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Driving risks of young drivers with symptoms of attention deficit hyperactivity disorder: association with the dopamine transporter gene VNTR polymorphism

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Abstract

Background
Road traffic injuries are a leading cause of death for young adults, and young drivers with higher expression of symptoms of attention deficit-hyperactivity disorder (ADHD) could pose an even greater risk in traffic. Dopaminergic dysfunction has been found to occur in ADHD, with the dopamine transporter (DAT) gene VNTR polymorphism (DAT1 VNTR; rs28363170) being one of the most consistent genetic markers. Thus, we aimed at clarifying how the ADHD symptoms and the DAT1 VNTR relate to risk-taking behaviour in traffic, impulsivity and driving anger in young drivers.

Method
We used data of two traffic behaviour study samples (n = 741, mean age = 23.3±7.2 years; n = 995, mean age = 22.9±8.1 years) and the Estonian Children Personality Behaviour and Health Study (ECPBHS; traffic behaviour data n = 1016, mean age = 25.2±2.1 years). ADHD symptoms were assessed by self-report with the Adult ADHD Self-Report Scale (ASRS v1.1) and impulsivity with the Adaptive and Maladaptive Impulsivity Scale. Traffic behavioural measures were either self-reported (Driver Behaviour Questionnaire, Driving Anger Scale) or obtained from databases (registered accidents and violations).

Results
Drivers with more self-reported ADHD symptoms also reported more risk-taking in traffic and had more of recorded traffic accidents and violations. DAT1 9R carriers had a higher probability of high traffic risk behaviour only if they also had ADHD symptoms.

Conclusion
Higher level of ADHD symptoms is a significant risk factor in traffic, and carrying of the DAT1 9R allele appears to aggravate these risks.
Keywords

ADHD; DAT1 VNTR; traffic behaviour; impulsivity

Introduction

Road traffic injuries are a leading cause of death for children and young adults aged 5–29 years (WHO, 2018). While pedestrians, cyclists and motorcyclists are most vulnerable, young novice drivers also belong to the highest traffic injury risk group. A variety of risk factors are related to traffic injuries, such as age, gender, driving experience, and a few personality measures (e.g. impulsivity and aggressiveness) (Burtäverde et al., 2017; McCartt et al., 2003; Regev et al., 2018; Paaver et al., 2006).

Attention deficit/hyperactivity disorder (ADHD) is associated with traffic accidents and violations, and the reported risk has been up to 4 times higher as compared with controls (Barkley et al., 1993; Barkley et al., 2002,; Brunkhorst-Kanaan et al., 2021; Fried et al., 2006; Kittel-Schneider et al., 2019; Vaa, 2014). ADHD is a neurodevelopmental disorder, characterized by a persistent and developmentally inappropriate level of inattention and/or hyperactivity and impulsivity, resulting in functional impairment (Buitelaar et al., 2011). With one of the core ADHD symptom domains being hyperactivity/impulsivity (DSM 5), it might seem obvious that ADHD drivers are more at risk due to their increased impulsivity, but previous research has also shown a relationship between ADHD symptoms and driving anger: adults with ADHD symptoms seem to express their emotions in more aggressive ways, which might be caused by lack of emotion control (King and Waschbusch, 2010; Ramirez et al., 1997). Symptoms of ADHD are not exclusively present in patients, but are dimensional, and exist as continuous measures within any population (Mulligan et al., 2009) and interfere with social functioning (Faraone and Larsson, 2019). ADHD as expressed at clinical level
and as subsyndromal are genetically strongly linked with each other, according to both twin studies and genome-wide analyses (Demontis et al., 2019; Larsson et al., 2012; Levy et al., 1997). Furthermore, symptoms of ADHD at the population level are in similar relationship with broad personality traits as in ADHD patients (Li et al., 2019). All this suggests that ADHD symptoms in general population could represent heightened traffic risk. Nevertheless, this possibility has not been studied directly, and what is known can be derived only from studies on diagnosed ADHD patients.

In terms of behaviour specific to traffic, ADHD drivers have been found to differ from controls in multiple ways. First, ADHD drivers seem to drive more than controls (Vaa, 2014). Second, ADHD drivers have more speeding violations, but generally no more drunk or reckless driving citations than drivers without ADHD (Vaa, 2014). Higher rates of alcohol and drug related traffic violations have been found in adolescents with ADHD in their first year of driving (Curry et al., 2019). There is also a distinction between driving errors and deliberate violations: The former are more known to be associated with accidents (Fuermaier et al., 2017; Vaa, 2014). ADHD drivers would have excess of both kinds of violations depending on their dominant symptoms. Driving errors are caused mainly by inattention that may lead to late detection of critical situations and by this means an increase in near-crashes/crashes, while deliberate violations would rather be produced by increased hyperactivity/impulsivity resulting in unsafe manoeuvres and speeding (Fuermaier et al., 2017).

Of the neural substrates of impulsive behaviour, the dopaminergic system has received much attention (e.g., Dalley and Roiser, 2012), and dopaminergic dysfunction indeed occurs also in people with attention-deficit/hyperactivity disorder (ADHD) (Ludolph et al., 2008; Thapar et
Altered dopamine transporter function is the most consistently observed neurochemical characteristic of ADHD (Krause et al., 2006). The dopamine transporter plays a critical role in terminating the effect of synaptically released dopamine by taking it up into neurons (Chen and Reith, 2000). Dopamine transporter is encoded by the DAT1 gene (SLC6A3) that bears a variable number of tandem repeats (VNTR) polymorphism (rs28363170) of a 40-base pair sequence in the 3′-untranslated region of the gene (Costa et al., 2011). It has been shown that the DAT1 VNTR 9-repeat (9R) allele carriers have higher striatal dopamine transporter availability than do the 10-repeat (10R) allele homozygotes (Faraone et al., 2014; Van De Giessen et al., 2009). The increased levels of DAT might lead to more efficient clearing of extracellular dopamine, resulting in lower extracellular levels and reduced dopamine signalling (Faraone et al., 2014). Being a 9R carrier has been associated with persistent ADHD in adults (Franke et al., 2012, 2010). This genotype can also be associated with traffic risks: We have previously found that male 9R carriers had more accidents in traffic by their own fault (Tokko et al., 2019) and, in another sample, a higher general traffic risk (Luht et al., 2019).

The aim of this study was to clarify how self-reported symptoms of ADHD are related to risk-taking behaviour in traffic, impulsivity and driving anger in young drivers, whether the possible association of ADHD symptoms and traffic behaviour is related to the DAT1 VNTR, and whether the presence of ADHD symptoms might affect the success of impulsivity-related intervention.
Methods

Samples/participants

Traffic study samples

We used two samples of the Estonian Psychobiological Study of Traffic Behaviour (EPSTB) that had self-reported ADHD symptom data available for some of the participants. Both samples comprised driving school students applying for a passenger car driving license and were from studies that had included an intervention arm with focus at the acknowledgement of the impulsivity factor in traffic. From the earlier study of the two, herewith labelled Traffic Study I (Eensoo et al., 2018; Paaver et al., 2013) a total of 741 (40% of the original sample, mean age=23.3±7.2 years) filled in the ADHD Self-report scale and were included in the present analysis. From the second intervention study, here labelled Traffic Study II (Luht et al., 2019) 995 (69% of the original sample, mean age=22.9±8.1 years) filled in the adult ADHD Self-report scale and were included in analysis.

Estonian Children Personality Behaviour and Health Study (ECPBHS)

The rationale and procedure of sample formation have been described elsewhere in detail (Harro et al., 2001; Joost et al., 2019; Luht et al., 2018; Tomson et al., 2011). In the current analysis, data of both birth cohorts of ECPBHS obtained at age 25 years were used. For subjects (~6%) who did not fill in respective questionnaires at age 25, data collected at age 33 years were used, if available. Altogether 1016 ECPBHS participants (82% of the original sample, mean age=25.2±2.1 years) were included in analyses.

Ethical standards

All procedures in EPSTB and ECPBHS have been approved by the Research Ethics Committee of the University of Tartu. All procedures contributing to this work complied with
the ethical standards of the relevant national and institutional authorities on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

Questionnaires

**ASRS v1.1**

The World Health Organization recommended questionnaire for ADHD screening, ASRS v1.1 (Kessler et al., 2005) was used. The ASRS v1.1 is an 18-item checklist that assesses symptoms of ADHD in adults. Subjects completed the full version of the ASRS indicating the frequency of symptom occurrence in the past 6 months: 0 (never), 1 (rarely), 2 (sometimes), 3 (often) or 4 (very often). Nine items measure inattention symptoms and 9 measure hyperactivity/impulsivity symptoms. The screening score of the ASRS, known as the ASRS v1.1 short form, is formed by counting the results of the six most predictive questions if reported at least “sometimes” (for questions 1-3) or “often” (for questions 4-6). In case 4 of those 6 symptoms are reported to be present at the described level, then the screener is considered positive (Kessler et al., 2005). In the present study, subjects were categorized by ADHD-related risks based on three indicators: 1) ADHD screener score ≥4 (ADHD screener positive) vs <4 (ADHD screener negative); 2) Hyperactivity/impulsivity low vs high (subscale score by 50th percentile); 3) Inattention low vs high (subscale score by 50th percentile).

**Driver Behaviour Questionnaire**

The Driver Behaviour Questionnaire (DBQ) (Lawton et al., 1997; Reason et al., 1990), previously shown to predict traffic accidents (De Winter and Dodou, 2010), was filled in at least 3 months after recruitment of the participants. The questionnaire is made up of 28 statements, which measure the frequency of different risky behaviours of drivers on a scale from 0 (never) to 5 (almost always). Reason and co-authors (Reason et al., 1990) have
demonstrated that driver errors and violations are two empirically distinct classes of behaviour. They defined errors as the failure of planned actions to achieve their intended consequences (e.g., not noticing traffic signs, choosing the wrong traffic lane), and violations as deliberate deviations from those practices believed necessary to maintain the safe operation of a potentially hazardous system (e.g., speeding, drunk driving). In this sample principal component analysis (PCA) of the DBQ showed two distinct classes of behaviour (driver errors and violations; Cronbach’s alpha 0.83 and 0.82 accordingly) that together explained 52% of the total variability (Eensoo et al., 2020). Driver errors factor (17 items, Cronbach’s alpha 0.83) had an eigenvalue of 11.9 and accounted for 30% of the variance in the data, and driver violations factor (11 items, Cronbach’s alpha 0.82) had an eigenvalue of 7.2 and accounted for a further of 22% of the variance. The lowest loadings of included items were 0.54 in either subscale (Errors - "Intending to drive to destination A, you “wake up” to find yourself on the road to destination B, perhaps because the latter is your more usual destination (2)"; Violations - "Become angered by another driver and give chase with the intention of giving him/her a piece of your mind"). PCA separated the the items of the two subscales entirely and the correlation between the subscale scores was r=0.47 that was statistically highly significant (p<0.001) but moderate in size. While all participants in the Traffic Studies were Estonian, there were Russian speaking subjects in ECPBHS sample who filled in the Russian version of the DBQ (13%, n = 106). Therefore z-scores were calculated for the questionnaire in both languages. Low and high scorers were separated by the 50th percentile cut-off value.

**Driving Anger Scale**

The Driving Anger Scale (Deffenbacher et al., 1994) was filled in by subjects on the web at least 3 months after intervention at the traffic school. It includes 33 potentially angering traffic situations and queries how much anger does each provoke on a scale from 0 to 4 (0 =
none at all, 1 = a little, 2 = some, 3 = much, 4 = very much). The Cronbach’s alpha for the scale was 0.93. In the analyses, z-scores were used.

**Impulsivity measures**

The Adaptive and Maladaptive Impulsivity Scale (AMIS) was used to measure different facets of impulsivity (fast decision making, thoughtlessness, disinhibition, excitement seeking) as previously described (Laas et al., 2010). AMIS is based on the concept of functional and dysfunctional impulsivity as described by Dickman (Dickman, 1990). Subjects were asked to assess how much the 24 impulsivity-related statements applied to them on a scale from 1 to 5.

**Mileage**

Subjects were also asked to report their mileage per previous year in kilometres. A 10,000 km/per year cut-off was used for categorization of driving activity.

**Databases**

Traffic insurance and police databases were used to obtain data about traffic collisions and traffic violations of the participants of traffic studies for the respective three-year period after the traffic school intervention. In addition, we formed a general traffic risk index (high traffic risk – occurrence of either a recorded traffic violation or a collision; low traffic risk – no recorded traffic violation or collision).

**Genotyping**

The \(DAT1\) \((SLC6A3)\) VNTR was genotyped following the analytical method by Anchordoquy et al. (2003) as previously described (Maksimov et al., 2015). Polymorphic regions were amplified using the primer rs28363170F: 5’/56-FAM/TGT GGT GTA GGG AAC GGC CTG AG 3’ and rs28363170R: 5’ CTT CCT GGA GGT CAC GGC TCA AGG 3’ for \(DAT1\) 3’UTR VNTR. The VNTR repeat numbers range from 6 to 11, with 9- and 10-repeat alleles being the most common. Genotype frequencies were in the Hardy–Weinberg
equilibrium. We compared the 9-repeat carriers (9R/9R and 9R/10R) and 10-repeat (10R/10R) homozygotes; subjects who had a rare VNTR genotype (10R/11R, 6R/10R) were excluded from the analysis. The number of subjects included was as follows: 1) Traffic Study I, n = 339 (9R – 38.1%, 10R/10R – 61.9%) 2) Traffic Study II, n = 917 (9R – 38.8%, 10R/10R – 61.2%) 3) ECPBHS, n = 1000 (9R – 37.0%, 10R/10R – 63.0%).

Statistical analysis

Data were analysed using SPSS (version 23.0 SPSS, Chicago, IL) and SAS (version 9.4 SAS Inc., Cary, NC) software. The Traffic Studies I and II were largely similar by design, so the data were first analysed pooled to increase statistical power, and then separately to examine reproducibility. Differences between groups regarding categorical variables were analysed with Pearson’s chi-square test and the post-hoc Fisher test, and for continuous variables with ANOVA. Logistic regression analyses were used for predicting high traffic risk and high violations scores. First, simple logistic regression analyses were conducted with each independent variable, and next, independent variables were adjusted by the ADHD screening score (high ADHD risk vs low ADHD risk), hyperactivity/impulsivity subscale score (high vs low) and inattention subscale score (high vs low). The p < 0.05 level was considered as statistically significant.

Results

ADHD symptoms and traffic behaviour

The proportion of subjects with positive ADHD screener was, by sample, as follows: Traffic Study I, 8.2% (n = 61); Traffic Study II, 18.4% (n = 183); Traffic Studies combined, 14% (n = 244); ECPBHS, 9.5% (n = 97). The mean scores of the ASRS were 13.4 (S.D. 4.9) and
11.5 (5.1); for inattention and hyperactivity/impulsivity, respectively, in the Traffic Studies I & II combined sample, and in ECPBHS, 12.8 (4.8) and 10.9 (5.1), respectively. Data on socio-demographic variables, impulsivity and driving anger measures, traffic behaviour and DAT1 VNTR in Traffic Studies I & II combined are presented in Table 1 and for the ECPBHS, in Table 2. Data of the Traffic Studies I and II are separately presented in Supplementary Tables 1 and 2, respectively.

Subjects in all of the ADHD screener positive/high symptom groups of the Traffic Studies sample were statistically significantly younger (Table 1). The ECPBHS sample was older than the Traffic Studies combined sample \( F (2, 2732) = 40.1, p < 0.001 \) and there was no age difference between the low and high ADHD symptom groups (Table 2). The gender balance was similar for the ADHD symptom groups in all samples.

In the Traffic Studies, subjects with positive ADHD screener or high inattention were more frequently in the high mileage group. This was not found in the ECPBHS sample where the proportion on higher mileage drivers was larger.

Subjects in positive ADHD screener group and high ADHD symptom groups had significantly higher scores in most of the impulsivity measures in all samples, except fast decision making, which was significantly lower in subjects with high inattention. The high inattention and low fast decision making association was significant in all samples.

Higher DBQ Violations and DBQ Errors scores were present in ADHD screener positive/high symptom drivers. Only in the ECPBHS sample did the ADHD screener
positives not significantly differ from the ADHD screener negatives by the DBQ Violations score.

We examined how many subjects with high self-reported DBQ Violations in the Traffic Studies samples had actual violations recorded by the traffic police. Of the subjects with a high DBQ Violations score (Traffic Studies I & II, n = 475), 24.8% had at least one actual traffic violation, whereas of the subjects with a low DBQ Violations score (Traffic Studies I & II, n = 575), only 10.8% had actual traffic violations ($\chi^2 = 36.2$, $p < 0.001$). So, a considerable number of high scorers in DBQ Violations also had a higher proportion of actual violations in traffic in a three-year period. Traffic accidents were also more related to DBQ Violations: 16.0% of high scorers vs 10.6% of low scorers had actual traffic accidents ($\chi^2 = 6.7$, $p = 0.01$). As to the DBQ Errors score and traffic accidents, similar trends were not significant: 14.8% of high scorers (n = 508) had had at least one accident vs 11.4% of low scorers (n = 542), and the proportion of violators was 19.1% vs 15.3%.

When comparing Driving Anger Scale scores between groups, ADHD screener positives, high hyperactivity/impulsivity and high inattention subjects in both samples had significantly higher scores in driving anger compared with the respective low scorers (Tables 1 and 2). Significant differences in traffic accidents, violations and traffic risk were found only between the hyperactivity/impulsivity groups, with a significantly higher prevalence of traffic violations and high traffic risk in the high hyperactivity/impulsivity group (Table 1). There were no differences in prevalence of the DAT1 VNTR alleles between the ADHD groups in any of the samples.

Predicting traffic risk and DBQ Violations
**Traffic Studies I & II**

Significant predictors of high traffic risk in the Traffic Studies combined sample were as follows: younger age, male gender, higher mileage, higher fast decision making or excitement seeking score, higher DBQ Violations score and higher hyperactivity/impulsivity (Table 3; Supplementary Tables 3 and 5). After adjustment with ADHD screener, hyperactivity/impulsivity and inattention, most of the predictors remained significant, but a higher driving anger and DBQ Errors score appeared as significant predictors only after adjustment with the screening score and inattention. In addition we adjusted one of the significant results in the focus of this study (hyperactivity/impulsivity) by mileage to see if this effect is independent of mileage, but the association remained significant: higher hyperactivity/impulsivity (OR = 1.52 (95% CI: 1.13, 2.04)).

Significant predictors for high scores in DBQ Violations in the Traffic Studies sample were as follows: younger age, male gender, higher mileage, higher scores in all impulsivity measures, higher driving anger score, higher DBQ Errors score, ADHD screener positive, higher hyperactivity/impulsivity and higher inattention (Table 4; Supplementary Tables 4 and 6). After adjustment with ADHD screener, hyperactivity/impulsivity and inattention, all of the predictors remained the same. In addition, we adjusted all statistically significant associations in the focus of this study (ADHD screener, hyperactivity/impulsivity and inattention) by mileage to ascertain that the effects were not directly owing to higher of mileage. All of these associations remained significant: ADHD screener positive (OR = 1.14 (95% CI: 1.09, 1.18)); higher hyperactivity/impulsivity (OR = 1.84 (95% CI: 1.42, 2.39)); higher inattention (OR = 1.71 (95% CI: 1.32, 2.23)).
Supplementary Tables (3 and 5) also include the predictive effect of the intervention in the traffic schools owing to methodological differences and large baseline differences. While the first intervention study significantly reduced speeding and drunk driving (Eensoo et al., 2018; Paaver et al., 2013), the intervention effect on the general traffic risk was not significant (Supplementary Table 3) with the given number of participants for whom all necessary data were available. However, in Traffic Study II (Luht et al., 2019), the intervention was predictive of high traffic risk, and the effect remained statistically significant after adjustment for ADHD screener, hyperactivity/impulsivity and inattention (Supplementary Table 5).

**ECPBHS**

Significant predictors for high scores in DBQ Violations in the ECPBHS sample were male gender, higher mileage, higher scores in all impulsivity measures, higher driving anger score, higher DBQ Errors score and higher hyperactivity/impulsivity (Table 5). After adjustment for ADHD screener, hyperactivity/impulsivity and inattention, all of the significant predictors remained significant. In addition, we adjusted the statistically significant association that is in the focus of this study (hyperactivity/impulsivity) by mileage; as in the other sample, the association remained a significant predictor: higher hyperactivity/impulsivity (OR = 2.12 (95% CI: 1.56, 2.87)).

**DAT1 VNTR genotype, ADHD symptoms and high traffic risk**

*DAT1* VNTR allele prevalence was similar in the ADHD symptom groups. However, a significantly higher proportion of *DAT1* 9R carriers was found in the Traffic Studies combined sample among subjects with co-occurring high traffic risk and ADHD screener positive (Fig. 1A, $\chi^2 = 5.3, p = 0.022$) or high hyperactivity/impulsivity (Fig. 1B, $\chi^2 = 6.2, p = $
0.013) or high inattention (Fig. 1C, $\chi^2 = 7.0$, $p = 0.008$). Considering the two traffic study samples separately, the genotype prevalence effect was statistically significant in the larger sample of Traffic Study II ($\chi^2 = 3.8$, $p = 0.05$; 7.6, $p = 0.006$ and $\chi^2 = 4.1$, $p = 0.04$ for comparison by ADHD screener, hyperactivity/impulsivity and inattention, respectively), but similar trends were present in Traffic Study I (Supplementary Figures 1, 2 and 3).

**Discussion**

Taking advantage of three population-derived samples with information about ADHD symptoms self-reported using the WHO-recommended instrument, we have examined the predictors of risks in traffic behaviour. Furthermore, we could analyse the impact of ADHD symptoms on objective outcomes, and examine the contribution of the DAT1 VNTR. The summary data of three different samples ($n = 2752$) revealed that there were approximately 12% of drivers with a score considered positive in ASRS screening (Kessler et al., 2005).

In all samples we could observe similarities in the groups with higher self-reported ADHD symptoms, such as error-prone driving, more frequent traffic law violations and high driving anger. Thus, previous association of ADHD with traffic accidents and violations (Barkley et al., 2002, 1993; Fried et al., 2006; Kittel-Schneider et al., 2019; Vaa, 2014) could be extended to ADHD symptoms below the clinical level. Further, high traffic risk (accidents + violations) was in particular associated with high hyperactivity/impulsivity, and especially owing to link to traffic violations, confirming the suggested particular association between increased hyperactivity/impulsivity and deliberate violations (Fuermaier et al., 2017).

A major difference between drivers with positive ADHD screener in the traffic studies and in the ECPBHS was that in the former sample the mileage driven past year was lower in the
ADHD screener positives. A meta-analysis has shown ADHD drivers driving more than controls (Vaa, 2014). Specifically, it was high inattention and not at all hyperactivity/impulsivity that was associated with lower mileage in our study. (It should be noted that the ASRS screening score has 4 inattention items and only 2 hyperactivity/impulsivity items.) The ECPBHS sample had higher overall driving experience that may explain the disappearance of the possible difference at the stage of limited background in driving. However, it appears that self-reported ADHD symptoms, at least in early years, is not characterized by extensive driving.

Higher mileage and gender were the most significant predictors of high traffic risk and DBQ violations. Therefore, we also checked if the associations of ADHD symptoms with high traffic risk and DBQ violations were directly owing to higher mileage, but after adjustments with mileage in regressions all the associations with ADHD symptom groups remained significant. This indicates that even though higher mileage was very much predictive of traffic risks, higher expression of self-perceived ADHD symptoms makes an independent contribution.

We expected drivers with self-reported ADHD symptoms to have both higher adaptive and maladaptive impulsivity. Indeed, the components of maladaptive impulsivity, thoughtlessness and disinhibition, as well as excitement seeking, an aspect of adaptive impulsivity, were higher in ADHD screener positives. However, another component of adaptive impulsivity, fast decision making, tended to be higher only in subjects with high hyperactivity/impulsivity, but instead lower in subjects with high inattention. It is of course adaptive to hold back fast decision making impulses if a tendency to be inattentive is present.
Almost all positive screener, high hyperactivity/impulsivity and high inattention groups in both samples had significantly higher scores in driving anger, showing that in addition to impulsive behaviour, some of the resulting behaviour might be due to lack of emotion control (King and Waschbusch, 2010; Ramirez et al., 1997).

Being a \textit{DAT1} 9R allele carrier has been associated with higher striatal dopamine transporter availability (Faraone et al., 2014; Van De Giessen et al., 2009) and adult ADHD (Franke et al., 2012). We did not observe any surplus of 9R in the ADHD screener positives, but the genotype together with positive screener presented an additive effect to traffic risk. This seems also in line with our previous smaller study on a different sample where 9R carriers had more accidents in traffic through their own fault (Tokko et al., 2019). It might be that the drivers with self-reported ADHD symptoms and the risk-allele of the dopamine transporter gene are more prone to traffic offences and/or collisions. An effect of \textit{DAT1} on cortical activation within a group of adult ADHD patients has been previously found (Dresler et al., 2010), more specifically a pattern of reduced NGA (NoGo anteriorization – topographical ERP parameter) in 9R allele carriers. \textit{DAT1} has been assumed to have a crucial role in regulating the cortical signal-to-noise ratio. First, it appears to have a direct effect through its influence on prefrontal pyramidal neurons through regulation of DA volume transmission on the surrounding GABA-inhibitory neurons. Second, it influences the cortical signal-to-noise ratio indirectly through effects in the striatum, which regulates activity within the cortico-striatal-thalamo-cortical pathway (Bertolino et al., 2006; Mattay et al., 2002; Newman and Grace, 1999). In addition, young people with ADHD and the \textit{DAT1} 10R/10R genotype have been shown to have significantly greater inhibitory control-related activation in the left striatum, right dorsal premotor cortex, and bilaterally in the temporoparietal cortical junction compared to 9R allele carriers, providing additional evidence that neural activity related to
inhibitory control may differ as a function of DAT1 genotype in subjects with ADHD (Bédard, et al., 2010). Recently Brown et al. (2017) found that also in healthy individuals DAT1 can alter the neural basis of emotional processing and response inhibition in go/no-go task, with 9R carriers having increased neural activation compared to 10R/10R homozygotes during emotional response inhibition, which could indicate that 9R carriers put forth more effort when inhibiting responses in emotional contexts (Brown et al., 2017). But 9R carriers still had the same accuracy and reaction time as 10R/10R homozygotes. Considering these previous findings, the risk genotype might be additive to ADHD tendencies. In our study the increased driving anger of ADHD screener positives and the result that there are more 9R carriers among those with positive screener and high traffic risk might show the resulting dysfunctional behaviour of 9R carriers with ADHD symptoms in failing to control their anger in everyday high risk traffic behaviour.

ADHD drivers are often not aware of their higher impulsivity and tend to overestimate their driving skills (Fabiano et al., 2018). Whether a driver with ADHD is aware of the condition or not, and is taking the necessary medication or using some behavioural strategies instead, is also something to consider. In male ADHD patients, medication has been associated with a 58% risk reduction (Chang et al., 2014) and we have previously shown that brief interventions in driving schools, guided by the affective neuroscience concept (Montag and Panksepp, 2017; Panksepp, 1998) and focusing on impulsive traffic behaviour, can have a long-term positive effect on traffic behaviour (Eensoo et al., 2018; Luht et al., 2019; Paaver et al., 2013) in terms of reducing speed limit exceeding, drunk driving occurrence of accidents and lowering general traffic risk. In the present analysis of the Traffic Study II data, the intervention effect did remain significant even for ADHD screener positives (Supplementary Table 5) while we had to use smaller sample size because of the limited
number of subjects who filled in the ASRS questionnaire. It appears as beneficial to include in the drivers’ training a discussion of the risks of ADHD and other related mental health issues and the threats they bring about in traffic.

The main strengths of this study were the possibility to take advantage of three population-derived samples with information about self-reported ADHD symptoms, and the impact of these symptoms on objective outcomes for two of the samples and the possibility to examine the contribution of the DAT1 VNTR. As to the limitations in the current study, much of the previous work on ADHD and traffic behaviour has been done on patients (Barkley et al., 1993; Barkley et al., 2002; Fried et al., 2006; Kittel-Schneider et al., 2019; Vaa, 2014), and it would have been useful to have information about any eventual clinical diagnoses, and also medication usage in our subjects. Whether the results of the present study are applicable to a patient group or not could not be studied. According to a recent analysis the world average adult ADHD prevalence is around 2.8% (Fayyad et al., 2017), so most likely the majority of our subjects in the positive screener group do not have actual ADHD. Another limitation was the use of self-reported data about traffic behaviour. While we could validate some of the results in the Traffic Studies sample by data about recorded accidents and violations, we did not have such data for the ECPBHS sample. Nevertheless, the finding that DBQ Violations does associate with the objective record of traffic violations while DBQ Errors does not lends some additional credibility to this scale, and to the proposed two-factor solution of DBQ.

In conclusion, having self-perceived ADHD symptoms is a significant risk factor in traffic, and carrying the DAT1 9R allele appears to aggravate these risks. Self-reported ADHD symptoms do however not impair efficacy of prevention of risk-taking driving behaviour.
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Declaration of conflicting interests

The authors declare that they have no conflicts of interest.

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Supplemental material

Supplementary data accompanying this paper: Supplementary tables 1 – 6, Supplementary figures 1-3.

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https://doi.org/10.1080/00140139008925335


https://doi.org/10.1016/j.jsr.2018.07.002


https://doi.org/10.1093/hmg/ddi263

https://doi.org/10.1016/j.pnpbp.2011.08.004

https://doi.org/10.1016/j.aap.2013.10.003

https://doi.org/10.2967/jnumed.108.053652

Table 1. Demographic and traffic behaviour variables by self-reported ADHD symptom levels in the Traffic Studies I & II combined.

<table>
<thead>
<tr>
<th>Variable</th>
<th>ADHD screener negative</th>
<th>ADHD screener positive</th>
<th>Low hyperactivity/impulsivity</th>
<th>High hyperactivity/impulsivity</th>
<th>Low inattention</th>
<th>High inattention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>23.3 (7.9)</td>
<td>21.1 (6.1)***</td>
<td>23.5 (8.0)</td>
<td>22.5 (7.4)***</td>
<td>23.6 (8.2)</td>
<td>22.3 (7.1)***</td>
</tr>
<tr>
<td>Gender, male % (n)</td>
<td>36.5 (545)</td>
<td>37.7 (92)</td>
<td>36.0 (339)</td>
<td>37.5 (298)</td>
<td>37.4 (367)</td>
<td>35.7 (270)</td>
</tr>
<tr>
<td>Mileage driven past year (n=988) &gt;10000 % (n)</td>
<td>18.9 (163)</td>
<td>8.7 (11)**</td>
<td>17.8 (96)</td>
<td>17.4 (78)</td>
<td>20.8 (119)</td>
<td>13.2 (55)**</td>
</tr>
<tr>
<td>Fast decision making (n=1717), mean (SD)</td>
<td>18.0 (4.2)</td>
<td>17.3 (4.6)*</td>
<td>17.7 (4.3)</td>
<td>18.1 (4.3)*</td>
<td>18.5 (4.1)</td>
<td>17.2 (4.4)**</td>
</tr>
<tr>
<td>Excitement seeking (n=1717), mean (SD)</td>
<td>18.7 (5.0)</td>
<td>20.4 (5.1)**</td>
<td>18.2 (4.9)</td>
<td>19.8 (4.9)**</td>
<td>18.5 (5.0)</td>
<td>19.5 (5.0)**</td>
</tr>
<tr>
<td>Thoughtlessness (n=1717), mean (SD)</td>
<td>15.0 (4.5)</td>
<td>18.0 (4.8)**</td>
<td>14.2 (4.4)</td>
<td>16.8 (4.6)**</td>
<td>14.3 (4.4)</td>
<td>16.8 (4.7)**</td>
</tr>
<tr>
<td>Disinhibition (n=1717), mean (SD)</td>
<td>17.0 (4.2)</td>
<td>19.8 (4.3)**</td>
<td>16.2 (4.3)</td>
<td>18.7 (4.0)**</td>
<td>16.2 (4.2)</td>
<td>18.9 (4.1)**</td>
</tr>
<tr>
<td>DBQ Violations (n=1050), z score mean (SD)</td>
<td>-0.03 (1.0)</td>
<td>0.2 (1.1)**</td>
<td>-0.2 (0.8)</td>
<td>0.2 (1.1)**</td>
<td>-0.1 (1.0)</td>
<td>0.1 (1.0)**</td>
</tr>
<tr>
<td>DBQ Errors (n=1050), z score mean (SD)</td>
<td>-0.1 (1.0)</td>
<td>0.4 (1.1)**</td>
<td>-0.2 (0.9)</td>
<td>0.3 (1.1)**</td>
<td>-0.2 (1.0)</td>
<td>0.2 (1.0)**</td>
</tr>
<tr>
<td>Driving Anger Scale (n=1028), z score mean (SD)</td>
<td>-0.1 (1.0)</td>
<td>0.4 (1.0)**</td>
<td>-0.2 (1.0)</td>
<td>0.2 (1.0)**</td>
<td>-0.1 (1.0)</td>
<td>0.2 (1.0)**</td>
</tr>
<tr>
<td>Traffic accidents, % (n)</td>
<td>10.9 (162)</td>
<td>9.4 (23)</td>
<td>10.2 (96)</td>
<td>11.2 (89)</td>
<td>11.5 (113)</td>
<td>9.5 (72)</td>
</tr>
<tr>
<td>Traffic violations, % (n)</td>
<td>14.6 (218)</td>
<td>18.0 (44)</td>
<td>12.0 (113)</td>
<td>18.8 (149)**</td>
<td>15.5 (152)</td>
<td>14.6 (110)</td>
</tr>
<tr>
<td>High traffic risk, % (n)</td>
<td>22.1 (330)</td>
<td>23.0 (56)</td>
<td>19.0 (179)</td>
<td>26.1 (207)**</td>
<td>23.3 (228)</td>
<td>20.9 (158)</td>
</tr>
<tr>
<td>DAT1 (n=1256), 9R allele carriers % (n)</td>
<td>39.2 (416)</td>
<td>35.6 (69)</td>
<td>39.5 (273)</td>
<td>37.6 (212)</td>
<td>39.0 (273)</td>
<td>38.1 (212)</td>
</tr>
</tbody>
</table>

n in brackets in the variable column is the number of subjects with all valid data, presented only for variables with data not available for all included subjects (n = 1736); * p < 0.05, ** p < 0.01, *** p < 0.001 statistically significant difference from the respective negative/low ADHD symptom group.
Table 2. Demographic and traffic behaviour variables by self-reported ADHD symptom levels in the Estonian Children Personality Behaviour and Health Study sample.

<table>
<thead>
<tr>
<th></th>
<th>ADHD screener negative</th>
<th>ADHD screener positive</th>
<th>Low hyperactivity/impulsivity</th>
<th>High hyperactivity/impulsivity</th>
<th>Low inattention</th>
<th>High inattention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD)</td>
<td>25.2 (2.1)</td>
<td>25.3 (2.1)</td>
<td>25.3 (2.1)</td>
<td>25.2 (2.0)</td>
<td>25.3 (2.2)</td>
<td>25.2 (2.0)</td>
</tr>
<tr>
<td>Gender, male, % (n)</td>
<td>42.7 (392)</td>
<td>44.3 (43)</td>
<td>40.5 (214)</td>
<td>45.3 (221)</td>
<td>44.3 (264)</td>
<td>40.7 (171)</td>
</tr>
<tr>
<td>Mileage driven past year (% (n)</td>
<td>42.1 (289)</td>
<td>40.0 (28)</td>
<td>40.7 (164)</td>
<td>43.3 (153)</td>
<td>44.4 (200)</td>
<td>38.2 (117)</td>
</tr>
<tr>
<td>Fast decision making (mean (SD))</td>
<td>18.7 (4.5)</td>
<td>17.5 (5.6)*</td>
<td>18.3 (4.3)</td>
<td>18.8 (4.9)</td>
<td>19.3 (4.3)</td>
<td>17.5 (4.9)**</td>
</tr>
<tr>
<td>Excitement seeking (mean (SD))</td>
<td>20.0 (4.8)</td>
<td>20.9 (5.2)</td>
<td>19.5 (4.7)</td>
<td>20.7 (4.9)**</td>
<td>20.3 (4.7)</td>
<td>19.8 (5.0)</td>
</tr>
<tr>
<td>Thoughtlessness (mean (SD))</td>
<td>15.8 (4.6)</td>
<td>18.9 (5.0)**</td>
<td>14.7 (4.5)</td>
<td>17.6 (4.5)**</td>
<td>15.2 (4.6)</td>
<td>17.4 (4.7)**</td>
</tr>
<tr>
<td>Disinhibition (mean (SD))</td>
<td>17.0 (4.1)</td>
<td>19.9 (4.1)**</td>
<td>16.0 (4.0)</td>
<td>18.7 (3.9)**</td>
<td>16.1 (3.9)</td>
<td>18.9 (4.0)**</td>
</tr>
<tr>
<td>DBQ Violations (z score mean (SD))</td>
<td>-0.02 (1.0)</td>
<td>0.2 (1.2)</td>
<td>-0.2 (0.9)</td>
<td>0.2 (1.1)**</td>
<td>-1.0 (0.9)</td>
<td>0.1 (1.1)**</td>
</tr>
<tr>
<td>DBQ Errors (z score mean (SD))</td>
<td>-0.03 (1.0)</td>
<td>0.3 (1.1)**</td>
<td>-0.2 (0.9)</td>
<td>0.2 (1.1)**</td>
<td>-0.2 (0.9)</td>
<td>0.3 (1.0)**</td>
</tr>
<tr>
<td>Driving Anger Scale (z score mean (SD))</td>
<td>-0.02 (1.0)</td>
<td>0.2 (1.2)*</td>
<td>-0.1 (1.0)</td>
<td>0.1 (1.0)**</td>
<td>-0.1 (1.0)</td>
<td>0.1 (1.0)**</td>
</tr>
<tr>
<td>DAT1 (9R allele carriers % (n))</td>
<td>37.1% (335)</td>
<td>36.5% (35)</td>
<td>38.5% (200)</td>
<td>35.4% (170)</td>
<td>35.3% (208)</td>
<td>39.4% (162)</td>
</tr>
</tbody>
</table>

n in brackets in the variable column is the number of subjects with all valid data, presented only for variables with data not available for all included subjects (n = 1016); * p < 0.05, ** p < 0.01, *** p < 0.001 statistically significant difference from the respective negative/low ADHD symptom group.
Table 3. Predictors of high traffic risk in the Traffic Studies I & II combined.

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.98 (0.96 – 0.99)</td>
<td>0.98 (0.96 – 0.99)</td>
<td>0.98 (0.96 – 1.00)</td>
<td>0.98 (0.96 – 0.99)</td>
</tr>
<tr>
<td>Gender, male vs female</td>
<td>2.84 (2.26 – 3.59)</td>
<td>2.84 (2.26 – 3.58)</td>
<td>2.85 (2.26 – 3.59)</td>
<td>2.84 (2.25 – 3.58)</td>
</tr>
<tr>
<td>Mileage driven past year &gt; 10 000 km vs less (n=988)</td>
<td>3.85 (2.73 – 5.42)</td>
<td>3.89 (2.74 – 5.46)</td>
<td>3.91 (2.77 – 5.52)</td>
<td>3.81 (2.70 – 5.37)</td>
</tr>
<tr>
<td>Fast decision making (n=1717)</td>
<td>1.06 (1.03 – 1.09)</td>
<td>1.06 (1.03 – 1.09)</td>
<td>1.06 (1.03 – 1.09)</td>
<td>1.06 (1.03 – 1.09)</td>
</tr>
<tr>
<td>Excitement seeking (n=1717)</td>
<td>1.06 (1.03 – 1.08)</td>
<td>1.06 (1.03 – 1.08)</td>
<td>1.05 (1.03 – 1.08)</td>
<td>1.06 (1.04 – 1.09)</td>
</tr>
<tr>
<td>Thoughtlessness (n=1717)</td>
<td>1.02 (1.00 – 1.05)</td>
<td>1.02 (1.00 – 1.05)</td>
<td>1.01 (0.98 – 1.03)</td>
<td>1.03 (1.00 – 1.05)</td>
</tr>
<tr>
<td>Disinhibition (n=1717)</td>
<td>1.00 (0.98 – 1.03)</td>
<td>1.00 (0.98 – 1.03)</td>
<td>0.99 (0.96 – 1.02)</td>
<td>1.01 (0.98 – 1.04)</td>
</tr>
<tr>
<td>Driving Anger Scale (n=1028)</td>
<td>1.01 (1.00 – 1.02)</td>
<td>1.01 (1.00 – 1.02)</td>
<td>1.01 (0.99 – 1.01)</td>
<td>1.01 (1.00 – 1.02)</td>
</tr>
<tr>
<td>DBQ Violations, high vs low (n=1050)</td>
<td>2.36 (1.77 – 3.13)</td>
<td>2.40 (1.80 – 3.19)</td>
<td>2.30 (1.73 – 3.06)</td>
<td>2.46 (1.85 – 3.27)</td>
</tr>
<tr>
<td>DBQ Errors, high vs low (n=1050)</td>
<td>1.32 (1.00 – 1.74)</td>
<td>1.34 (1.01 – 1.78)</td>
<td>1.26 (0.95 – 1.67)</td>
<td>1.39 (1.50 – 1.85)</td>
</tr>
<tr>
<td>ADHD screener, positive vs negative</td>
<td>1.05 (0.76 – 1.45)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ADHD hyperactivity/impulsivity, high vs low</td>
<td>1.50 (1.20 – 1.89)</td>
<td>-</td>
<td>-</td>
<td>1.66 (1.31 – 2.12)</td>
</tr>
<tr>
<td>ADHD inattention, high vs low</td>
<td>0.87 (0.69 – 1.10)</td>
<td>-</td>
<td>0.73 (0.58 – 0.94)</td>
<td>-</td>
</tr>
</tbody>
</table>

n in brackets in the variable column is the number of subjects with all valid data, presented only for variables with data not available for all included subjects (n = 1736); 1 adjusted for ADHD screener 0 vs 1; 2 adjusted for hyperactivity/impulsivity 50th percentile high vs low; 3 adjusted for inattention 50th percentile high vs low. Bold - significant predictor; odds ratio (OR) with 95 percent confidence intervals (CI).
Table 4. Predictors of high DBQ Violations scores in the Traffic Studies I & II combined.

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.94 (0.92 – 0.96)</td>
<td>0.94 (0.92 – 0.96)</td>
<td>0.94 (0.92 – 0.96)</td>
<td>0.94 (0.92 – 0.96)</td>
</tr>
<tr>
<td>Gender, male vs female</td>
<td>1.71 (1.32 – 2.20)</td>
<td>1.72 (1.33 – 2.22)</td>
<td>1.74 (1.35 – 2.25)</td>
<td>1.73 (1.34 – 2.24)</td>
</tr>
<tr>
<td>Mileage driven past year &gt; 10 000 km vs less (n=968)</td>
<td>2.46 (1.75 – 3.46)</td>
<td>2.61 (1.85 – 3.68)</td>
<td>2.52 (1.78 – 3.56)</td>
<td>2.68 (1.89 – 3.80)</td>
</tr>
<tr>
<td>Fast decision making (n=1041)</td>
<td>1.04 (1.01 – 1.07)</td>
<td>1.05 (1.02 – 1.08)</td>
<td>1.04 (1.01 – 1.07)</td>
<td>1.06 (1.02 – 1.09)</td>
</tr>
<tr>
<td>Excitement seeking (n=1041)</td>
<td>1.08 (1.06 – 1.11)</td>
<td>1.08 (1.05 – 1.11)</td>
<td>1.08 (1.05 – 1.11)</td>
<td>1.08 (1.05 – 1.11)</td>
</tr>
<tr>
<td>Thoughtlessness (n=1041)</td>
<td>1.04 (1.02 – 1.07)</td>
<td>1.03 (1.01 – 1.06)</td>
<td>1.03 (1.00 – 1.06)</td>
<td>1.03 (1.01 – 1.06)</td>
</tr>
<tr>
<td>Disinhibition (n=1041)</td>
<td>1.07 (1.04 – 1.10)</td>
<td>1.06 (1.03 – 1.10)</td>
<td>1.06 (1.03 – 1.09)</td>
<td>1.06 (1.03 – 1.09)</td>
</tr>
<tr>
<td>Driving Anger Scale (n=1019)</td>
<td>1.04 (1.03 – 1.04)</td>
<td>1.03 (1.03 – 1.04)</td>
<td>1.03 (1.03 – 1.04)</td>
<td>1.03 (1.03 – 1.04)</td>
</tr>
<tr>
<td>DBQ Errors, high vs low</td>
<td>3.52 (2.73 – 4.54)</td>
<td>3.41 (2.64 – 4.41)</td>
<td>3.30 (2.55 – 4.27)</td>
<td>3.37 (2.60 – 4.36)</td>
</tr>
<tr>
<td>ADHD screener, positive vs negative</td>
<td>1.88 (1.30 – 2.71)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ADHD hyperactivity/impulsivity, high vs low</td>
<td>1.81 (1.41 – 2.31)</td>
<td>-</td>
<td>-</td>
<td>1.65 (1.28 – 2.13)</td>
</tr>
<tr>
<td>ADHD inattention, high vs low</td>
<td>1.58 (1.24 – 2.03)</td>
<td>-</td>
<td>1.37 (1.06 – 1.77)</td>
<td>-</td>
</tr>
</tbody>
</table>

n in brackets in the variable column is the number of subjects with all valid data, presented only for variables with data not available for all included subjects (n = 1050); ^1 adjusted for ADHD screener 0 vs 1; ^2 adjusted for hyperactivity/ impulsivity 50th percentile high vs low; ^3 adjusted for inattention 50th percentile high vs low. Bold - significant predictor; odds ratio (OR) with 95 percent confidence intervals (CI).
Table 5. Predictors of high DBQ Violations scores in the Estonian Children Personality Behaviour and Health Study sample.

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
<th>Adjusted OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.96 (0.90 – 1.03)</td>
<td>0.96 (0.90 – 1.03)</td>
<td>0.97 (0.90 – 1.04)</td>
<td>0.96 (0.90 – 1.03)</td>
</tr>
<tr>
<td>Gender, male vs female</td>
<td>3.47 (2.60 – 4.64)</td>
<td>3.47 (2.60 – 4.64)</td>
<td>3.55 (2.64 – 4.76)</td>
<td>3.54 (2.65 – 4.75)</td>
</tr>
<tr>
<td>Mileage driven past year &gt; 10 000 km vs less(n=736)</td>
<td>2.78 (2.05 – 3.77)</td>
<td>2.79 (2.06 – 3.78)</td>
<td>2.83 (2.08 – 3.86)</td>
<td>2.88 (2.12 – 3.91)</td>
</tr>
<tr>
<td>Fast decision making (n=810)</td>
<td>1.07 (1.04 – 1.10)</td>
<td>1.07 (1.04 – 1.10)</td>
<td>1.07 (1.03 – 1.10)</td>
<td>1.08 (1.04 – 1.11)</td>
</tr>
<tr>
<td>Excitement seeking (n=810)</td>
<td>1.14 (1.10 – 1.18)</td>
<td>1.14 (1.10 – 1.18)</td>
<td>1.14 (1.10 – 1.17)</td>
<td>1.14 (1.10 – 1.18)</td>
</tr>
<tr>
<td>Thoughtlessness (n=810)</td>
<td>1.05 (1.02 – 1.08)</td>
<td>1.05 (1.02 – 1.08)</td>
<td>1.03 (1.00 – 1.07)</td>
<td>1.05 (1.02 – 1.08)</td>
</tr>
<tr>
<td>Disinhibition (n=810)</td>
<td>1.06 (1.03 – 1.10)</td>
<td>1.07 (1.03 – 1.10)</td>
<td>1.04 (1.01 – 1.08)</td>
<td>1.06 (1.02 – 1.10)</td>
</tr>
<tr>
<td>Driving Anger Scale (n=796)</td>
<td>1.02 (1.02 – 1.03)</td>
<td>1.02 (1.02 – 1.03)</td>
<td>1.02 (1.02 – 1.03)</td>
<td>1.02 (1.02 – 1.03)</td>
</tr>
<tr>
<td>DBQ Errors, high vs low</td>
<td>6.70 (4.92 – 9.11)</td>
<td>6.83 (5.00 – 9.32)</td>
<td>6.35 (4.65 – 8.67)</td>
<td>7.08 (5.14 – 9.76)</td>
</tr>
<tr>
<td>ADHD screener, positive vs negative</td>
<td>1.06 (0.67 – 1.68)</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>ADHD hyperactivity/impulsivity, high vs low</td>
<td>1.84 (1.39 – 2.43)</td>
<td>-</td>
<td>-</td>
<td>1.84 (1.36 – 2.49)</td>
</tr>
<tr>
<td>ADHD inattention, high vs low</td>
<td>1.27 (0.96 – 1.68)</td>
<td>-</td>
<td>1.00 (0.73 – 1.36)</td>
<td>-</td>
</tr>
</tbody>
</table>

n in brackets in the variable column is the number of subjects with all valid data, presented only for variables with data not available for all included subjects (n = 812); ¹ adjusted for ADHD screener 0 vs 1; ² adjusted for hyperactivity/ impulsivity 50th percentile high vs low; ³ adjusted for inattention 50th percentile high vs low. Bold - significant predictor; odds ratio (OR) with 95 percent confidence intervals (CI).
Figure Legends:

Figure 1. Prevalence of high traffic risk by self-reported ADHD measures and DAT1 VNTR genotype.
Subjects with high traffic risk - occurrence of accident and/or violation, n = 261 (21% of all the participants); (A) ADHD screener, (B) hyperactivity/impulsivity, (C) inattention.

Supplementary Figure 1. Prevalence of high traffic risk by ADHD screener and DAT1 VNTR genotype. High traffic risk - occurrence of accident and/or violation; (A) Traffic Study I, (B) Traffic Study II.

Supplementary Figure 2. Prevalence of high traffic risk by hyperactivity/impulsivity and DAT1 VNTR genotype. High traffic risk - occurrence of accident and/or violation; (A) Traffic Study I, (B) Traffic Study II.

Supplementary Figure 3. Prevalence of high traffic risk by inattention and DAT1 VNTR genotype. High traffic risk - occurrence of accident and/or violation; (A) Traffic Study I, (B) Traffic Study II.