	.40	.21	.04
Beta	Speed-versus-accuracy trade-off	**−.53**	.41	−.09	−.05
Variability	Within-respondent response speed consistency	−.06	**.79**	.23	.18
HRT SE	Response speed consistency across duration of test	−.29	**.77**	.41	.11
Perseverations	Responses made in less than 100 milliseconds	.14	**.67**	−.10	.12
Omission Errors	Missed targets (i.e., non-Xs)	.03	**.63**	−.11	−.22
HRT ISI Change	Changes in reaction time as a function of changes across 1, 2, and 4 second ISIs	−.03	.06	**.84**	−.03
HSE ISI Change	Change in response consistency as a function of changes across 1, 2, and 4 second ISIs	−.06	−.01	**.84**	.06
HRT Block Change	Change in reaction time across duration of test	.04	.04	−.01	**.86**
HRT SE Block Change	Changes in response consistency across duration of test	.07	.04	.05	**.86**
	Eigenvalue	3.18	2.33	1.54	1.36
	% of variance accounted	26.47	19.38	12.81	11.29

Note: Factor loadings ≥ .45 are bolded; HRT= Hit Reaction Time; SE= Standard Error; ISI= Interstimulus Interval. The final factor structure was broadly consistent with the updated normative sample factor structure of the CPT-3 (Conners, 2014), with minor exceptions: Detectability (*d'*) loaded onto the Disinhibition (Impulsivity; CPT-3) rather than the Endogenous Orienting (Inattention; CPT-3) factor, and Perseverations loaded onto the Endogenous Orienting (Inattention; CPT-3) rather than the Disinhibition (Impulsivity; CPT-3) factor. Lastly, Response Style (Beta) loaded onto the Disinhibition factor in the current study but was not included in the CPT-3 (Conners, 2014). Factors loadings within the CPT-3's Sustained Attention and Vigilance factors were consistent with our Vigilance Decrement (HRT Block Change, HRT SE Block Change) and Arousal Decrement (e.g., HRT ISI, HSE ISI) factor loadings, with the exception that the CPT-3 factors included additional variables that index changes in Omissions and Commissions by Block and ISI (not provided by the CPT-II output).

Table 3.

Partial correlations (correcting for covariates including age, sex, education, marital status, time in study, and miles driven)

		1	2	3	4	5	6	7	8	9	10
1	Disinhibition	1									
2	Endogenous Orienting Deficits	.061**	1								
3	Arousal Decrements	.015	−.012	1							
4	Vigilance Decrements	−.004	.000	.004	1						
5	Crash Count	.004	.043*	−.024	−.013	1					
6	Near-Crash Count	.008	.034	−.014	−.040*	.246**	1				
7	Fault Count	.004	.045*	−.014	−.025	.737**	.732**	1			
8	Crash Fault Count	.002	.038*	−.021	−.009	.958**	.231**	.748**	1		
9	Near-Crash Fault Count	.005	.034	−.004	−.029	.291**	.874**	.843**	.274**	1	
10	BAQS Score	.070**	.086**	.043*	−.014	.058**	.054**	.072**	.061**	.055**	1

Note.

*=p< .05,

**=p<.001.