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UNIVERSITY OF TARTU
Press
1632

Institute of Psychology, University of Tartu, Estonia

The dissertation has been accepted for the commencement of the degree of Doctor of Philosophy (in Psychology) on April 3, 2023 by the Council of the Institute of Psychology, University of Tartu.

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Commencement: June 1, 2023 at 16.00, in Näituse 2, Tartu

This work was supported by the Estonian Research Council [grant number PRG1213], and also received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 667302 (CoCa) and No 728018 (Eat2beNICE).

ISSN 1024-3291 (print)
ISBN 978-9916-27-183-4 (print)
ISSN 2806-2531 (pdf)
ISBN 978-9916-27-184-1 (pdf)

University of Tartu Press
www.tyk.ee

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LIST OF ORIGINAL PUBLICATIONS

Paper I: Tokko, T., Eensoo, D., & Harro, J. (2023). Driving anger dimensions as predictors of dangerous situations in traffic. In: Martin, C., Preedy, V. R., Patel, V. B. (eds) Handbook of Anger, Aggression, and Violence. Springer, Cham. https://doi.org/10.1007/978-3-030-98711-4_60-1

Paper II: Tokko, T., Eensoo, D., Vaht, M., Lesch, K. P., Reif, A., & Harro, J. (2019). Relapse of drunk driving and association with traffic accidents, alcohol-related problems and biomarkers of impulsivity. *Acta Neuropsychiatrica,* 31(2), 84–92.

Paper III: Tokko, T., Miškinyte, G., Eensoo, D., & Harro, J. (2022). Driving risks of young drivers with symptoms of attention deficit hyperactivity disorder: association with the dopamine transporter gene VNTR polymorphism. *Nordic Journal of Psychiatry*, 76(8), 575–583.

Paper IV: Tokko, T., Eensoo, D., Luht-Kallas, K., & Harro, J. (2022). Risk-taking in traffic is associated with unhealthy lifestyle: Contribution of aggressiveness and the serotonin transporter genotype. *Neuroscience Applied*, 1, 100110.

Paper V: Luht, K., Tokko, T., Eensoo, D., Vaht, M., & Harro, J. (2019). Efficacy of intervention at traffic schools reducing impulsive action, and association with candidate gene variants. *Acta Neuropsychiatrica*, 31(3), 159–166.

Contribution of the author

For **Paper I** author of the dissertation conducted literature search for the narrative review, carried out data analysis, and wrote the review chapter as the main author in cooperation with other authors. For **Papers II–IV** author of the dissertation formulated research hypotheses, conducted the data analysis, and wrote the manuscript as the main author in cooperation with other authors. For **Paper V** the author participated in formulating the research question, data analysis, and writing the paper in cooperation with other authors. The author of the dissertation also participated in the EPSTB and ECPBHS data collection during the years of his PhD studies.

ABBREVIATIONS

5-HTTLPR	–	serotonin transporter gene-linked polymorphic region
ADHD	–	attention deficit hyperactivity disorder
AMIS	–	Adaptive and Maladaptive Impulsivity Scale
ASRS	–	Adult ADHD Self Report Scale
AUDIT	–	Alcohol Use Disorders Identification Test
BMI	–	body mass index
BPAQ	–	Buss-Perry Aggression Questionnaire
CFI	–	Comparative Fit Index
DAS	–	Driving Anger Scale
DAT1	–	dopamine transporter gene
DBQ	–	Driver Behaviour Questionnaire
DWI	–	driving while impaired by alcohol
ECPBHS	–	Estonian Children Personality Behaviour and Health Study
EPSTB	–	Estonian Psychobiological Study of Traffic Behaviour
NPSR1	–	Neuropeptide S receptor gene
PPPE	–	problematic practice of physical exercise
RMSEA	–	root mean square error of approximation
SNP	–	single nucleotide polymorphism
TLI	–	Tucker Lewis index
VNTR	–	variable number of tandem repeats

1. INTRODUCTION AND REVIEW OF LITERATURE

1.1. Public health issues of traffic behaviour

More than 1 million lives are lost and approximately 50 million are injured each year due to road traffic injuries worldwide. Road traffic injuries are a leading cause of death for children and young adults aged 5–29 years (WHO, 2018). Besides enormous personal suffering for victims and their families, the impact is financial as well and is estimated to cost countries ~3% of annual gross domestic product. Pedestrians, cyclists, and motorcyclists are most vulnerable in traffic, whereas car/bus/truck drivers propose the biggest threat. Factors such as road design and vehicle safety do play a critical role in crash prevention and injury reduction (WHO, 2018), but behaviour of drivers (e.g., excessive speed, not wearing a seatbelt, using a mobile phone while driving) contributes to 90–95% of motor vehicle crashes and to the magnitude of the consequences. A variety of risk factors are related to increased traffic risk/injuries, such as age, gender, driving experience, and personality measures (e.g., impulsivity and aggressiveness) (Antić et al., 2018; Biçaksiz & Özkan, 2016; Burtăverde et al., 2017; Constantinou et al., 2011; McCartt et al., 2003; Paaver et al., 2006; Regev et al., 2018).

1.2. Risk-taking behaviour in traffic

There are multiple possible risk-taking behaviours in traffic – speeding, tailgating, not coming to stop at a stop sign, drunk driving, not using safety equipment (e.g., seat belt), being distracted while driving (using a mobile phone) etc. In this thesis, speeding, drunk driving, as well as traffic accidents are in focus, as speeding and drunk driving are two of the most significant risk-taking behaviours in traffic, and it is possible to obtain objective data (from police/traffic insurance databases) about these violations and accidents.

1.2.1. Speeding

Speeding in traffic has been shown to contribute significantly to the risk of traffic injury, and not only to the increased likelihood of being in an accident due to higher speed and less time for reacting; if a crash does occur, higher speed brings about larger impact, being therefore an important contributor to injury severity (Elvik, 2012). Every 1% increase in mean speed produces a 4% increase in the fatal crash risk and a 3% increase in the serious crash risk, and the death risk for pedestrians hit by front of the car rises rapidly (4.5 times from 50 km/h to 65 km/h) (WHO, 2022).

1.2.2. Drunk driving

Driving while impaired by alcohol (DWI), also known as drunk driving, is among the key behavioural risk factors in traffic that increases the risk of accidents with severe injuries (Hels et al., 2013). With alcohol consumption as a prerequisite for DWI, it is not surprising that drunk drivers have been shown to have higher rates of alcohol use disorders (Lapham et al., 2001). Recklessness in traffic is further associated with drunk driving, as engaging in different types of risky driving (e.g., speeding, distracted and fatigued driving, using the phone while driving) is more common in people with a history of DWI violation (Brown et al., 2020; Li et al., 2013).

1.2.3. Safety equipment usage

The behaviour of drivers encompasses using or not using safety equipment while driving. While there are numerous options for increasing drivers' safety with newer car models (e.g., traction control, brake assist, lane departure and blind spot warning) one of the most important safety measures is still using a seatbelt. Wearing a seatbelt reduces the risk of death among drivers and front seat occupants by 45–50%, and the risk of death and serious injuries among rear seat occupants by 25% (WHO, 2022).

1.2.4. Mobile phones and driving

Being in traffic situation, which is already a high-risk environment, is especially sensitive to distractions. With a second of not looking at the road equalling to ~14 m travelled at 50 km/h and 25 m at 90 km/h, distractions can have severe consequences. Using mobile phones while driving slows reaction times, makes it difficult to keep in the correct lane and keep the correct distance and leads to approximately 4 times increased odds of being involved in a crash, as compared to drivers not using a mobile phone (WHO, 2022).

1.3. Personality factors in traffic behaviour

1.3.1. Impulsivity

Impulsivity (or impulsiveness) has been defined as "unconscious risk taking" (Eysenck, 1993), "acting without thinking" (Barratt, 1994) or "inclining to act on impulse rather than thought" (American Heritage Dictionary, 2022). Regardless of the exact definition, it is generally agreed that impulsivity in traffic situations can be very dangerous due to the riskier environment in comparison with impulsivity in other aspects of our daily lives. Therefore, impulsivity is a widely studied personality factor in risky driving behaviour (Biçaksiz and Özkan, 2016).

Impulsive behaviour could be beneficial in some situations, but it can be dangerous and inappropriate in others. Dickman (1990) differentiated functional (adaptive) and dysfunctional (maladaptive) impulsivity, first is the willingness and ability to take risks in situations where it is beneficial, and the second is the tendency for thoughtlessness, inability to plan and leading to negative consequences. The concept of functional and dysfunctional impulsivity applied to traffic suggests that different types of violations may be associated with distinct aspects of impulsivity. For example, speeding has been found to be associated more with adaptive impulsivity, whereas DWI has mostly been associated with maladaptive impulsivity (Eensoo et al., 2004; Paaver et al., 2006).

1.3.2. Anger and aggressiveness

In addition to impulsivity, anger and aggressiveness are important constructs of potentially dangerous traffic behaviour. Higher anger drivers are more aggressive in their driving behaviour (Deffenbacher et al., 2016) and increased risk for crash and injury of high anger drivers and those affected by them has also been shown (Dahlen et al., 2005; Deffenbacher et al., 2003; Fei et al., 2019).

1.3.2.1. Trait anger and driving anger

Anger is included in personality inventories as a major independent component (e.g., Davis and Panksepp, 2011) or a facet of a main factor (e.g., angry hostility in the Big Five; Lui et al., 2022). Trait anger predicts the expression of angry states and aggression because people with high trait anger have the tendency to interpret situations as hostile and are less capable of keeping their hostile feelings and thoughts under control (Veenstra et al., 2018). If trait anger reflects a broad predisposition to anger across situations, then driving anger has been defined as more frequent and intense anger while operating a motor vehicle. Deffenbacher et al. (1994) suggested an analogy to anxiety, in which case context-specific anxieties are in correlation with overall trait anxiety but are more predictive of emotional responses and behaviours within corresponding specific contexts. Another, related term for aggressiveness in traffic is road rage, defined as “a motorist’s uncontrolled anger that is usually provoked by another motorist’s irritating act and is expressed in aggressive or violent behaviour” (Merriam-Webster, 2022).

1.4. Background factors affecting traffic behaviour

1.4.1. Cultural and socioeconomic differences

More than 90% of road traffic deaths occur in low- and middle-income countries, and even within high-income countries, people from lower socioeconomic backgrounds are more likely to be involved in road traffic crashes (WHO, 2022).

There are several relevant differences in traffic culture between developed and developing countries. Developed countries have advanced traffic culture supported by better roads, higher quality vehicles, well established and enforced traffic laws, and higher socio-economic status of the drivers that contributes to general well-being and better mental health. However, several studies have identified socio-economic stratification as a traffic risk factor within a single country, in case of both generally affluent and developing countries. For example, increased risk of crash-related hospitalisation of young drivers from areas with low socio-economic index compared to drivers from areas with high socioeconomic index (independent of driving exposure) has been reported (Chen et al., 2010) and lower socioeconomic status has been associated with increased incidence and mortality rate of traffic accidents (Sehat et al., 2012).

1.4.2. Age and gender

Younger age and male sex have been consistently found to be higher risk indicators in traffic behaviour (Ouimet et al., 2015; Regev et al., 2018). Traits such as impulsivity and disinhibition that are associated with high risk in traffic have been found to peak among young male drivers (Constantinou et al., 2011) and driving anger has been shown to be a stronger predictor of risky driving among young drivers (Herrero-Fernández, 2011; Li et al., 2014; Sullman et al., 2014). In addition to age, driving experience has been shown to moderate the relationship between anger and impulsive/aggressive driving, with inexperienced drivers being more aggressive when they experience anger (Bogdan et al., 2016). It is difficult however to separate age from driving experience as mostly people learn to drive at a young age and obtain more and more experience as they get older.

1.4.3. Mental health

Higher impulsivity/anger and the resulting risk-taking behaviour in traffic might be a sign of underlying psychopathological disorders, and if these are not identified and treated, then traditional learning/intervention strategies and punitive measures would only partially affect the problematic behaviour in traffic (Valero et al., 2017). Increased risk of injury that is associated with some psychiatric disorders (e.g., anxiety or mood disorder) has been found to be related to increased risk of collisions in traffic (Wickens et al., 2013).

In a study with the sample formed of people who lost their driving licence due to some traffic violation (mostly DWI/speeding), around 72% of the drivers had at least one psychiatric disorder at the time of the study or in the past, pointing out the significance of taking mental health status into account in the prevention of traffic injuries (Valero et al., 2017). The most prevalent disorders were substance abuse (58%), ADHD (24%), depression (11%) and anxiety (9%), with ADHD being the most discriminant disorder distinguishing between people at high vs

low risk while driving. In the Valero et al., (2017) study an approximately six times larger proportion of people was affected by ADHD as compared to the expected prevalence of ADHD in general population (2–3%) (Dobrosavljevic et al., 2020; Faraone and Biederman, 2005; Polanczyk et al., 2015).

1.4.3.1. Depression and anxiety

Depression and anxiety have been found to increase the risk for road traffic accidents 2.4- and 2.7 – fold, respectively (Alavi et al., 2017). Depression also has been identified as a risk factor in deaths by traffic accident (Crump et al., 2013), suggestive of the more serious nature of accidents. Subjects with high anxiety have been shown more frequently engaged in drunk driving and to cause significantly more crashes (Dula et al., 2010).

1.4.3.2. ADHD

While depression has been associated with being injured as a result of the accident then attention deficit/hyperactivity disorder (ADHD) has been associated more strongly with multiple traffic accidents and violations (Aduen et al., 2015). The reported risk of traffic accidents and violations by drivers with ADHD 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. Thus, 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 present exclusively in patients, but are dimensional, 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 a 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 thought to be known about ADHD and traffic is derived mostly 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 related mainly to 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).

Significant improvements in driving performance and reduction of accidents in ADHD drivers can be observed when pharmacological treatments have been provided (Barkley and Cox, 2007; Boland et al., 2020).

1.5. Lifestyle factors affecting risk-taking behaviour in traffic

1.5.1 Diet and alcohol consumption

Impulsive and aggressive behaviour has been found associated with health behaviours like exercising and maintaining a healthy diet. For example, impulsivity has consistently been found related to not only overeating and food addiction (Loxton, 2018) but also to greater fast-food consumption (Garza et al., 2016). Fast-food/junk food is any food, which is low in essential nutrients and high in everything else – in particular, calories and sodium (Segen's Medical Dictionary, 2022). Consumption of energy drinks, which contain high amounts of caffeine and sugar, is another example of questionable diet: Associations have been found between energy drink consumption and higher perceived stress, smoking and alcohol abuse, poor quality of sleep, increased blood pressure, and risk of obesity and type 2 diabetes (Ali et al., 2015; Al-Shaar et al., 2017; Gunja and Brown, 2012; Wolk et al., 2012).

Regarding the potential biological foundation of these associations, animal studies strongly suggest that poor dietary choices may influence neurodevelopmental trajectories during adolescence (Reichelt and Rank, 2017). More specifically, alterations in dopamine-mediated reward signalling and GABA-ergic inhibitory neurotransmission can occur and predispose individuals to dysregulated eating and impulsive behaviours. Diets high in processed fat and sugar induce impulsive choice behaviour (Steele et al., 2017). Evidence also indicates that the relationship between somatic markers (body fat percentage, insulin, and inflammation) and impulsive choice is indeed moderated by diet, and the combination of such bodily measures and diet is most predictive of an impulsive choice (Steele, 2019). Some evidence exists that diets poor in the serotonin precursor

tryptophan may induce depression (Shabbir et al., 2013), and acute tryptophan depletion has been shown to increase impulsivity in males (Dougherty et al., 2010; Walderhaug et al., 2002). For those who wish to be certain that the possibly low tryptophan level in their diet has no undesirable consequences, examples of foods rich in tryptophan could be pointed out: these include dairy products, tuna, chicken, oats, nuts, and seeds.

1.5.2. Physical activity

While physical exercise brings about multiple benefits to health, problematic practice of physical exercise (PPPE, also known as exercise addiction or exercise dependence) is a maladaptive pattern of excessive exercise behaviour that manifests in physiological, psychosocial, and cognitive symptoms (Hausenblas and Downs, 2002). Associations of PPPE with negative urgency and sensation seeking have also been found, and it has been suggested that PPPE serves to regulate or alleviate negative affect or aversive emotional states (Kotbagi et al., 2017).

1.6. Biological factors in traffic behaviour

Obviously, individual biological predisposition should play a role in risky driving behaviour. In dissection of the biological underpinnings of complex behaviours that likely largely result from gene-environment interactions, the candidate gene approach has so far been more useful in implicating specific mechanisms. While there is a vast literature on the neurobiology of traits like impulsivity and aggressiveness, such studies have largely been conducted in clinical settings, and little is known on how such findings apply to everyday behaviours involving higher risk. In this regard traffic behaviour is an excellent research area because of the high risk that is present in traffic situations, there is no shortage of subjects as most people are drivers in their everyday life, and for some consequences there is available an objective record.

1.6.1. Serotonin system

Impulsive behaviour has been consistently associated with low capacity of the central serotonergic system (Evenden, 1999; Fairbanks et al., 2001). The central serotonergic system refers to a network of neurons that produce and release the neurotransmitter serotonin that is involved in various functions, including attention, mood regulation, aggression, impulse control, sleep, and appetite (Lucki, 1998). In the brain, cell bodies of the neurons that use serotonin are located in the brainstem, more specifically in the raphe nuclei that send long axons to innervate much of the CNS (Berger et al., 2009).

1.6.1.1. Platelet monoamine oxidase (MAO) activity

Platelet MAO activity is a reliable peripheral marker of serotonergic activity in the CNS, and low levels of platelet MAO activity have been associated with social maladaptation, impulsivity, sensation seeking and monotony avoidance (Oreland, 1993; Oreland, 2004; von Knorring et al., 1984). Drunk drivers have been found to have lower platelet MAO activity (Eensoo et al., 2004; Eensoo et al., 2005). It should however be noted that platelet MAO activity can associate differently with the impulsivity profiles in e.g., drunk drivers vs speed limit exceeders, with the former having lower and the latter higher platelet MAO activity (Paaver et al., 2006). Platelet MAO activity is also lower in alcohol-dependent subjects (von Knorring and Oreland, 1996). Furthermore, low platelet MAO activity has also been associated with criminal behaviour and suicidality, especially in adolescents who come from an unfavourable psychosocial environment (Jokinen et al., 2018; Oreland et al., 2007; Stalenheim, 2004). Importantly, low platelet MAO is common in victims of severe trauma of any origin (Sabre et al., 2016). Despite the large body of evidence on the association of platelet MAO activity and low serotonergic activity with impulsivity (Harro and Oreland, 2016), evidence for everyday life significance of such measures from longitudinal observations is scarce.

1.6.1.2. The serotonin transporter gene promoter polymorphism (5-HTTLPR; rs25531)

Serotonin transporter plays a crucial role in serotonergic neurotransmission as a major regulatory mechanism controlling extracellular serotonin levels. Most of modern antidepressant drugs target serotonin transporters, and those of the most frequent use do so selectively. Thus, serotonin transporter was an obvious target in search of candidate genes for depression and anxiety. The serotonin transporter gene promoter polymorphism (5-HTTLPR) reported by Klaus-Peter Lesch and co-workers (Heils et al., 1995) has since its discovery been associated with several variables relevant to traffic behaviour, such as impulsivity (Paaver et al., 2008; Steiger et al., 2005), aggression (Gerra et al., 2005; Gonda et al., 2009), alcohol use (de Oliveira et al., 2016; Merenäkk et al., 2011; Vaht et al., 2014), and suicide (Gonda et al., 2011) and indeed also with speed limit exceeding and traffic accidents (Eensoo et al., 2018).

Carrying the short (s) allele of 5-HTTLPR and tryptophan depletion have been reported to be independently and additively associated with impulsivity (Walderhaug et al., 2010). The s-allele of 5-HTTLPR has been found to be a marker of less efficient serotonergic functioning, but being a common variant, it is not surprising that besides the risk behaviours it also confers higher adaptivity to the environment (Homberg and Lesch, 2011). Further, a review describing the effects of serotonergic variation and pharmacological manipulations on behaviours like parental attachment and caregiving, social play, aggressiveness, cooperation, and sexual behaviour has shown that serotonin is correlated with sensitivity to social factors (with higher sensitivity in s-allele carriers) and modulates social

behaviour dependant on the nature of these social factors, but the behavioural responses also influence the serotonergic system, leading to a bidirectional interaction (Kiser et al., 2012). Therefore, in a stressful social environment the s-allele carriers would likely have an increased risk of adverse outcomes but in a positive environment they would also benefit more, whereas l-allele carriers would be more resilient to the stressors but also less able to benefit if the situation was reversed. Regarding traffic behaviour 5-HTTLPR s'-allele carriers have been shown to have less violations and accidents in traffic, compared to l'/l' homozygotes (Eensoo et al., 2018).

1.6.2. Dopamine system, the dopamine transporter gene (*DAT1*) and the VNTR polymorphism (rs28363170F)

Of the neural substrates of impulsive behaviour, the dopaminergic system has received much attention as well (e.g., Dalley and Roiser, 2012). Dopaminergic system refers to the network of dopamine-producing neurons and their projections in the brain. Dopamine is produced in several areas of the brain, but the two main areas of origin are the cell bodies in substantia nigra and the ventral tegmental area, with their extensive projections innervating the striatum, the prefrontal cortex, the limbic system, and the hypothalamus (van den Heuvel and Pasterkamp, 2008).

Dopaminergic system is critically involved in behavioural activation, motivated behaviour and reward processing (Ikemoto and Panksepp, 1999). Human subjects with elevated dopaminergic functioning behave more impulsively (Bergh et al., 1997) and dopaminergic dysfunction occurs in patients with ADHD (Ludolph et al., 2008; Thapar et al., 2005). Altered dopamine transporter function is the most consistently observed neurochemical characteristic of ADHD (Krause et al., 2006).

Different pharmacological, biochemical, lesion and knockout studies in animals provide evidence that impulsivity is causally related to striatal dopamine function (Puumala and Sirviö, 1998; Winstanley et al., 2006). Pharmacological studies in healthy humans have provided similar results (de Wit et al., 2002; Friedel, 2004). The dopamine transporter (DAT) plays a critical role in terminating dopamine neurotransmission and in maintaining dopamine homeostasis in the CNS by taking up synaptic dopamine into neurons (Chen and Reith, 2000). The *DAT1* gene (SLC6A3) bears a rather widely studied variable number of tandem repeats (VNTR) polymorphism 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 DAT availability than do the 10-repeat (10R) allele homozygotes (Faraone et al., 2014; van de Giessen et al., 2009), and the over-supply of dopamine in striatum might weaken inhibitory pathways (Colzato et al., 2010). A significant association of 9R carriers has been found with alcohol dependence, withdrawal seizures and delirium tremens (Ma et al., 2016). Although being a 10R homozygote is thought

to be a risk factor for ADHD in children, a differential association of *DAT1* with ADHD has been suggested in children and adults, and being a 9R homozygote has been associated with persistent ADHD in adults (Franke et al., 2010), which might be explained by different functional consequences of the polymorphism in the matured dopamine system. In addition, compared to 10R homozygotes, 9R carriers have reported higher impulsivity in studies on adult healthy subjects (e.g., Forbes et al., 2009). Because impulsive behaviour has been associated with traffic accidents and violations (Pearson et al., 2013), the 9R carriers may also be more inclined towards risk-taking behaviour in traffic.

1.6.3. Neuropeptide S and the neuropeptide S receptor gene (*NPSR1*) A/T polymorphism (Asn107Ile)

Another candidate for regulation of impulse control and alcohol use is the neuropeptide S (NPS) system (Ghazal, 2016) that can stimulate dopaminergic neurotransmission (Si et al., 2010). NPS cell bodies are located in the pericoerulear region and the lateral parabrachial area including the Kölliker–Fuse nucleus (Clark et al., 2011; Reinscheid and Ruzza, 2021; Xu et al., 2004; Xu et al., 2007) with widespread projections of NPS-immunopositive fibers throughout the brain and highest densities in hypothalamus, thalamus, and structures of the extended amygdala (Clark et al., 2011; Liu et al., 2011). Much of the research regarding NPS system has been carried out in animals, but in humans, a functional polymorphism of the gene that encodes for the neuropeptide S receptor 1 (*NPSR1*) (Dannlowski et al., 2011) has been associated with the development of personality, hyperactivity, alcohol use and alcohol use disorders (Laas et al., 2014a; Laas et al., 2014b; Laas et al., 2015a; Laas et al., 2015b). *NPSR1* gene carries a functional A/T single-nucleotide polymorphism (SNP, rs324981) coding for an Asn-Ile exchange at position 107. NPS has up to 10 times higher potency at the receptor encoded by the T-allele (107Ile) compared to the A-allele-encoded receptor, leading to more effective signal transduction with mobilization of intracellular Ca^{2+}, stimulation of cyclic adenosine monophosphate synthesis and induction of mitogen-activated protein kinases phosphorylation (Reinscheid et al., 2005). Taranov and colleagues (2017) found in a study with bus drivers that *NPSR1* was associated with increased risk of a road accident. The T-allele of the *NPSR1* rs324981 polymorphism has been associated with increased impulsivity and ADHD-related traits (Laas et al., 2014a; Laas et al., 2015b). Further, an impulsivity-related early-onset pathway to alcohol use disorder has been revealed in male T-allele carriers, particularly in T/T homozygotes: already in adolescence, they exhibit more ADHD symptoms and impulsivity that could make them more vulnerable to alcohol use (Laas et al., 2015a).

1.7. Interventions

As with other kinds of dysfunctional and dangerous behaviours different types of interventions have been tried for reducing risk-taking behaviour in traffic. Interventions can be carried out as a form of rehabilitation/punishment, for example to people who have been caught driving drunk or speeding excessively, or interventions can be completely preventive and carried out already in driving schools as a part of education for young drivers.

As a part of the longitudinal Estonian Psychobiological Study of Traffic Behaviour (EPSTB) a brief intervention in driving schools was carried out by psychologists. The intervention was guided by the affective neuroscience concept (Panksepp, 1998) and had a focus on the acknowledgement of personal risks of impulsive traffic behaviour. It has been shown to have a diminishing effect on traffic violations and accidents for at least up to 4 years after the intervention (Eensoo et al., 2018; Paaver et al., 2013).

One of the aims of this thesis was to reproduce this intervention but if conducted by driving school teachers instead of psychologists, and investigate further whether the possible positive effects of the intervention could be dependent on subjects' individual factors such as high level of ADHD symptoms.

2. AIMS OF THE STUDY

The aims of the dissertation were to examine the association between risky traffic behaviour and personality factors like impulsivity and aggressiveness, and sub-syndromal ADHD; to find out if the contribution of impulsive/aggressive tendencies to different lifestyles is associated with dangerous traffic behaviour; to investigate the effectiveness of a driving school intervention and its dependence on individual aspects of risk-taking; and to clarify the potential underlying biological mechanisms of risk-taking in traffic.

To reach these aims, the following **research questions** were posed:

1. What are the associations between driving anger, impulsivity, and impulsive/aggressive behaviour in traffic? (**Paper I**)
2. Are young drivers' self-reported symptoms of ADHD related to their risk-taking behaviour in traffic? (**Paper III**)
3. Are there associations between risky traffic behaviour and lifestyle? (**Paper IV**)
4. Is drunk driving recurrent and can relapses be associated with risk driving and candidate gene variants? (**Paper II**)
5. Are other kinds of risk-taking behaviour in traffic associated with candidate genes of impulsive/aggressive behaviour? (**Paper II – V**)
6. Are the associations between impulsivity, lifestyle, and risky driving behaviour mediated and/or moderated by 5-HTTLPR polymorphism? (**Paper IV**)
7. What is the long-term effect of a brief psychological intervention in driving schools and what are the possible moderators of a successful intervention? (**Paper V**)
8. Does the presence of ADHD symptoms affect the success of an impulsivity awareness intervention? (**Paper III**)
9. Can the effect of the intervention be moderated by 5-HTTLPR and/or *DAT1* (**Paper V**)?

3. MATERIALS AND METHODS

3.1. Subjects

Sub-samples of EPSTB and ECPBHS were used in **Papers I–V**. All stages of both studies were approved by the Research Ethics Committee of the University of Tartu.

3.1.1. EPSTB

The longitudinal Estonian Psychobiological Study of Traffic Behaviour (EPSTB) comprises of samples from four originally independent projects starting from 2001. The last data collection included in this thesis started in 2019, when a link to a web-based questionnaire was sent by e-mail to all subjects of the EPSTB. Description of the samples is as follows:

1) Sampling of drunk drivers (described in detail by Eensoo et al., 2005) was conducted in 2001. The group of drunk drivers comprised male subjects who were identified by the police as driving drunk at least once during the previous year (n = 203; mean age (SD) = 33.0 (11.0) years). The control group was formed of the male subjects in the driving licence database of the Estonian Motor Vehicle Registration Centre and consisted of 211 individuals (mean age (SD) = 36(12.0) years). Data of this sample was included in **Papers I and II.** Subjects of this sample were sent by e-mail a link to a questionnaire in 2019 and these who responded are included in the study group of **Paper IV.**

2) Sampling of speed limit exceeders (described in detail by Paaver et al., 2006) was conducted in 2002–2003 and comprised male subjects with previous speeding violation as well as a control group formed again as described above. Similarly, subjects of this sample were asked to fill out new questionnaires in 2019, and those who participated in this data collection are included in the study group of **Paper IV.**

3) The sample of the first driving school based study (described in detail by Paaver et al., 2013) comprised driving school students applying for a passenger car driving license. This was a study including an intervention arm with focus on the acknowledgement of the impulsivity factor in traffic. Sampling was conducted in 2007 (n = 1866; mean age (SD) = 23.0 (7.2) years). Data of the sample is used in analyses included in **Papers I, III and IV**. As the focus in **Paper III** was on subsyndromal ADHD, we included only subjects who had self-reported ADHD symptom data by the ASRS v1.1 screening tool at sampling: n = 741 (40% of the original sample, mean age (SD) =23.3 (7.2) years). In **Paper IV**, data of participants in this sample were, again, used if they filled out new questionnaires in 2019.

4) The sample of the second driving school study (for details see **Paper V**). This sampling was carried out in 2013/2014. The total sample was 1441 participants (mean age (SD) = 22.5 (7.9) years). This sample is the basis for **Paper V**. It was also included in analyses described in **Papers I, III** and **IV**. For the analyses in **Paper III**, we included only subjects who had self-reported ADHD symptom data available (collected at sampling): n = 995 subjects (69% of the original sample, mean age (SD) =22.9 (8.1) years), and for those in **Paper IV** we included subjects if they had filled out the new questionnaire in 2019.

The presently last stage of the EPSTB was conducted in 2019 when a link to a web-based questionnaire on lifestyle was sent by e-mail to **all subjects of the EPSTB.** Data of those who agreed to participate were included in analyses reported in **Papers I and IV** (n = 817, mean age (SD) = 31.4 (10.0) years); 49.2% males and 50.8% females). The proportion of EPSTB subjects in this last stage by sampling were as follows: 2001–2003, n = 183 (22%); 2007, n = 285 (35%); 2013–2014, n = 349 (43%).

3.1.2. ECPBHS

In addition to EPSTB samples, we used data of the Estonian Children Personality Behaviour and Health Study (ECPBHS). The rationale and procedure of sample formation and follow-ups have been described in detail elsewhere (Harro et al., 2001; Joost et al., 2019; Luht et al., 2018; Tomson et al., 2011). Specifically, data of the two birth cohorts of ECPBHS obtained at age 25 years were included (**Papers I and III**). For subjects who did not fill in respective questionnaires at age 25 (~6%), data collected at age 33 years were used, if available. Altogether there were 1016 ECPBHS participants (82% of the original sample, mean age (SD) = 25.2 (2.1) years) in the analyses. ECPBHS sample data was used in analyses either separately (**Paper III**) or combined with the two driving school samples of the EPSTB (**Papers I and III**).

3.2. Measurements

3.2.1. Socio-economic background

Subjects reported their socio-economic status (**Papers II and V**). Questions about socio-economic background included education, monthly income, and relationship status. Monthly income was dichotomized according to the mean income in the country at the time of data collection.

3.2.2. (Excessive) alcohol consumption and tobacco smoking

Subjects reported their alcohol use habits and tobacco smoking in a self-report questionnaire. The Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993) was used to identify subjects with problematic alcohol consumption (**Papers II, IV, V**). The test contains 10 multiple choice questions on quantity and frequency of alcohol consumption, drinking behaviour, and alcohol-related problems or reactions (scale 0–4). The frequency of using alcoholic drinks was used in data analysis both as a categorical (at least once a week vs. less than once a week) (**Paper II**) and as a continuous variable (**Papers IV and V**). Tobacco smoking status was categorized to non-smokers, ex-smokers, ≤10 cigarettes/day, 11–19 cigarettes/day, ≥20 cigarettes/day (Eensoo et al., 2004) and was used in data analysis both as a categorical variable (current smoker vs. non-smoker) and as a continuous variable (5-point scale).

3.2.3. Healthy eating questionnaire

Subjects answered questions about the frequency (1–7, never – several times in a day) of eating different foods and drinking energy drinks (**Paper IV**) in the past few weeks. We made use of the mean score of eating of French fries and hamburgers as an indicator of fast/unhealthy eating (junk food consumption). As an indicator of healthy eating, we used the question about the frequency of eating vegetables. In addition, we included the frequency of energy drink consumption (at least once a week vs less than once a week) in the analysis.

3.2.4. Physical activity and body mass index

Subjects were asked about their physical activity (**Paper IV**) in the past week. We used "vigorous physical activity" as a measure. Vigorous physical activity was defined as activities that require great physical energy and make the subject breathe a lot faster than regularly (e.g., doing hard work, exercising). The activity had to last at least 10 min consecutively, and it was asked how much time the participants spent in the past week on this kind of activities (hours per week). Body mass index (BMI) was calculated from data provided by the subjects about their weight and height (BMI = kg/m^2).

3.2.5. Measurement of impulsivity and aggressiveness

Adaptive and Maladaptive Impulsivity Scale (AMIS) was used to measure facets of impulsivity (fast decision-making, thoughtlessness, disinhibition and excitement seeking) (**Papers II–V**) as described in detail elsewhere (Paaver et al., 2006; Laas et al., 2010). AMIS is based on the concept of functional and dysfunctional impulsivity by Dickman (Dickman, 1990). Subjects were asked to assess how much the 24 different impulsivity-related statements applied to them on a scale of 1 to 5.

The Buss–Perry Aggression Questionnaire (BPAQ) (Buss and Perry, 1992) was used to assess aggressiveness (**Papers I and IV**). The 29-item instrument assesses four aspects of aggressive behaviour: Physical aggression, Verbal aggression, Anger, and Hostility. Participants rated each statement on a 5-point Likert scale (uncharacteristic = 1, characteristic = 5).

3.2.6. ADHD symptoms, ASRS v1.1

The World Health Organization recommended questionnaire for ADHD screening, ASRS v1.1 (Kessler et al., 2005) was used (**Paper III**). 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 **Paper III** 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).

3.2.7. Mileage

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

3.2.8. Driver Behaviour Questionnaire

The Driver Behaviour Questionnaire (DBQ) (Lawton et al., 1997; Reason et al., 1990) that has been shown to predict traffic accidents (de Winter and Dodou, 2010), was filled in at least 3 months after recruitment of the participants (**Papers I and III**). 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). In the sample of (**Paper III**) 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 items of the two subscales entirely; the correlation between the subscale scores was r=0.47 that was statistically 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.

3.2.9. Driving Anger Scale

The Driving Anger Scale (Deffenbacher et al., 1994) was filled in by subjects (**Papers I, III and IV**). 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.

3.2.10. Databases

Data on violations of traffic law, traffic accidents and the status of subjects' driver's licences (valid or withdrawn) were obtained from databases maintained by the traffic police, the traffic insurance fund and the Estonian Road Administration for the period of January 1, 2002, to December 31, 2011 (**Paper II**), for the respective three-year period after the driving school intervention (**Papers III and V**), or for the respective five-year period since the recruitment of each sample (**Paper IV**). The traffic behaviour measures were as follows: speeding (penalties for exceeding the speed limit), DWI (penalties for drunk driving with an estimated blood alcohol level of 0.2‰ or more) and other traffic violations (all the traffic violations besides speeding and DWI). For speeding violations and drunk driving (DWI) we also used the sum of violations in this five-year period (**Paper IV**), so that the score could be from 0 (no speeding tickets/drunk driving violations) to 5 (received at least one speeding/drunk driving violation ticket every year). The accidents where the subject was at fault were classified as active accidents, and other accidents as passive accidents. 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).

3.2.11. Platelet MAO activity

Platelet MAO activity was analysed in platelet-rich plasma by a radioenzymatic method with β-phenylethylamine as the substrate according to the procedure described by Hallman et al. (1987) after modification (Harro et al., 2001). Platelet MAO activity was analysed in 405 subjects (**Paper II**) and used both as a categorical variable (low 25th percentile vs. high) and as a continuous variable.

3.2.12. Genotyping

3.2.12.1. 5-HTTLPR

Genotyping for the triallelic classification of the 5-HTTLPR polymorphism was performed according to the method of Anchordoquy et al. (2003) as described in detail elsewhere (Tomson et al., 2011; **Papers IV and V**). In brief, genotyping was performed in two stages. First, all subjects were genotyped for the 5-HTTLPR VNTR polymorphism, then for the single nucleotide polymorphism (SNP) rs25531 (A/G). The polymorphic region was amplified using the primers 5-HTTLPR-F: 5′-6FAM-ATG CCA GCA CCT AAC CCC TAA TGT-3′ and 5-HTTLPR-R: 5′-GGA CCG CAA GGT GGG CGG GA-3′. As the next step the SNP rs25531 (A→G) was genotyped. Triallelic 5-HTTLPR genotypes were categorised into groups according to the effectiveness at the transcriptional level as follows: l_G/l_G, l_G/s, and s/s were designated as s'/s'; l_A/s and l_A/l_G as l'/s'; and l_A/l_A as l'/l'. Genotype frequencies were in the Hardy–Weinberg equilibrium. We compared the s' allele carriers with the l'/l' homozygotes (**Paper IV** – n = 644 (s'/s' and l'/s' – 68.0%, l'/l' – 32.0%); **Paper V** – n = 1339 (s'/s' and l'/s' – 66.8%, l'/l' – 33.2%)).

3.2.12.2. *DAT1*

The *DAT1* (*SLC6A3*) VNTR was genotyped following the analytical method by Anchordoquy et al. (2003) as described in detail elsewhere (Maksimov et al., 2015) (**Papers II, III, V**). 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 ranged 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: **Paper II** – n = 391 (9R – 36,3%, 10R/10R – 63.7%); **Paper III** – n = 2256 (9R – 37.9%, 10R/10R – 62.1%); **Paper V** – n = 1312 (9R – 38.3%, 10R/10R – 61.7%).

3.2.12.3. *NPSR1*

NPSR1 rs324981 was genotyped by routine polymer chain reaction followed by restriction enzyme digest and gel electrophoresis as described in detail by Domschke and colleagues (2011). *NPSR1* was successfully genotyped in 402 subjects (**Paper II**). Genotype distribution was as follows: A/A 28.1%, A/T 46.3% and T/T 25.6%. Genotype frequencies were in Hardy–Weinberg equilibrium.

3.2.13. Intervention procedure

The intervention 'Reducing Impulsive Action in Traffic' (Eensoo et al., 2018; Paaver et al., 2013;) consisted of a lecture (45 min) and group work (45 min) (**Papers III and V**). The intervention was theoretically guided by the affective neuroscience concept (Panksepp, 1998), according to which emotions are biologically based phenomena that play a crucial role in regulating behaviour and adapting to the environment. Emotions can be triggered by certain stimuli, such as sensory inputs or memories, and emerge from the activation of specific brain regions and circuits that can lead to characteristic behavioural responses. These systems are individually different and thought to influence human personality and behaviour in a bottom–up fashion, and together with individual differences in top-down regulation capacities they result in unique personality/behavioural patterns (Montag and Panksepp, 2017). It is the essence of the activity of emotive systems that they operate automatically (Panksepp, 1998). The subthreshold activity in emotive systems may remain non-conscious; importantly, individuals greatly differ for genetic and developmental reasons in their emotive activities but may neither acknowledge their vulnerability nor their own countermeasures by the adaptive self-organization of the brain (Harro, 2010).

The intervention was aimed at acknowledgement of personal impulsive tendencies, so that the subjects could build their own strategies to reduce personal risk. Lectures of the intervention were carried out and the group work conducted by regular teachers of the driving schools, who had previously been trained in a tailor-made 2 European Credit Transfer and Accumulation System point course at the University of Tartu to carry out the intervention. The intervention process is described in detail by Paaver and colleagues (2013), but in brief, main topics of the intervention lecture were: 1) impulsivity as a personality feature and information processing style that is partly biologically determined and can lead to risky behaviour in traffic; 2) different types of impulsivity, their association to risk-taking and recognizing impulsive tendencies in oneself; 3) potential situational factors triggering impulsive behaviour and encouragement of subjects to note situations in which they behave impulsively or take risks. Importantly, participants received feedback on their own level of impulsivity based on the AMIS scale. For group work participants were given tasks to identify the psychological factors involved in real-life traffic accident cases, estimate their own risk for this kind of traffic accident and generate ideas of ways to decrease this risk.

3.3. Statistical analysis

Data were analysed using SPSS (version 23.0 SPSS, Chicago, IL) and SAS (version 9.4 SAS Inc., Cary, NC) software.

Differences between groups regarding categorical variables were analysed with Pearson's chi-square test and the post-hoc Fisher's test (**Papers II – V**), and for continuous variables with ANOVA (**Papers II and III**) or Student's T-test (**Papers I, IV and V**) for variables following normal distribution, and Kruskal-Wallis test (**Paper II**) or Mann-Whitney U-Test (**Paper IV**) for nonparametric data. Normality of data was assessed visually by box plots, Q-Q plots and also by skewness and kurtosis. Survival analysis and Cox regression analyses were used to investigate the effect of different variables upon the time subjects committed DWI after the initial recruitment to the study (**Paper II**) and traffic accidents and general traffic risk (**Paper V**). Logistic regression analyses were used for predicting the occurrence of active traffic accidents in 2002–2011 (**Paper II**), high traffic risk and high DBQ violations scores (**Paper III**), speeding (**Paper IV**). Logistic regression analyses were adjusted if necessary – 1) by occurrence of DWI (**Paper II**) 2) ADHD screening and subscale scores (**Paper III**) 3) gender (**Paper IV**). To obtain insight into the complex relationship between traffic violations and accidents, genotype, impulsivity, eating and physical health behaviour, structural equation modelling (SEM) was used (**Paper IV**). For multiple imputation of missing cases Full Information Maximum Likelihood Estimation (FIML) was used. Fit indices and their acceptable thresholds used values as Root Mean Square Error of Approximation (RMSEA) less than 0.07 (Steiger, 2007), Comparative Fit Index (CFI) and Tucker Lewis index TLI greater than 0.95 (Sharma et al., 2005). The $p < 0.05$ level was considered as statistically significant in all the analyses.

4. RESULTS AND DISCUSSION

4.1. Association of driving anger with impulsivity (Papers I, II and IV)

Driving anger and impulsivity are often independently studied but apparently related constructs both potentially increasing risk-taking behaviour in traffic. We examined the association between these two constructs and whilst driving in traffic to uncover their specific roles in risk-taking traffic behaviour (**Papers I, II and IV**).

Driving anger as measured with questionnaires tailor-made for that specific purpose has been found to exert a more direct impact on driving behaviour as compared to trait anger and to partially mediate the effect of trait anger on risky driving behaviour (Ge et al., 2017). We found different traffic violations like speeding, drunk driving, and self-reported risk in traffic (DBQ questionnaire) to be associated with higher driving anger, whereas anger (BPAQ subscale) was associated only a little with self-reported risk, and not at all with the actual traffic violations (**Paper I**, Fig.1).

Impulsivity has been shown to be related to emotional and instrumental aspects of aggressiveness, especially physical aggression (Vigil-Colet et al., 2008). We could observe higher physical aggression among subjects with speeding violations, drunk driving violations and higher DBQ scores (**Paper I**, Fig. 1) showing the associations of physical aggression with impulsivity by the example of these objective and self-reported risk-taking behaviours in traffic. Impulsivity has been hypothesized to mediate the aggression expression (Vigil-Colet et al., 2008). While inhibition deficits related to impulsivity have been suggested to lead to anger and aggressive behaviours, specifically dysfunctional/maladaptive impulsivity has been shown to be correlated with aggression, whereas functional/adaptive impulsivity had no significant relationship (Vigil-Colet and Codorniu-Raga 2004). Subjects who had drunk driving violation differed in their higher maladaptive impulsivity, higher aggressiveness and driving anger from controls (**Papers I and II)**. As to adaptive impulsivity it might not be correlated with aggressiveness but can still be dangerous in traffic by the example of speed limit exceeding. Consistent with the study by Paaver and colleagues (2006) we found speeding to be associated with fast decision making and excitement seeking (aspects of adaptive impulsivity) in a sample that included some of the participants of the earlier study, but these formed only one fifth of the whole sample (**Paper IV**).

Trait anger, driving anger, aggressiveness, and aspects of impulsivity are partially but not entirely overlapping constructs all significant in understanding risky driving behaviour. Based on literature review and our own findings we have proposed a schematic representation of the associations between trait anger, driving anger, aggressiveness, impulsivity, and risky driving behaviour (Figure 1). Trait anger and driving anger precede the aggressive behaviour in traffic, and driving anger is largely based on trait anger, although some drivers do become angered

in traffic situations yet not being high in trait anger. There is the tendency of people with high trait/driving anger to interpret situations as hostile which leads to aggressiveness and risky driving behaviour, and in addition, if the person has impulsive tendencies, then this can contribute to the true expression of anger and aggressiveness in the form of risk-taking in traffic.

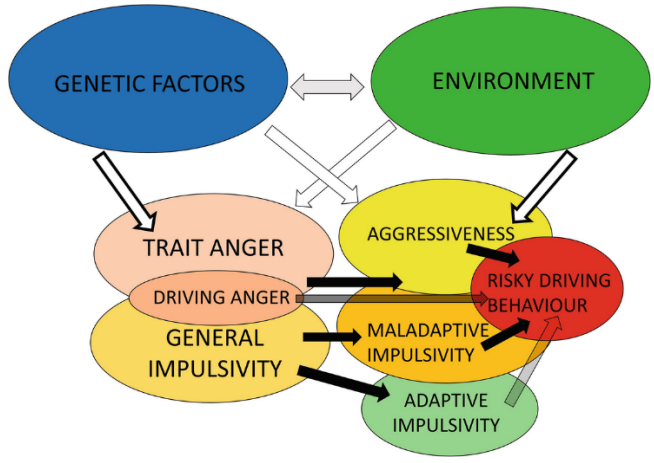

Figure 1. Emotional and behavioural tendencies leading to taking risks in traffic. Illustration of the contribution of genetic factors and environment manifested through trait anger, driving anger, aggressiveness, and aspects of impulsivity to risky driving behaviour. (**Paper I**)

4.2. Symptoms of ADHD and traffic behaviour (Paper III)

When investigating the potential significant threat of self-reported ADHD symptoms in traffic, the summary data of three different samples (n = 2752) revealed (**Paper III**) that there were approximately 12% of drivers with a score considered positive in ASRS screening (Kessler et al., 2005). This is much higher than the expected prevalence of ADHD in general adult population (2–3%; Faraone and Biederman, 2005), but then again ASRS is not a diagnostic tool but a sensitive screening measure that yields normally distributed data. In all the samples included in **Paper III** 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 the link to traffic violations, confirming the suggested particular association between increased hyperactivity/impulsivity and deliberate violations (Fuermaier et al., 2017).

One of the major differences between drivers with positive ADHD screener in the EPSTB and in the ECPBHS was that in the former sample the mileage driven past year was lower in the ADHD screener positives (**Paper III**, Table 1). 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 (**Paper III)**. 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 (**Paper III**, Tables 1 and 2). It is of course adaptive to learn 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 (**Paper III**, Tables 1 and 2), 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).

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). When not medicated ADHD drivers have been shown to have significantly different pre-crash performance (velocity, brake force, steering movement etc) as compared to controls, and this could be detected by in-vehicle safety systems (Barragan and Lee, 2018).

4.3. Lifestyle and traffic behaviour (Paper IV)

Exceeding speed limits, which is a good example of behaviour resulting from potentially impulsive decision-making in traffic that has possible dangerous outcomes (Elvik, 2012; Theofilatos and Yannis, 2014), was consistently associated with both personality factors and health behaviours (**Paper IV**). Speed limit exceeders had higher adaptive impulsivity and physical and verbal aggression compared to subjects with no speeding tickets in the 5-year period, but they were not significantly different in maladaptive impulsivity (thoughtlessness and disinhibition), anger or hostility. Speeding subjects also had higher AUDIT score, they reported doing more vigorous physical activity and consuming energy drinks more often. Speed limit exceeders also had significantly more violations of other type, and higher prevalence of traffic accidents (**Paper IV**, Table 2).

While speed limit exceeding was not directly associated with eating habits, drunk drivers reported eating less healthy foods by the example of vegetable consumption (**Paper IV**, Table 4). Interestingly, speed limit exceeding was also associated with higher involvement in vigorous physical activity, both directly and mediated by physical aggression (**Paper IV**, Fig. 2). Animal experiments have shown that exercise can effectively alleviate ADHD-like symptoms through enhancing dopamine D_2 receptor expression in the brain (Cho et al., 2014), and it has been suggested that exercise may serve as a way of counteractive regulation of impulsive behaviours (Racine, 2012). It has also been shown that adults with ADHD engaging in frequent aerobic physical activity report significantly less behavioural impulsivity compared to subjects with low activity (Abramovitch et al., 2013). As a matter of fact, dopamine D_2 receptor availability is affected by exercise in also humans, at least in the context of preventing the decline that comes with aging (Dang et al., 2017). In addition, Joseph et al. (2011), have concluded that increased physical activity may help compensate and suppress the hedonic drive to over-eat. The results of **Paper IV** do not suggest that speeding is a behaviour truly compensated by physical activity; rather exercising appears as being fuelled at least in part by similar mechanisms as the risky behaviour of speeding, and it is generally descriptive of the profile of someone with higher adaptive impulsivity.

Associations of lifestyle with aggressive behaviour have also been shown before, with higher aggression levels among subjects with unhealthy lifestyles (sleeping less, poor eating habits, drinking, smoking, not working out) (Kim et al., 2020; Pouyamanesh, 2013; Rao et al., 2015). In a study using the Buss – Perry Aggression Questionnaire, the subjects with poor lifestyle had higher physical aggression, hostility, and anger, but those with healthier habits reported higher verbal aggression (Pouyamanesh, 2013). In **Paper IV** both physical and verbal aggression predicted speeding behaviour, the former being a significant mediator for speeding in males and the latter in females. Even though we did not find a direct link between higher physical aggression, speeding and accidents in males, we did find that males had significantly more accidents in traffic as compared to females (**Paper IV**, Table 1). Gender differences of physical vs verbal aggression in

speeding behaviour appear consistent with previous studies which have found that females express their anger in traffic more constructively than males (e.g., doing things to calm down like taking deep breaths, turning on radio/music or paying even closer attention to others' driving to avoid accidents) (González-Iglesias et al., 2012; Hernández-Hernández et al., 2019; Sullman, 2015). Thus, speeding in males and females is in part differently mediated and possibly for this reason more often resulting in accidents in males, because in traffic, higher inclination to physical aggressiveness represents potentially more dangerous behavioural tendencies.

4.4. Recurrence of drunk driving and association with other risky driving behaviour (Paper II)

In addition to speeding, DWI (drunk driving) is one of the most alarming dangerous behaviours in traffic. Approximately one-third of those arrested for DWI are repeat offenders (Warren-Kygenyi and Coleman, 2014) which is consistent with our findings (**Paper II**) – 33% of former drunk drivers had committed another DWI within the next 10 years after the original recruitment, which is significantly more compared to controls. Only a few people of the original drunk drivers group were reintroduced into police records as drunk drivers during the second half of the observation period, so the probability of committing DWI again was highest in the 6 years following the initial violation (**Paper II**, Fig. 1). Although drunk drivers with repeat DWI group had the most subjects whose driver's licence was withdrawn before and during the observation period and also the longest period of time without a valid driver's licence per person, it seems that it was not enough for preventing their new DWI or other traffic violations.

In addition to their DWI violation, repeat offenders had more of other traffic violations than subjects in all the other groups. There were also significantly more other traffic violations in the original control group that had a later DWI record and in drunk drivers without further DWI, compared to controls without DWI. Therefore, by their other traffic violations, subjects who had committed DWI at least once were more hazardous drivers in traffic than controls without DWI, and subjects with repeat DWI were more hazardous than those who did not repeat drunk driving in the 10-year period.

4.5. Association of candidate gene variants and platelet MAO activity with risk-taking behaviour in traffic and other areas of life (Papers II–V)

4.5.1. Platelet MAO activity and DWI

Platelet MAO activity is suggested to reflect the capacity of the central serotonergic system (Oreland, 2004), possibly owing to the developmental differences brought about during the foetal stage, eventually leading to higher risk-taking and impaired decision-making in adulthood, in particular if intoxicated by alcohol (Harro and Oreland, 2016). Significant differences between the DWI subgroups were found in platelet MAO activity (**Paper II,** Table 1): drunk drivers without further DWI and drunk drivers with repeat DWI had lower platelet MAO activity compared to controls without DWI. Since cigarette smokers have been shown to have reduced MAO activity (Fowler et al., 2003), we controlled for smoking status in the statistical analyses of MAO and with smoking as a covariate significant difference between the subgroups in platelet MAO activity disappeared ($p = 0.19$). When it was taken into account that the effect of smoking on platelet MAO activity is dose-dependent and not observable at low levels of smoking (Eensoo et al., 2004), then it was found that among subjects smoking less than 10 cigarettes per day, there was however a significantly higher proportion of subjects with low-platelet MAO activity. This was observed in drunk drivers without further DWI compared to controls without DWI, and a tendency for higher proportion of subjects with low platelet MAO activity in drunk drivers with repeat DWI compared to controls without DWI was also present. It has been shown in a longitudinal study that subjects with lower than average platelet MAO activity are more likely to become a smoker. This suggests that smoking is associated with low platelet MAO activity not only because of the direct inhibitory effect of tobacco constituents on the enzyme but also because subjects with low platelet MAO activity are more likely to become smokers (Harro et al., 2004). Indeed, separate consideration of low-intensity smoking, not likely to have any significant impact on MAO activity, suggested that higher smoking prevalence in drunk drivers does not explain this association.

4.5.2. 5-HTTLPR association with lifestyle and speeding (Paper IV)

The s'-allele of the 5-HTTLPR is another marker of less efficient serotonergic functioning but the effect of the carrier status of this allele on behaviour has been found to be more dependent on the environment, the l'/l' homozygotes expressing less malleability or flexibility (Homberg and Lesch, 2011; Kiser et al., 2012). We hypothesized that the s'-allele carriers would have higher maladaptive impulsivity accompanied by unhealthier eating habits, higher alcohol consumption and possibly also drunk driving, while 5-HTTLPR l'/l' homozygotes might be prone to

speeding. As there was no simple association between 5-HTTLPR and traffic behaviour measures we conducted a structural equation modelling path analysis to uncover any indirect association of 5-HTTLPR with risky traffic behaviour and health behaviour measures. No direct association of the genotype with traffic behaviour was found in this model; however, a path for the l'/l' homozygotes leading through problematic alcohol use to drunk driving could be established (**Paper IV**, Fig. 3). Those 5-HTTLPR l'/l' homozygotes who had more problematic alcohol consumption and a higher score in physical aggression were more likely to speed in traffic (**Paper IV**, Fig. 2A). Those 5-HTTLPR s'-allele carriers who eat junk food more often joined the path as consuming more alcohol, and were also more likely to speed in traffic. Admittedly, the sample of subjects, especially with DWI, was small for this type of an analysis. However, the path analysis models suggested that the 5-HTTLPR genotypes have both common and unique aspects in the path to traffic violations, contributing indirectly. The path of both l'/l' homozygotes and s'-allele carriers leading to exceeding the speed limits included excessive use of alcohol and tendency of physical aggression, while the speeding s'-allele carriers also had less healthy dietary habits. Therefore, speeding, and drunk driving by l'/l' homozygotes may occur in other contexts as of the s'-allele carriers, given their difference in aspects of impulsive and compulsive behaviour (Hong et al., 2018; Paaver et al., 2008; Sinopoli et al., 2019; Walderhaug et al., 2007). It should be noted that while drunk drivers had higher frequency of other violations, they did not differ significantly in terms of involvement in traffic accidents.

Considering the notable differences in risk-taking behaviour between males and females (**Paper IV**, Tables 1 and 3), path models for males and females were also constructed separately. In the path analysis for males, among l'/l' homozygotes speeding was still associated with higher AUDIT scores, driving anger and physical aggression (**Paper IV**, Fig. 2B), but in females energy drink consumption appeared as a significant predictor for speed limit exceeding on its own as well as mediated through a higher AUDIT score and verbal aggression. No relationship of speeding to the 5-HTTLPR genotype was apparent in females (**Paper IV, Fig. 2C**).

Altogether, the 5-HTTLPR l'/l' homozygotes who had a record of either drunk driving or speeding were likely to be abusers of alcohol, and this was observable in males. The 5-HTTLPR s'-allele carriers had a speeding record if they also presented further aspects of unhealthy lifestyle, here in the form of junk food eating. These findings are consistent with the view that the 5-HTTLPR l'/l' homozygotes are behaviourally less flexible while the s'-allele carriers have higher sensitivity to the environmental context (Homberg and Lesch, 2011); on the other hand, behaviour of the 5-HTTLPR l'/l' homozygotes may become more controlled by alcohol (Kapitau et al., 2019). The reported association between consumption of energy drinks and high-risk behaviour (Hamilton et al., 2013) was also observable in the **Paper IV**, and the association was stronger among those with higher AUDIT scores. That energy drink consumption is linked to high-risk behaviour particularly when combined with alcohol has also been observed (Breda et al., 2014). In traffic behaviour the co-use of these beverages appeared particularly

significant in females. It has been found that high habitual caffeine consumers report greater trait-wise motor impulsivity, but acute caffeine intake did not influence response inhibition or impulsive, risky, or aggressive behaviour in high or low habitual caffeine consumers (Giles et al., 2017). So, it may be hypothesized that the consumption of energy drinks does not by itself induce risk-taking behaviour in traffic but is a behavioural tendency that accompanies those who take more risks.

4.5.3. The association of *DAT1* with DWI, higher traffic risk, accidents and ADHD related risks

Carrying the 9R allele of *DAT1* VNTR genotype has been associated with higher impulsivity (Forbes et al., 2009) and we expected to see more traffic violations among them as compared to 10R/10R homozygotes. While there was no significant difference in genotype frequencies between the DWI subgroups in the study reported in **Paper II**, then in the younger sample (**Paper V**) male 9R allele carriers had indeed been driving drunk more frequently **(Paper V)**. This points out a possible differential association of drunk driving and *DAT1* based on age or driving experience. There were further significant differences with respect to *DAT1* VNTR when we compared the occurrence of active traffic accidents (**Paper II, Figure 2**): a significantly higher proportion of 9R carriers had been involved in active accidents in 2002–2011. Moreover, male 9R allele carriers were more likely to have higher general traffic risk (**Paper V**). This in addition to more DWI and more accidents in *DAT1* VNTR 9R carriers is consistent with the significant role of dopaminergic system in impulse control and risk-taking behaviour (Congdon et al., 2008; de Wit et al., 2002) and the potentially higher risk in *DAT1* VNTR 9R allele carriers (van de Giessen et al., 2009; Forbes et al., 2009).

Being a *DAT1* 9R allele carrier has also been associated with adult ADHD (Franke et al., 2012). In **Paper III** we did not observe any surplus of 9R in the ADHD screener positives, but there was a significantly higher proportion of *DAT1* 9R carriers in the EPSTB sample among subjects with co-occurring high traffic risk and ADHD screener positive or high hyperactivity/impulsivity or high inattention (**Paper III**, Fig. 1). This seems also in line with 9R carriers having more accidents in traffic through their own fault (**Paper II**). 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 *DAT1* on cortical activation within a group of adult ADHD patients has been found (Dresler et al., 2010), more specifically a pattern of reduced NGA (NoGo anteriorization – topographical ERP parameter) in 9R allele carriers. *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-ergic 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 subjects with ADHD and the *DAT1* 10R/10R genotype 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). More recently Brown et al. (2017) found that also in healthy individuals *DAT1* genotype can alter the neural basis of emotional processing and response inhibition in the 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 and our results, the risk genotype effect 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 further show the resulting dysfunctional behaviour of 9R carriers with ADHD symptoms in failing to control their anger in everyday high risk traffic behaviour.

4.5.4. The *NPSR1* association with DWI (Paper II)

We expected to see more DWI in the T-allele carriers of the *NPSR1* rs324981 polymorphism, which has been associated with increased impulsivity, ADHD-related traits (Laas et al., 2014a; Laas et al., 2015b), earlier and higher alcohol use and higher probability of developing alcohol use disorder (Laas et al., 2015a).

The following variables predicted DWI in a 10-year period independently: age, education, AMIS disinhibition, AMIS thoughtlessness, frequency of using light alcoholic beverages, alcohol-related problems, tobacco smoking, *NPSR1* genotype, active traffic accidents and other traffic violations (in 2002–2011) and drunk driving before recruitment. (**Paper II,** Table 2). After adjusting these associations by drunk driving before recruitment, models with age, education, alcohol-related problems, tobacco smoking, *NPSR1* and other traffic violations predicted DWI in 2002–2011 significantly. Next, we ran Cox regression models with *NPSR1* and other significant variables to see which variables predict DWI best together with *NPSR1*. The final model included drunk driving before recruitment, committing other traffic violations and being an *NPSR1* T-allele carrier. Therefore, being a T-allele carrier contributed significantly to the risk of repeatedly committing DWI, showing indeed increased impulsivity and more serious alcohol-related problems among T-allele carriers.

In a study of bus drivers, *NPSR1* A/A homozygotes had higher incidence of self-reported traffic accidents (Dorokhov et al., 2017; Taranov et al., 2017). We did not find any association between *NPSR1* and traffic accidents (**Paper II)**, but our study was also highly different with regard to the sample formation: bus drivers are highly trained and experienced professionals, and it is unlikely that their involvement in accidents could be largely driven by impulsivity. Indeed, we have demonstrated that intervention aimed at reducing impulsivity in driving schools does diminish the involvement in traffic accidents (Eensoo et al., 2018; Paaver et al., 2013). Somewhat speculatively, one may propose that the findings in the bus drivers' study suggest that the higher innate anxiety of the A/A homozygotes, liable of developing into maladaptive traits under environmental pressures (Laas et al., 2014a), could have contributed to their higher proneness to accidents.

4.6. Efficacy of the intervention, and its moderators (Papers III and V)

Evaluation of the effect of a previously successful brief psychological intervention (Paaver et al., 2013) in a new sample and conducted by appropriately trained driving school teachers showed that during the 3-year study period a significant impact of intervention on traffic safety was indeed present (**Paper V**). Similar to the previous study, the intervention had a beneficial effect on the involvement in traffic offenses and accidents. Participants of the intervention group appeared significantly less often in the general traffic risk group and, specifically, less involved in traffic accidents compared to controls during the 3-year study period (**Paper V**, Fig. 1). This suggests that the training the trainers approach is adequate for conducting this impulsivity-focussed intervention.

4.6.1. Efficacy by biological and personality factors

It is plausible that some subjects may be more receptive to the intervention compared to others. Thus we investigated whether the *DAT1* VNTR and the 5-HTTLPR polymorphisms have any diminishing or additive effect on the success of the intervention. While the intervention effect was independent of the 5-HTTLPR genotype in males, then consistently with the first intervention study (Eensoo et al., 2018), the female s'-allele carriers of the intervention group were the safest drivers in traffic (**Paper V**, Table 4). This fits well with the 5-HTTLPR s'-allele being associated with higher adaptivity to the environment (Homberg and Lesch, 2011), and with gender differences that have been reported in how carrying the s'-allele of the 5-HTTLPR affects susceptibility to stress (Brummett et al., 2008; Eley et al., 2004; Priess-Groben and Hyde, 2013). Therefore, it is not surprising that these differences are observable also in how an intervention might affect males and females differently.

In females, being a *DAT1* VNTR 9R allele carrier had no significant association with traffic behaviour at baseline but prevented the intervention effect to occur (**Paper V**, Fig. 2). The lack of similar association in males can result from gender differences in traffic behaviour and differential striatal dopamine release, re-uptake and responses during cognitive and motor function tasks in males and females (Riccardi et al., 2011). The lower intervention effect among female *DAT1* 9R carriers (P**aper V)** suggests that under certain environmental conditions the 9R allele might appear as a risk allele in females, and that other type of interventions may be more adequate for this group.

As to the possibility of ADHD symptoms to affect the success of the impulsivity-related intervention, the decreased likelihood of subjects of the intervention group to be in high traffic risk group remained statistically significant even after adjustment for ADHD screener, hyperactivity/impulsivity and inattention (**Paper III,** Supplementary Table 5).

Considering the contribution of individual differences on the efficacy of the intervention, and the wide array of different kinds of risk-taking behaviours in traffic, it is desirable that in future the selection of intervention methods would be informed by the subjects' profile – e.g. are personal risks associated more with inattention and driving errors or impulsivity and deliberate violations; is trait anger the most prominent psychological risk in traffic, does anger/impulsivity manifest only in traffic situations and is it rather persistent while driving or only present in specific driving situations (e.g., progress impediment); might there be some underlying psychiatric disorders that can affect traffic behaviour, and if there are, then is the person aware of this and taking the necessary medication etc**.**

5. CONCLUSIONS

This dissertation aimed to clarify associations of risky traffic behaviour with personality factors, lifestyle and biological predispositions and a driving school intervention, and **Papers I–V** have led to the following answers to the research questions:

1. Trait anger, driving anger, aggressiveness, and aspects of impulsivity are partially but not entirely overlapping constructs that are all significant in understanding risky driving behaviour (**Papers I, II and IV**).
2. Self-perceived ADHD symptoms were revealed as a significant risk factor in traffic, suggesting that sub-syndromal psychiatric disorders are an issue in traffic safety (**Paper III**). It would be beneficial to include a discussion in the drivers' training about the risks of ADHD and other related mental health issues and the threats they present in traffic so that people would be more aware of their mental health status in relation to driving.
3. Several aspects of lifestyle like junk food consumption and physical activity were associated with risk-taking and traffic law violations (**Paper IV**). These maladaptive behavioural patterns appear to form around impulsive tendencies and develop along distinct patterns that in part vary by genetically encoded differences in neural circuits and include the gender factor. It might be beneficial to consider the constellations of the larger spectrum of health-related behaviours and the diversity of their mediating mechanisms in future interventions.
4. Drunk driving is a serious violation reoccurring in approximately one third of subjects (**Paper II**). Repeat offenders had more other traffic violations than subjects in all the other groups, and committing DWI identified subjects as more hazardous drivers in traffic. Being a *NPSR1* T-allele carrier contributed to the risk of repeatedly committing DWI, and *DAT1* 9R allele carriers had been driving drunk more frequently (**Paper V**). Therefore, established biological markers of alcohol use and impulsivity can be reliably associated with everyday traffic behaviour and help in contributing to the understanding of the need for more personalized prevention activities.
5. Different candidate genes of impulsive/aggressive behaviour might influence behaviour in traffic (**Papers II – IV**). Significantly higher proportion of *DAT1* VNTR 9R carriers had been involved in active accidents (**Paper II)**, and they were more likely to have higher traffic risk (**Paper V**), which supports the significant role of dopaminergic system in impulse control and risk-taking behaviour. We did not observe any surplus of 9R in the ADHD screener positives, but a significantly higher proportion of *DAT1* 9 R carriers was found in the EPSTB sample among subjects with co-occurring high traffic risk and ADHD screener positive (**Paper III**).

6. Variation in the serotonergic system appears as a mediating and moderating factor of the associations between risky traffic behaviour and aspects of lifestyle such as consumption of alcohol or junk food or energy drinks, as well as engagement in vigorous physical activity (**Paper IV**).
7. The brief intervention conducted by driving school teachers had a significant impact on traffic safety similarly to previous intervention conducted by psychologists. The effect has been shown to be present for up to 3 years (**Paper V**). Future studies should address the potential of online training instruments for intervention as more and more studying and courses take place online not face to face.
8. Self-reported ADHD symptoms did not impair the efficacy of the intervention (**Paper III**).
9. Female 5-HTTLPR s'-allele carriers who were in the intervention group were the safest drivers and carrying the *DAT1* VNTR 9R allele prevented the intervention effect to occur (**Paper V**). This suggests that genetic variation could play a role in the successfulness of the intervention and that different type of interventions may be considered for some groups of people.

6. ACKNOWLEDGEMENTS

I am grateful to the EPSTB and ECPBHS participants, and the Estonian Road Administration (for cooperation and support in years 2001 – 2015). Special gratitude goes out to my supervisors Professor Jaanus Harro and Diva Eensoo for their guidance and support. I also wish to thank the study teams of EPSTB and ECPBHS, my co-workers and co-authors. Finally, I would like to thank my family and friends for their encouragement and support.

This work was supported by the Estonian Research Council [grant number PRG1213], and also received funding from the European Union's Horizon 2020 research and innovation programme under grant agreement No 667302 (CoCa) and No 728018 (Eat2beNICE).

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8. SUMMARY IN ESTONIAN

Riskeeriva liikluskäitumise seosed isiksuse, elustiili ja bioloogilise eelsoodumusega ning impulsiivsuseteadlikkusele suunatud sekkumine autokoolides

Sõidukijuhtide riskeeriv käitumine liikluses on endiselt aktuaalne oht kõikidele liiklejatele. Kahtlemata on isiksusel suur roll liikluskäitumises, seejuures kõige olulisemad konstruktid sellise riskikäitumise mõistmiseks on impulsiivsus ja viha. Käesolevas väitekirjas uurisime Eesti Psühhobioloogilise Liikluskäitumise Uuringu (EPBLU) ja Eesti Laste Isiksuse, Käitumise ja Tervise Uuringu (ELIKTU) valimite põhjal riskeeriva liikluskäitumise seoseid isiksuse näitajatega ning ka aktiivsus- ja tähelepanuhäire (ATH) sümptomaatikaga. Tahtsime lisaks teada kas impulsiivsuse ja agressiivsusega seostatud kehvemad elustiilivalikud on samuti seotud käitumisega liikluses. Selleks, et arendada edasi liiklusohtudega seotud ennetusvaldkonda uurisime EPBLU raames autokoolides läbi viidud sekkumisprogrammi pikemaajalist efektiivsust ning seda, et millised individuaalsed tegurid võivad efektiivsust mõjutada. Väitekirjas tahtsime lisaks selgitada võimalikku bioloogilist eelsoodumust riskikäitumiste suhtes, uurides selleks impulsiivsuse ja agressiivsuse kandidaatgeene.

Kirjanduse analüüsist ja meie enda tulemustest ilmnesid erinevad viha ja impulsiivsuse omavahelised seosed liiklusalase riskikäitumise mõjutamisel. Viha isiksuseomadusena ning spetsiifilisem sõidukijuhtimisega seonduv viha on eelduseks agressiivsele käitumisele liikluses. Sõidukijuhtimisega seonduv viha (*driving anger*) kattub suuresti üldise vihaga (trait anger), kuid on sõidukijuhte kes vihastuvad liiklusolukordades, aga muudes eluvaldkondades mitte. Kõrge vihaga inimestel on kalduvus tõlgendada olukordasid vaenulikkudena, mis liikluse kontekstis viib agressiivsema ja riskeerivama käitumiseni ning kui sellele lisaks on tegemist kõrgema impulsiivsusega indiviidiga, siis panustab see omakorda viha väljendumisele riskeeriva liikluskäitumise kujul. Kokkuvõtlikult võib öelda, et üldine viha, sõidukijuhtimisega seonduv viha, agressiivsus ning impulsiivsuse aspektid on osaliselt kattuvad konstruktid mis on kõik olulised selleks, et mõista riskeeriva liikluskäitumise põhjuseid.

Selleks, et uurida geenide ja keskkonna vahelisest koosmõjust tingitud mitmetahulise liikluskäitumise bioloogilisi aluseid kasutasime kandidaatgeenide lähenemist. Impulsiivne käitumine on seostatud serotonergilise süsteemi madalama aktiivsusega ning vereliistakute monoamiinide oksüdaasi (MAO) aktiivsus on tuntud pärilik serotonergilise aktiivsuse ja impulsiivsuse marker. Just madal MAO aktiivsus on seostatud vähesema sotsiaalse kohanemise, impulsiivsuse ja elamustejanuga. Serotoniini transporti ajus kontrollib serotoniini transporter (5-HTT) ja enam on uuringutes kasutatud 5-HTT geeni (*SLC6A4*) promootorregiooni funktsionaalset polümorfismi (5-HTTLPR), millel on kaks alleelivarianti: lühike alleel (s-alleel) ja pikk alleel (l-alleel). S-alleeli kandlus on seostatud madalama serotonergilise aktiivsusega, kuid samas ka kõrgema kohanemisvõimega keskkonna suhtes. Dopamiinergiline süsteem on samuti olulise rolliga

impulsiivse käitumise bioloogiliste mehhanismide juures. Kõrgem dopamiinergiline aktiivsus on seostatud impulsiivsuse ning ka ATH-ga. Inimese dopamiini transporteri geen (*DAT1*) sisaldab varieeruva arvuga tandeemse kordusega (R) polümorfisme (rs28363170F), levinuimad on 9R ja 10R. 9R alleeli kandlust on seostatud suurema riskeerimisega. Veel üks kandidaat impulsside kontrolli ja ka alkoholitarvitamise regulatsioonis on Neuropeptiid S (NPS) süsteem, mis omakorda võib stimuleerida dopamiinergilist aktiivsust. Inimesel on NPS retseptori geenis NPSR1 funktsionaalne A>T polümorfism (rs324981), mille T-alleeli kooditud retseptorvalk on signaali vahendamisel tõhusam ning just T-alleeli kandlus on seostatud kõrgema impulsiivsusega.

Mootorsõiduki juhtimine alkoholijoobe seisundis on tõsine liiklusrikkumine mida umbes kolmandik rikkujatest tulevikus kordab. Kui uurisime selle rikkumise bioloogilisi mehhanisme siis selgus, et alkoholijoobes juhtinutel oli madalam vereliistakute MAO aktiivsus ja *NPSR1* T-alleeli kandlus suurendas riski korduvalt alkoholijoobe seisundis mootorsõiduki juhtimiseks ning *DAT1* 9R alleeli kandjad olid suurema tõenäosusega joobes juhtimise eest karistatud. Lisaks joobes juhtimise seosele, oli *DAT1* 9R alleeli kandjate seas rohkem neid kes olid põhjustanud liiklusõnnetusi ning nad olid suurema tõenäosusega kõrgema üldise liiklusriskiga (esinenud vähemalt üks liiklusõnnetus/rikkumine). Need tulemused toetavad dopamiinergilise süsteemi olulist rolli impulsside kontrollimisel ja riskeeriva käitumise puhul. Lisaks, ATH seiretestis positiivse tulemuse saanute seas ei olnud oluliselt rohkem 9R kandlusega uuritavaid, kuid neid oli statistiliselt oluliselt rohkem uuritavate seas kellel oli nii positiivne seiretest kui ka kõrgem liiklusrisk.

Kindlasti võivad erinevad vaimse tervise häired nagu depressioon, ärevus ning aktiivsus ja tähelepanuhäire mõjutada käitumist liikluses. Käesolevas väitekirjas uurisime täpsemalt ATH sümptomite mõju liikluskäitumisele, sest ATH on seostatud kõrgema impulsiivsusega ning rohkemate õnnetustega liikluses. Tulemused näitasid, et enese raporteeritud ATH sümptomid on liikluskäitumise puhul oluline riskitegur. See omakorda viitab alaläviste vaimsete häirete olulisusele liiklusohtude teemal. Kindlasti tuleks kasuks autokoolides sõidukijuhtide õppesse kaasata arutelu ATH ja muude adekvaatset liikluskäitumist ohustavate häiretega kaasnevate riskide teemal, et tulevased sõidukijuhid oleksid teadlikumad enda vaimse tervise olulisusest autojuhtimise kontekstis.

Elustiiliga seotud näitajad nagu toitumisharjumised, alkoholi tarvitamine, füüsiline aktiivsus näitavad mitmeid erinevaid inimese arengu ja sotsiaalse staatusega seotud keskkondlike tegurite ning bioloogiliste eelsoodumuste koosmõjust tingitud otsuste tulemust. Seega ühe uurimisküsimusena tahtsime välja selgitada kas riskeerivam käitumine elustiili puhul väljendub ka riskeerivamas käitumises liiklusolukordades. Mitmed elustiili aspektid nagu rämpstoidu söömine ja füüsiline aktiivsus seostusid riskeeriva käitumise ja otseselt ka liiklusrikkumistega ning erinevus serotonergilises süsteemis 5-HTTLPR-i järgi oli neid seoseid vahendavaks teguriks. Tulevastes autokoolide ja muudes sarnastes sekkumistes võib olla kasulik võtta vaatluse alla tervisekäitumine laiemalt ning keskenduda võimalike vahendavate mehhanismide variatiivsusele.

Sekkumine autokoolides, mille läbiviijateks olid sõiduõpetajad, omas sarnaselt eelnevale psühholoogide poolt läbiviidud sekkumisele liiklusohutust suurendavat mõju. Seejuures mõju uuritavate liikluskäitumisele on uuringujärgselt kestnud kuni 3 aastat. Sekkumiste metoodika arenedes peaksid tulevased uuringud keskenduma veebikeskkonnas läbiviidavatele sekkumise instrumentidele, et selgitada kas efektiivsust on võimalik ka kaugõppe formaadis tagada.

Uurisime lisaks võimalust, et individuaalsed erinevused nagu ATH sümptomite olemasolu või bioloogiline eelsoodumus võivad mõjutavad impulsiivsuse alase sekkumise edukust. Selgus, et sekkumisrühma uuritavate vähesem tõenäosus olla kõrge liiklusriski grupis püsis statistiliselt oluline isegi pärast seda kui lisasime ATH sümptomeid hindava testi tulemused liiklusriski prognoosivasse statistilisse mudelisse. Bioloogiliste markerite osas esines võimalikke mõjusid ainult naissoost uuritavate puhul – 5-HTTLPR s'-alleeli kandjad kes olid sekkumisgrupis olid kõige ohutumad juhid üldse; samas *DAT1* 9R kandlus oli sekkumise efekti vähendavaks teguriks.

Alkoholi tarvitamise, kõrgema impulsiivsuse ja agressiivsusega seostatud bioloogilised markerid seonduvad usaldusväärselt igapäevase liikluskäitumisega ning alalävised vaimsed häired ATH näitel on liiklusriskide teemal olulisel kohal. Väitekirjas näidati ka elustiili ja riskeeriva liikluskäitumise vahelisi seoseid mis suunab tähelepanu käitumise olulisusele väljaspool liikluse konteksti. Tulemused viitavad ka sellele, et geneetilised erinevused võivad mängida rolli sekkumisprogrammide efektiivsuses ning kõigile ei pruugi sobida sama tüüpi sekkumisprogrammid. Veel, oleks kasulik lisada autojuhtide õppesse arutelu ATH ja muude liikluskäitumist mõjutada võivate vaimsete häirete üle, et inimesed oleksid rohkem teadlikud endast ja teistest tulenevate ohtude osas.

9. PUBLICATIONS

10. CURRICULUM VITAE

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Publications:

Tokko, T., Eensoo, D., Vaht, M., Lesch, K. P., Reif, A., & Harro, J. (2019). Relapse of drunk driving and association with traffic accidents, alcohol-related problems and biomarkers of impulsivity. *Acta Neuropsychiatrica,* 31(2), 84–92.

Luht, K., Tokko, T., Eensoo, D., Vaht, M., & Harro, J. (2019). Efficacy of intervention at traffic schools reducing impulsive action, and association with candidate gene variants. *Acta Neuropsychiatrica*, 31(3), 159–166.

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Tokko, T., Eensoo, D., Luht-Kallas, K., & Harro, J. (2022). Risk-taking in traffic is associated with unhealthy lifestyle: Contribution of aggressiveness and the serotonin transporter genotype. *Neuroscience Applied*, 1, 100110.

Tokko, T., Eensoo, D., & Harro, J. (2023). Driving anger dimensions as predictors of dangerous situations in traffic. In: Martin, C., Preedy, V. R., Patel, V. B. (eds) Handbook of Anger, Aggression, and Violence. Springer, Cham.

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Teaduspublikatsioonid:

Tokko, T., Eensoo, D., Vaht, M., Lesch, K. P., Reif, A., & Harro, J. (2019). Relapse of drunk driving and association with traffic accidents, alcohol-related problems and biomarkers of impulsivity. *Acta Neuropsychiatrica,* 31(2), 84–92.

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Tokko, T., Miškinyte, G., Eensoo, D., & Harro, J. (2022). Driving risks of young drivers with symptoms of attention deficit hyperactivity disorder: association with the dopamine transporter gene VNTR polymorphism. *Nordic Journal of Psychiatry*, 76(8), 575–583.

Tokko, T., Eensoo, D., Luht-Kallas, K., & Harro, J. (2022). Risk-taking in traffic is associated with unhealthy lifestyle: Contribution of aggressiveness and the serotonin transporter genotype. *Neuroscience Applied*, 1, 100110.

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