DISSERTATIONES MEDICINAE UNIVERSITATIS TARTUENSIS
131

DISSERTATIONES MEDICINAE UNIVERSITATIS TARTUENSIS
131

RISK-TAKING IN TRAFFIC AND MARKERS OF RISK-TAKING BEHAVIOUR IN SCHOOLCHILDREN AND CAR DRIVERS

DIVA EENSOO

TARTU UNIVERSITY
PRESS

Department of Public Health, University of Tartu, Estonia

Department of Psychology, University of Tartu, Estonia

Supervisor: Jaanus Harro, MD, PhD, Professor of Psychophysiology, University of Tartu, Estonia

Reviewers: Heidi-Ingrid Maaroos, MD, PhD, Professor of Policlinic and Family Medicine, University of Tartu, Estonia
Lembit Mehilane, MD, PhD, Consulting Psychiatrist, Tartu University Hospital, University of Tartu, Estonia

Dissertation is accepted for the commencement of the degree of Doctor of Philosophy in Medicine on December 6, 2006 by the Council of Medical Faculty

Opponent: Mati Heidmets, PhD, Professor of Social and Environmental Psychology, Tallinn University, Estonia

Commencement: February 1, 2007

Publication of this dissertation is granted by the University of Tartu

ISSN 1024–395X
ISBN 978–9949–11–528–0 (trükis)
ISBN 978–9949–11–529–7 (PDF)

Autoriõigus Diva Eensoo, 2007

Tartu Ülikooli Kirjastus
www.tyk.ee
Tellimus nr 690

CONTENTS

LIST OF ORIGINAL PUBLICATIONS

The thesis is based on the following original papers, which will be referred to in the text by Roman numerals (I–V):

I. Harro, M., Eensoo, D., Kiive, E., Merenäkk, L., Alep, J., Oreland, L., & Harro, J. (2001). Platelet monoamine oxidase in healthy 9- and 15-year-old children: the effect of gender, smoking and puberty. Progress in Neuro-Psychopharmacology and Biological Psychiatry 25, 1497–1511.

II. Eensoo, D., Harro, M., Pullmann, H., Allik, J., & Harro, J. Association of traffic behavior with personality and platelet monoamine oxidase activity in schoolchildren. Journal of Adolescent Health (in press).

III. Eensoo, D., Paaver, M., Pulver, A., Harro, M., & Harro, J. (2004). Low platelet MAO activity associated with high dysfunctional impulsivity and antisocial behavior: evidence from drunk drivers. Psychopharmacology 172, 356–358.

IV. Eensoo, D., Paaver, M., Harro, M., & Harro, J. (2005). Predicting drunk driving: contribution of alcohol use and related problems, traffic behaviour, personality and platelet monoamine oxidase (MAO) activity. Alcohol & Alcoholism 40, 140–146.

V. Paaver, M., Eensoo, D., Pulver, A., & Harro, J. (2006). Adaptive and maladaptive impulsivity, platelet monoamine oxidase (MAO) activity and risk-admitting in different types of risky drivers. Psychopharmacology 186, 32–40.

ABBREVIATIONS

ANOVA	analysis of variance
CI	confidence interval
DAD	driving after drinking
DWI	driving while impaired by alcohol
SD	standard deviation
MAO	monoamine oxidase
OR	odds ratio
PEA	phenylethylamine
PCR	polymerase chain reaction
SSRIs	selective serotonin reuptake inhibitors
5-HIAA	5-hydroxyindoleacetic acid
5-HT	serotonin
5-HTT	serotonin transporter
5-HTTLPR	serotonin transporter gene-linked polymorphic region

1. INTRODUCTION

Road traffic accidents are judged to be a public health issue worldwide. It is expected that by 2020 road traffic accidents will rank third after ischaemic heart disease and unipolar major depression among top causes of the burden of premature death and disability in the world (Murray and Lopez, 1997). Motor vehicle crashes are the leading cause of death for every age from 1 through 34 years in the United States (Moffat, 2001; Horan and Mallonee, 2003). Among the young people aged 15–24 years residing in the fifteen old member states of the European Union, unintentional injuries caused 183,308 deaths between 1984 and 1993, of which 84% were motor vehicle crashes (Morrison et al., 2000). Overall comparisons of road risk levels have most validity for countries with similar vehicle ownership ratios while an increased number of road motor vehicles increases the likelihood of accidents. Comparing some old and new European Union countries and the United States regarding road risk level in 2002 (Table 1.1), one can see that in the Slovak Republic, Hungary, Estonia, Latvia, and Lithuania the fatality rate of motor vehicle accidents was high while the number of motor vehicles per one thousand inhabitants was lower. At the same time, in the Netherlands, Sweden, the United Kingdom, Germany, Denmark, Finland, and Ireland the fatality rate was lower while the number of motor vehicles per one thousand inhabitants was higher. In the United States both indicators were high (European Conference of Ministers of Transport, 2004). The trend in the years 1990 to 2002 shows a decrease in the number of fatalities in the European Union countries, which is more favourable than a small decrease (4%) in the United States. One has to conclude that the new European Union countries have a worse traffic culture compared to the old Europe and the United States.

Most traffic fatalities are caused by risky traffic behaviour such as alcohol use by drivers, non-use of safety belts, child occupant restraints (Satcher, 2001), or reflectors on road in the dark (European Transport Safety Council, 2006), speeding (Shinar et al., 2001; Golias and Karlaftis, 2002), reckless riding on bicycles (Wesson et al., 2000) or motorbikes (Bastos et al., 2005; Lace and Goldstein, 2005), and unsafe crossing of roads (Tabibi and Pfeffer, 2003). If people with high risk traffic behaviour could be identified using markers of risk-taking behaviour, such as personality measures and corresponding biological markers, preventive programmes could be better targeted and more effective.

Table 1.1. Road risk levels in different countries

	Fatalities per million inhabitants in 2002	Number of road motor vehicles per one thousand inhabitants in 2002	Number of fatalities in 1990	Number of fatalities in 2002	Change in number of fatalities 2002/1990 (%)
Germany	83	589	11046	6842	–37.1
Denmark	86	428	634	463	–27.0
Estonia	164	358	436	223	–48.9
Finland	80	488	649	415	–36.1
Hungary	141	300	2432	1429	–41.3
Ireland	96	445	478	376	–21.4
Lithuania	201	386	933	697	–25.3
Latvia	222	315	946	559	–41.0
Netherlands	61	504	1376	987	–28.3
Sweden	63	500	772	560	–27.5
Slovak Republic	116	291	731	625	–14.6
United Kingdom	60	533	5217	3431	–34.3
United States	148	807	44599	42815	–4.1

2. REVIEW OF LITERATURE

2.1. Public health issues in traffic behaviour

Most traffic fatalities are caused by risky traffic behaviour, such as drunk driving, non-use of safety belts or child occupant restraints (Satcher, 2001), speeding (Shinar et al., 2001; Golias and Karlaftis, 2002; Mesken et al., 2002), not making oneself visible at night and in poor visibility (European Transport Safety Council, 2006), reckless riding on bicycles (Wesson et al., 2000) or motorbikes (Bastos et al., 2005; Lace and Goldstein, 2005), and unsafe crossing of roads (Tabibi and Pfeffer, 2003).

Although drunk driving has decreased in many countries during the past twenty years, alcohol consumption is still one of the main factors in road traffic accidents (European Transport Safety Council, 2001). In the United States approximately 40% of all traffic fatalities in 2001–2002 (Chou et al., 2006) and in Estonia one fifth of all traffic fatalities in 2001–2005 (Maanteeamet, 2006a; Maanteeamet, 2006b) were alcohol-related. An epidemiological survey found that 11.3% of American adults had been engaged in at least one drinking and driving behaviour (driving after drinking, driving after having drunk too much; riding with a drinking driver; riding as a passenger while drinking) (Chou et al., 2006). It has been shown that interventions among convicted drunk drivers reduced motor vehicle crashes and injuries (Dinh-Zarr et al., 1999), and such activities are needed.

Riding with a drinking driver was reported to have declined in the United States the 1980s, but the decline did not continue through the 1990s (Everett et al., 2001). The fact that among children who died in alcohol related crashes nearly two thirds were riding with a drunk driver (Quinlan et al., 2000; Shults, 2004) emphasizes the need for interventions to reduce this transport-related risk behaviour among young people.

It has been reported that overall safety belt use has risen over the past two decades: for example, in 2005, 82% of Americans wore safety belts while driving or riding in vehicles on sampled roadways (Glassbrenner, 2005), and in Japan seat-belt use among motor vehicle drivers was about 90–95% in 2000–2003 (Nakahara et al., 2006). Nevertheless, certain groups (e.g. teenagers, drunk drivers) consistently report lower than average usage rates (Dinn-Zarr et al., 2001). Among north-western American Indian children aged 8 years and younger, only 59% were restrained in vehicles (Lapidus et al., 2005). Young adults generally have the lowest seat belt use rates. The seat-belt use of children depends on the habits of adults: children are 3–4 times as likely to be unrestrained when riding with an unbuckled driver as when driving with a buckled driver (Glassbrenner et al., 2004). Safety belt use on the back seat is not much lower than on the front seat in some countries (e.g. 74–81% vs 81–95% in Finland) (Inseneribüroo “Stratum”, 2003; Helakorpi et al., 2005), but in others

the difference may be high (e.g. 1.4% vs 80–90% in Oman) (MciLvenny et al., 2004). In Estonia, the use of safety belts on front seat vs back seat has been reported to be 76–89% vs 23–24%, respectively (Inseneribüroo "Stratum", 2003; National Institute for Health Development, 2005). Experience of the United States has shown that safety belt campaigns and safety belt law have both steadily increased the use of seat belts (Glassbrenner et al., 2004), and further activities are needed focusing on the young people and seat belt use on the back seat.

Whether a driver speeds depends on whether there is an opportunity for speeding (e.g. do the vehicle capabilities, the road layout, and the conditions currently allow this), whether driving fast at this point would fit the lifestyle obligations being met by the trip agenda (e.g. "I am on my way to an important business appointment, and I am running late"; "I am conveying my own and other's children to a leisure activity and have a clear duty of care to these precious passengers"), and on the individual driver's attitudes, beliefs, and values driving the inclination to speed (e.g. "Yes! Driving fast just makes me feel good!"; "No! I do not feel in control if things are happening too quickly") (Stradling et al., 2004). A study has shown that 35% of car drivers who had been penalized for speeding in the last three years also reported having been involved in an accident, compared to 22% of those who had not been penalized (Stradling et al., 2004). Speeding violations are most often reported in young drivers (Shinar et al., 2001; Golias and Karlaftis, 2002; Mesken et al., 2002; Stradling et al., 2004), and women are more observant of traffic regulations than men (Shinar et al., 2001; Whissell and Bigelow, 2003). Young male drivers are more likely to be driving with their peers, and it is plausible that this peer association contributes to accidents. It has been suggested that it would be useful to direct the action on safe passengering as well as safe driving in driver training (Whissell and Bigelow, 2003).

Pedestrians and cyclists can be poorly seen in the road traffic, especially at night and poorly visible weather conditions. Without wearing something reflective, a pedestrian or cyclist is in low beam headlights likely to be visible only 30 meters away. By wearing something reflective they would become visible at 150 meters. This gives drivers five times the distance to notice them and to avoid them (European Transport Safety Council, 2006).

Driving a bicycle or a motorbike is common both for recreation and for transportation (Johansson and Drott, 2001; Pomerantz et al., 2005), and the risk that the use of motorbikes presents to health is increasing (Lace and Goldenstein, 2005). Of behaviours reported on routine health risk assessments, the strongest predictor of subsequent fatal accidents was motorcycle use (Wilson et al., 2003).

Studies of road-crossing behaviour have shown that children make decisions in road crossing in the same way as adults, but children are less good in judgement, and there were large individual differences (Pitcairn and Edlmann, 2000). The ability to identify safe and dangerous road-crossing sites increases

with age (Tabibi and Pfeffer, 2003). It has been suggested that overall differences in road-crossing behaviour in adults and children may be better explained by perceptual-motor abilities (adults have more experience with traffic) than by cognitive differences in their understanding of time and speed (Huber et al., 2003). Also children would be more sensitive to distractions than adults if the amount of irrelevant information increases in heavy traffic situations (Dempster and Corkill, 1999).

2.2. Personality and risk-taking in traffic

Estimations, attitudes, and behavioural decisions in everyday life, including traffic-related situations, are influenced by personality traits. Amongst the personality traits, risky behaviour is most consistently associated with impulsivity. The relationship between impulsivity and traffic behaviour has been studied since the late 1980s (Jonah, 1997), but not all of the accumulated evidence is consistent. However, it has not always been considered that impulsivity is a multifactorial construct and includes such facets as Novelty Seeking, Excitement Seeking, Sensation Seeking, Venturesomeness, Hyperactivity, Self-control and Disinhibition (Evenden, 1999). Impulsivity is defined by a scope of various tendencies including rapid and thoughtless action (Dickman, 1993; Barratt, 1993), risk-taking (Eysenck, 1993), low self-control, and inability to hold back one's desires (Buss et al., 1973; Costa and McCrae, 1989). In the context of everyday life, the approach of Dickman (1990), which differentiates Dysfunctional Impulsivity (tendency to act with less forethought than most people which leads the subject to difficulties) and Functional Impulsivity (tendency to act with little forethought when such a style is optimal), may bear particular significance.

Jonah (1997) reviewed studies done in the late 1980s and the early 1990s on the relationship between sensation seeking and risky driving (e.g. driving while impaired, speeding, following too closely, not wearing a seat belt), as well as its consequences (e.g. collisions, violations). The vast majority of the 40 studies reviewed showed a positive relationship between sensation seeking and risky driving. Among these studies the subscales of Zuckerman's Sensation Seeking Scale, Thrill and Adventure Seeking appears to have the strongest relationship to risky driving. Also in the later studies it has been shown that sensation seeking is positively associated with risky driving, such as speeding (Iversen and Rundmo, 2002), driving after drinking, and not wearing the seat belt (Jonah et al., 2001). Risk-taking in driving habits has shown a positive correlation also with the Aggression-Hostility trait according the Zuckerman-Kuhlman Personality Questionnaire (Zuckerman and Kuhlman, 2000) and with Anger (Parker et al., 2002; Deffenbacher et al., 2003). Several studies established an asso-

ciation between impulsivity and risky driving and motor vehicle crash injuries (Hilakivi et al., 1989; Wilson, 1992; Cherpitel and Tam, 2000).

Studies comparing drunk drivers and high-risk drivers with multiple non-alcohol related traffic violations concluded that there are distinct subgroups of people with risky driving habits (Donovan et al., 1985; Wilson, 1992). Regarding personality, they shared similar traits such as Impulsivity and Sensation Seeking, and drunk drivers were reported to be more deviant with regard to these features.

While impulsivity in car drivers has received considerable attention, the contribution of impulsivity to traffic accidents and law violations in cyclists and pedestrians has been studied less. In particular, there are only few studies on the association of personality and traffic behaviour in adolescents, but the available evidence suggests that impulsivity plays a significant role (Pitcairn and Edlmann, 2000). Rosenbloom and Wolf (2002) studied the association between road-crossing practices of 7-, 13- and 22-year-old subjects and facets of sensation seeking and found that among males the association between Thrill and Adventure Seeking and risky road-crossing was significant in all age groups. The strength of correlations increased with an increase in age. Females showed a strong association with Thrill and Adventure Seeking in 7- and 22-year-old subjects but not in the teenager group. It has also been found that children injured as bicyclists and pedestrians have higher scores in Impulsivity and Hyperactivity than controls (Pless et al., 1995).

2.3. Markers of risk-taking behaviour in the serotonin system

For a better understanding of the neurobiological basis of certain personality traits, such as impulsivity, sensation seeking, monotony avoidance, aggression, behavioural deviations (hyperactivity disorder, suicidality), attention has been focused on the serotonin (5-HT) system. Serotonergic activity of the central nervous system correlates inversely with human impulsive and aggressive behaviour. Markers of the serotonergic system in human and non-human tissues have been widely used in psychiatric and psychopharmacological research: tryptophan (LeMarquand et al., 1998; Lauterbach et al., 2006), several 5-HT receptor subtypes (de Boer and Koolhaas, 2005; Lauterbach et al., 2006), 5-HIAA concentration (Fahlke et al., 2002; Sher et al., 2005), enzyme monoamine oxidase (MAO) activity (Lesch and Merschdorf, 2000; Fahlke et al, 2002; Oreland, 2004), serotonin transporter (5-HTT) (Praschak-Rieder et al., 2005; Alexandre et al., 2006), 5-HTT gene polymorphism (Lesch et al., 1996; Retz et al., 2004; Beitchman et al., 2006).

2.3.1. Monoamine oxidase (MAO)

Monoamine oxidase is a protein in outer mitochondrial membranes, and there are two MAO isoenzymes, named type A and B (Chen and Shih, 1998). MAOs oxidatively deaminate the neurotransmitters dopamine, noradrenaline, and serotonin, as well as exogenous monoamines. The two isoenzymes have different inhibitor and substrate specificities: MAO-A is selectively inhibited by, fo example, clorgyline and has 5-HT and noradrenaline as preferred substrates, and MAO-B is selectively inhibited by L-deprenyl (selegiline), and dopamine and a number of exogenous monoamines such as phenylethylamine (PEA) as preferred substrates (Oreland, 1993). Most human tissues, including the brain, contain both isoenzymes, but platelet MAO is exclusively of the B-type and has the same amino acid sequence as MAO-B in the brain (Chen et al., 1993).

2.3.1.1. Platelet MAO activity, personality traits, and risk-taking

It has been shown that low platelet MAO activity is associated with several behavioural and psychiatric problems (Murphy and Weiss, 1972; Meltzer and Stahl, 974; Zureick and Meltzer, 1988), including tendencies of impulsive behaviour, alcohol dependence, and antisocial behaviour (Buchsbaum et al., 1976; von Knorring and Oreland, 1996; Zuckerman and Kuhlman, 2000; Oreland, 2004). According to the vulnerability hypothesis, low platelet MAO activity is associated with personality traits that increase the vulnerability to several psychiatric disorders and maladaptive behaviours such as drug abuse and social maladjustment (Buchbaum et al., 1976). Such personality traits as Impulsiveness, Sensation Seeking and Monotony Avoidance have been correlated with a low level of platelet MAO activity (von Knorring et al., 1984; Cloninger et al., 1988; von Knorring and Oreland, 1996; Oreland, 2004). Many studies have found low platelet MAO activity in highly risk-taking groups, such as gambling-dependent subjects, mountaineers, and criminals (Zuckerman and Kuhlman, 2000; Longato-Stadler et al., 2002; Oreland, 2004). Studies describing higher Impulsiveness in subjects with low MAO activity have often been carried out in subjects abusing drugs and alcohol (Longato-Stadler et al., 2002; Oreland, 2004). Some of the studies that have accounted for the inhibiting effect of smoking on platelet MAO activity have failed to confirm the relationship between impulsive behaviour and lower platelet MAO activity (Ward et al., 1987; Kiive et al., 2002). Because smoking prevalence in the subjects with risky health behaviour (drug and alcohol abusers, gambling-dependent subjects) might be rather high, smoking may confound the analysis. Furthermore, a longitudinal study has shown that subjects with low but also high platelet MAO activity are more likely to become smokers (Harro et al., 2004). Risky behaviours that are not related to alcohol or drug abuse are not so well studied with regard to their relation to platelet MAO activity or show no clear asso-

ciation with low platelet MAO activity. For example, air force pilots, despite their higher Impulsiveness score, had a similar MAO activity compared to the controls (af Klinteberg et al., 1992).

2.3.1.2. Gender, age, and smoking-related differences in platelet MAO activity

Platelet MAO activity has been reported to be on average 10–27% higher in adult females than in males (Murphy et al., 1976; Bridge et al., 1985; Oreland 1993, Snell et al., 2002; Coccini et al., 2005). In children the results have been inconclusive. Some authors have found no gender difference in platelet MAO activity (Shekim et al., 1982; Shekim et al., 1984) while others have found higher MAO activity in girls in comparison with boys (Murphy et al., 1976; Roth et al., 1976; Young et al., 1980). In adults platelet MAO activity is characterized by considerable stability within individuals over time, with a possible increase after the age of 40 (Murpy et al., 1976; Bridge et al., 1985; Bagdy and Rihmer, 1986). In children conclusive results have not been obtained yet. Some authors have not found any age-related differences in platelet MAO activity during the childhood and adolescence (Murphy et al., 1976; Shekim et al., 1984), others have shown that the level of platelet MAO activity is higher in children than in adults (Young et al., 1980), with the highest level in prepubescent children and an abrupt decrease in enzyme activity during the puberty to adult levels (Roth et al., 1976). It has been found that smokers have a significantly lower platelet MAO activity than non-smokers (Oreland et al., 1981; Norman et al., 1987), and some components in tobacco smoke inhibit MAO activity (Yu and Boulton, 1987; Fowler et al., 1996) in a dose-related manner (Whitfield et al., 2000). Therefore the effect of smoking has to be considered as a confounding factor in investigations on platelet MAO activity.

2.3.2. Serotonin transporter gene

The serotonin transporter (5-HTT), which is encoded by a single gene (SLC6A4) on chromosome 17q12, regulates the magnitude and duration of serotonergic signalling by governing the reuptake of serotonin from the synaptic junction. In humans, transcriptional activity of the 5-HTT gene is modulated by a polymorphic repetitive element (5-HTT gene-linked polymorphic region, 5-HTTLPR) located upstream of the transcription start site (Lesch et al., 1996). 5-HTT is the molecular site of action of many antidepressant drugs and several potentially neurotoxic compounds, such as Ecstasy. Serotonergic antidepressants, such as the tricyclic clomipramine and the selective 5-HT reuptake inhibitors (SSRIs) fluoxetine, fluvoxamine, paroxetine, citalopram, and sertraline occupy several pharmacologically distinct sites in the 5-HTT molecule overlapping at least partially the substrate binding site. These agents are widely

used in the treatment of depression, anxiety, and impulse control disorders, as well as substance abuse including alcoholism. SSRIs are uniquely efficacious in treating obsessive-compulsive disorders (Lesch and Gutknecht, 2005).

2.3.2.1. 5-HT transporter and its coding gene

The promoter region of the serotonin transporter gene contains a 44-base-pair insertion/deletion polymorphism that is located ~1 kb upstream of the transcription initiation site and influences the expression of this locus at the transcriptional level. Experiments have shown that the long allele of the 5-HTT gene has a more efficient promoter than the short allele, and that l/l genotype cells produce more 5-HTT mRNA and take up more serotonin from the medium than l/s or s/s genotype cells (Lesch et al., 1996).

2.3.2.2. Polymorphism of the 5-HTT gene, personality, and risk-taking

The 5-HTT gene plays an important role in violent and impulsive behaviour, and suicide, suggesting it as a candidate gene for borderline personality disorder (Retz et al., 2004; Ni et al., 2006). The association of 5-HTT gene polymorphism and personality has been explored in population and family-based genetic studies. 5-HTT gene polymorphism was originally shown to be associated with anxiety-related personality traits (Lesch et al., 1996). Individuals and siblings with s/l and s/s genotypes have scored higher in Neuroticism than individuals with l/l genotypes (Ricketts et al., 1998; Jang et al., 2001). Du et al. (2000) were able to replicate the finding of an association between 5-HTT gene polymorphism and Neuroticism only in a male population. Some studies, however have failed to find association between 5-HTT gene polymorphism and Neuroticism (Ebstein et al., 1997; Brummett et al., 2003). The individuals heterozygous or homozygous for the s allele have also been shown to be lower in Agreeableness (Lesch and Merschdorf, 2000; Jang et al., 2001), and this association was stronger in the female sample. The reasons for gender-related differences in the association between 5-HTT gene polymorphism and personality traits have been discussed, and possible effects of gonadal steroids on 5-HTT expression have been suggested (Fink et al., 1999; McQueen et al., 1999). A recent meta-analysis concluded, however, that if only healthy subjects are included, 5-HTT promoter polymorphism is not significantly associated with anxiety-related personality traits (Munafò et al., 2005a).

Among health-related behaviours, smoking has been studied mostly in association with 5-HTT gene polymorphism. The frequency of s/s genotype was significantly higher among smokers than in non-smokers (Gerra et al., 2005). To analyse the effects of 5-HTT gene polymorphism and Neuroticism on smoking behaviour, a study in a population of 759 never, current, and former smokers was carried out in North America (Hu et al., 2000). Smoking cessation was negatively associated with s/l and s/s genotypes and more prevalent with the l/l genotype. Individuals with s/l and s/s genotypes and a high level of

Neuroticism had the greatest difficulty in quitting smoking. This study showed that smoking behaviour is more strongly influenced by the combination of 5-HTT gene polymorphism and Neuroticism than by either factor alone. Also, Lerman et al. (2000) and Munafò et al. (2005b) found that smokers who scored high in Neuroticism had more frequently the short promoter variant.

3. AIMS OF THE PRESENT STUDY

The aims of the present study are listed as follows:

1. To characterize platelet MAO activity, a marker of CNS serotonergic neurotransmission, and the effect of smoking on platelet MAO activity in schoolchildren and in car drivers.
2. To examine the associations between traffic behaviour, personality, platelet MAO activity, and the polymorphism of the 5-HTT gene in adolescents.
3. To study impulsivity and platelet MAO activity among people with alcohol-related and non-alcohol-related driving violations.
4. To characterize the predictive value of socio-demographic data, alcohol consumption measures, smoking, platelet MAO activity, traffic behaviour habits, and impulsivity measures for drunk driving.

4. MATERIALS AND METHODS

4.1. Subjects

The dissertation is based on the data from two studies. The study on school-children used the sample of the European Youth Heart Study, which was incorporated into the longitudinal Estonian Children Personality Behaviour and Health Study (Papers I and II). Adult male subjects driving a car were studied in a project funded by the Estonian Road Administration to investigate the role of personality traits on traffic behaviour (Papers III, IV, and V). The author of the thesis participated in both studies during the preparatory stage, data collection, data analysis, and writing the papers.

4.1.1. Schoolchildren

The data of two groups of children, 9 years old (just before puberty) and 15 years old (in the last stages of their puberty), living in Tartu County were collected in 1998/1999 for a study of platelet MAO activity (Paper I). The main sampling unit was a school. Headmasters of 54 schools out of 56 with 9- and 15-year-old children in Tartu County agreed to participate in the study. A random sample of 25 schools was selected using cluster sampling (urban and rural schools with younger and older children from Estonian and Russian language schools) and probability proportional to school size. At each sampled school, all 9- and 15-year-old children were asked to participate in the study. Parents and children gave their written consent. Of all subjects invited to participate (n=1486), 79% of children (n=1176) and their parents agreed. Paper I includes the data from subjects whose platelet MAO activity was measured, 545 younger (264 boys and 281 girls) and 584 older (257 boys and 327 girls) children with mean age 9.6±0.5 (SD) and 15.5±0.6 years, respectively. The second study wave was performed with the group of younger children (9 years old in 1998/1999) six years later in 2004/2005 when they were 15 years old. Data on traffic behaviour and personality traits were collected, and blood samples drawn (Paper II). Of the 583 children who participated in the first study wave, 47 could not be contacted because they had changed schools and/or their addresses. Thus 536 adolescents were invited to participate and of them 90.1% agreed. Altogether, 483 children (222 boys and 261 girls) with the mean age of 15.3±0.5 years participated. Parents and children gave their written consent.

In the study on schoolchildren the subjects were divided according to the 75-percentile value of the personal traffic risks score (2.2 for boys and 2.1 for girls) into low (n=161 in boys, n=193 in girls) and high (n=54 in boys, n=64 in girls) risk subjects for the analysis (Paper II).

4.1.2. Car drivers

The data on adult male subjects driving a car were collected in 2001 (Papers III, IV, and V) and in 2002–2003 (Paper V) for studying traffic behaviour, impulsivity and platelet MAO activity according to different driving violations. The groups of drunk and speeding drivers were formed from the male subjects from the police database of driving violations. The control groups were formed from male subjects with a driving licence, who were derived from the driving-licence database of the Estonian Motor Vehicle Registration Centre by computerized random choice, and their police records were checked. The subjects were contacted by telephone, and the description and aims of the study were provided. Four hundred sixteen men (27% of the contacted people) agreed to participate in the study on drunk driving. Two men dropped out of the study at the stage of completing the questionnaires. Eight subjects recruited as controls had an earlier record of drunk driving in the police database and were moved to the drunk drivers group. The group of driving while impaired by alcohol (DWI) consisted of persons who were caught by the police driving drunk at least once during previous year (n=203, mean age±SD 33±11 years). The respective control group consisted of 211 persons, with a mean age of 36±12 years. In the study on speeding drivers, 610 men (33% of the contacted people) agreed to participate. One subject dropped out of the study at the stage of filling in the questionnaires. After checking the subjects for additional violations in the police database, thirteen subjects who had repeatedly exceeded speed limits, but additionally had been driving while drunk, were removed from the database. Speeding drivers were divided into two groups: 1) speeding drivers, subjects exceeding the speed limits less than 20 km/h at least twice or more than 20 km/h once during the previous year (n=127, mean age 38±11); 2) high-risk drivers, subjects exceeding speed limits over 20 km/h at least twice during the previous year (n=165, mean age 34±11 years). The size of the control group was 304 subjects, mean age 38±11 years. The control groups of the study on drunk driving and the study on speeding drivers were merged for comparative analyses; six persons from the control groups participated in both studies, their data for the years 2002–2003 were used. The size of the final merged control group was 509 subjects, mean age 37±12 years.

For a specific analysis the subjects were divided into six subgroups according to admitting driving too fast as a driving risk: 1) speeding drivers admitting the risk of speeding (n=38, mean age 34±10 years), 2) speeding drivers denying the risk of speeding (n=85, mean age 40±11 years), 3) high-risk drivers admitting the risk of speeding (n=58, mean age 31±8 years), 4) high-risk drivers denying the risk of speeding (n=104, mean age 36±12 years), 5) controls admitting the risk of speeding (n=72, mean age 36±12 years) and 6) controls denying the risk of speeding (n=220, mean age 38±11 years) (Paper V).

In order to analyse predictors of drunk driving (Paper IV) and to obtain more homogenous groups analysis, the subjects were divided into four subgroups

according to self-reported driving after drinking (DAD): Control I (n=129, mean age 36±13 years) – random choice from the driving-licence database who denied DAD during the previous year; Control II (n=81, mean age 34±10 years) – random choice from the driving-licence database who reported DAD some times or often per year; DWI I (n=45, mean age 37±14 years) – DWI subjects from the police database of driving violations who denied DAD during the previous year; DWI II (n=157, mean age 31±10 years) – DWI subjects from the police database of driving violations who reported DAD some times per year or often.

4.2. Methods

4.2.1. Assessment of traffic behaviour

Traffic behaviour was assessed in adolescents in the second study wave in 2004/2005 (Paper II) and in the studies with car drivers (Papers III, IV and V) by self-administered questionnaires.

The traffic behaviour questionnaire for the schoolchildren consisted of five items: (1) "frequency of using a seat belt in the front seat", (2) "frequency of using a seat belt in the back seat", (3) "frequency of using a reflector while moving on streets and roads in darkness", (4) "frequency of using pedestrian crossings on the way to school", and (5) "frequency of bicycle racing or motor-bike racing with cars in traffic". Participants responded on a five-point scale ranging from "1" – "always" to "5" – "never". In addition, a question about "riding with a drunk driver" was included, and the subjects responded "1" – "no", "2" – "do not know for sure" or "3" – "yes". We also asked about motor-bike driving ("1" – "no" or "2" – "yes"). Traffic risk-taking behaviours have been viewed as a separate health behaviour cluster (Wasylkiw & Fekken, 1999); therefore all 7 items were included in the traffic risk score. All items were standardized into z-scores [(Y – mean (Y))/SD (Y)] (separately for boys and girls), and they were summed. Responses to the question on racing were reversed. In case some activity missed during the past year (e.g. a considerable number of children had no need to cross a street on their way to school), the subject received "0" for that. On the basis of the 75-percentile value of the traffic risks score (separately for boys and girls), we divided the subjects into low and high traffic risk groups.

The traffic behaviour questionnaire in the drunk drivers study (Paper III, IV, and V) included questions about the frequency of car driving, using the seat belt, breaking the speed limits, paying for parking, stopping before pedestrians' crossings and overtaking the preceding car, all during the previous year. Also the duration of holding a driving licence was recorded. All the subjects were

asked about driving while impaired by alcohol during the previous year. Also their knowledge about time limits for safe drinking before driving was checked.

In the study with speeding drivers (Paper V) more specific questions about high-risk driving were added. Similarly to Iversen and Rundmo (2002), occurrence of driving faster to catch up on an appointment was included. For the polytomous logistic regression analysis, traffic behaviour habits were grouped into "never or rarely", "occasionally" and "more often". Answers to the question "on average how close to the actual speed limit do you usually drive?" (as in Bell et al., 2000) were categorized into "less than 11 km/h", "11–20 km/h" and "over 20 km/h". The frequency of exceeding the speed limits in built-up areas and on country roads (Iversen and Rundmo, 2002) was established separately as follows: 1) up to 20 km/h, 2) 21–40 km/h, 3) more than 40 km/h over the speed limit were scored separately for built-up areas and for country roads. Distracting activities during driving (five point scale from "never" to "very often") (minor modification of Hatakka, 1998) were recorded (I have competed in traffic, I have driven while tired, I have listened loud and rhythmic music while driving, I have made mobile-phone (not handsfree) calls while driving, I have gone for a drive to let off steam, I have been annoyed when another driver was driving slower than the speed-limit allowed) and scored. A list of several possible driving hazards (21 items included insufficient knowledge of traffic regulations, driving too fast, falling asleep while driving, overcareful driving, willingness to compete, too short following distance, carelessness; excitement or irritation, overselfconfidence, use of alcohol, insufficient vehicle handling skills, ignorance of traffic regulations, willingness to show off, forgetting to fasten seatbelt, tendency to get nervous, being hasty, insufficient knowledge of risks, willingness to take risks, playing with car, being short-tempered, being too insecure), based on the perceived driving risks questionnaire by Hatakka (1998), with "yes" or "no" answers were scored, leaving out the hazard of "driving too fast". Lifetime incidence of traffic accidents, where the driver was guilty, was recorded.

4.2.2. Collection of socio-demographic data

Socio-demographic data were collected in the studies with car drivers by a self-reported questionnaire (Papers III, IV, and V). The questionnaire included questions about age, marital status, education, monthly income, occupation and religion. For analysis we used the following categories: "single" and "living as a couple" for marital status, "elementary to high school" and "university" for education, "lower income" as monthly income below 10000 EEK (~641 EUR) and "higher income" as monthly income above 10000 EEK (~641 EUR), "white collar", "blue collar" and "other" for occupational status, "yes" and "no" for religious beliefs.

4.2.3. Assessment of smoking

Smoking was assessed in schoolchildren (Papers I and II) and in car drivers (Papers III, IV and V) using self-administered questionnaires.

Children (Paper I) reported whether they had ever tried a cigarette ("yes" or "no"), whether they currently smoked ("yes" or "no"), how often they smoked (I do not smoke, I smoke occasionally, I smoke once per week, I smoke almost every day or every day), and whether they considered themselves to be a non-smoker, occasional, or a daily smoker. A child was considered a smoker if he/she reported to smoke at least occasionally.

In the second study wave of children in 2004/2005 (Paper II), the number of smoked cigarettes during the previous seven days was recorded. According to the questions describing the frequency of smoking, the subjects were divided into non-smokers (had never smoked or had tried, but did not smoke currently) and smokers.

In the study on drunk driving (Papers III and IV) all the subjects were divided on the basis of self-reports into non-smokers, ex-smokers, and smokers smoking 10 or fewes cigarettes per day, 11–19 cigarettes per day, and 20 or more cigarettes per day.

The study on different driving violations (Paper V) coded smoking status as "smokers" and "non-smokers" (subjects who had never smoked and ex-smokers).

4.2.4. Assessment of alcohol use habits

Alcohol use habits were assessed in the study on drunk driving (Papers III and IV) using a self-administered questionnaire. The amount of alcohol consumed during the past week was reported separately for different alcoholic drinks and was expressed in grams of pure alcohol. The score of alcohol-related problems was obtained by summing five questions based on DSM-IV diagnostic criteria for alcohol abuse concerning specific life events (having "turned aggressive while drunk", having "had longer periods of alcohol use", having "had conflicts with friends and family", having "been absent from work", and having "lost one's job"; reported as present or not, total score 0–5) (Papers III and IV). Questions concerned the frequency of using strong and light alcoholic drinks during the past year on a six-point scale (none, some times per year, one to three times per month, one to two times per week, three to four times per week, almost every day) were used (Paper V).

4.2.5. Personality measures

Personality measures were assessed in adolescents (Paper II) and in car drivers (Papers III, IV and V) using self-administered inventories.

Adolescents (Paper II) filled in the Estonian Personality Item Pool NEO (EPIP-NEO; Mõttus et al., 2006), which is a 240-item measure of the five major personality domains: Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness. Russian-speaking students filled in the 240-item Russian version of the Revised NEO Personality Inventory (Martin et al., 2002). The two inventories measure the same domains very similarly (Gow et al., 2005; Mõttus et al., 2006), and the personality data were unified by means of z-scores computed separately by gender.

Different facets of impulsivity were measured with a short instrument based on the Dickman Impulsivity Inventory (Dickman, 1990) and impulsivity-related subscales of the NEO Personality Inventory (NEO-PI, Costa and McCrae, 1989, adapted for Estonian by Pulver et al., 1995) (Papers II, III, IV, and V). Altogether four scales were created: Thoughtlessness and Fast Decision Making based on the Dickman Inventory and Disinhibition and Excitement Seeking based on NEO-PI subscales. Each of the four scales consisted of 6 items*.

4.2.6. Measurement of platelet MAO activity

Platelet MAO activity was measured in platelet-rich plasma by a radioenzymatic method with [^{14}C]-β-phenylethylamine (β-PEA) ("Amersham") as substrate, as described by Hallman et al. (1987) (Papers I, II, III, IV and V). Blood samples were collected by antecubital venepuncture into 4.5 ml Vacutainer® tubes containing EDTA as an anticoagulant. The samples were centrifuged (Jouan BR4i) for 10 min with 800 rpm, obtaining platelet-rich plasma. Part of the obtained plasma (100 µl) was used for counting platelets with Sysmex SE-9000 in a certified clinical laboratory of the Clinicum of the University of Tartu. One ml of platelet-rich plasma was stored at –80°C until the measurement of MAO activity. After melting the platelet-rich plasma on ice, platelets were sonicated with Bandelin Sonopuls Ultrasonic Homogenizer HD2070 4 x 10 s with intervals for 5 s at 4°C. Then, 50 µl of 0.1 mM [^{14}C]-β-PEA was mixed with 50 µl of sonicated plasma, following 4 min incubation in 37°C water bath. After that, 30 µl of 1.0 M HCl was added to stop the reaction and all the tubes were put onto an ice bath for another 10 minutes. After adding 750 µl solution of toluene and ethylacetate (1:1), all

* In the earlier stages of the studies on impulsivity, Thoughtlessness was referred to as Dysfunctional Impulsivity (as in Papers III, IV, and V), Fast Decision Making as Functional Impulsivity (as in Papers III, IV, and V), Disinhibition as Impulsivity (as in Papers III and IV), and Disinhibition as Impulsiveness (as in Paper V).

the samples were mixed on a shaker (Vibromax 110, Heidolph) for 30 s at 1700 rpm, and thereafter centrifuged for 5 min at 2000 rpm. From the organic phase 500 µl was pipetted into vials with 8 ml of scintillation liquid (Optiphase "HiSafe"3, Wallac). For standard samples 50 µl of 0.1 mM [^{14}C]-β-PEA was added to 8 ml of scintillation cocktail. All the samples were analysed in duplicate and blindly and corrected using a reference sample. Radioactivity was measured in a β-counter (Wallac Guardian 1414 Liquid Scintillation Counter). MAO activity was calculated using the following formula: [the amount of the substrate (nmol) x β-count of the sample (cpm) x 1.5]/[β-count of the standard (cpm) x incubation time (min) x the count of platelets in 50 µl of platelet-rich plasma (10^{10} of platelets)], and expressed as nmol of substrate oxidized per 10^{10} platelets per min (nmol x min^{-1} x 10^{10} $platelets^{-1}$). Platelet MAO activity was standardized into z-scores for regression analysis (Paper II). After logarithmic transformation of platelet MAO activity for normality, parametrical statistical methods were used (Papers III and IV).

4.2.7. Genotyping of the 5-HTT gene

DNA was extracted from peripheral blood and genotype of the promoter region of 5-HTT was determined by polymerase chain reaction (PCR) amplification and agarose gel electrophoresis as described previously (Nilsson et al., 2005). The polymorphic region was amplified using the primers: 5-HTTLPR-F: CAA CCT CCC AGC AAC TCC CTG TA, 5-HTTLPR-R: GAG GGA CTG AGC TGG ACA ACC AC, where the forward primer was labelled with a 5'-FAM. Reagents and conditions for the PCR included 1 x PCR buffer (Perkin Elmer, AmpliTaq Gold buffer II), 200 µM dNTP with 50% of dGTP replaced with 7-deaza-dGTP, 2 mM $MgCl_2$, 1 µM of each primer, 1U Taq polymerase (Perkin Elmer, AmpliTaq Gold), and 20 ng genomic DNA, in a total reaction volume of 10 µl. The reaction started with 10 min at 95ºC, followed by 35 cycles with 30 s at 95ºC, 30 s at 59ºC, 30 s at 72ºC, and ended with 10 min at 72ºC. PCR products were then run on an ABI 3100 Genetic analyser (Applied biosystems), and scored using the software GeneMapper 1.5 (SoftGenetics). All genotypes were manually checked on chromatograms to detect inconsistencies, and where needed, amplified and scored a second time. Genotype frequencies were in Hardy – Weinberg equilibrium. The subjects were grouped according to whether they were homozygous for the long allele (n=194, 43.1%) or were carriers of the short allele (n=256, 56.9%).

4.2.8. Statistical analysis

Statistical analysis was performed using StatView (version 4.5) (Papers I, II, and IV), SAS (version 9.1) (Papers II, III, IV and V) and STATISTICA (version 4.12) (Papers III and V).

Nominal variables were described using frequency tables, and Pearson's Chi-Square test with post hoc analysis of individual cell contributions was used to compare different groups (Papers II and IV). Continuous variables are expressed as means and standard deviations (SD) or 95% confidence intervals (CI) for variables normally distributed, or as median and range for variables not in normal distribution. The Spearman correlation analysis (Paper IV) and the t-test procedure were used to compare different groups (Papers II and III). If a significant difference was found with analysis of variance (ANOVA), Scheffe's (Paper V) or Fisher's least-significant-difference (LSD) (Papers I, IV, and V) post-hoc test was used. Multivariate analysis of variance was used for comparing the groups by two variables (Papers I, III, IV, and V). The Kruskal-Wallis (Papers I, IV, and V) and the Mann-Whitney U-test (Papers I and III) were used for comparing variables not in normal distribution. Simple and multiple logistic regression analysis were used for computing odds ratios belonging to the high traffic risk group (Paper II). To identify the combination of quantitative variables predicting driving while impaired by alcohol, discriminant analysis was performed (Paper IV). Polytomous logistic regression was used to analyse traffic behaviour and socio-demographic variables in different traffic risk groups (Paper V).

Values of $p<0.05$ were considered statistically significant.

5. RESULTS AND DISCUSSION

5.1. Effect of gender, age, and smoking on platelet MAO activity (Papers I and III)

Platelet MAO activity was measured in children and adolescents (Papers I and II) and in car drivers (Papers III, IV and V).

Paper I describes gender differences in platelet MAO activity. Platelet MAO activity was significantly lower in boys than in girls in both younger and older children. Previously, similar gender-related differences in platelet MAO activity were found in adults (Murphy et al., 1976; Bridge et al., 1985; Oreland, 1993, Snell et al., 2002; Coccini et al., 2005). Some studies reported similar gender differences in children (Murphy et al., 1976; Roth et al., 1976; Young et al., 1980) while others did not find any differences between boys and girls (Shekim et al., 1982; Shekim et al., 1984). All previous studies concerning MAO activity in children and adolescents had been carried out on very small samples of subjects (Murphy et al., 1976; Roth et al., 1976; Shekim et al., 1984), and often with psychiatric or other patients (Shekim et al., 1982; Young et al., 1980; Rogeness et al., 1985; Filinger et al., 1987; Cederblad et al., 1992). The results of the present study were obtained in a randomly selected large sample of healthy children and adolescents and suggest that the gender differences of early age are reliable.

The effect of age on platelet MAO activity was found only in girls, whereas 9-year-old girls had a significantly lower platelet MAO activity than 15-year-old girls. When the smokers were excluded from the analysis, the difference in platelet MAO activity between younger and older boys also became significant (Paper I). These data suggest that there is a slight increase in platelet MAO activity between 9 to 15 years. A significant increase in platelet MAO activity was observed from the age of 15 to 18 years in the longitudinal study by Kiive et al. (2005), but it may have been caused by methodological issues. Some authors found positive correlations between age and MAO activity (Robinson, 1975; Berlin et al., 2000), but other authors consider that platelet MAO activity is stable within individuals over time (Murphy et al., 1976; Bridge et al., 1985; Bagdy and Rihmer, 1986). It has been claimed that the variation in platelet MAO activity of the same individual over time is rather small, whereas in these studies the time between two platelet MAO activity measurements ranged from 8 to 10 weeks. Furthermore, as these studies of platelet MAO activity were done on adults, there is a possibility that MAO activity does not reach stability during adolescence.

In 9-year-old children, no statistically significant difference was found in MAO activity between smokers and non-smokers. While smoking among 9-year-old children was very low (2.4%), and the children who smoked at least occasionally were considered to be smokers, it is not surprising that the effect of

smoking was not sufficient to reduce platelet MAO activity significantly. In 15-year-old children, a significant effect of smoking on MAO activity was found – both in older boys and girls smoking reduced significantly platelet MAO activity. Among 15-year-old children, 24.5% were smokers (Paper I). In adult male subjects smoking had a dose-dependent effect on platelet MAO activity (Paper III). However, one can not exclude that in adolescents platelet MAO activity is more sensitive to smoking than in adults. It is known that some components of tobacco smoke inhibit MAO activity, and thus the reduced MAO activity in smokers reflects a pharmacological effect not related to the intrinsic characteristics of the 5-HT-ergic system (Yu and Boulton, 1987; Fowler et al., 1996). At the same time, subjects with low platelet MAO activity are more likely to become smokers (Harro et al., 2004). One can conclude that the association between smoking and platelet MAO activity includes two independent aspects: 1) among the subjects who have not started to smoke or smoke occasionally or with low intensity, lower platelet MAO activity reflect their proneness to smoke, and 2) if subjects smoke with high intensity, the tobacco smoke inhibits platelet MAO activity directly, but this effect is limited.

5.2. Traffic behaviour in schoolchildren (Paper II)

For teenagers not wearing a seat belt (Dinn-Zarr et al., 2001), driving with a drunk driver, reckless riding on a bicycle or a motorbike (Everett et al., 2001), unsafe crossing of roads (Pitcairn and Edlmann, 2000, Rosenbloom and Wolf, 2002), and non-use of reflectors on road in the dark (European Transport Safety Council, 2006) are considerable traffic risk factors.

Table 1 of Paper II presents an overview of traffic behaviour of the sample. There were no differences between boys and girls in the frequency of using seat belts in either seat, in using a reflector while moving on streets and highways in darkness, or in using pedestrian crossings on their way to school. However, a significant difference appeared in the frequency of racing with cars in traffic on a bicycle or motorbike, the boys having more frequently raced in traffic.

Seat belt use was roughly similar to the previous studies of adolescents in other countries (Grunbaum et al., 2000; Everett et al., 2001; Glassbrenner et al., 2004; Ruangkanchanasetr et al., 2005). It was found that seat belt use on the front vs back seats was remarkably different in adolescents – while 77% of the participants reported front seat belt use “always” or “almost always”, only 46% used seat belts on the back seat consistently. Seat belt use in the sample was also similar to the data of Finbalt Health Monitoring in Estonia in 2004 (National Institute for Health Development, 2005). Both front and back seat belt use were lower than in Finland (Helakorpi et al., 2005) but higher than in Latvia (Pudule et al., 2005) or Lithuania (Grabauskas et al., 2005). Regional differences in seat belt use at the population level have clearly been shown to be

significantly associated with effective seat belt safety interventions, including legislation, but the efficacy of such preventive measures in influencing seat belt use in teenagers has been found less reliable (Dinn-Zarr et al., 2001; Glass-brenner et al., 2004).

During the darkness periods pedestrians are not sufficiently visible to drivers, and traffic accidents are more probable where pedestrians move along the roads without a reflector. In Finland in 2003 more than half of all the accidents with pedestrians occured during dark hours. It has been estimated that at least six of the 55 pedestrian lives would have been saved had they worn a reflector (European Transport Safety Council, 2006). In Latvia, 65% of the vulnerable road users' deaths occurred in darkness, and the frequency of serious accidents in darkness was twice higher than in daylight (European Transport Safety Council, 2006). In the present study 44% of the teenagers reported using a reflector regularly, which is more than in youngest study groups in the national samples in both Estonia and Finland, and much more than in Latvia and Lithuania. A possible reason for this high prevalence is that the study was carried out in a region with a relatively high level of education of the population.

In this study 10% of the subjects reported that they "never" or "mostly not" use pedestrian crossings on their way to school, and boys tended to be more risk-taking than girls. It is consistent with a study of pedestrians, which found that up to one tenth of them cross the streets at undesignated locations, males committing significantly more violations than females (Rosenbloom et al., 2004).

Driving with a drunk driver during the previous year showed a similar proportion in both gender groups 28.9%, n=63 and 28.0%, n=73 for boys and girls, respectively. Higher prevalence was found in the United States where this behaviour was reported by 36.6% of 9 – 12-grade students (Everett et al., 2001), and in Thailand 18.8% students in grades 7 to 12 reported this behaviour (Ruangkanchanasetr et al., 2005) during the previous month. Riding with a drinking driver was reported to have declined in 1980s, but the decline did not continue through the 1990s (Everett et al., 2001). The fact that among children who died in alcohol-related crashes nearly two thirds of them had riddden with a drinking driver (Quinlan et al., 2000; Shults, 2004) emphasizes the need for interventions to reduce this transport-related risk behaviour among young prople.

During the previous year many more boys than girls had ridden a motorbike (60.3%, n=132 and 36.5%, n=95, respectively; χ^2=26.86, p<0.0001). About two thirds of boys and one third of girls had been driving a motorbike. Also more boys than girls engaged significantly frequently in such high-risk behaviour as bicycle or motorbike racing with cars in traffic. Taking into account that more than 40% of bicycle-related fatalities and injuries are among the youth aged 10–19 years (Everett et al., 2001) safety campaigns among teenagers and

policies targeting safer traffic behaviour are needed, and they should consider gender (Shinar et al., 2001; Goloias and Karlaftis, 2002).

5.3. Association of traffic behaviour in schoolchildren with personality, platelet MAO activity (Paper II) and a polymorphism of the 5-HTT gene (unpublished data)

According to the reported traffic behaviour, schoolchildren were divided into low (n=354) and high (n=118) risk subjects. The high traffic risk group was more likely to have higher Disinhibition, Excitement Seeking, and Thoughtlessness, and lower Openness, Agreeableness, and Conscientiousness than the low personal traffic risk group according to simple logistic regression analysis (Paper II, Table 2). Significantly lower Openness and higher Disinhibition were found in high-risk boys but not in girls, whereas only high-risk girls had higher Excitement Seeking and Fast Decision Making. These results show that girls with high-risk traffic behaviour were more likely to have higher scores in all facets of impulsivity. In boys, high-risk traffic behaviour was positively associated with Disinhibition and Thoughtlessness, the traits that describe the maladaptive side of impulsivity and have been characterized as inability to plan and think one's actions through, which leads to negative consequences (Dickman, 1993).

High traffic risk girls were more likely to have lower platelet MAO activity, also after checking the effect of smoking. The association between high traffic risk and low platelet MAO activity did not appear in boys (Paper II). Many studies have found low platelet MAO activity in risk groups, such as gambling-dependent persons, mountaineers, criminals, and subjects with psychiatric disorders (Zuckerman and Kuhlman, 2000; Oreland, 2004). Some, but not all of these findings could be related to the direct effect of smoking on enzyme activity.

The association between traffic risk and the promoter polymorphism of the 5-HTT gene did not appear in children in overall simple logistic regression analysis (OR, 95%CI: 1.23, 0.79–1.92) or in boys and girls analysed separately (OR, 95%CI: 1.69, 0.87–3.27 and 0.95, 0.52–1.73, respectively). The results show that there is no direct association between traffic risk behaviour and 5-HTT gene polymorphism.

In multiple logistic regression analysis, when adjusting for smoking, high-risk girls were more likely to have lower platelet MAO activity and higher Excitement Seeking (Paper II, Table 3). High-risk boys were more likely to have lower Agreeableness (OR, 95%CI: 0.48, 0.30–0.79) and higher Thoughtlessness (OR, 95%CI: 1.74, 1.12–2.70), and the short allele of the 5-HTT gene was overrepresented in this group (OR, 95%CI: 2.48, 1.08–5.70). The analysis shows that boys and girls have different biological substrates associated with

risky traffic behaviour. Platelet MAO activity, one of the markers of the capacity of the 5-HT-ergic system, was associated with risky traffic behaviour in girls, whereas the promoter polymorphism of the 5-HTT gene was associated with risky traffic behaviour in boys. There are many studies on associations between polymorphism of the 5-HTT gene, Neuroticism measured with the five-factor model, and health-related behaviour such as smoking. It has been found that smoking behaviour or difficulty in quitting smoking is more strongly influenced by the combination of the s/l and s/s genotypes and a high level of Neuroticism than by either factor alone (Hu et al., 2000; Lerman et al., 2000; Munafò et al., 2005b). The reason why this study showed a significant association in the combination Agreeableness, Thoughtlessness, and traffic risk in boys may be explained by the significantly negative correlation between personality traits Neuroticism and Agreeableness (r=–0.31, p<0.0001), and a positive correlation between Neuroticism and Thoughtlessness (r=0.31, p<0.0001). Different studies have described lower platelet MAO activity and higher Excitement Seeking or Sensation Seeking in different risk-taking behaviours (Zuckerman and Kuhlman, 2000; Longato-Stadler et al., 2002; Oreland, 2004). It has been suggested that males and females carrying the short 5-HTT gene allele may react differently to the environmental stimuli (Sjöberg et al., 2006).

5.4. Traffic behaviour in car drivers (Papers IV and V)

When comparing the traffic behaviour of different driving violation groups such speeding, high-risk, and drunk drivers with the controls, no differences were found in the duration of holding the driving licence, paying for parking, and stopping before a zebra crossing (data not shown). Statistically significant differences in odds ratios appeared for frequencies of car driving and using the seat belt; speeding exceeders and high-risk drivers differed from the controls by the frequency of catching up the preceding car even when it kept to the appropriate speed (Table 1 of Paper V). High-risk drivers, as well as drunk drivers, were more likely to use the seat belt rarely than the controls. High-risk drivers, similarly to speeding drivers, were more likely to drive more frequently and to catch up preceding cars more often compared to the controls. Thus, the study showed that different types of risky driving could be differentiated by violation patterns. Speeding and high-risk drivers reported more frequent catching up the preceeding car, which confirms that police-referred high-risk drivers actually exceed speed limits more often.

More specific questions about high-risk driving were added to the questionnaire in the study on speeding drivers, and the traffic behaviour of driving violation groups of speeding and high-risk drivers could thus be further characterized. Both risk groups were more likely to exceed the speed limit to a higher

extent than the controls. High-risk drivers, when compared to the controls, were more likely to break the speed limit more frequently on country roads, to engage more frequently in distracting activities during car driving, and to have more lifetime incidences of traffic accidents where the driver was guilty. The risk groups did not differentiate from the controls by the frequency of exceeding the speed limits in built-up areas, by the frequency of driving faster to catch up on an appointment, and by perceived individual risks (Table 5.1). These data show the relationship of self-reported traffic behaviour and the police database – speeding drivers were subjects who exceeded the speed limits less than 20 km/h at least twice or more than 20 km/h once during the previous year, and they did not differentiate from the controls by self-reported traffic violations as much as the high-risk drivers, who exceeded speed limits over 20 km/h at least twice during the previous year.

Table 5.1. Likelihood of being a penalized speeding driver or a high-risk driver vs control depending on self-reported traffic behaviour

	Adjusted odds ratio (95% CI)	
	Speeders	High-risk drivers
Extent of exceeding the speed limit	**1.30 (1.03–1.64)**	**1.26 (1.01–1.56)**
Frequency of exceeding the speed limits in built-up areas	0.92 (0.52–1.64)	0.99 (0.59–1.69)
Frequency of exceeding the speed limits on country roads	1.23 (0.69–2.19)	**1.82 (1.07–3.08)**
Frequency of distracting activities during driving	1.29 (0.79–2.13)	**1.80 (1.13–2.85)**
Lifetime incidence of traffic accidents, where the driver was guilty	0.95 (0.75–1.21)	**1.25 (1.03–1.52)**
Frequency of driving faster to catch up on an appointment	1.21 (0.96–1.50)	1.06 (0.87–1.30)
Perceived individual risks	0.72 (0.45–1.17)	0.90 (0.58–1.40)

Significant differences compared to the controls are in bold type.

The next research question was whether traffic behaviour might differentiate high-risk drivers and the corresponding controls according to admitting the risk of "driving too fast". In this analysis the subgroup of controls denying the risk of speeding were used as the reference group (Table 2 of Paper V). Two thirds of the drivers who had been repeatedly caught by the police violating traffic rules denied the risk accompanying this kind of behaviour. The groups did not differ regarding the duration of holding the driving licence, the frequencies of exceeding the speed limits, catching up the preceding car, paying for parking, stopping before a zebra crossing, and exceeding the speed limits in built-up areas (data not shown). Statistically significant odds ratios appeared for

frequencies of driving, using the seat belt, and exceeding the speed limit on country roads and for the sake of catching up on an appointment, the extent of exceeding the speed limits, and the score of acknowledged personal driving risks. All three groups were more likely (or had such a tendency) to drive more often and to exceed the speed limits to a higher extent compared to the controls denying the risk of speeding. Risk-admitting high-risk drivers were more likely to exceed the speed limits more frequently on country roads and for the sake of catching up on an appointment. High-risk drivers denying the risk of speeding were less likely to use the seat belt compared to the risk-denying controls, which may signal about their less perceived accident risk due to driving too fast. Risk-admitters (of driving too fast) were about three to four times more likely to have a higher score of the acknowledgement of (other) personal driving risks. The Kruskal-Wallis analysis of variance of self-reported traffic accidents revealed a significant group difference (H=10.87, p<0.05) in the number of accidents on their own fault, with medians as follows: risk-denying controls, median=1 (range 0–4, mean=0.67, SD=0.79), risk-admitting controls, median=1 (range 0–4, mean=1.00, SD=1.02), risk-denying high-risk drivers, median=1 (range 0–4, mean=0.97, SD=1.13, and risk-admitting high-risk drivers, median=1 (range 0–8, mean=1.34, SD=1.63). The further pair-wise post-hoc tests revealed that risk-admitting high-risk drivers and risk-admitting controls had a higher number of life-time incidence of accidents on their own fault, compared to the risk-denying controls (H=6.58, p<0.05 and H=5.53, p<0.05, respectively). Indeed, it appeared that risk-denying high-risk drivers had not experienced as many accidents as risk-admitting high-risk drivers. Among the risk-denying high-risk drivers the score of acknowledged individual driving risks was similar to the controls denying the risk of speeding. At the same time risk-denying high-risk drivers violated the traffic rules more often by police records and self-reports than the controls denying the risk of speeding.

Next it was checked whether traffic behaviour may differentiate drunk drivers and the controls according self-reported driving after drinking (Paper IV). Both police database and self-reports were used to identify people who had been driving while drunk during the previous year. It is well known that in the case of violating rules, people might want to show themselves in a socially desirable way, thus the research based only on self-reported questionnaires for behaviour in traffic might not be fully reliable. At the same time not all the drunk drivers are caught by the police. Therefore, the controls and the drunk drivers were separated according to whether they reported or denied drunk driving. The four groups did not differ by frequency of stopping before a zebra crossing to allow the pedestrians to cross the road and the frequency of over-taking (data not shown). Statistically significant differences between the groups appeared in the frequency of driving, duration of holding the driving licence, frequency of using the seat belt, frequency of exceeding the speed limits, frequency of paying for parking, and knowledge of time limits for safe drinking before driving (Table 2 of Paper IV). Most of the studied men drove almost

every day. A few men in the DWI groups reported that they had stopped to drive or that they drive without a driving licence. In DWI II the number of subjects having held the driving licence for two years or less was three or more times higher than in other groups. Regarding the frequency of using the seat belt, the subjects of the DWI II group answered more often “mostly not” and the Control I group more often “almost always” compared to the other groups. As to the frequency of exceeding the speed limits, the subjects of the Control I and also the DWI I group answered more often “mostly not”, and the subjects of the DWI II group answered more often “occasionally”. Regarding the frequency of paying for parking, the subjects of the Control I group answered more often “almost always”, and the subjects of the DWI II group answered more often “occasionally”. Subjects who were caught driving while impaired and reported it in the questionnaire (DWI II group), reported more often their generally careless traffic behaviour and violating other traffic rules as well. They reported using the seat belt and paying for parking more rarely, and exceeding the speed limits more often. This group also included more subjects who had been driving without a valid driving licence, and more novice drivers. Similar associations between drunk driving and low seat belt use, speeding (Golias and Karlaftis, 2002), driving without a driving licence (Baum, 2000; Begg et al., 2003), and novice driving (Cooper et al., 1995) have been described by the other investigators.

Altogether two thirds of subjects did not know about safe time limits for drinking before driving. The difference between the groups was minimal, but this knowledge was somewhat better among the subjects of the DWI II group. Knowledge of safe time limits for drinking before driving was rather low, which suggests that such information should be included in DWI prevention programmes. A survey of drunk-driving offenders has also shown that the knowledge of safe drinking levels for open licence holders was low, less than 50% of the respondents of both offender and community samples providing correct answers (Baum, 2000). It has been shown that knowledge of drunk driving improved after participating in a DWI prevention programme (Kayser et al., 1995). In the present study, the DWI I group subjects had a somewhat lower level of knowledge of time limits for safe drinking before driving than the other groups, which might be the reason for underreporting their drunk driving. By contrast, in the DWI II group the knowledge of time limits for safe drinking before driving was slightly better in this study compared to the other groups. It might be explained by the fact that more subjects had held a driving licence for two years or less in the DWI II group, and these drivers might remember the time limits for safe drinking before driving from their studies. However, this knowledge is not reflected in their actual driving habits.

5.5. Impulsivity in car drivers (Papers IV and V)

Figure 5.1 Part A presents impulsivity data standardized into z-scores in drunk drivers, speeding drivers, high-risk drivers, and the controls. It is known that younger people in general score slightly higher in impulsivity (Eysenck et al., 1985). As in most analyses the covariate effect of age was significant, the effect of age is already taken into account in the data given on the figures. There were significant differences between the groups in Disinhibition, Thoughtlessness, Excitement Seeking and Fast Decision Making ($F_{3,1000}$=9.19, $F_{3,1000}$=9.42, $F_{3,1000}$=9.67, and $F_{3,1000}$=7.17, respectively, p<0.0001 for all the differences). The covariate effect of age was not significant for Thoughtlessness, but it was significant for Disinhibition (ß=–0.22), and after taking into account the effect of age the group difference in Disinhibition remained significant ($F_{3,1000}$=8.30, p<0.0001). There was a strong covariate effect of age on the group effect on Excitement Seeking (ß=–0.40) and a weak effect on Fast Decision Making (ß=–0.09). After considering the effect of age the difference between the groups in Fast Decision Making and in Excitement Seeking were still significant ($F_{3,1000}$=8.69, p<0.0001; $F_{3,1000}$=6.00, p<0.001, respectively). The maladaptive aspects of impulsivity Disinhibition and Thoughtlessness were higher in drunk drivers and Thoughtlessness in high-risk drivers as compared to the controls. The adaptive facets of impulsivity, Fast Decision Making, and Excitement Seeking were higher only in high-risk drivers. Thus high-risk drivers had higher scores in both adaptive impulsivity (Fast Decision Making and Excitement Seeking) and also in maladaptive impulsivity (Thoughtlessness). Risky driving has been associated with several problematic traits such as Normlessness, Driver Anger (Iversen and Rundmo, 2002), and Disinhibition (Jonah, 1997). It has been suggested earlier that the variety inside the risky drivers group is high; thus risk-takers need to be divided into subgroups, for example, according to sex, age, or personality (Wilson, 1992). The present study showed remarkable variety among traffic risk-takers, where drunk drivers, although similar to high-risk drivers in Thoughtlessness, differed from the latter in Fast Decision Making and Excitement Seeking. In earlier studies, which found that drunk drivers and high-risk drivers share personality traits, the focus was not on different aspects of impulsivity (Donovan et al., 1985, Wilson, 1992). Higher Excitement Seeking among high-risk drivers can be compared with the earlier research on higher Sensation Seeking in high-risk drivers (Zuckerman, 1994).

The next question analysing impulsivity in car drivers was whether differences in impulsivity in the different groups of drivers would depend on risk admission. The following analysis dealt with high-risk drivers and the corresponding controls, omitting the speeders who did not differ by impulsivity from the control group. Figure 5.1 Part B shows the standardized impulsivity scores of high-risk drivers and the controls denying and admitting the risk of

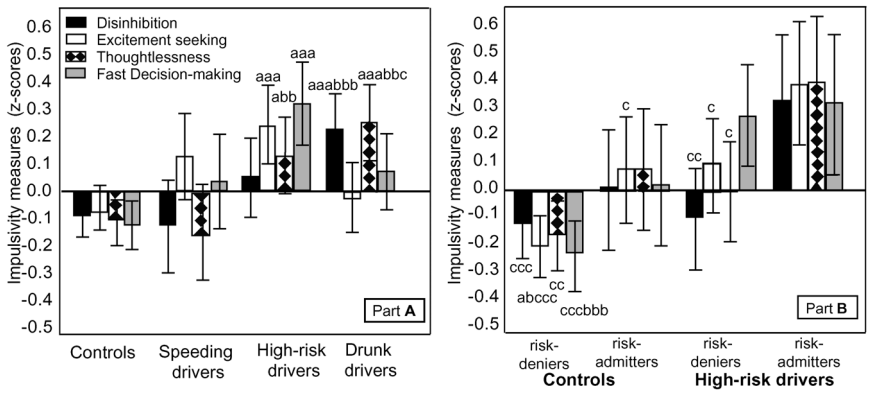

Figure 5.1.

Part A. The types of impulsivity in speeding drivers, high-risk drivers, and drunk drivers compared to controls.

The difference from controls: $^{a}p<0.05$, $^{aa}p<0.01$, $^{aaa}p<0.001$. The difference from speeding drivers: $^{bb}p<0.01$, $^{bbb}p<0.001$. The difference from high-risk drivers: $^{c}p<0.05$.

Part B. The types of impulsivity in high-risk drivers and controls denying and admitting the risk of driving too fast. The difference from risk-admitting controls: $^{a}p<0.05$. The difference from risk-denying high-risk drivers: $^{b}p<0.05$, $^{bbb}p<0.001$. The difference from risk-admitting high-risk drivers: $^{c}p<0.05$, $^{cc}p<0.01$, $^{ccc}p<0.001$.

Whiskers mark 95% confidence intervals.

driving too fast. Risk-admitting high-risk drivers showed a higher score than the sample's mean in all impulsivity facets, the difference extending approximately to half of the standard deviation. The groups were significantly different by Disinhibition ($F_{3,450}$=5.89, p<0.001), Thoughtlessness ($F_{3,450}$=5.60, p<0.001), Excitement Seeking ($F_{3,450}$=13.25, p<0.0001), and Fast Decision Making ($F_{3,450}$=10.52, p<0.0001). The covariate effects of age were significant for the group effect on Excitement Seeking (ß=–0.36), Disinhibition (ß=–0.18), and Fast Decision Making (ß=–0.12), but the differences remained significant after correction ($F_{3,450}$=8.26, p<0.0001, $F_{3,450}$=5.25, p<0.01, and $F_{3,450}$=8.85, p<0.0001, respectively). Risk-admitting high-risk drivers had a higher score than all the other groups in Excitement Seeking. Both risk-denying and risk-admitting high-risk drivers had higher Fast Decision Making compared to the risk-denying controls. Risk-admitting high-risk drivers had higher Disinhibition and Thoughtlessness than the risk-denying controls and higher Disinhibition than risk-denying high-risk drivers. The risk-admitting controls and risk-denying high-risk drivers had higher Excitement Seeking than the risk-denying controls. Thus, two thirds of drivers who had repeatedly been caught violating the traffic rules by the police denied the risk accompanying this kind of behaviour. Both adaptive aspects of impulsivity were higher among risk-denying high-risk drivers compared to the risk-denying controls. One third of the high-risk drivers exceed speed limits despite acknowledging the risk

involved. It has been concluded that the knowledge of personal driving risks is not a sufficient reason for avoiding them (Hatakka, 1998). Safety problems may exist not only because drivers are unaware of the risk but because of time pressure, ego gratification, inattention, tension relief (Gabany et al., 1997), and also different levels of subjectively perceived and objective crash-risks (Harre, 2000). Maladaptive impulsivity and Excitement Seeking were far more deviant in risk-admitting high-risk drivers. It has been claimed that risk-taking behaviour is most probable in people combining high sensation seeking and impulsive decision-making style (Donohew et al., 2000). The combination of different types of impulsivity may lead to acknowledged risk-taking cumulatively – Excitement Seeking is responsible for one's need for new and exciting stimuli while an impulsive cognitive style does not allow the person inhibiting these needs. Earlier studies compared two groups of angry drivers – the ones who report their reckless driving as a problem and the ones who do not – and in the first group anger was more intense and frequent (Deffenbacher et al., 2003). It has been found that drivers with the intention to speed are characterized rather by general deviance than a tendency to underestimate the negative consequences (Lawton et al., 1997).

While studying impulsivity in drunk drivers the question was whether drivers differ by impulsivity on the basis of self-reported driving after drinking among DWI subjects and the controls (Paper IV). The groups did not differ significantly in Fast Decision Making, which showed only a tendency to be higher in DWI groups compared to the control groups. Statistically significant differences between the groups appeared in the measures of Thoughtlessness ($F_{3,408}$=8.55, p<0.0001), Disinhibition ($F_{3,408}$=8.37, p<0.0001), and Excitement Seeking ($F_{3,408}$=3.69, p=0.012) (Table 5.2). The scores of Thoughtlessness, Disinhibition and Excitement Seeking were higher in the DWI II group compared to other groups. The subjects of the DWI II group were younger than in other groups. Age had a significant covariate effect ($F_{3,408}$=19.29, p<0.0001) on Disinhibition, but the group effect remained significant ($F_{3,408}$=6.41, p<0.0001). The significant difference between the groups in Excitement Seeking disappeared after taking age into account (the covariate effect of age on Excitement Seeking was $F_{3,408}$=77.95, p<0.0001, and the group effect was not significant $F_{3,408}$=1.35, p=0.3). Drunk drivers had higher Thoughtlessness and Disinhibition, which both represent the maladaptive side of impulsivity. Thoughtlessness is a feature of cognitive style supporting quick and thoughtless action in complex situations, where this kind of information processing leads to errors and inaccuracy (Dickman, 1993). This information-processing style might bring about their inadequate evaluation of the risk when deciding to drive after drinking. Fast Decision Making defined as adaptive and situation-specific was not associated with drinking and driving. The difference between the groups in Disinhibition, described by problems with controlling one's wishes and impulses, decreased after taking age into account. The difference between

the groups in Excitement Seeking appeared to be mostly due to the younger average age of the group of drunk drivers.

Table 5.2. The types of impulsivity (mean ± SD) according the self-reported driving after drinking in convicted drunk drivers and control groups

	Control I (n=129)	Control II (n=81)	DWI I (n=45)	DWI II (n=157)
Disinhibition	16.3±4.6	17.8±4.0	17.9±4.9	18.8±4.0 [a]
Excitement Seeking	20.0±4.9	20.6±5.5	18.7±5.7	21.4±5.2 [a b]
Thoughtlessness	13.9±4.6	15.4±4.5	15.5± .6	16.8±4.9 [a]
Fast Decision Making	19.6±4.8	19.7±4.7	20.4±3.6	20.3±4.2

[a] significant difference from the group Control I $p<0.05$, [b] significant difference from the group DWI I $p<0.05$; Fisher's LSD post hoc test was used.

5.6. Predicting drunk driving by socio-demographic data, alcohol consumption measures, smoking, platelet MAO activity, traffic behaviour habits, and impulsivity measures (Paper IV)

To identify the combination of quantitative variables that differentiate drunk drivers from the controls the most distinct groups (Control I group and DWI II group) were used in discriminant analysis (Paper IV). The analysis included socio-demographic data, alcohol consumption measures, smoking, traffic behaviour habits, impulsivity measures (Paper IV, Tables 1 and 2), and platelet MAO activity – the variables that showed significant differences between drunk drivers and the control groups by Pearson's Chi Square test or by ANOVA. By the final model alcohol related problems, the frequency of using strong and light alcoholic drinks during the previous year, seat belt use, smoking, paying for parking, Thoughtlessness, platelet MAO activity, age, and the amount of consumed alcohol in past seven days had significant loadings in the discriminant function (Paper IV, Table 3). Canonical discriminant analysis revealed the emergence of Wilks's Lambda 0.57, $F_{9,268}=22.8$, $p<0.0001$. The number of observations and the proportion of cases classified correctly into the first class (Control I group) was 105 (83%) and for the second class (DWI II group) 123 (81%). The drunk drivers and the controls were classified correctly using this discriminant function for the groups Control II and DWI I 60 (76%) and 36 (80%) observations, respectively. Most of the variables describing socio-demographic background did not appear as independent predictors of drunk driving in discriminant analysis, which suggests that their significance is closely related to other factors. Several studies have found that drunk driving is associated with lower income (Baum, 2000; Golias and Karlaftis, 2002), lower

education, and is more frequent among blue-collar workers (Baum, 2000). However, some studies did not find any association between drunk driving and socio-economic measures (Wilson and Jonah, 1985; Grunewald, 1996). In the present study younger age predicted drunk driving compared to the controls. Studies of drunk drivers who had caused traffic accidents, as well as population studies, show that younger drivers tend to drive after drinking more often (Wilson and Jonah, 1985; Grunewald, 1996; Johnson et al., 1998; Golias and Karlaftis, 2002; Chou et al., 2006). Among alcohol-related variables the best discriminating indicators appeared to be alcohol-related problems and the frequency of consuming alcohol. Alcohol impairs driver's reaction time and ability to estimate risks adequately, and drunk driving is considered a serious violation of traffic law (Tzambazis and Stough, 2000). Quantity (Wilson and Jonah, 1985) or frequency (Grunewald et al., 1996; Baum, 2000) of alcohol consumption or both (Johnson et al., 1998) are related to drunk driving and the probability of alcohol-related injury. It has been found, that drunk driving incidents are more frequent among drivers having alcohol-related problems (Del Rio et al., 2001).

Among the traffic behaviour variables drunk drivers and the controls were discriminated significantly by seat belt use and paying for parking. Lower seat belt use among drunk drivers has been described by Golias and Karlaftis (2002). The association between drunk driving and paying for parking has rarely been described; however, drunk drivers have been found to break other traffic rules more frequently (Wilson and Jonah, 1985; Baum, 2000; Golias and Karlaftis, 2002; Begg et al., 2003) so it is not surprising that drunk drivers nip paying for parking more often than the controls.

Thoughtlessness, which appeared to be the best predictor of drunk driving, is a feature of cognitive style supporting quick and thoughtless action in complex situations, where this kind of information processing leads to errors and inaccuracy (Dickman, 1990). This information-processing style might bring about inadequate estimation of the risk when deciding to drive after drinking. In previous studies Thoughtlessness that is similar to trait Impulsivity appeared to be associated with risk-taking and rule breaking as well as alcohol-related problems (von Knorring and Oreland, 1996). Impulsivity is defined by a scope of various tendencies including rapid and thoughtless action (Dickman, 1993; Barratt, 1993), risk-taking (Eysenck, 1993), low self-control, and inability to hold back one's desires (Buss et al., 1973; Costa and McCrae, 1989). The discriminant analysis identified smoking and platelet MAO activity as separate significant factors in predicting drinking and driving. Platelet MAO activity is directly reduced by tobacco smoking (Norman et al, 1987; Yu and Boulton, 1987; Whitfield et al., 2000), and the present analysis considered the effect of smoking. At the same time smoking was an independent predictor of drunk driving. It is known that several types of risky behaviour may be significantly correlated (Caspi et al., 1997; Zuckerman and Kuhlman, 2000). Previous studies have shown a link between platelet MAO activity and risky behavioural

tendencies (Fowler et al., 1980), social maladaptation (Oreland, 2004), and alcohol dependence (von Knorring and Oreland, 1996). Studies on alcohol-dependent subjects have found that low MAO activity in platelets is associated with type 2 alcoholism (Cloninger et al., 1988; von Knorring and Oreland, 1996), which is described by several social complications and an early debut of the abuse (Oreland, 2004). The ability of platelet MAO activity to predict central serotonergic activity was recently further confirmed by the finding that there is a strong correlation between cerebrospinal fluid levels of the serotonin metabolite 5-HIAA and platelet MAO activity (Fahlke et al., 2002). These authors also found that rhesus macaques with low platelet MAO activity were less competent socially and consumed alcohol to excess. In the present study platelet MAO activity was negatively correlated (r=–0.20, p<0.0001) with alcohol-related problems but not with any measures of alcohol consumption. The findings of this study also lead to an assumption that MAO activity is more strongly related to the tendency for alcohol-related problem behaviour than to the tendency to consume more alcohol.

Discriminating the drunk drivers and controls by the pattern of alcohol-related problems, the frequency of using strong and light alcoholic drinks during the previous year, safety-belt use, smoking, paying for parking, Thoughtlessness, platelet MAO activity, age, and alcohol consumption suggests that drunk driving is a resulting combination of various behavioural, biological, and personality-related risk factors.

5.7. Association of platelet MAO activity with alcohol-related and non-alcohol-related driving violations (Paper V)

The mean values and standard deviations of platelet MAO activity in the groups were as follows: speeding 7.85±3.0 and high-risk drivers 7.46±3.4, drunk drivers 6.60±4.1, and controls 7.76±3.4 nanomoles of substrate oxidized per 10^{10} platelets per minute. The mean MAO activity of speeding and high-risk drivers did not differ from the controls. MAO activity was significantly lower compared to the controls in drunk drivers ($F_{3,976}$=5.69, p<0.001), and the difference remained significant among non-smokers ($F_{3,561}$=4.00, p<0.01). Platelet MAO activity is a peripheral marker of central serotonergic activity (Fahlke et al., 2002; Oreland 2004). Central serotonergic activity is associated with alcohol abuse (LeMarquand et al., 1994) as well as with impulsive behavioural tendencies (Evenden, 1999). Knowing the strong interrelation between of impulsivity and alcohol abuse, one may infer that while low platelet MAO activity is clearly associated with alcohol-related impulsivity or impulsivity leading to alcohol abuse, non-alcohol-related impulsive behaviour does not have a clear association with MAO activity. The present study found that

other types of high-risk drivers with multiple non-alcohol-related driving violations, while being more impulsive than the controls, shared a similar MAO activity with the controls, and drunk drivers had a lower platelet MAO activity. At the same time there were no significant linear correlations between platelet MAO activity and Excitement Seeking, Disinhibition, Fast Decision Making, or Thoughtlessness. The fairly large sample of the present study was derived from a normal healthy population, unlike the majority of studies carried out on psychiatric populations with impulsivity as one symptom in their pathology. Af Klinteberg et al. (1992), observing higher levels of self-reported impulsivity in pilots, showed that at the same time they did not present any deviances in neuropsychological tests of disinhibition or platelet MAO activity. In another study, Longato-Stadler et al. (2002) revealed that criminal offenders with accompanying personality disorders and problems of drug abuse had a lower platelet MAO activity while platelet MAO activity other criminal offenders was similar to the controls. All the available evidence suggests that platelet MAO activity is linked with impulsivity in a sub-population of people with drug problems.

High-risk drivers admitting the risk of driving too fast were more deviant in all types of impulsivity as well as self-reported traffic behavioural indices than the risk-deniers. Analysis of high-risk drivers, speeding drivers, and controls according to their admission of traffic risk having driven too fast revealed a significant interaction effect on platelet MAO activity between the groups ($F_{5,558}$=3.40, p=0.005). The effect remained significant among non-smokers ($F_{5,352}$=2.54, p<0.05) (Figure 2, Paper V). Post-hoc analysis revealed that risk-admitting high-risk drivers and risk-admitting speeding drivers had a higher MAO activity than the corresponding risk-denying group and both risk-admitting and risk-denying controls. A previous study described in a small (n=40) sample with undefined smoking status that subjects with intermediate platelet MAO activity are more conforming while high and low MAO activity subjects have low social desirability (Schalling et al., 1987). However it remains unclear how such tendencies would influence self-acknowledgement of risks. Another possibility is to interpret high platelet MAO activity in risk-admitting high-risk drivers in the context of previous research suggesting an association of high platelet MAO activity with anxiety (Irving et al., 1989; Davidson et al., 1980) and neuroticism (Kirk et al., 2001) and obsessive-compulsive disorder (Cath et al., 2001). Anxiety in certain cases brings along impulsive behaviour (Wallace et al., 1991) as people may engage in risky behaviours to relieve their anxiety. Ulleberg (2001) identified two types of traffic risk-takers on the basis of different personality profiles – one high in Sensation Seeking and low in Anxiety, and the other high in both. These subtypes were also different by risk-evaluations – the latter group perceiving the traffic risks higher. Thus, the risk-admitting high-risk drivers with higher

platelet MAO activity might have been similar to the subgroup of Anxious Sensation-Seekers in Ulleberg's study (2001).

The finding that risk-taking and law-breaking behaviour such as drunk driving, repetitive speeding, and other driving violations can be related to lower as well as higher platelet MAO activity is in accordance with the previous studies showing non-linear associations of behaviour or personality traits and monoaminergic functioning. Several items of the Eysenck impulsivity scale (I_7) were shown to be associated with platelet MAO activity either lower or higher than average (Schalling et al., 1988). Similarly to Impulsivity, the association between platelet MAO activity and Anxiety shows a tendency for non-linearity (Schalling et al., 1987) – for example, subjects with high MAO activity being the most anxious and subjects with low MAO activity being slightly less anxious, but more anxious than subjects with medium MAO activity (Irving et al., 1989). Harro et al. (2004) have demonstrated in a prospective longitudinal study that the likelihood of starting to smoke is higger among adolescents with lower and higher MAO activity. Af Klinteberg et al. (1987) showed that male subjects with lower as well as higher platelet MAO activity show more disinherited responses in neuropsychological tests (low MAO subjects responding faster to new stimuli and high MAO subjects spending less time inspecting the labyrinth). This is also in accordance with the animal studies showing that serotonergic activity is associated with impulsivity in several, sometimes opposite, ways (Evenden, 1999). In the study by Kirk and colleagues (2001) the positive association between MAO activity and Neuroticism was strengthened when adjusted to the effect of smoking. Therefore, actual associations between such traits as Neuroticism and high platelet MAO activity may actually have remained hidden in many studies by the direct effect of smoking.

It has been found earlier that while some performance measures of impulsivity (e.g. commission errors in the vigilance task and delay errors in the delay task) are associated with higher platelet MAO activity in boys with disruptive behaviour disorders, the self-report scales of impulsivity show no associations with MAO activity (Stoff et al., 1989). While laboratory performance tests measure narrow cognitive tendency to respond quickly and make mistakes, questionnaire measures contain a much wider meaning, including, for example, sociability, emotional and motivational dimensions. The finding that behavioural impulsivity – driving at high speeds and admitting the associated risk – was related to higher platelet MAO activity, while results of the self-reported impulsivity scales did not, is comparable with the afore-mentioned result and suggests that behavioural measures can be more reliable in further studies on the relationship between serotonergic activity and impulsivity.

6. CONCLUSIONS

The main conclusions of present study are listed as follows:

1. A marker of CNS serotonergic neurotransmission, platelet MAO activity was significantly lower in boys than in girls. There was a slight increase in platelet MAO activity during adolescence. Smoking had inhibiting effect on platelet MAO activity.
2. Risky traffic behaviour in schoolchildren was associated with basic personality dimensions, most consistently with higher Agreeableness.
3. In girls with a high traffic risk platelet MAO activity was lower if compared to girls with a low traffic risk.
4. In boys with a high traffic risk the short allele of the 5-HTT gene when associated with lower Agreeableness and higher Thoughtlessness was overrepresented if compared to boys with a low traffic risk.
5. Drunk driving was associated with higher scores of maladaptive types of impulsivity (Thoughtlessness and Disinhibition) and high-risk driving more with higher scores of adaptive types of impulsivity (Fast Decision Making and Excitement Seeking). Speeding drivers did not differ from the controls by impulsivity measures.
6. Drunk drivers had lower platelet MAO activity, speeding drivers and high-risk drivers shared similar platelet MAO activity with the controls. Risk-admitting of "driving too fast" high-risk drivers and risk-admitting speeding drivers had a higher MAO activity than the corresponding risk-denying groups and both risk-admitting and risk-denying control goups.
7. Drunk drivers were discriminated from control group by higher score of alcohol-related problems, more frequent alcohol use, bigger amount of alcohol consumed, more frequent smoking, lower seat belt use, rarely paying for parking, higher score of Thoughtlessness, lower platelet MAO activity and younger age.

7. REFERENCES

Alexandre, C., Popa, D., Fabre, V., Bouali, S. Venault, P., Lesch, K.P., Hamon, M., & Adrien, J. (2006). Early life blockade of 5-hydroxytryptamine 1A receptors normalizes sleep and depression-like behavior in adult knock-out mice lacking the serotonin transporter. Journal of Neuroscience 26, 5554–5564.

Bagdy, G., & Rihmer, Z. (1986). Measurement of platelet monoamine oxidase activity in healthy human volunteers. Acta Physiologica Hungarica 68, 19–24.

Barratt, E.S. Impulsivity: integrating cognitive, behavioral, biological and environmental data. In: McCown, W.B., Johnson, J.L., & Shure, M.B., editors. The Impulsive Client: Theory, Research and Treatment. Washington, D.C.: American Psychological Association, 1993, pp 39–56.

Bastos, Y.G.L., de Andrade, S.M., Soares, D.A., Matsuo, T. (2005). Seat belt and helmet use among victims of traffic accidents in a city of southern Brazil, 1997–2000. Public Health 119, 930–932.

Baum, S. (2000). Drink driving as a social problem: comparing the attitudes and knowledge of drink driving offenders and the general community. Accident Analysis and Prevention 32, 689–694.

Begg, D.J., Langley, J.D., & Stephenson, S. (2003). Identifying factors that predict persistent driving after drinking, and driving after using cannabis among young adults. Accident Analysis and Prevention 35, 669–675.

Beitchman, J., Baldassarra, L., Mik, H., De Luca, V., King, N., Bender, D., Ehtesham, S., & Kennedy, J. (2006). Serotonin transporter polymorphisms and persistent, pervasive childhood aggression. Amceican Journal of Psychiatry 163, 1103–1105.

Bell, N.S., Amoroso, P.J., Yore, M.M., Smith, G.S., & Jones, B.H. (2000). Self-reported risk-taking behaviors and hospitalization for motor vehicle injury among active duty army personnel. American Journal of Preventive Medicine 18(3S), 85–95.

Berlin, I., Spreux-Voroquaux, O., & Launy, J.M. (2000). Platelet monoamine oxidase B activity in inversely associated with plasma cotinine concentration. Nicotine and Tobacco Research 2, 243–236.

de Boer, S.F., & Koolhaas, J.M. (2005). 5-HT_{1a} and 5-HT_{1B} receptor agonists and aggression: a pharmacological challenge of the serotonin deficiency hypothesis. European Journal of Pharmacology 526, 125–139.

Bridge, T.P., Soldo, B.J., Phelps, B.H., Wise, C.D., Franak, M.J., & Wyatt, R.J. (1985). Platelet monoamine oxidase activity: demographic characteristics contribute to enzyme activity variability. Journal of Gerontology 40, 23–28.

Brummett, B.H., Siegler, I.C., McQuoid, D.R., Svenson, I.K., Marchuk, D.A., & Steffens, D.C. (2003). Associations among the NEO Personality Inventory, revised and the serotonin transporter gene-linked polymorphic region in elders: effects of depression and gender. Psychiatric Genetics 13, 13–18.

Buchsbaum, M.S., Coursey, R.D., & Murphy, D.L. (1976). The biochemical high-risk paradigm: behavioural and familial correlates of low platelet monoamine oxidase activity. Science 194, 339–341.

Buss, A.H.U., Plomin, R., & Willerman, L. (1973). The inheritance of temperaments. Journal of Personality 41, 513–524.

Caspi, A., Begg, D., Dickson, N., Harrington, H., Langley, J., Moffitt, T.E., & Silva, P.A. (1997). Personality differences predict health-risk behaviors in young adult-

hood: evidence from a longitudinal study. Journal of Personality and Social Psycholology 73, 1052–1063.
Cath, D.C., Spinhoven, P., Landman, A.D., & Kempen, G.M.J. (2001). Psychopathology and personality characteristics in relation to blood serotonin in Tourette's syndrome and obsessive-compulsive disorder. Journal of Psychopharmacology 15, 111–120.
Cederblad, M., Oreland, L., & Zachrisson, E. (1992). Thrombocyte monoamine oxidase activity and behavior deviances in adolescence. Developmental Pharmacology and Therapeutics 18, 184–190.
Chen, K., Wu, H.F., & Shih, J.C. (1993). The deduced amino acid sequences of human platelet and frontal cortex monoamine oxidase B are identical. Journal of Neurochemistry 61, 187–190.
Chen, K., & Shih, J.C. (1998). Monoamine oxidase A and B: structure, function and behaviour. Advances in Pharmacology 42, 292–296.
Cherpitel, C.J., & Tam, T.W. (2000). Variables associated with DUI offender status among whites and Mexican Americans. Journal of Studies on Alcohol 61, 698–703.
Chou, S.P., Dawson, D.A., Stinson, F.S., Huang, B., Pickering, R.P., Zhou, Y., & Grant, B.F. (2006). The prevalence of drinking and driving in the United States, 2001–2002: results from the national epidemiological survey on alcohol and related conditions. Drug and Alcohol Dependence 83, 137–146.
Cloninger, C.R., Sigvardsson, S., & Bohman, M. (1988). Childhood personality predicts alcohol abuse in young adults. Alcoholism: Clinical and Experimental Research 12, 494–505.
Coccini, T., Randine, G., Castoldi, A.F., Balloni, L., Baiardi, P., & Manzo, L. (2005). Lymphocyte muscarinic receptors and platelet monoamine oxidase-B as biomarkers of CNS function: effects of age and gender in healthy humans. Environmental Toxicology and Pharmacology 19, 715–720.
Cooper, P.J., Pinili, M., & Chen, W. (1995). An examination of the crash involvement rates of novice drivers aged 16 to 55. Accident Analysis and Prevention 27, 89–104.
Costa Jr, P.T., & McCrae, R.R. The NEO-PI/NEO-FFI Manual Supplement. Odessa, F.L.: Psychological Assessment Resources, 1989.
Davidson, J.R.T., McLeod, M.N., Turnbull, C.D., White, H.L., & Feuer, E.J. (1980). Platelet monoamine oxidase activity and the classification of depression. Archives of General Psychiatry 37, 771–773.
Deffenbacher, J.L., Lynch, R.S., Filetti, L.B., Dahlen, E.R., & Oetting, E.R. (2003). Anger, aggression, risky behaviour, and crash-related outcomes in three groups of drivers. Behaviour Research and Therapy 41, 333–349.
Del Rio, M.C., Gonzalez-Luque, J. C., & Alvarez, F.J. (2001). Alcohol-related problems and fitness to drive. Alcohol and Alcoholism 36, 256–261.
Dempster, F.N., & Corkill, A.J. Neo-interference research and the development of intelligence. In: Anderson, M., editor. The Development of Intelligence. Hove, UK: Psychology Press, 1999.
Dickman, S.J. (1990). Functional and dysfunctional impulsivity: personality and cognitive correlates. Journal of Personality and Social Psychology 58, 95–102.
Dickman, S.J. Impulsivity and information processing. In: McCown, W.B., Johnson, J.L., & Shure, M.B., editors. The Impulsive Client: Theory, Research and Treatment. Washington, D.C.: American Psychological Association, 1993, pp 151–184.

Dinh-Zarr, T., Diguiseppi, C., Heitman, E., & Roberts, I. (1999). Preventing injuries through interventions for problem drinking: a systematic review of randomized controlled trials. Alcohol & Alcoholism 34, 609–621.

Dinh-Zarr, T.B., Sleet D.A, Shults R.A, Zaza, S., Elder, R.W., Nichols, J.L., Thompson, R.S., Sosin, D.M., & the Task Force on Community Preventive Services (2001). Reviews of evidence regarding interventions to increase the use of safety belts. American Journal of Preventive Medicine 21(4S), 48–65.

Donohew, L., Zimmerman, R., Cupp, P.S., Novak, S., Colon, S., & Abell, R. (2000). Sensation Seeking, impulsive decision-making and risky sex: implications for risk-taking and design of interventions. Personality and Individual Differences 28, 1079–1091.

Donovan, D.M., Queisser, H.R., Salzberg, P.M., & Umlauf, R.L. (1985). Intoxicated and bad drivers: subgroups within the same population of high-risk men drivers. Journal of Studies on Alcohol 46, 375–382.

Du, L., Bakish, D., & Hrdina, P.D. (2000). Gender differences in association between serotonin transporter gene polymorphism and personality traits. Psychiatric Genetics 10, 159–164.

Ebstein, R.P., Gritsenko, I., Nemanov, L., Frisch, A., Osher, Y., & Belmaker, R.H. (1997). No association between the serotonin transporter gene regulatory region polymorphism and the tridimensional personality questionnaire (TPQ) temperament of harm avoidance. Molecular Psychiatry 2, 224–226.

European Conference of Ministers of Transport. Road accident statistical information, 2002. European Conference of Ministers of Transport, 2004. Available at: http://www.cemt.org/index.htm. Accessed March 15, 2006.

European Transport Safety Council. Priorities for EU Motor Vehicle Safety Design. Brussels: European Transport Safety Council, 2001.

European Transport Safety Council. ETSC fact sheet: Conspicuity and roads safety. Available at: http://www.etsc.be/documents/FINAL_Fact_Sheet_Conspicuity.pdf. Accessed March 15, 2006.

Evenden, J.L. (1999). Varieties of impulsivity. Psychopharmacology 146, 348–361.

Everett, S.A., Shults, R., Barrios, L.C., Sacks, J.J., Lowry, R., & Oeltmann, J. (2001). Trends and subgroup differences in transportation-related injury risk and safety behaviors among high school students, 1991–1997. Journal of Adolescent Health 28, 228–234.

Eysenck, S.B.G., Pearson, P.R., Easting, G., & Allsopp, J.F. (1985). Age norms for impulsiveness, venturesomenes and empathy in adults. Personality and Individual Differences 6, 613–619.

Eysenck, S.G.B. The 17: development of a measure of impulsivity and its relationship to superfactors of personality. In: McCown, W.B., Johnson, J.L., & Shure, M.B., editors. The Impulsive Client: Theory, Research and Treatment. Washington, D.C.: American Psychological Association, 1993, pp 39–56.

Fahlke, C., Garpenstrand, H., Oreland, L., Suomi, S. J., & Dee Higley, J. (2002). Platelet monoamine oxidase activity in a nonhuman primate model of type 2 excessive alcohol consumption. American Journal of Psychiatry 159, 2107–2109.

Filinger, E.J., Garcia-Cotto, M.A., Vila, S., Gerbaldo, H., & Jerez, D. (1987). Possible relationship between pervasive developmental disorders and platelet monoamine oxidase activity. Brazilian Journal of Medical and Biological Research 20, 161–164.

Fink, G., Sumner, B., Rosie, R., Wilson, H., & McQueen J. (1999). Androgen actions on central serotonin neurotransmission: relevanc for mood, mental state and memory. Behavioural Brain Resarch 105, 53–68.

Fowler, C.J., von Knorring, L., & Oreland, L. (1980). Platelet monoamine oxidase activity in sensation seekers. Psychiatry Research 3, 273–279.

Fowler, J.S., Volkow, N.D., Wang, G.J., Pappas, N., Logan, J., MadGregor, R., Alexoff, D., Shea, C., Schlyer, D., Wolf, A.P., Warner, D., Zezulkova, I., & Cilento, R. (1996). Inhibition of monoamine oxidase B in the brain of smokers. Nature 379, 733–736.

Gabany, S.G., Plummer, P., & Grigg, P. (1997). Why drivers speed: the speeding perception inventory. Journal of Safety Research 28, 29–36.

Gerra, G., Garofano, L., Zaimovic, A., Moi, G., Bfanchi, B., Bussandri, M., Brambilla, F., & Donnini, C. (2005). Association of the serotonin transporter promoter polymorphism with smoking behaviour among adolescents. American Journal of Medical Genetics. Part B, Neuropsychiatric Genetics 135, 73–78.

Glassbrenner, D., Carra, J.S., & Nichols, J. (2004). Recent esrimates of safety belt use. Journal of Safety Research 35, 237–244.

Glassbrenner D. Safety belt use in 2005. National Center for Statistics and Analysis, 2005. Available at: http://www-nrd.nhtsa.dot.gov/pdf/nrd-30/NCSA/Rnotes/2005/809932.pdf. Accessed March 15, 2006.

Golias, I., & Karlaftis, M.G. (2002). An international comparative study of self-reported driver behavior. Transportation Research Part F: Traffic Psychology and Behaviour 4, 243–256.

Gow, A.J., Whiteman, M.C., Pattie, A., & Deary, I.J. (2005). Goldberg's IPIP' Big-Five factor markers: International consistency and concurrent validation in Scotland. Personality and Individual Differences 39, 317–329.

Grabauskas, V., Klumbinė, J., Petkevičienė, J., Katvickis, A., Šačkutė, A., Helasoja, V., & Paalanen, L. Health Behaviour among Lithuanian Adult Population, 2004. Helsinki: National Public Health Institute, 2005.

Grunbaum, J.A., Lowry, R., Kann, L., & Pateman, B. (2000). Prevalence of health risk behaviors among Asian American/Pacific Islander high school students. Journal of Adolescent Health 27, 322–330.

Grunewald, P.J., Mitchell, P.R., & Treno, A.J. (1996). Drinking and driving: drinking patterns and drinking problems. Addiction 91, 1637–1649.

Hallman, J., Oreland, L., Edman, G. & Schalling, D. (1987). Thrombocyte monoamine oxidase activity and personality traits in women with severe premenstrual syndrome. Acta Psychiatrica Scandinavica 76, 225–234.

Harre, N. (2000). Risk evaluation, driving, and adolescents: a typology. Developmental Review 20, 206–226.

Harro, J., Fischer, K., Vansteelandt, S., & Harro, M. (2004). Both low and high activities of platelet monoamine oxidase increase the probability of becoming a smoker. European Neuropsychopharmacology 14, 65–69.

Hatakka, M. Novice drivers' risk- and self-evaluations. Turku, Finland: Department of Psychology, University of Turku, Painosalama Oy, 1998.

Helakorpi, S., Patja, K., Prättälä, R., Aro, A.R., & Uutela, A. Health Behaviour and Health among the Finnish Adult Population, Spring 2004. Helsinki: National Public Health Institute, 2005.

Hilakivi, I., Veilahti, J., Asplund, P., Sinivuo, J., Laitinen, L., & Koskenvuo, K. (1989). A sixteen-factor personality test for predicting automobile driving accidents of young drivers. Accident Analysis and Prevention 21, 413–418.

Horan, J.M., & Mallonee, S. (2003). Injury Surveillance. Epidemiologic Reviews 25, 24–42.

Hu, S., Brody C.L., Fisher, C., Gunzerath, L., Nelson, M.L., Sabol, S.Z., Sirota, L.A., Marcus, S.E., Greenberg, B.D., Murphy, D.L., & Hamer, D.H. (2000). Interaction between the serotonin transporter gene and neuroticism in cigarette smoking behaviour. Molecular Psychiatry 5, 181–188.

Huber, S., Krist, H., & Wilkening, F. (2003). Judgement and action knowledge in speed adjustment tasks: Experiments in a virtual environment. Developmental Science 6, 197–210.

Inseneribüroo "Stratum". Monitorig the Traffic Behaviour in 2003 (Liikluskäitumise monitooring 2003.a.). Tallinn: Inseneribüroo "Stratum", 2003. Available at: http://www.mnt.ee/atp/. Accessed March 15, 2006.

International Road Traffic and Accident Data. Brief Overview. Available at: http://www.bast.de/htdocs/fachthemen/irtad/english/englisch.html. Accessed March 15, 2006.

Irving, J.B., Coursey, R.D., Buchsbaum, M.S., & Murphy, D.L. (1989). Platelet monoamine oxidase activity and life stress as predictors of psychopathology and coping in a community sample. Psychological Medicine 19, 79–90.

Iversen, R., & Rundmo, T. (2002). Personality, risky driving and accident involvement among Norwegian drivers. Personality and Individual Differences 33, 1251–1264.

Jang, K.L., Livesley, W.J., Riemann, R., Vernon, P.A., Hu, S., Angleitner, A., Ando, J., Ono, Y., & Hamer, D.H. (2001). Covariance structure of neuroticism and agreeableness: a twin and molecular genetic analysis of the role the serotonin transporter gene. Journal of Personality and Social Psychology 81, 295–304.

Johansson, B., & Drott, P. (2001). Informal parental traffic education and children's bicycling behaviour. Upsala Journal of Medical Sciences 106, 133–144.

Johnson, F.W., Gruenewald, P.J, & Treno, A.J. (1998). Age-related differences in risks of drinking and driving in gender and ethnic groups. Alcoholism: Clinical and Experimental Research 22, 2013–2022.

Jonah, B.A. (1997). Sensation seeking and risky driving: a review and synthesis of the literature. Accident Analysis and Prevention 29, 651–665.

Jonah, B.A., Thiessen, R., & Au-Yeung, E. (2001). Sensation seeking, risky driving and behavioural adaptation. Accident Analysis and Prevention 33, 679–684.

Kayser, R.E., Schippers, G.M., & Van Der Staak, C.P. (1995). Evaluation of a Dutch educational "Driving While Intoxicated (DWI)" prevention program for driving schools. Journal of Drug Education 25, 379–393.

Kiive, E., Eensoo, D., Harro, M., & Harro, J. (2002). Platelet monoamine oxidase activity in association with childhood aggressive and hyperactive behaviour: the effect of smoking? Personality and Individual Differences 33, 355–364.

Kiive, E., Merenäkk, L., Harro, M., & Harro, J. (2005), Changes in platelet monoamine oxidase activity, cholesterol levels and hyperactive behaviour in adolescents over the period of three years. Neuorscience Letters 384, 310–315.

Kirk, K.M., Whitfield, J.B., Pang, D., Heath, A.C., & Martin, N.G. (2001). Genetic covariation of neuroticism with monoamine oxidase activity and smoking. American Journal of Medical Genetics105, 700–706.

af Klinteberg, B., Levander, S.E., Oreland, L., Asberg, M., & Schalling, D. (1987). Neuropsychological correlates of platelet monoamine oxidase (MAO) activity in female and male subjects. Biological Psychology 24, 237–252.

af Klinteberg, B., Hallman, J., Oreland, L., & Wirsen, A. (1992). Exploring the connections between platelet monoamine oxidase activity and behavior: II. Impulsive personality without neuropsychological signs of disinhibition in Air Force pilot recruits. Neuropsychobiology 26,136–145.

von Knorring, L., Oreland, L., & Winblad, B. (1984). Personality traits related to monoamine oxidase activity in platelets. Psychiatry Research 12, 11–26.

von Knorring, L., & Oreland, L. (1996). Platelet MAO activity in type I/ type II alcoholics. Alcoholism – Clinical and Experimental Reseach 20(S), 224A–230A.

Lace, J.K., & Goldstein, B. (2005). Kids and motorbikes: the need for speed. Pediatrics 115, 1085–1086.

Lapidus, J.A., Smith, N.H., Ebel, B.E., & Romero, F.C. (2005). Restraint use among northwest American Indian children travelling in motor vehicles. American Journal of Public Health 95, 1982–1988.

Lauterbach, E., Brunner, J., Hawallek, B., Lewitzka, U., Ising, M., Bondy, B., Rao, M.L., Frahnert, C., Rujescu, D., Müller-Oerlinghausen, B., Schley, J., Heuser, I., Maier, W., Hohangen, F., Felber, W., & Bronisch, T. (2006). Platelet 5-HT_{2a} receptor binding and tryptophan availability in depression are not associated with recent history of suicide attempts but with personality traits characteristic for suicidal behavior. Journal of Affective Disorders 91, 57–62.

Lawton, R., Parker, D., Stradling, S.G., & Manstead, A.S.R. (1997). Self-reported attitude towards speeding and its possible consequences in five different road contexts. Journal of Community & Applied Social Psychology 7, 153–165.

LeMarquand, D.G., Pihl, R.O., Young, S.N., Tremblay, R.E., Séguin, J.R., Palmour, R.M., & Benkelfat, C. (1998). Tryptophan depletion, executive functions, and disinhibition in aggressive, adolescent males. Neuropsychopharmacology 19, 333–341.

LeMarquand, D., Pihl, R.O., & Benkelfat, C. (1994). Serotonin and alcohol intake, abuse, and dependence: Clinical evidence. Biological Psychiatry 36, 326–337.

Lerman, C., Caporaso, N.E., Audrain, J., Main, D., Boyd, N.R., & Shields, P.G. (2000). Interacting effects of the serotonin transporter gene and neuroticism in smoking practices and nicotine dependence. Molecular Psychiatry 5, 189–192.

Lesch, K.P., Bengel, D., Heils, A., Sabol, S.Z., Greenberg, B.D., Petri, S., Benjamin, J., Müller, C.R., Hamer, D.H., & Murphy, D.L. (1996). Association of anxiety-related traits with a polymorphism in the serotonin transporter gene regulatory region. Science 274, 1527–1531.

Lesch, K.P., & Merschdorf, U. (2000). Impulsivity, aggression, and serotonin: a molecular psychobiological perspective. Behavioral Sciences and the Law 18, 581–604.

Lesch, K.P., & Gutknecht, L. (2005). Pharmacogenetics of the serotonin transporter. Progress in Neuro-Psychopharmacology & Biological Psychiatry 29, 1062–1073.

Longato-Stadler, E., af Klinteberg, B., Garpenstrand, H., Oreland, L. & Hallman, J. (2002). Personality traits and platelet monoamine oxidase activity in Swedish male criminal population. Neuropsychobiology 46, 202–208.

Maanteeamet. Liiklusõnnetused Eestis 1975–2005. Available at: http://www.mnt.ee. Accessed March 15, 2006a.

Maanteeamet. Liiklusõnnetuste dünaamika 1995–2005. Available at: http://www.mnt.ee. Accessed March 15, 2006b.

Martin, T.A., Costa, P.T., Oryol, V.E. Rukavishnikov, A.A., & Senin, I.G. Applications of the Russian NEO-PI-R. In: McCrae, R.R., & Allik, J., editors. The Five-Factor model of Personality Across Cultures. New York, NY: Kluwer Academic/ Plenum Publishers, 2002, pp 261–277.

MciLvenny, S., Al Mahrouqi, F., Al Busaidi, T., Al Nabhani, A., Al Hikmani, F., Al Kharousi, Z., Al Mammari, S., Al Hoti, A., Al Shihi, A., Al Lawati, A., & Al Kharousi, I. (2004). Rear seat belt use as an indicator of safe road behavior in a rapidly developing country. Journal of the Royal Society for the Promotion of Health 124, 280–283.

McQueen, J.K., Wilson, H., Summer, B.E.H., & Fink, G. (1999). Serotonin transporter (SERT) mRNA and binding site densities in male rat brain affected by sex steroids. Molecular Brain Resarch 63, 241–247.

Meltzer, H.Y., & Stahl, S.M. (1974). Platelet monoamine oxidase activity and substrate preferences in schizophrenic patients. Research Communications in Chemical Pathology and Pharmacology 7, 419–431.

Mesken, J., Lajunen, T., & Summala, H. (2002). Interpersonal violations, speeding violations and their relation to accident involvement in Finland. Eronomics 45, 469–483.

Moffat, J. (2001). Motor vehicle occupant injury prevention, the States' perspective. American Journal of Preventive Medicine, 21(4S), 5–6.

Morrison, A., Stone, D.H., & the EURORISC Working Group. (2000). Trends in injury mortality among young people in the European Union: a report from the EURORISC Working Group. Journal of Adolescent Health 27, 130–135.

Mõttus, R., Pullmann, H., & Allik, J. (2006). Towards more readable Big Five personality inventories. European Journal of Psychological Assessment 22, 149–157.

Munafò, M.R., Clark, T., & Flint, J. (2005a). Does measurement instrument moderate the association between the serotonin transporter gene and anxiety-related personality? A meta-analysis. Molecular Psychiatry 10, 415–419.

Munafò, M.R., Roberts, K., Johnstone, E.C., Walton, R.T., & Yudkin, P.L. (2005b). Association of serotonin transporter gene polymorphism with nicotine dependence: no evidence fof an interaction with trait neuroticism. Personality and Individual Differences 38, 843–850.

Murphy, D.L., & Weiss, R. (1972). Reduced monoamine oxidase activity in blood platelets fom bipolar depressed patients. American Journal of Psychiatry 128, 1351–1357.

Murphy, D.L., Wrught, C., Buchsbaum, M., Nichols, A., Costa,.J.L., & Wyatt, R.L. (1976). Platelet and plasma monoamine oxidase activity in 680 normals: sex and age differences and stability over time. Biochemical Medicine 16, 254–256.Murray, C.J., & Lopez, A.D. (1997). Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. Lancet 349, 1498–1504.

Nakahara, S., Kawamura, T., Ichikawa, M., & Wakai, S. (2006). Mathematical models assuming selective recruitment fitted do data for driver mortality and seat belt use in Japan. Accident Analysis and Prevention 38, 175–184.

National Institute for Health Development. Health Behaviour among Estonian Adult Population, 2004. Tallinn: National Institute for Health Development, 2005.

Ni, X., Chang, K., Bulgin, N., Sicard, T., Bismil, R., McMain, S. & Kennedy, J.L. (2006). Association between serotonin transporter gene and borderline personality disorder. Journal of Psychiatric Reseach 40, 448–453.

Nilsson, K.W., Sjoberg, R.L., Damberg, M., Alm, P.O., Ohvrik, J., Leppert, J., Lindstrom, L., & Oreland, L. (2005). Role of the serotonin transporter gene and family function in adolescent alcohol consumption. Alcoholism – Clinical and Experimental Reseach 29, 564–570.

Norman, T.R., Chamberlain, K.G., & French, M.A. (1987). Platelet monoamine oxidase: low activity in cigarette smokers. Psychiatry Research 20, 199–205.

Oreland, L., Fowler, C.J., & Schalling, D. (1981). Low platelet monoamine oxidase activity in cigarette smokers. Life Sciences 29, 2511–2518.

Oreland, L. Monoamine oxidase in neuropsychiatric disorders. In: Yasuhara, H., Parvez, S.H., Oguchi, K., Sandler, M., & Nagatsu, T., editors. Monoamine Oxidase: Basis and Clinical Aspects. Ultrecht, The Netherlands: VSP press, 1993, pp 219–247.

Oreland, L. (2004). Platelet monoamine oxidase, personality and alcoholism: the rise, fall and resurrection. Neurotoxicology 25, 79–89.

Parker, D., Lajunen, T., & Summala, H. (2002). Anger and aggression among drivers in three European countries. Accident Analysis and Prevention 34, 229–235.

Pitcairn, T.K., & Edlmann, T. (2000). Individual differences in road crossing ability in young children and adults. British Journal of Psychology 91, 391–410.

Pless, I.B., Taylor, H.G., & Arsenault, L. (1995). The relationship between vigilance deficits and traffic injuries involving children. Pediatrics 95, 219–224.

Pomerantz, W.J., Gittelman, M.A., & Smith, G.A. (2005). No license required: severe pediatric motorbike-related injuries in Ohio. Pediatrics 115, 704–709.

Praschak-Rieder, N., Wilson, A.A., Hussey, D., Carella, A., Wei, C., Ginovart, N., Schwartz, M.J., Zach, J., Houle, S., & Meyer, J.H. (2005). Effects of tryptophan depletion on the serotonin transporter in healthy humans. Biological Psychiatry 58, 825–830.

Pudule, I., Jece, I., Velika, B., Grīnberga, D., Villeruša, A., Dzerve, V., Zīle, S., Helasoja, V., Paalanen, L., & Prättäla, R. Health Behaviour among Latvian Adult Population, 2004. Helsinki, Finland: National Public Health Institute, 2005.

Pulver, A., Allik, J., Pulkkinen, L., & Hämäläinen, M. (1995). A Big Five personality inventory in two non-Indo-European languages. European Journal of Personality 9, 109–l24.

Quinlan, K.P., Brewer, R.D., Sleet, D.A., & Dellinger, A.M. (2000). Characteristics of child passenger deaths and injuries involving drinking drivers. The Journal of the American Medical Association 283, 22249–2252.

Retz, W., Retz.Junginger, P., Supprian, T., Thome, J., & Rösler, M. (2004). Association of serotonin transporter promoter gene polymorphism with violence: relation with personality disorders, impulsivity, and childhood ADHD psychopathology. Behavioural Sciences and the Law 22, 415–425.

Ricketts, M.H., Hamer, R.M., Sage, J.I., Manowitz, P., Feng, F., & Menza, M.A. (1998). Association of a serotonin transporter gene promoter polymorphism with harm avoidance behaviour in an elderly population. Psychiatr Genet 8, 41–44.

Robinson, D.S. (1975). Changes in monoamine oxidase and monoamines with human development and aging. Federation Proceedings 34, 103–107.

Rogeness, G.A., Mitchell, E.L., Custer, G.J., & Harris, W.R. (1985). Comparison of whole blood serotonin and platelet MAO in children with schizophrenia and major depressive disorder. Biological Psychiatry 20, 270–275.

Rosenbloom, T., & Wolf, Y. (2002). Sensation seeking and detection of risky road signals: a developmental perspective. Accident Analysis and Prevention 34, 569–580.

Rosenbloom, T., Nemrodov, D., & Barkan, H. (2004). For heaven's sake follow the rules: pedestrians' behavior in an ultra-orthodox and a non-orthodox city. Transportation Research Part F 7, 395–404.

Roth, J.A., Young, J.G., & Cohen, D.J. (1976). Platelet monoamine oxidase in children and adolescents. Life Sciences 18, 919–924.

Ruangkanchanasetr, S., Plitponkarnpim, A., Hetrakul, P., & Kongsakon, R. (2005). Youth risk behavior survey: Bangkok, Thailand. Journal of Adolescent Health 36, 227–235.

Satcher, D. (2001). Note from the surgeon general. American Journal of Preventive Medicine 21(4S), 1–2.

Schalling, D., Asberg, M., Edman, G., & Oreland, L. (1987). Markers for vulnerability to psychopathology: temperament traits associated with platelet MAO activity. Acta Psychiatrica Scandinavica 176, 172–182.

Schalling, D., Edman, G., Asberg, M., & Oreland, L. (1988). Platelet MAO activity associated with impulsivity and aggressivity. Personality and Individual Differences 9, 597–605.

Shekim, W.O., Davis, L.G., Bylund, D.B., Brunngraber, E., Fikes, L., & Lanham, J. (1982). Platelet MAO in children with attention deficit disorder and hyperactivity: a pilot study. American Journal of Psychiatry 139, 936–938.

Shekim, W.O., Hodges, K., Horwitz, E., Glaser, R.D., Davis, L.G., & Bylund, D.B. (1984). Psychoeducational and impulsivity correlates of platelet MAO in normal children. Psychiatry Res 11, 99–106.

Sher, L., Oquendo, M.A., Li, S., Burke, A.K., Grunebaum, M.F., Zalsman, G., Huang, Y., & Mann, J.J. (2005). Higher cerebrospinal fluid homovanillic acid levels in depressed patients with comorbid posttraumatic stress disorder. European Neuropsychopharmacology 15, 203–209.

Shinar, D., Schechtman, E., & Compton, R. (2001). Self-reports of safe driving behaviors in relationship to sex, age, education and income in the US adult driving population. Accident Analysis and Prevention 33, 111–116.

Shults, R.A. (2004). Child passenger deaths involving drinking drivers–United States, 1997–2002. The Journal of the American Medical Association 291, 934–935.

Sjöberg, R.L., Nilsson, K.W., Nordquist, N., Öhrvik, J., Leppert, J., Lindström, L., & Oreland, L. (2006). Development of depression: sex and the interaction between environment and a promoter polymorphism of the serotonin transporter gene. International Journal of Neuropsychopharmacology 9, 443–449.

Snell, L.D., Glanz, J., & Tabakoff, B. (2002). Relationships between effects of smoking, gender, and alcohol dependence on platelet monoamine oxidase-B: activity, affinity labelling, and protein measurements. Alcoholism, Clinical and Experimental Research 26, 1105–1113.

Stoff, D.M., Friedman, E., Pollock, L., Vitiello, B., Kendall, P.C., & Bridger, W. (1989). Elevated platelet MAO is related to impulsivity in disruptive behaviour

disorder. Journal of the American Academy of Child and Adolescent Psychiatry 28, 754–760.

Stradling, S., Meadows, M., & Beatty, S. Characteristics and crash-involvement of speeding, violating and thrill-seeking drivers. In: Rothengatter, T., & Huguenin, R.D., editors. Traffic and Transport Psychology: Theory and Application. Oxford, United Kingdom: Elsevier Ltd, 2004, pp 177–192.

Tabibi, Z., & Pfeffer, K. (2003). Choosing a safe place to cross the road: the relationship between attention and identification of safe and dangerous road-crossing sites. Child: Care, Health & Development 29, 237–244.

Tzambazis, K., & Stough, C. (2000). Alcohol impairs speed of information processing and simple and choice reaction time and differentially impairs higher-order cognitive abilities. Alcohol and Alcoholism 35, 197–201.

Ulleberg, P. (2001). Personality subtypes of young drivers. Relationship to risk-taking preferences, accident involvement, and response to a traffic safety campaign. Transportation Research Part F: Traffic Psychology and Behaviour 4, 279–297.

Wallace, J.F., Newman, J.P., & Bachorowski, J. (1991). Failures of response modulation: impulsive behavior in anxious and impulsive individuals. Journal of Research in Personality 25, 23–44

Ward, P.B., Catts, S.V., Norman, T.R., Burros, G.D., & McGonaghy, N. (1987). Low platelet monoamine oxidase and sensation seeking in males: an established relationship? Acta Psychiatrica Scandinavica 75, 86–90.

Wasylkiw, L., & Fekken, G.C. (1999). The dimensionality of health behaviors. Journal of Social Behavior and Personality 14, 585–96.

Wesson, D., Spence, L., Hu, X., & Parkin, P. (2000). Trends in bicycling-related head injuries in children after implementation of a community-based bike helmet campaign. Journal of Pediatric Surgery 35, 688–689.

Whissell, R.W., & Bigelow, B.J. (2003). The Speeding Attitude Scale and the role of Sensation Seeking in profiling young drivers at risk. Risk Analysis 23, 811–820.

Whitfield, J.B., Pang, D., Bucholz, K.K., Madden, P.A.F., Heath, A.C., Statham, D.J., & Martin, N.G. (2000). Monoamine oxidase: associations with alcohol dependence, smoking and other measures of psychopathology. Psychological Medicine 30, 443–454.

Wilson, J., & Jonah, B.A. (1985). Identifying impaired drivers among the general driving population. Journal of Studies on Alcohol 46, 531–537.

Wilson, R.J. (1992). Convicted impaired drivers and high-risk drivers: how similar are they? Journal of Studies on Alcohol 53, 335–344.

Wilson, A.L.G., Lange, J.L., Brundage, J.F., & Frommelt, R.A. (2003). Behavioral, demographic, and prior morbidity risk factors for accidental death among men: a case-control study of soldiers. Preventive Medicine 36, 124–130.

Young, J.G., Cohen, D.J., Waldo, M.C., Feiz, R., & Roth, J.A. (1980). Platelet monoamime oxidase activity in children and adolescents with psychiatric disorder. Schizophrenia Bulletin 6, 324–333.

Yu, P.H., & Boulton, A.A. (1987). Irreversible inhibition of monoamine oxidase by some components of cigarette smoke. Life Sciences 41, 675–682.

Zuckerman, M. Risk taking. In: Zuckerman, M., editor. Behavioral expressions and biosocial bases of sensation seeking. Cambridge: University of Cambridge Press, 1994, pp 124–154.

Zuckerman, M., & Kuhlman, M.D. (2000). Personality and risk-taking: common biosocial factors. Journal of Personality 68, 999–1029.
Zureick, J.L., & Meltzer, H.Y. (1988). Platelet MAO activity in hallucinating and paranoid schizophrenics: a review and meta-analysis. Biological Psychiatry 24, 63–78.

SUMMARY IN ESTONIAN

Riskeeriv liikluskäitumine ning riskikäitumise markerid kooliõpilastel ja sõidukijuhtidel

Liiklusõnnetused on oluline rahvatervise probleem kogu maailmas. Uurides riskeerivat käitumist liikluses ja seda mõjutavaid tegureid, näiteks isiksuseomadusi ja riskikäitumise bioloogilisi markereid, saab neid teadmisi rakendades paremini suunata liiklusohutuse-alast ennetavat tegevust.

Käesoleva töö eesmärgid on järgmiselt:

1. Iseloomustada aju serotoniinergilise süsteemi bioloogilise markeri vereliistakute monoamiinide oksüdaasi (MAO) aktiivsust ja selle seost suitsetamisega kooliõpilastel ning sõidukijuhtidel.
2. Uurida riskeeriva liikluskäitumise seost isiksuseomaduste, vereliistakute MAO aktiivsuse ja serotoniini transporter-geeni polümorfismiga kooliõpilastel.
3. Uurida politsei poolt alkoholijoobes juhtimise ja kiiruspiirangute ületamise tõttu kinnipeetud sõidukijuhtidel impulsiivsuse eri tahke ning vereliistakute MAO aktiivsust.
4. Kirjeldada sotsiaal-demograafiliste näitajate, alkoholitarbimise, suitsetamise, impulsiivsuse, vereliistakute MAO aktiivsuse ning liikluskäitumisega seotud näitajate kaudu alkoholijoobes juhtimise ennustatavust.

Vereliistakute MAO aktiivsus oli oluliselt madalam poistel võrreldes tüdrukutega nii 9- kui ka 15-aastaste kooliõpilaste hulgas. Võrreldes vanuse mõju vereliistakute MAO aktiivsusele selgus, et 9-aastastel tüdrukutel oli oluliselt madalam vereliistakute MAO aktiivsus kui 15-aastastel tüdrukutel. Poistel ilmnes vanusega seotud erinevus analoogiliselt tüdrukutega vaid mittesuitsetajate hulgas (Artikkel I). Võrreldes 9- ja 15-aastasi lapsi võib täheldada, et vereliistakute MAO aktiivsus võib mõnevõrra tõusta teismeliseeas. Suitsetamise mõju vereliistakute MAO aktiivsusele 9-aastaste laste hulgas ei ilmnenud, küll aga oli 15-aastaste kooliõpilaste hulgas suitsetajate vereliistakute MAO aktiivsus oluliselt madalam kui mittesuitsetajatel, seda nii poistel kui ka tüdrukutel (Artikkel I). Täiskasvanud meessoost sõidukijuhtidel ilmnes seos vereliistakute MAO aktiivsuse ja suitsetamise määra vahel (Artikkel III). Tulemused kinnitavad, et kõrge intensiivsetel suitsetajatel tubakasuitsus olevad komponendid pärsivad vereliistakute MAO aktiivsust.

Riskeeriv liikluskäitumine 15-aastastel kooliõpilastel oli isiksuseomadustest seotud kõige kindlamini Sotsiaalsusega isiksuse viie-faktorilise mudelist ning impulsiivsuse erinevate tahkudega. Madal vereliistakute MAO aktiivsus seostus riskeeriva liikluskäitumisega vaid tütarlastel. Riskeeriv liikluskäitumine tüdrukutel võib seega olla seotud aju serotoniinergilise aktiivsusega (Artikkel II).

Poistel seostus riskeeriv liikluskäitumine 5-HTT geeni lühikese alleeli kõrgema esinemissagedusega kombinatsioonis madala Sotsiaalsuse ja kõrge Mõtlematusega. Otsene seos riskeeriva liikluskäitumise ja 5-HTT geeni polümorfismi vahel puudus.

Sõidukijuhtidel seostusid impulsiivsuse näitajad oluliselt õigusrikkumistega – kui politsei poolt kinnipeetud alkoholijoobes autojuhtidel olid kõrgemad skoorid impulsiivsuse maladaptiivsetes impulsiivsuse näitajates (Mõtlematus, Pidurdamatus), siis politsei poolt mitmekordselt kinnipeetud ja kiiruspiiranguid üle 20 km/h ületanud sõidukijuhtidel olid kõrgemad skoorid impulsiivsuse adaptiivsetes näitajates (Kiire Otsustamisstiil, Elamustejanu). Alla 20 km/h kiiruspiirangute ületamise tõttu kinnipeetud sõidukijuhtidel impulsiivsuse näitajad ei erinenud kontrollrühmast. Alkoholijoobes autojuhtidel oli oluliselt madalam vereliistakute MAO aktiivsus, aga kiirusepiirangute ületajate vereliistakute MAO aktiivsus oli sarnane kontrollrühma vereliistakute MAO aktiivsusega. Samas kihutamisest tuleneva riski teadvustanud kiirust ületavatel sõidukijuhtidel olid kõrged kõik uuritud impulsiivsusnäitajad ja ka kõrgem vereliistakute MAO aktiivsus võrreldes kontrollrühmaga. Riski eitanud kiirust ületavatel sõidukijuhtidel oli kõrgem vaid Kiire Otsustamisstiil võrreldes kontrollrühmaga (Artikkel V).

Diskriminantanalüüsi alusel eristusid politsei poolt kinnipeetud alkoholijoobes sõidukijuhid kontrollrühmast järgmiste näitajate poolest: noorem vanus, sagedasem ja suuremas koguses alkoholitarbimine, alkoholitarbimisega seotud probleemid, suitsetamine, kõrgem Mõtlematuse skoor, madalam vereliistakute MAO aktiivsus, harvem turvavöö kasutamine ning parkimise eest tasumine (Artikkel IV). Tulemustest ilmneb, et alkoholijoobes juhtimist mõjutavad kombinatsioonis mitmesugused isiksuslikud, käitumuslikud ning bioloogilised tegurid.

ACKNOWLEDGEMENTS

I would like to especially thank Prof. Jaanus Harro, the supervisor of my dissertation, for inspiration, constructive discussions about scientific problems and helpful comments on various aspects of this thesis.

I am also grateful to the co-authors of the research papers Prof. Maarike Harro, Prof. Jüri Allik, Prof. Aleksander Pulver, Marika Paaver, and Helle Pullmann for collaboration, comments, and suggestions concerning the studies of this dissertation;

Dr Toomas Ernits and his colleagues at the Estonian Road Administration for their help in conducting this study;

Prof. Lars Oreland and Niklas Nordquist from the University of Uppsala for cooperation work studying the polymorphism of the serotonin transporter gene;

Krista Fischer for statistical advice;

Enn Veldi for editing the English language of the thesis;

My colleagues at the Department of Public Health and Department of Psychology for cooperation in collecting data and supporting me with an encouraging smile and kind words;

All the people who participated in these experiments.

Last but not least, thanks go to my family for their support and encouragement throughout my life. Special thanks go to Margus, Indrek, Madis, and Maria for their love and understanding.

This study was supported by the Estonian Science Foundation (grants 3277, 3934, 3945, 5209, and 5450), the Estonian Ministry of Education and Science (grants 0814, 0820, and 2643), and the Estonian Road Administration (grants LSOPH01030 and LSOPH02038).

PUBLICATIONS

Harro, M., Eensoo, D., Kiive, E., Merenäkk, L., Alep, J., Oreland, L., & Harro, J. (2001). Platelet monoamine oxidase in healthy 9- and 15-year-old children: the effect of gender, smoking and puberty. Progress in Neuro-Psychopharmacology and Biological Psychiatry 25, 1497–1511.

Eensoo, D., Harro, M., Pullmann, H., Allik, J., & Harro, J. Association of traffic behavior with personality and platelet monoamine oxidase activity in schoolchildren. Journal of Adolescent Health (in press).

Eensoo, D., Paaver, M., Pulver, A., Harro, M., & Harro, J. (2004). Low platelet MAO activity associated with high dysfunctional impulsivity and antisocial behavior: evidence from drunk drivers. Psychopharmacology 172, 356–358.

Eensoo, D., Paaver, M., Harro, M., & Harro, J. (2005). Predicting drunk driving: contribution of alcohol use and related problems, traffic behaviour, personality and platelet monoamine oxidase (MAO) activity. Alcohol & Alcoholism 40, 140–146.

Paaver, M., Eensoo, D., Pulver, A., & Harro, J. (2006). Adaptive and maladaptive impulsivity, platelet monoamine oxidase (MAO) activity and risk-admitting in different types of risky drivers. Psychopharmacology 186, 32–40.

CURRICULUM VITAE

Diva Eensoo

Citizenship: Estonian
Date of birth: December 14, 1963
Address: Department of Public Health, University of Tartu,
Ravila 19, Tartu 50411
Telephone: +372 737 4196
Fax: +372 737 4192
E-mail: diva.eensoo@ut.ee

Education

1971–1982	The 1st Secondary School of Valga
1982–1987	University of Tartu, Faculty of Medicine, pharmacy
1996–1998	University of Tartu, Master studies in biomedicine (Public Health)
2000	Master of Science in Public Health, University of Tartu

Professional employment

1987–2000	Senior laboratory assistant, Department of Public Health, University of Tartu
Since 2000	Researcher, Department of Public Health, University of Tartu

Scientific Activity

Main research areas are 1) health-related behaviour, association with biological indicators and personality traits, 2) family socio-economic status and health-related behaviour in children, 3) environmental risks and their influence on health.

Membership in professional organizations

Estonian Society of Biological Psychiatry, member

CURRICULUM VITAE

Diva Eensoo

Kodakondsus: Eesti
Sünniaeg: 14.detsember 1963
Aadress: Tervishoiu instituut, Tartu Ülikool, Ravila 19, Tartu 50411
Telefon: 737 4196
Faks: 737 4192
E-mail: diva.eensoo@ut.ee

Haridus

1971–1982	Valga 1. Keskkool
1982–1987	Tartu Ülikool, Arstiteaduskond, farmaatsiaosakond
1996–1998	Tartu Ülikool, Arstiteaduskonna biomeditsiini magistratuur (rahvatervis)
2000	Magister scientarium (rahvatervis), Tartu Ülikool

Erialane teenistuskäik

1987–2000	Vanemlaborant, tervishoiu instituut, Tartu Ülikool
Alates 2000	Teadur, tervishoiu instituut, Tartu Ülikool

Teadustegevus

Peamisteks uurimisvaldkonnad on 1) tervist mõjutav käitumine, seos bioloogiliste markerite ja isiksuseomadustega, 2) seos pere sotsiaalmajandusliku olukorra ja tervisega seotud käitumise vahel lastel, 3) keskkonnast tulenevad riskid tervisele.

Erialaorganisatsioonid

Eesti Psühhiaatrite Selts, liige