DISSERTATIONES MEDICINAE UNIVERSITATIS TARTUENSIS
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INGA VILLA

Cardiovascular health-related nutrition, physical activity and fitness in Estonia

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To my family

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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–VI):

I. Villa, I., Alep, J., & Harro, M. (2002). Eesti koolilaste toitumine viimasel 15 aastal. Eesti Arst 81(1), 9–18.

II. Harro, M., Villa, I., Liiv, K., Aru, J., & Alep, J. (2005). Nutrition-related health indicators and their major determinants in the new member states: case of Estonia. Journal of Public Health 13, 111–119.

III. Harro, M., Oja, L., Tekkel, M., Aru, J., Villa, I., Liiv, K., Jürimäe, T., Prättälä, L., & Pudule, I. (2006). Monitoring physical activity in Baltic countries: the FINBALT study, HBSC and other surveys in young people. Journal of Public Health 14, 103–109.

IV. Villa, I., Yngve, A., Poortvliet, E., Grjibovski, A., Liiv, K., Sjöström, M., & Harro, M. (2007). Dietary intake among under-, normal- and overweight 9- and 15-year-old Estonian and Swedish schoolchildren. Public Health Nutrition 10, 311–322.

V. Ruiz, J.R., Ortega, F.B., Rizzo, N.S., Villa, I., Hurtig-Wennlöf, A., Oja, L., & Sjöström, M. (2007). High cardiovascular fitness is associated with low metabolic risk score in children; The European Youth Heart Study. Pediatric Research 61, 350–355.

VI. Mäestu, J., Villa, I., Parik, J., Paaver, M., Merenäkk, L., Eensoo, D., Harro, M., & Harro, J. (2007). Human adrenergic α_{2A} receptor C-1291G polymorphism leads to higher consumption of sweet food products. Molecular Psychiatry 12, 520–521.

My contribution to the articles in the current thesis is as follows:

Paper I:	collecting data, data analysis, writing the paper
Papers II and III:	collecting the overview data, participation in writing the papers
Paper IV:	collecting data, data analysis, writing the paper
Paper V:	collecting data, participation in writing the paper
Paper VI:	collecting data, data analysis, participation in writing the paper

ABBREVIATIONS

ADRA2A	α_{2A}-adrenoceptor gene
ANOVA	analysis of variance
BMI	body mass index
CHD	coronary heart disease
CI	confidence interval
CNS	central nervous system
CVD	cardiovascular disease
CVF	cardiovascular fitness
EYHS	European Youth Heart Study
GL	glucose
HDL-C	high density lipoprotein cholesterol
IOTF	International Obesity Task Force
LDL-C	low density lipoprotein cholesterol
MVPA	moderate-to-vigorous physical activity
OR	odds ratio
PA	physical activity
PCR	polymerase chain reaction
ROC	receiver operating characteristic
SD	standard deviation
SFA	saturated fatty acids
TG	triglycerides
VO_2max	maximum oxygen uptake
WHO	World Health Organization

I. INTRODUCTION

Chronic (non-communicable) diseases, including cardiovascular diseases (CVD), diabetes and cancer, accounted for 59% of global mortality in 2001; for nearly 54% of deaths in low- and middle-income countries, and 87% of deaths in high-income countries (Lopez et al., 2006). CVD remain a leading cause of global mortality resulting in nearly 17.5 million deaths worldwide in 2005 (Smith et al., 2006). Cancers accounted for over 7 million deaths (13% of total mortality) and there were more than 10 million new cancer cases worldwide in 2000 (Shibuya et al., 2002). Diabetes is expected to double from 171 million to 366 million cases over the period 2000–2030 (Wild et al., 2004). Due to rising rates of CVD, diabetes and cancer, the deaths from these chronic diseases are expected to increase by 17% over the period 2006–2015, accounting for nearly 70% of global deaths by 2030 (Mathers et al., 2006). Some population studies have shown that up to 80% of cases of CVD, and up to 90% cases of type II diabetes, could potentially be avoided through changing lifestyle, and about one-third of cancers could be avoided by healthy eating, sufficient physical activity and maintaining normal weight (Stampfer et al., 2000; Hu et al., 2001; Key et al., 2002).

As the roots of cardiovascular diseases have been found in childhood (Strong et al., 1992; Berenson et al., 1998, McGill et al., 2000), lifestyle modification during this period may be particularly effective in lowering CVD risk in adulthood.

CVD risk factors can be divided into major risk factors (high blood pressure, age, smoking, high serum LDL cholesterol, low serum HDL cholesterol, elevated glucose), underlying risk factors (overweight/obesity, physical inactivity, atherogenic diet, stress, heredity) and emerging risk factors (triglycerides, insulin resistance, proinflammatory and prothrombotic markers (Smith et al., 2004). Although both underlying and emerging risk factors likely add an independent component to total CVD risk, their use in clinical practice must be individualized and they should not be given more priority in risk assessment than that given to the major risk factors. Nonetheless, several risk factors expressed in a moderate degree that occur together often incur a greater total CVD risk in the short term than does a single, while quantitatively impressive, risk factor.

Previous studies have evaluated the strength of the association between risk factors and CVD across varying lengths of follow-up in adult population (Yarnell et al., 2000; Navas-Nacher et al., 2001; Menotti & Lanti, 2003; Daviglus et al., 2004, Yan et al., 2006). There is strong evidence that lifestyle risk factors such as unhealthy diet and physical inactivity are the risk factors for many diseases including CVD (Stampfer et al., 2000; Hu et al., 2001; Thompson et al., 2003; Hung et al., 2004; Dontas & Yiannakopoulos, 2007). It is now universally recognised that a diet which is rich in fat, salt and free sugars and low in complex carbohydrates, fruit and vegetables increases the risk of chronic diseases – particularly CVD and cancer (WHO, 2003). Several clinical trials

have also demonstrated the effectiveness of lifestyle (diet and physical activity) modifications in risk factor reduction at the individual level (Sacks et al., 2001; Vollmer et al., 2001; Knowler et al., 2002; Elmer et al., 2006).

Furthermore, unhealthy diet and physical inactivity are not risk factors for only CVD as such, but they can also aggravate other (pathophysiological) risk factors (e.g. overweight/obesity). Overweight and obesity prevalence are increasing in both adults and children worldwide and it is considered a global epidemic (WHO, 1998; Koletzko et al., 2002; Frye & Heinrich, 2003; Lobstein & Frelut, 2003; Petersen et al., 2003, Hedley et al., 2004; Matsushita et al., 2004; Ogden et al., 2004; Sundquist et al., 2004). This increase has been related to changing dietary habits as well as physical inactivity not only in affluent countries, but also in developing countries and in countries in economic transition (Drewnowski & Popkin, 1997; Popkin & Doak, 1998; Koletzko et al., 2002; Popkin, 2006). Overweight/obesity is commonly associated with insulin resistance, hypertension (high blood pressure), coronary artery disease, and cholesterol abnormalities, and can be a component of the condition that is called the metabolic syndrome.

The present study was designed to assess the prevalence of CVD lifestyle risk factors such as diet and physical activity in children and adolescents as well as to investigate the associations of these factors with overweight/obesity and the associations of cardiovascular fitness with metabolic risk.

2. REVIEW OF THE LITERATURE

2.1. Cardiovascular health and nutrition-related health indicators

Each year CVD causes over 4.3 million deaths in Europe and over 1.9 million deaths in the European Union. The main forms of CVD are coronary heart disease (CHD) and stroke (Petersen et al., 2005). An increasing trend is obvious in the incidence of new cases of CVD (European Health for All database, WHO).

CVD is the main cause of the disease burden (illness and death) in Europe (23% of all disease burden). The World Health Report 2002 estimated that around 4% of all disease burden in developed countries is caused by low fruit and vegetable consumption: 30% of CHD and almost 20% of stroke is due to fruit and vegetable consumption below 600 g/day. Three percentages of all disease burden is caused by physical inactivity: 20% of CHD and 10% of stroke in developed countries is due to physical inactivity defined as less than 2.5 hours per week of moderate exercise or 1 hour per week of vigorous exercise (WHO, 2002). The dietary patterns across Europe are now converging. In many Northern and Western European countries there have been a slight reduction in fat intake and an increase in fruit and vegetable consumption over the past 20 years. However, in Southern, Eastern and Central European countries fat intake is increasing and fruit and vegetable consumption is declining (FAO, 2004). Global availability of cheap vegetable oils and fats resulted in greatly increased fat consumption among low-income nations (Drewnowski & Popkin, 1997).

The process of transition towards market economy has had a great impact on population health in the Baltic countries, including Estonia. During the first years of transition between 1990 and 1994, life expectancy decreased and total mortality increased dramatically in Baltic countries (European Health for All database, WHO). Diseases and health problems associated with nutrition have been described as alarming in Estonia. In 2002 54% of all deaths in Estonia were caused by cardiovascular diseases. However, it was almost one third less than in the mid-1990s (Petersen et al., 2005). For example, Estonia had 220 total deaths from CVD per 100 000 inhabitants before the age of 65 in 1994 compared to 162/100 000 in 2002 (National Strategy for Prevention of CVD 2005–2020, 2005). In comparison, an average 49% of all deaths in European countries and 42% in the EU countries were caused by CVD (Petersen et al., 2005).

Overweight is an independent risk factor that increases the risk of CVD, but is also a major risk factor for raised blood cholesterol, high blood pressure, diabetes and impaired glucose tolerance (WHO, 2000). The majority of European populations have experienced an increase in average body mass index (BMI) between the mid-1980s and mid-1990s (WHO Monica Project, 2003). It

has been estimated that over 7% of all disease burden in developed countries is caused by raised BMI (WHO, 2002).

Relationships between obesity and cardiovascular and metabolic risk factors have been clearly established in adults (Must et al., 1999; Haffner, 2000). Although the monitoring of independent CVD risk factors is statistically very important, it has been suggested that in younger people clustering of cardiovascular disease risk factors is a better measure of cardiovascular health than single risk factors, and that composite risk score could compensate for day-to-day fluctuations in the single risk factors (Andersen et al., 2006).

Clustering of CVD risk factors, including obesity, hypertension, dyslipidemia and insulin resistance, is closely associated with CVD and type II diabetes (Haffner, 2002; Lakka et al., 2002). The extent of coronary atherosclerosis in children and adolescents increases remarkably with the number of multiple risk factors (Berenson et al., 1998). The cross-sectional study carried out among 1018 Estonian 9-, 12- and 15-year-old schoolchildren to assess the occurrence of conventional cardiovascular risk factors after the socioeconomic changes of the early 1990s revealed that 11–24% of the children had higher total cholesterol levels, 3–5% were considered to be obese and 6–12% had higher blood pressure. The occurrence of 3 or more risk factors simultaneously characterized only the older age group, in which 2.3% of girls and 3.5% of boys exhibited clustering of potential cardiovascular risk factors (Grünberg & Thetloff, 1998). Obesity in childhood causes increased blood clotting tendency, hypertension, dyslipidaemia, hyperinsulinaemia, chronic inflammation and endothelial dysfunction (Ferguson et al., 1998; Freedman et al., 1999a; Srinivasan et al., 1999; Ford et al., 2001; Tounian et al., 2001). The clustering of insulin resistance, obesity, hypertension, dyslipidemia, and atherosclerosis has been referred to as the insulin resistance syndrome, the metabolic syndrome, or syndrome X (Reaven, 1988). For the metabolic syndrome there is no standard paediatric definition. Several attempts to describe metabolic syndrome in adolescents have been made by using criteria analogous to Adult Treatment Panel (ATP) III (Cook et al., 2003; de Ferranti et al., 2004; Shaibi et al., 2005). Another approach is to assess metabolic risk by computing the clustering of metabolic risk factors (Brage et al., 2004). Reduced insulin sensitivity and other components of metabolic syndrome have been identified in children as young as 5 years of age (Young-Hyman et al., 2001). The Bogalusa Heart Study showed that the association between age and the degree of clustering of cardiovascular risk variables of metabolic syndrome varied during childhood and young adulthood and was likely influenced by the age-related changes in BMI and the attendant insulin resistance (Chen et al., 2000). Several authors have shown that obesity is an important factor in the development of metabolic syndrome (Chen et al., 2000; Maison et al., 2001; Srinivasan et al., 2002; Cook et al., 2003; Weiss et al., 2004; Chen et al., 2007) and therefore the prevention of childhood obesity is particularly important.

2.2. Overweight and obesity in schoolchildren

Obesity has been defined as an excessive deposition of fat in the body that is associated with adverse consequences for metabolic parameters and short- and long-term physical health, as well as with significant psychosocial problems (WHO, 1998). The criteria for overweight and obesity in children and adolescents vary between epidemiological studies and the classification is more problematic than in adults. This makes the international comparisons of cross-sectional prevalence data difficult.

The prevalence of overweight and obesity is commonly assessed by using the body mass index (BMI). The BMI is widely used in adult populations and it is defined as the weight in kilograms divided by the square of the height in metres (kg/m^2). The BMI is usually accepted as the standard measure for overweight and obesity because it tends to correlate better with body fat mass than relative weight (Troiano & Flegal, 1998). In terms of this index, overweight is defined as a BMI of 25–30, and a BMI above 30 is recognized internationally as a definition of obesity (WHO, 1995). However, in childhood BMI changes substantially with age (Rolland-Cachera et al., 1982; Cole et al., 1995). Therefore, BMI should be used differently in children and a cut-off point related to age is needed to define childhood obesity, using reference percentiles which allow comparison with children of the same age and gender (Power et al., 1997a). Children with a BMI between the 85th and 95th percentile are considered to be overweight and those with a BMI above the 95th percentile are considered obese (Barlow & Dietz, 1998).

International Obesity Task Force (IOTF) has proposed an international classification (criteria) for overweight and obesity in childhood (age- and gender-specific BMI cut-off points), based on pooled international data for BMI and linked to the widely used adult cut-off points of a BMI of 25 and 30 kg/m^2. These cut-off points are recommended to use in international comparisons of prevalence of overweight and obesity (Cole et al., 2000). Therefore, the criteria developed by the IOTF to define overweight and obesity among children and adolescents aged 2 to 18 years should be used in epidemiological studies, whereas national BMI centiles should be used in clinical practice.

Skinfold measurements are also considered to be good indicators and have been widely used for assessing overweight/obesity, but they are open to numerous random and systematic errors. Moreover, while skinfold measurements correlate quite well with total body fat, the size of the correlation is site and sex dependent (Rolland-Cachera et al., 1989).

In children, waist circumference has found to be correlated well with abdominal fat, as well as with other cardiovascular risk factors (Freedman et al., 1999b; Savva et al., 2000; Taylor et al., 2000). It has been shown that children with a waist circumference greater than the 90th percentile are more likely to have multiple risk factors than children with a waist circumference that is less than or equal to the 90th percentile (Maffeis et al., 2001). Nationally developed

waist circumference centiles should be used for clinical purposes as there are no internationally accepted criteria for high- or low-risk waist circumference.

The prevalence of overweight and obesity among children and adolescents has increased dramatically worldwide in the last twenty years. At least 155 million school-age children are overweight or obese. The worldwide prevalence of overweight in children and young people aged 5 to 17 years has been estimated approximately 10%, whereas the prevalence of obesity 2 to 3% (Lobstein et al., 2004). The prevalence of overweight in Europe has risen from <10 % in the 1980s to >20% on current estimates. In some countries prevalence rates of overweight above 30% have been found (IOTF, 2005). For example in the UK, the prevalence of overweight children aged 7–11 years rose from 8% in 1984 to 20% in 1998 (Lobstein et al., 2003). In Spain the prevalence among children aged 6–7 years rose from 23% in 1985–1986 to 35% in 1995–1996 (Moreno et al., 2002). In the European Union the number of overweight children is increasing an average 400 000 per year, of which 85 000 are obese (Lobstein, 2004). Shifts in diet and physical activity are consistent with these changes, but little systematic work has been done to understand all the factors contributing to these high levels. There is a trend that the prevalence of overweight is higher in the Southern European countries, especially those outside of the former Eastern bloc (the prevalence rates vary between 20–40%, while those in northern areas show rates in the range of 10–20%) (Lobstein & Frelut, 2003). The report of the IOTF showed that around one third of young children in Italy, Greece and Portugal were overweight or obese (Lobstein et al., 2004). According to a study of 6–13 year old children from the north of Sweden, the prevalence of overweight doubled from 11.5% in 1986 to 22.2% in 2001 (Petersen et al., 2003).

In Estonia the prevalence of overweight and obesity has decreased during the socio-economic transition period in the 1990s. The prevalence of obesity among Estonian children and adolescents aged 7–18 years has declined from 19% (16–23% dependent on the age and gender) in the beginning of 1980s (Silla & Teoste, 1989) to 2–8% at the end of 1980s (Tur et al., 1994) and to 3–5% in the middle of the 1990s (Grünberg & Thetloff, 1998).

As the prevalence of obesity tends to increase with age, the increase in the prevalence of overweight among young children is a significant concern. Overweight children have a 1.5 to twofold higher risk for becoming overweight adults (Guo et al., 1994). Several investigators have reported that up to two thirds of all obese children become obese adults (Serdula et al., 1993; Power et al., 1997b; He & Karlberg, 1999; Must & Strauss, 1999).

2.3. Associations of overweight/obesity with dietary intake and physical activity

Body weight is regulated by many physiological mechanisms that maintain balance between energy intake and energy expenditure (Lustig, 2001). The development of overweight and obesity is characterized by energy imbalance whereby energy intake (caloric consumption) exceeds energy expenditure and thus any factor that raises energy intake or decreases energy expenditure may cause obesity in the long term (Hill & Melanson, 1999; Schrauwen & Westerterp, 2000).

The increase in the prevalence of overweight and obesity is often attributed to the changing lifestyle in westernized societies, particularly to the increased consumption of high-fat diets and decreased physical activity. However, the evidence linking dietary factors and physical activity patterns with the development of overweight and obesity in childhood is controversial and inconclusive.

Moreno & Rodríguez (2007) have concluded that lack of breastfeeding, high energy intake and high intake of sugar-sweetened beverages may be the main dietary factors contributing to the development of obesity in childhood.

Several authors have found that breastfeeding was a protective factor for later development of obesity (Dewey, 2003; Arenz et al., 2004; Harder et al., 2005; Owen et al., 2005). Arenz et al. (2004) have shown, for example, that the duration of breastfeeding was inversely associated with the risk of overweight (1 month of breastfeeding was associated with 4% decrease in the risk).

In terms of food intake, several cross-sectional and longitudinal studies of children and adolescents have found clear positive associations between the consumption of sugar-sweetened beverages, particularly carbonated soft drinks, and overweight or obesity (Troiano et al., 2000; Ludwig et al., 2001; Giammattei et al., 2003; Nicklas et al., 2003; Berkey et al., 2004; Welsh et al., 2005; Malik et al., 2006). Consumption of sugar-sweetened soft drinks increases energy intake and may promote excessive weight gain because of their high glycaemic index (Ludwig et al., 2001). It has also been shown that consumption of meals composed predominately of high glycaemic index foods induces a sequence of hormonal events that stimulate hunger, and causes overeating (Ludwig et al., 1999a; Roberts, 2000). Harnack et al. (1999) showed that total energy intake was about 10% greater among schoolchildren who consumed soft drinks than in those who did not.

The relationship between increased fat intake and obesity has been shown by several authors (Lissner & Heitmann, 1995; Maffeis et al., 1996; Tucker et al., 1997; McGloin et al., 2002). However, some other epidemiological studies have not found any association between obesity and dietary fat intake in children and young adults (Ludwig et al., 1999b; Atkin & Davies, 2000). French et al. (2001) have found a positive association between fast-food consumption and higher total energy and fat intake but no association with overweight status among adolescents. Moreover, several studies have reported a trend of a decreased fat

consumption in United States, while obesity prevalence is rising (Willett, 1998; Cavadini et al., 2000; Troiano et al., 2000).

Physical activity (PA) of a person affects total energy expenditure and thus energy balance. Low physical activity levels and sedentary behaviours can be associated with overweight and obesity in children and adolescents and may be both the cause and consequence of overweight (Klesges et al., 1995; Moore et al., 1995; Maffeis et al., 1998; Sallis et al., 2000). Johnson et al. (2000) provided strong evidence that lower cardiovascular fitness (CVF) results in greater adiposity gain in pre-pubertal children and it is known that CVF is positively associated with PA. A study among Swedish adolescents between 1974 and 1995 showed that the increase in body weight was due to the declining overall daily physical activity and the lack of specific muscle endurance training (Westerstahl et al., 2003). Prentice & Jebb (1995) and Troiano et al. (2000) found that the increase in BMI was caused more by a decrease in PA rather than by an increase in energy intake. Patrick et al. (2004) examined both dietary and physical activity variables in a cross-sectional study of adolescents and found that only insufficient vigorous physical activity was the risk factor for higher BMI.

The obesity risk of a child has also been correlated to time spent viewing television (Dietz & Gortmaker, 1985; Klesges et al., 1993; Deheeger et al., 1997; Giammattei et al., 2003; Marshall et al., 2004). Television viewing is thought to promote overweight and obesity not only by displacing physical activity, but also by increasing energy intake (Robinson, 1998; Matheson et al., 2004). Wiecha et al. (2006) showed that the increase in television viewing was associated with increased calorie intake among youth and this was mediated by increasing consumption of calorie-dense low-nutrient foods frequently advertised on television. Moreover, television viewing during mealtime is inversely associated with consumption of products not typically advertised, such as fruits and vegetables (Coon et al., 2001).

2.4. Dietary intake in schoolchildren

A diet which is rich in fat, salt and free sugars, and low in complex carbohydrates, fruit and vegetables, has been recognised to increase the risk of several chronic diseases (WHO, 2003). Although most of the studies on these associations have been focused on adults, some have also examined the influence of childhood diet on diseases in later life. It has been found that higher levels of energy intake in childhood may increase the risk of later development of cancer (Frankel et al., 1998) and childhood fruit consumption may be protective against cancer in adulthood (Maynard et al., 2003). In a study of over 20 000 children aged 7–11 in six European countries, low intake of fish, fruits and vegetables were found to be the predictors of poor respiratory health (Antova et al., 2003). At the same time both children and adults in most regions of the world are not meeting the minimum suggested fruit and vegetable

consumption goals of 400 g/day (WHO, 2003). Dietary habits are one of the lifestyle-related determinants of CVD, but also the determinants of obesity and high blood cholesterol levels. The process of atherosclerosis, which may begin already in childhood, is accelerated by the presence of a prolonged elevation of LDL-cholesterol (Wynder et al., 1989). It has been shown that on a "duration of exposure" basis, dietary fat and serum cholesterol levels in childhood may directly influence the risk of CVD later in adult life (Law, 2000). That is why childhood is an important period in modifying children's food patterns and nutrient intakes toward expected values. Evidence suggests that adolescents´ food consumption patterns track into young adulthood (Lien et al., 2001), and this could at least in part explain the impact of childhood nutrition on health in adult age.

Many surveys of dietary intake in children and adolescents have been carried out over the past two decades. The assessment of nutritional adequacy of the diets and making comparisons between countries and regions is very valuable, but has been quite limited. The review by Lambert et al. (2004), which included 79 dietary surveys on children and adolescents from 23 European countries, showed that the main reasons why surveys could not be easily compared are: 1) different methods for measuring intake; 2) different age cut-off points; 3) use of a variety of food composition tables based on different analytical techniques for measuring food composition; 4) failure to exclude under-reporters; and 5) a small number of truly nationally representative samples. Despite these limitations mentioned, still some common trends can be observed in dietary intake in children and adolescents in the European countries (Lambert et al., 2004). Reported energy intake was quite similar across Europe. In children up to 12 years there were no differences in the energy intake between boys and girls. In adolescent boys the reported energy intake increased until the age of 18, but in girls it declined in late adolescence. The intake of carbohydrate, total sugars and sucrose tended to be the lowest in Southern European countries and the highest in the Central and Eastern countries. The lowest fat intakes were recorded in Northern Europe (Norway and Sweden, except Finland where SFA intakes were the greatest), and the highest of total fat (more than 40% of energy) was in Southern European countries, particularly Spain and Greece. Reported intakes of vitamins and minerals were inconsistent and no clear regional trends were noticed, except for vitamin D and iron intake, which were the greatest in Northern countries (Lambert et al., 2004). The latter may be related to the higher consumption of milk products and also food fortification with these supplements.

A study on dietary habits and nutritional status of adolescents in Southern European countries has shown that there exists a risk of micronutrient deficiency, particularly for calcium, iron and zinc. In addition, there was over-consumption of total fat (around 40% of the energy intake) and SFA (around 13% of the energy intake), which means that two important characteristics of the Mediterranean diet, a low consumption of SFA and a high intake of carbohydrates, have been lost (Cruz, 2000). This can be taken as an example

that major changes have occurred in the historical patterns of diet, especially among the youth.

There have been only a couple of large dietary studies in schoolchildren in Estonia. One of them was conducted in the 1980s with 10–15 years old children (Saava et al., 1995) and the other in the early 1990s with 12- and 15-year-olds (Grünberg et al., 1997). Both of them showed higher total fat intake (36–38% of the total energy intake) and SFA intake and lower intakes of vitamins C and D, calcium and zinc than national recommendations (Kuivjõgi et al., 1995).

2.5. Genetic predispositions in body composition and food preferences

Several studies have shown that total adiposity, fat distribution and visceral fat are influenced by genetic factors (Bouchard et al., 1996; Rice et al., 1997; Katzmarzyk et al., 2000; Pérusse et al., 2000; Ukkola et al., 2000; Garenc et al., 2002). Several genes responsible for monogenic obesity in people have been identified, such as leptin, leptin receptor, prohormone convertase 1 (PC1) and pro-opiomelanocortin (POMC) genes. All of them are associated with hypothalamic and pituitary disorders. The mutation in the melanocortin-4 receptor (MC4-R) gene causes a non-syndromic phenotype of morbid obesity (Vaisse et al., 2000; Lee et al., 2001; Farooqi, 2005). However, the common forms of obesity are polygenic, being determined by the interaction of several genes which may each have a relatively small effect and will work in combination with environmental factors, such as nutrition and physical activity. This genetic approach of polygenic obesity has so far been less successful (Boutin & Froguel, 2001).

Some studies have found a genetic component in the response of catecholamine-stimulated lipolysis of abdominal subcutaneous fat cells to prolonged overfeeding (Mauriège et al., 1992), very-low-calorie diet (Stich et al., 1997) or exercise training (Tremblay et al., 1997). Therefore it has been suggested that adrenergic receptor genes may be reasonable candidates to account for such genetic effects (Garenc et al., 2002). Adrenergic receptors form the interface between the endogenous catecholamines adrenaline and noradrenaline and a wide array of target cells in the body to mediate signals in the sympathetic nervous system (Philipp et al., 2002). α_{2A}-Adrenoceptors are G protein-coupled receptors that mediate important physiologic responses, particularly in the cardiovascular and central nervous systems and therefore directly or indirectly participate in all aspects of stress and arousal, including cognitive functions, cardiovascular responses, and metabolic effects (Lafontan & Berlan, 1993). In the CNS, α_{2A}-adrenoceptors are particularly strategically located to control the activity of all monoaminergic neurotransmitter systems (Harro & Oreland, 2001). It has been shown that α_{2A}-adrenoceptor knockout mice have higher concentrations of noradrenaline and higher blood pressure and heart rate (Devedjian et al., 2000). Furthermore, an increased expression of α_{2A}-

adrenoceptors may cause alteration in the regulation of insulin and glucose secretion (Devedjian et al., 2000). This evidence supports the role of the α_{2A}-adrenoceptor gene (*ADRA2A*) as a candidate gene for adiposity and fat distribution. The human *ADRA2A* gene is located at 10q23-q25 (Lario et al., 1997). Several polymorphisms have been detected in the *ADRA2A* gene (Kurnik et al., 2006). Lario et al. (1997) described a single nucleotide polymorphism (SNP) in the promoter region of the *ADRA2A*. The substitution C–G at position -1291 results in an MspI restriction site (Lario et al., 1997). This polymorphism is located in the promoter region and therefore could alter gene expression and receptor density.

In humans, it has been shown that carrying the G allele for C-1291G polymorphism resulted in lowered glucose and diastolic blood pressure and increased triglyceride levels in middle-aged men (Rosmond et al., 2002). Other researchers have found no differences in the selected cardiovascular variables (including blood pressure, resting heart rate, plasma noradrenalin) between subjects with different *ADRA2A* genetic variants in a small group of 85 white subjects (Kurnik et al., 2006). Garenc et al. (2002) studied the connection between the C-1291G polymorphism and total adiposity and fat distribution in black and white adult subjects. They concluded that the impact of the *ADRA2A* polymorphism was marginal in white subjects, while in black subjects the G allele had an effect on fat distribution. It should, however, be considered that among Caucasians the distribution of alleles is such that GG homozygocity is rare in small samples. Besides a possible direct effect of genotype on metabolic measures, it should not be excluded that variants of genes highly expressed in the brain have effects on behavioural aspects of nutrition, such as food intake habits. The possibility that there might be a genetic basis for food preference was recently highlighted by the finding that a polymorphism of a serotonin receptor gene is associated with higher consumption of beef and essential amino acids (Prado-Lima et al., 2006).

2.6. Physical activity in schoolchildren

Physical activity (PA) is an important determinant of health and it has been defined as “bodily movement produced by skeletal muscles that requires energy expenditure and produces progressive health benefits” (Caspersen et al., 1985). PA consists of such dimensions as duration (e.g., hours, minutes), frequency (e.g., times per week), intensity (e.g., percentage of maximal aerobic power output or maximal heart rate) and mode (e.g., type of activity to be performed) (Montoye et al., 1996).

Some positive effects of PA on health outcomes to the health of young people have been identified, such as cardiovascular fitness (CVF), blood lipids, blood pressure, musculoskeletal health and psychological well-being (Riddoch, 1998, Strong et al., 2005). The establishment of healthy patterns of PA during childhood and adolescence is important as the benefits of PA carry over into

adulthood, so that an active child is more likely to be a physically active adult (Kelder et al., 1994; Malina, 1996; Hallal et al., 2006). Furthermore, many conditions associated with a lack of sufficient PA (such as obesity, cardiovascular risk, poor skeletal health, metabolic syndrome) develop in childhood and may result in chronic illness in adulthood (Biddle et al., 2004; Hallal et al., 2006). According to the guidelines for PA, children should participate in at least 60 minutes of moderate-to-vigorous PA (MVPA) daily and activities improving muscular strength, flexibility and bone health should be undertaken on two or more days a week (Biddle et al., 1998; Strong et al., 2005).

The levels of PA among children and adolescents have declined dramatically in the westernized countries during the past few decades due to increasing sedentary lifestyle (Harsha, 1995; Telama & Yang, 2000; van Mechelen et al., 2000; Lotan et al., 2005) and many youngsters do not meet established recommendations for daily MVPA (Sallis et al., 2000; Strong et al., 2005). It has been shown that actual PA at suggested levels declines during the preadolescent and adolescent years (Sallis et al., 2000; Biddle et al., 2004). Males are more active than females and this remains so as age increases (Vilhjalmsson & Kristjansdottir, 2003; Biddle et al., 2004).

The PA level of children can be related to living conditions and socioeconomic status, peer pressure and the degree of PA of their parents (Pérusse et al., 1989; Terre et al., 1990; Gordon-Larsen et al., 2000; Simonen et al., 2002; Mo et al., 2005; Humbert et al., 2006). Sallis et al. (2000) have pointed out the key determinants of PA, such as demographic factors (greater likelihood of activity in younger people, especially boys), social factors (encouragement from peers and parents), psychological factors (perceived competence and enjoyment) and the physical environment (availability of different facilities).

2.7. Associations between physical activity, cardiovascular fitness and cardiovascular risk factors

Regular PA plays an important role in the prevention of CVD (Paffenbarger et al., 1986; Blair, 1994; Thomas et al., 2003). Lack of PA is considered to be a major risk factor for the development of CVD (Powell et al., 1987; Fletcher et al., 1992). PA may result in CVD through various physiological mechanisms, which relate partly to the detrimental effects on blood pressure, serum lipoprotein profiles, as well as insulin and glucose metabolism (Chandrashekhar & Anand, 1991). Among the mechanisms that mediate the effect of PA and help to decrease the rate of the atherosclerosis process are increased insulin sensitivity; a non-insulin-dependent glucose uptake, which causes lower insulin release; an improved ratio between HDL and LDL cholesterol because of increased activity of lipoprotein lipase, and improved function of other metabolic hormones and enzymes for fat metabolism (Froberg & Andersen, 2005). So it has been shown that lean inactive children may later become overweight because of insulin resistance (Froberg & Andersen, 2005).

CVF is a direct marker of physiological status and reflects the overall capacity of the cardiovascular and respiratory systems, and the ability to carry out prolonged physical exercise (Taylor et al., 1955). LaMonte & Blair (2006) have shown that high levels of CVF provide strong and independent prognostic information about the overall risk of illness and death, especially related to cardiovascular causes. It has been suggested that up to 40% of variation in the level of CVF is attributable to genetic factors (Bouchard et al., 1986; Wolfarth et al., 2005). PA and CVF are closely related: the level of CVF is mainly determined by PA patterns over recent weeks or months. In children and adolescents, there is a positive association between objectively measured PA and CVF (Brage et al., 2004; Gutin et al., 2005; Andersen et al., 2006). The standard for the measurement of CVF is the maximum rate of oxygen uptake (VO_2max). CVF is influenced by several factors including age, gender, health status and genetics.

Previous studies have also shown associations between CVF and several cardiovascular risk factors (body fatness, serum lipid profile, fasting glycaemia) in children and adolescents (Twisk et al., 2002; Reed et al., 2005; Mesa et al., 2006; Ruiz et al., 2006). It has been shown that those children who perform better on standardized fitness tests have more favourable body composition and lipid profiles (Harsha, 1995). Maximum oxygen uptake (VO_2max) has been associated with lower levels of cardiovascular risk factors in a longitudinal study from adolescence to early adulthood (Andersen & Haraldsdottir, 1993). An eight-year follow-up study indicated that the changes in the levels of PA and physical fitness, and especially the changes in CVF, between adolescence and young adulthood seemed to be the best predictor of CVD risk factor levels in young adulthood, especially in men (Hasselstrøm et al., 2002). Carnethon et al. (2005) have found low CVF in adolescents and adults to be associated with an increased prevalence of CVD risk factors (such as overweight/obesity, high systolic blood pressure, high total serum cholesterol and low serum HDL cholesterol).

3. AIMS OF THE PRESENT STUDY

The aims of the present study are listed as follows:

1. To characterize nutrient adequacy in 9- and 15-year-old children in Estonia on the basis of national recommendations, and to describe the trends in dietary intake among 15-year-old children over the time period from 1984/85 to 1998/99.
2. To describe nutrition-related health indicators and their major determinants in Estonia and to compare them with other European countries.
3. To describe the trends in physical activity in the Baltic countries and in Finland and the association between physical activity, BMI and socio-economic determinants.
4. To study the differences in macronutrient and food group contribution to total food and energy intake between Estonian and Swedish under-, normal- and overweight schoolchildren and to estimate the association between diet and BMI.
5. To examine whether cardiovascular fitness (CVF) identifies children with a high or low metabolic risk score (MRS) and if so, to determine the CVF level that corresponds to a low metabolic risk.
6. To investigate whether the adrenergic α_{2A} receptor gene (*ADRA2A*) C-1291G polymorphism is associated with glucose metabolism and dietary habits.

4. MATERIALS AND METHODS

4.1. Epidemiological overview

Trends in nutrition-related health indicators for Estonia were compared with the respective European average using relevant international surveys. The prevalence of overweight/obese adults and the food consumption patterns in the Baltic countries and Finland was compared using the data from the *Baltic Nutrition and Health Survey* for adults aged 19–64 years (Pomerleau et al., 2000a) and the *FINBALT Health Monitoring Project* for persons aged 16–64 years (Helakorpi et al., 2002; Grabauskas et al., 2003; Kasmel et al., 2003; Pudule et al., 2003). The data of *NORBAGREEN 2002 study* (2003) for persons aged 15–74 was used to examine the consumption frequency of certain foods. *The HBSC (Health Behaviour in School-aged Children) survey* for 11 years, 13 years, and 15 years old school children was used to compare the overweight prevalence in children and adolescents (Currie et al., 2004). The data presented on overweight and obesity has been derived from self-reported height and weight information used to calculate BMI and so need to be treated with some caution.

The *FINBALT Health Monitoring Project* for adults and the *HBSC survey* for children and adolescents were the only relevant surveys to make international comparisons in physical activity trends between the Baltic countries and Finland (Puska et al., 2003; Currie et al., 2000; Currie et al., 2004).

Table 1. Study designs of epidemiological overview studies (Papers II and III)

	Paper II				Paper III	
Studies	Baltic Nutrition and Health Survey	FINBALT Health Monitoring Project	NORBA-GREEN 2002 study	The HBSC (Health Behaviour in School-aged Children) survey	FINBALT Health Monitoring Project	The HBSC (Health Behaviour in School-aged Children) survey
Age of the subjects	19–64 y	16–64 y	15–74 y	11, 13, 15 y	16–64 y	11, 13, 15 y
Sample size	Estonia: n=2108, Latvia: n=2308, Lithuania: n=2153	n=3000–5000 from each country (Estonia, Latvia, Lithuania, Finland)	Total n=8397 (approx. 1000 from each country, such as Finland, Sweden, Norway, Denmark, Iceland, Estonia, Latvia, Lithuania)	n=1500 from each age group	n=3000–5000 from each country (Estonia, Latvia, Lithuania, Finland)	n=1500 from each age group
Time of data collection	Summer 1997	Every year in Finland since 1978; every 2nd year in Estonia since 1990, in Lithuania since 1994; in Latvia since 1998	April–May 2002	Every 4th year since 1983/84 in Finland. Latvia since 1989/90, Estonia and Lithuania since 1993/94	Every year in Finland since 1978; every 2nd year in Estonia since 1990, in Lithuania since 1994; in Latvia since 1998	Every 4th year since 1983/84 in Finland. Latvia since 1989/90, Estonia and Lithuania since 1993/94
Measure-ments	24-hour recall of dietary intake; standar-dized questionnaire (demographic charac-teristics, eating habits and health behaviours; height and weight mea-surements	Mailed questionnaire (socio-demographic background, health, smoking, food habits, height, weight and physical activity	Computer assisted telephone interview in the Nordic countries; paper assisted personal interview in the Baltic countries	Questionnaire (back-ground factors; indi-vidual and social re-sources, health beha-viours and health outcomes)	Mailed questionnaire (socio-demographic background, health, smoking, food habits, height, weight and physical activity	Questionnaire (background factors; individual and social resources, health behaviours and health outcomes)

4.2. Empirical studies: The EYHS

4.2.1. Subjects of EYHS

The subjects were apparently healthy children and adolescents who participated in the European Youth Heart Study (EYHS) in Estonia and in Sweden. The number of subjects in both countries was planned to be 1000, including 500 9-year-old and 500 15-year-old children. In Estonia, the city of Tartu and its surrounding rural areas was the geographical sampling area. In Sweden, two areas in central Sweden were chosen for data collection (Södertörn and Örebro). The main sampling unit was a school. Schools (25 from Estonia and 42 from Sweden) were sampled using probability proportional to the school size and cluster sampling (urban and rural schools from Estonian and Russian language schools in Estonia). From each school, all 9-year-old children (grade 3) and 15-year-old children (grade 9) were invited to participate in the study. The age of children was selected on the basis of sexual maturation (9-year-old – just before the puberty and 15-year-old – in the last stages of their puberty). Parents and children gave their written consent. The participation rate was 79%[i] in Estonia and 50% in Sweden. From Estonia 1176 and from Sweden 1132 children and their parents agreed to participate. Mean age of younger children was 9.6±0.5 (9.6±0.5 in Estonian and 9.5±0.4 in Swedish children) and of older children 15.5±0.6 years (15.5±0.6 in Estonian and 15.6±0.5 in Swedish children).

Table 2. Description of subjects in EYHS (Papers I, IV, V and VI)

	Paper I	Paper IV	Paper V	Paper VI
Age of the subjects	9-year-old and 15-year-old children from Estonia	9-year-old and 15-year-old children from Estonia and Sweden	9-year-old children from Estonia and Sweden	9-year-old and 15-year-old children from Estonia
Sample size	Total n=1176 (9-year-olds: n= 583 (278 boys and 305 girls) 15-year-olds: n= 593 (260 boys and 333 girls)), of which 1090 provided a complete data set	Total n= 2308 (1176 from Estonia and 1132 from Sweden), of which 1098 from Estonia and 1084 from Sweden provided a complete data set	Total n=1140, of which 873 (539 from Estonia and 334 from Sweden) provided a complete data set	Total n=1176, of which 1171 (536 boys and 635 girls) provided a complete data

[i] In Papers I and IV, participation rate is given 76%. This was based on the first paper on the study (Harro M, Eensoo D, Kiive E, Merenäkk L, Alep J, Oreland L, Harro J. Platelet monoamine oxidase in healthy 9- and 15-years old children: the effect of gender, smoking and puberty. Prog Neuropsychopharmacol Biol Psychiatry 2001;25: 1497–1511). In this paper, the participation rate was calculated specifically on the basis of samples available for measurement of platelet MAO activity, which was 1129.

Data were collected between the beginning of September 1998 and the end of June 1999. The local research ethics committees approved the study (Örebro City Council no. 690/98, Huddinge University Hospital no. 474/98, and University of Tartu no. 49/30-1997).

4.2.2. Methods

4.2.2.1. Assessment of dietary intake

Dietary 24-hour-recall of food intake was used (Papers I, IV, VI). Children completed a food record at home during the day before the study, the younger children with support from their parents if necessary. A face-to-face interactive interview was performed on the next day. The interview data was compared with the record data and differences were discussed with the participant. Portion size that was not indicated on the food record was estimated using pictures of portion sizes (Haapa et al., 1985). A quality rating of the interview was also recorded with 1=very good to 5=very poor. Diet interviews with a quality rating score 3 or above were excluded from further analysis.

Nutrient intake data was analysed in Sweden using the Swedish food composition database *PC-kost* (maintained by the Swedish National Food Administration) (Paper IV) and in Estonia with the Finnish food composition database *Micro-Nutrica* 2.0 (modified and translated into the Estonian language at Tallinn University of Technology, Department of Food Processing) (Papers I, IV and VI). Hakala and co-workers have indicated that for a dominant part of the nutrients, the estimated intakes calculated by means of standardised procedures using the *PC-kost* and *Micro-Nutrica* databases are comparable (Hakala et al., 2003).

4.2.2.2. Physical examination

Height and weight were measured in light clothing by standardized procedures. Body weight was measured to the nearest 0.1 kg (SECA digital balance beam) and height to the nearest 0.5 cm (Harpenden transportable stadiometer). BMI was calculated as weight / height squared $(kg/m)^2$. Based on BMI, all children were grouped into underweight, normal weight and overweight groups. The cut-off points for underweight were taken as the age-adjusted 10^{th} percentile according to population reference standards in Estonia (Grünberg et al., 1998) and in Sweden (Lindgren et al., 1995). Criteria for overweight and obesity are based on data following the IOTF proposed gender- and age-specific BMI cut-off points (Cole et al., 2000). Skinfold thickness was measured with a Harpenden calliper at the biceps, triceps, subscapular, suprailiac and triceps surae areas on the left side of the body (Lohman, 1991). All measurements were taken twice and in rotation and the means were calculated. Pubertal maturation of participants was assessed by male and female investigators using the 5-stage scale according to Tanner (Tanner & Whitehouse, 1976). Tanner score was

calculated by summing pubic hair stage with breast development stage in girls and testis development stage in boys. The height and weight of parents were self-reported.

4.2.2.3. Cardiovascular fitness (cardiorespiratory endurance)

In Paper V CVF was determined by a maximum cycle-ergometer test (Hansen et al., 1989). The workload was preprogrammed on a computerized cycle ergometer (Monark 829E Ergomedic, Vansbro, Sweden) to increase every third minute until the subject reached exhaustion. Heart rate was registered continuously by telemetry (Polar Sport Tester, Kempele, Finland). The criteria for exhaustion were a heart rate ≥185 beats per minute, failure to maintain a pedalling frequency of ≥30 revolutions per minute and a subjective judgement by the observer that the child could no longer keep up, even after encouragement. The power output was calculated as $W_1 + (W_2 \times t/180)$, where W_1 is the work rate at the last fully completed stage, W_2 is the work rate increment at the final incomplete stage, and t is the time in seconds at the final incomplete stage. CVF was expressed as the maximal power output per kilogram body mass (W/kg). The test used to measure CVF was previously validated in children of the same age (Riddoch et al., 2005). The "Hansen formula" for calculated VO2max in mL/min was equal to 12 x calculated power output + 5 x body weight in kilograms (Hansen et al., 1989).

4.2.2.4. Blood pressure

The resting systolic and diastolic blood pressures were measured with an automatic oscillometric method (Dinamap model XL, Critikron, Inc., Tampa, FL) (Papers V and VI). The equipment has been validated in children (Park & Menard, 1987). The subjects were in a sitting, relaxed position and the recordings were made from the left arm every 2 min for at least 10 min with the aim of obtaining a set of systolic recordings not varying by more than 5 mmHg. The mean value of the last three recordings was used as the resting systolic and diastolic blood pressures, in mmHg.

4.2.2.5. Blood biochemistry

Blood samples for the assessment of TG, HDL-C, glucose and insulin were taken by antecubital venipuncture after an overnight fast, using vacuum tubes (Paper V and VI). All the following analyses were measured on an Olympus AU600 autoanalyser (Olympus Diagnostica GmbH, Hamburg, Germany): serum concentrations of TG were measured using the lipase/glycerol kinase/ glycerol phosphate oxidase enzymatic method, HDL-C was measured using the homogeneous polyanion/cholesterol esterase/oxidase enzymatic method and glucose using the hexokinase method. The insulin for Estonian subjects was analyzed with an enzyme immunoassay (DAKO Diagnostics Ltd., Ely, England). All these analyses were performed at Bristol Royal Infirmary, UK, with the exception of insulin for Swedish subjects, which was performed at Huddinge University Hospital, Sweden (Elecsys, Roche Diagnostics GmbH,

Mannheim, Germany). The HOMA was calculated: fasting insulin (mU/L x fasting glucose (mmol/L) / 22.5 (Matthews et al., 1985).

4.2.2.6. Clustering of metabolic risk factors

The clustering of metabolic factors (Paper V) was computed from the following variables: TG, HDL-C, insulin, glucose, skinfold thickness, and blood pressure (systolic blood pressure and diastolic blood pressure). Each of these variables was standardized as follows: standardized value = (value – mean)/SD. The HDL-C standardized value was multiplied by –1 to confer higher risk with increasing value for the purpose of calculating the metabolic risk score. The mean of the standardized values of systolic blood pressure and diastolic blood pressure was calculated. The metabolic risk score was calculated as the mean of the six standardized scores separately for boys and girls. Children being below the 75th percentile of the score were defined as having a low metabolic risk and children being at or above the 75th percentile of the score were defined as having a high risk. The same percentile has been used in different health-related variables (waist circumference, insulin levels, systolic blood pressure, etc.) in a number of population-based studies to define subjects at low (<75th) or high (≥75th) risk (Chu et al., 2000; Wyszynski et al., 2005).

4.2.2.7. Genotyping of the *ADRA2A* gene

DNA was extracted from blood and genotype of C-1219G polymorphism in the promoter region of the α_{2A}-adrenoceptor gene (*ADRA2A*) was determined by polymerase chain reaction (PCR) amplification using the primers and protocols as described by Lario et al. (1997) (Paper VI). DNA was amplified by using the following primers: the forward primer was 5′-TCA CAC CGG AGG TTA CTT CCC TCG-3′ and the reverse primer was 5′-TCC GAC GAC AGC GCG AGT T-3′. These primers generated the product of 552 bp. PCR amplification was carried out in a volume of 20 μL containing 150 ng DNA. The PCR conditions were: an initial denaturation step at 94° C for 3 minutes, followed by 35 cycles of denaturation at 94°C for 30 seconds, annealing at 60° C for 45 seconds, and extension at 72° C for 45 seconds. After each amplification, the PCR reaction product was digested overnight at 37°C after adding 6 U of the restriction enzyme MspI (New England Biolabs, Inc., Beverly, MA) to the PCR mixture that cut the product into five fragments (5, 62, 116, 165, and 174 bp). The 174 bp fragment was cut into two bands in the presence of the G-1291 allele. Resulting fragments were separated by electrophoresis in 3% agarose gels. Each gel was run for 30 minutes at 180 V, stained with ethidium bromide and then photographed under UV transmitted light. The allele without the MspI restriction site is designated here as C-1291 allele and G-1291 allele is with the restriction of the MspI site.

In total, 1171 subjects were genotyped for the C-1291G polymorphism and they were categorized as CC, CG or GG genotype. Genotype frequencies were in Hardy-Weinberg equilibrium.

4.2.2.8. Statistical analysis

Statistical analysis was performed using StatView (version 4.0) (Paper I) and SPSS (versions 11.0 and 13.0) (Papers IV, V and VI).

All variables were checked for normality of distribution before the analysis. Crude differences in average values of the studied factors between different groups were calculated using analysis of variance (ANOVA) (Papers I, IV, V and VI). Associations between metabolic risk factors and CVF quartiles were assessed by ANOVA (Paper V). Continuous variables were expressed as means and standard deviations (SD) or 95% confidence intervals (CI). Nominal data were compared using chi-square tests and Fisher's exact tests. (Papers I, IV and VI). A chi-square test was performed to determine whether the genotype frequencies of the C-1291G were in Hardy-Weinberg equilibrium. The General Linear Model with the Fisher's least-significant-difference (LSD) post-hoc test was used to detect the effects of *ADRA2A* C-1291G polymorphism on different phenotypes (Paper VI). Hochberg's GT2 tests and Games-Howell tests were used for post-hoc comparisons for situations with equal and unequal variances, respectively (Paper IV).

Differences of metabolic risk factors among CVF quartiles were assessed by Tukey´s test. The CVF threshold to discriminate between either a low or high metabolic risk was calculated by receiver operating characteristic (ROC) curve (Zweig & Campbell, 1993). The AUC (area under the curve) and 95% CI were calculated. Binary logistic regression was used to study the relationship between CVF and metabolic risk (Paper V).

Kruskal-Wallis tests and Mann-Whitney tests with Bonferroni correction were used for non-normally distributed data. Individual effects of the studied factors on the risk of being overweight/obese were studied by multiple logistic regressions. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. Multiple linear regressions were performed to study independent influences of the studied characteristics on the whole BMI distribution. Due to a relatively low number of participants in each age group and a large number of variables, forward stepwise procedures were performed (Paper IV).

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

5. RESULTS AND DISCUSSION

5.1. Dietary intake in Estonian schoolchildren (Paper I)

The study on dietary intake of Estonian schoolchildren (Paper I) showed that 60% of 9-year-old children and 68% of 15-year-old children received a high proportion of energy from fat, especially from saturated fatty acids (34–38% and 13% of total energy intake, respectively), although the daily energy intake was according to national recommendations (Tables 3 and 4). The consumption of polyunsaturated fatty acids was below the recommendation in 80% of children. The mean intake of calcium, vitamin D and vitamin B_2 (riboflavin) in all children, fibre in 9-year-old children, magnesium and zinc in 15-year-old children and iron in 15-year-old girls was below the amount suggested in recommendations. The reported intake of vitamin C was higher in 9-year-old urban than rural children ($p<0.05$ boys; $p<0.0001$ girls) and the intake of calcium was higher in 15-year-old urban compared with rural girls ($p<0.001$). The lower intake of vitamin C and calcium in rural children might be due to the poor socio-economical status of the families that did not allow buying a sufficient amount of milk products, fruits and vegetables. Comparing the Estonian speaking and Russian speaking children and adolescents, some differences in dietary intake were found only in boys. The Estonian speaking boys of both age groups consumed more fibre, vitamin B_6 and iron ($p<0.01$). Although the mean intake of energy was found to be higher in 15-year-old Estonian speaking boys than in Russian speaking boys (11.9±3.4 vs. 10.4±3.4 MJ/day; $p<0.01$), it can be said that Estonian speaking boys might need more energy, because they were significantly taller and heavier.

Comparing the results of dietary intake of 15-year-olds in this study with previous studies (Saava et al., 1995; Grünberg et al., 1997), we can see tendencies of increase in daily energy intake and consumption of polyunsaturated fatty acids (PUFA) between 1984–1999, and decreasing tendencies in the consumption of fat, SFA and cholesterol, which is a very positive change (Figure 5 of Paper I). Therefore, the consumption of calcium, vitamin D, zinc and magnesium has been quite stable and below the recommendations all this period (Figure 6 of Paper I).

Although healthy and positive tendencies in dietary intake occurred in schoolchildren in Estonia, still an overconsumption of fats was observed over the time period from 1984 to 1999. On the other hand, according to the EYHS study in 1998/99, the consumption of calcium, vitamin D and riboflavin in all children, fibre in 9-year-old children and magnesium and zinc in 15-year-old children was below suggested recommendations.

Table 3. Mean ± SD of energy, macro- and micronutrients intake for 9-year-old boys and girls in Estonia in 1998/99

	RDA[1]	Boys (n=256)		Girls (n=278)	
		Urban (n=179)	Rural (n=77)	Urban (n=193)	Rural (n=85)
Energy intake (MJ/day)	5.9–10.5	8.4±2.7	8.6±3.3	7.9±2.3	7.9±2.4
Protein (%)	10–15	12.6±3.0	12.0±2.6	12.4±2.8	12.1±2.4
Fat (%)	30–32	35.4±8.6	34.5±8.1	34.2±8.2	33.5±8.1
Carbohydrate (%)	52–60	51.8±9.1	53.3±9.1	53.2±8.8	54.3±9.0
Protein (g/day)	49–73	61±22	60±24	57±19	55±18
Fat (g/day)		80±37	80±42	73±30	70±30
Carbohydrate (g/day)		254±84	265±101	245±75	250±83
SFA (%)	10–12	13.4±3.7	13.2±4.0	12.8±3.7	12.6±4
MUFA (%)	10	11.7±3.7	11.3±3.2	11.3±3.4	11.1±3.8
PUFA (%)	10	7.4±3.6	7.3±3.5	7.4±3.4	7.0±2.9
Cholesterol (mg/day)	≤300	227±163	257±213	209±134	198±138
Fibre (g/day)	20–30	18±9	19±10	18±8	19±10
Vitamin A (mg-eqv/day)	0.7	1.8±5.2	0.8±1.3	1.4±3.3	0.6±0.8
Vitamin D (µg/day)	5	2.0±2.1	2.0±1.8	1.9±2.2	2.0±1.9
Vitamin E (mg/day)	7	11.1±8.5	11.6±10.3	10.6±6.9	10.3±6.4
Thiamin (mg/day)	1.0	1.0±0.5	0.9±0.5	0.9±0.4	0.9±0.4
Riboflavin (mg/day)	1.2	1.2±0.6	1.0±0.5	1.0±0.5	0.9±0.4
Niacin (mg-eqv/day)	13	22.7±8.8	21.4±8.4	20.8±7.3	20.1±7.3
Vitamin B6 (mg/day)	1.4	1.5±0.9	1.6±0.7	1.4±0.7	1.4±0.8
Vitamin B12 (µg/day)	3.0	9.0±25.3	4.8±6.1	7.4±15.9	3.5±4.3
Folate (µg/day)	80	187 ±95	176±76	179±70	169±79
Vitamin C (mg/day)	45	69±76 #	47±52	72±63 **	41±30

	RDA[1]	Boys (n=256)		Girls (n=278)	
		Urban (n=179)	Rural (n=77)	Urban (n=193)	Rural (n=85)
Potassium (mg/day)	1000	2928±1275	3006±1114	2854±991	2909±1148
Calcium (mg/day)	800	701±342	654±309	664±313	635±310
Magnesium (mg/day)	250	264±111	277±115	257±103	267±115
Phosphorus (mg/day)	800	1178±397	1191±447	1111±367	1118±385
Iron (mg/day)	10	13.2±6.3	13.4±6.8	12.6±6.6	11.8±4.4
Zinc (mg/day)	7	9.2±3.6	9.3±3.9	8.6±3.3	8.9±3.3
Manganese (mg/day)	2.5	3.6±2.2	3.8±2.5	3.4±2.2 *	3.9±2.4
Iodine (μg/day)	120	197±92	219±132	185±89	195±80
Selenium (μg/day)	15–30	64±30	65±31	60±24	58±21

[1] Recommended Dietary Allowance (Kuivjõgi et al., 1995)

SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids

#$p<0.05$ difference between urban and rural boys;

*$p<0.05$; **$p<0.0001$ difference between urban and rural girls.

Table 4. Mean ± SD of energy, macro- and micronutrients intake for 15-year-old boys and girls from Estonia in 1998/99

	RDA[1]	Boys (n=246)		Girls (n=310)	
		Urban (n=181)	Rural (n=65)	Urban (n=240)	Rural (n=70)
Energy intake (MJ/day)	Boys: 8.2–14.9 Girls: 6.5–11.6	11.4±3.6	11.8±3.1	8.5±2.8	8.3±2.2
Protein (%)	10–14	12.5±2.8	12.8±2.4	12.9±3.4	12.7±2.9
Fat (%)	30–32	37.5±8.6	35.1±8.2	35.5±8.4	36.1±9.1
Carbohydrate (%)	52–60	49.9±9.3	52.0±9.2	51.5±9.1	51.0±9.9
Protein g/day	Boys: 69–96	83±31	90±31	64±26	62±22
Fat g/day	Girls: 54–75	113±47	110±42	82±38	79±29
Carbohydrate g/day		332±121	356±105	256±87	249±86
SFA (%)	10–12	13.4±4.0	12.5±4.2	13.1±3.8	12.8±4.0
MUFA (%)	10	12.7±3.5	12.2±3.2	12.0±3.6	12.2±3.8
PUFA (%)	10	8.0±3.8	7.6±2.9	7.2±3.2	7.9±4.0
Cholesterol (mg/day)	≤300	357±209	341±199	271±192	252±166
Fibre (g/day)	20–30	27±16	28±13	19±10	22±10
Vitamin A (mg-eqv/day)	Boys: 1.0 Girls: 0.8	2.0±4.7	1.9±4.8	1.8±4.5	1.3±4.2
Vitamin D (µg/day)	5	4.1±4.1	3.6±3.7	3.3±5.7	2.7±3.1
Vitamin E (mg/day)	Boys: 10 Girls: 8	16.4±11.4	15.1±7.0	11.2±6.9	12.4±11.9
Thiamin (mg/day)	Boys: 1.4 Girls: 1.1	1.5±0.8	1.6±0.5	1.1±0.4	1.2±0.8
Riboflavin (mg/day)	Boys: 1.7 Girls: 1.3	1.5±0.9	1.4±0.8	1.2±0.6***	1.0±0.7
Niacin (mg-eqv/day)	Boys: 18 Girls: 14	30.5±12.4	33.7±12.3#	23.8±10.1	24.6±12.3

	RDA[1]	Boys (n=246)		Girls (n=310)	
		Urban (n=181)	Rural (n=65)	Urban (n=240)	Rural (n=70)
Vitamin B6 (mg/day)	Boys: 2.0 Girls: 1.6	2.0±1.1	2.5±1.2##	1.6 ±0.8	1.6±0.9
Vitamin B12 (µg/day)	3.0	8.7±16.6	8.4±16.5	8.1±16.7	5.1±7.1
Folate (µg/day)	200	249±115	269±119	203±91**	175±79
Vitamin C (mg/day)	60	68±71	68±79	62±62	56±47
Potassium (mg/day)	1900	3901±1676	4520±1726##	2992±1095	2970±1150
Calcium (mg/day)	1000	905±517	835±467	706±372***	546±308
Magnesium (mg/day)	400	367±172	393±147	273±101	291±137
Phosphorus (mg/day)	1000	1609±650	1705±599	1222±444	1164±434
Iron (mg/day)	Boys: 12 Girls: 18	18.9±9.3	19.3±8.7	14.5±7.6	15.1±9.2
Zink (mg/day)	15	13.1±5.5	14.2±5.7	9.5±3.8	9.8±4.0
Manganese (mg/day)	5	5.7±5.1	6.1±3.6	3.7±2.2	4.3±2.3*
Iodine (µg/day)	150	306±140	308±137	218±111	212±104
Selenium (µg/day)	30–60	88±37	95±34	67±34	67±34

[1]Recommended Dietary Allowance (Kuivjõgi et al., 1995)

SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids

*p<0.05; **p<0.01; ***p<0.001 difference between urban and rural girls;

#p<0.05; ##p<0.01 difference between urban and rural boys.

5.2. Nutrition-related health indicators and trends for Estonia during the years of economic and political transition (Paper II)

The prevalence of overweight and obesity can serve as an indicator of the dynamics associated with nutrition and health. The decrease in the prevalence of overweight and obesity has been obvious in the socio-economical transition period in 1990s in Estonia. This phenomenon has been found both in adult men and women according to the *FINBALT Health Monitoring Project* (Figure 5 of Paper II). If compared to other Baltic countries and Finland, the prevalence of overweight in men was higher in Lithuanian and Finnish men than in Estonian and Latvian men; in women the prevalence of overweight was quite the same (Figure 6 of Paper II). Obesity of adults showed a very small increasing trend in almost all countries in the 2000s, but the prevalence of overweight and obesity according to the *HBSC (Health Behaviour in School-aged Children) survey* in children in Baltic countries was found to be significantly lower than in other European countries (Figure 7 of Paper II). One reason for the decrease of the mean BMI in 1990s in Estonian might be the decline in the purchasing power of families. The other possible reason for the decrease in BMI might also be an increase in the desirability of a slim body. This interpretation is supported by the high popularity of miss and model contests in Estonia during the 1990s. Thus, it is important to find the exact mechanism behind this lower prevalence of overweight and obesity.

Availability of different foods has probably had an influence on eating habits. During the economic transition period the availability of food choices in Estonia increased enormously and dietary habits have changed towards a more "western" style. Both more healthy choices like fresh fruits, vegetables and low-fat alternatives, and less healthy choices like chips, burgers, crisps and processed food choices, can be made around the year. According to the *FINBALT Health Monitoring Project,* the positive changes in eating habits of Estonian adults were the replacement of animal fat with vegetable oil and an increased consumption of fruits and vegetables (Figures 9 and 10 of Paper II). Nevertheless, data from *NORBAGREEN Study* showed that the proportion of daily consumers of vegetables was the lowest in Estonia (35%) if compared to Sweden (78%) and Finland (61%). This might also be attributable to traditions in eating habits, food availability and the purchasing power of families.

The food in Estonia and other Baltic countries has traditionally been rich in fat. The *Baltic Nutrition and Health Survey* conducted in 1997 showed that the intake of fat as a percentage from daily calories both by men and women in Estonia was lower than in Latvia and Lithuania but still remained above the recommended level of 30% out of daily calories (Figure 11 of Paper II). At the same time, the average number of total calories (kcal) available per person per day in Estonia was markedly lower than in other European countries. Based on data from the WHO European Health for All database, the European average of

calories consumed per person per day was between 3400 and 3500 kcal in the time period from 1986 to 2001 with a slightly increasing trend. Estonia had the lowest level in the years 1990–1994 with approximately 2500 kcal, but this increased to about 3000 kcal per person per day by the year 2001.

It was also evident that Estonia is a country where the consumption of alcohol is relatively high. The data of the *FINBALT Health Monitoring Project* showed that the frequency of consuming spirit beverages increased in both men and women in the middle of the 1990s, but then reduced by the end of the 1990s (Figure 13 of Paper II). At the same time, the consumption of beer increased rapidly in Estonia (Figure 12 of Paper II). This phenomenon might be explained by the increased choice of high quality brands of beer during this time period.

Conclusively, it can be said that socio-economic changes during the transition period have had a clear impact on the nutrition and nutrition-related health status of people.

5.3. The trends in physical activity in the Baltic countries and Finland and the associations of physical activity with body mass index and socio-economic status (Paper III)

The *FINBALT Health Monitoring Project* data indicated that PA (participation in physical exercise during leisure time at least twice a week) among adults in the Baltic countries was lower if compared to Finland (Figure 1). The greatest differences between the Baltic countries and Finland were in age groups starting from 35–44 years (Figure 1). If in Latvia and Lithuania men were more active than women, then this was not the case in Estonia (since 2002) where no difference existed, and in Finland, where women were more active than men (Figure 2).

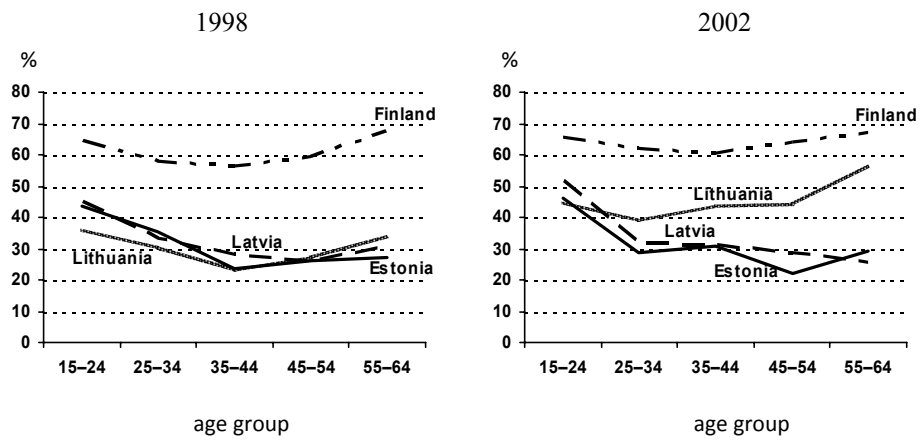

Figure 1. Percentage of men and women from the Baltic countries and Finland in different age groups who participated in leisure time PA at least twice per week in 1998 and 2002

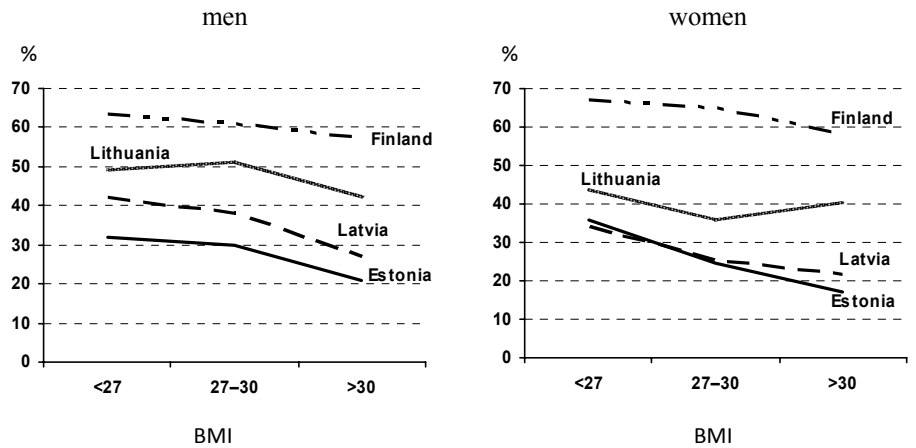

Figure 2. Percentage of adult men and women who were at least twice per week physically active among normal weight, overweight and obese groups, 2002[ii].

The *HBSC (Health Behaviour in School-aged Children) survey* indicated PA trends among schoolchildren in the Baltic countries and Finland. If to compare data from the *HBSC* 1997/98 and 2001/02 surveys, then the percentage of children who took part in PA at leisure time had increased in all countries with the exception of Estonian children who had lower prevalence rates in meeting the MVPA guidelines on physical activity if compared to the peers from other Baltic countries and Finland (Figures 2 and 3 of Paper III). The difference can be due to real changes in PA or due to changes in the question in the questionnaire. In the 1997/98 *HBSC* survey PA was assessed by a question "Outside school hours, how often do you usually exercise in your free time so much that you get out of breath or sweat?" In 2001/2002 this question was replaced with "Over past days, on how many days were you physically active for a total of at least 60 minutes per day".

The results of the HBSC 2001/02 survey also clearly showed in all countries and across all three age groups that boys were more likely than girls to meet the current guidelines on recommended frequency (40% and 27% respectively), and the proportions of children meeting the guidelines declined with age. These findings were similar with the results of other studies (Sallis et al., 2000; Vilhjalmsson & Kristjansdottir, 2003; Biddle et al., 2004; Nader et al., 2008) and suggest that much more effort and work are needed to improve and increase the levels of PA to maximize the potential health benefits in the future.

The current overview indicated that both overweight and obese adults (Figure 2) and schoolchildren (Figure 5 of Paper III) in the Baltic countries tended to be physically less active than their normal weight peers. This was also

[ii] In Paper III as printed, Figure 4 erroneously contains only the data of women.

found in the study among 4–19 years old children and adolescents (Butte et al., 2007). On the other hand, a study carried out among adults in the Baltic countries in 1997 found no association between leisure time PA and obesity (Pomerleau et al., 2000b). However, Ball et al. (2000) observed that obese adults were less active than normal or even overweight people as 22.6% of obese individuals reported that „being too fat" was a barrier to increase their level of PA with the respective numbers of 5% and 0.7% for overweight and normal weight individuals. It means that more efforts should be made and special attention to be given to weight-related physical activity barriers that can help public health strategies to increase PA among those who are overweight and obese.

Schoolchildren and adults from more affluent families were physically more active than those in less affluent families (Figures 6 and 7 of Paper III). In Estonia, men in the highest income group were physically more active than women with the same income level (Figure 6 of Paper III). Comparing this with the data of the *Baltic Nutrition and Health Survey,* it was observed that in men from the Baltic countries, reported income was inversely related with the likelihood of engaging or not in vigorous exercise, and respondents with higher income were 40–60% more likely to engage in vigorous activities compared with men in the lowest income category (Pomerleau et al., 2000c). It can be said that belonging to the highest income group was a protective factor only for men. Mutunga et al. (2006) found that 12- and 15-year-old adolescents of higher socio-economic status had significantly higher levels of habitual physical activity and also higher CVF. However, Voss et al. (2008) have reported that social inequalities have little impact on PA in young children as children from low-income families may have less access to sports facilities but they are not less physically active, and concluded that improving provisions for sports may not lead to the expected rise in PA levels in young children.

In order to better explain and compare the trends and the determinants of PA in European countries, a regular monitoring system with common methodology is needed.

5.4. Overweight/obesity and associations of body mass index with dietary intake in Estonian and Swedish schoolchildren (Paper IV)

Overweight was more prevalent among younger girls in Sweden and underweight among girls of both age groups in Estonia (Figures 3 and 4).

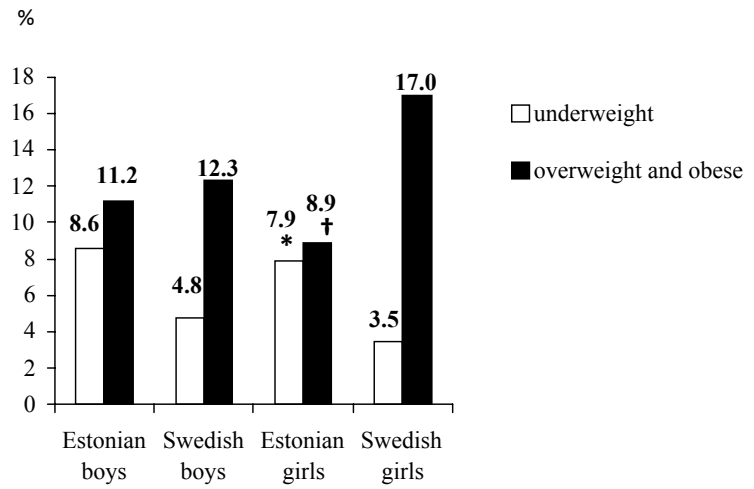

Figure 3. Distribution (%) of 9-year-old Estonian and Swedish children into groups according to BMI (data for normal-weight children not shown).
*Significant difference from underweight Swedish girls (p=0.002); †significant difference from overweight/obese Swedish girls (p=0.002)

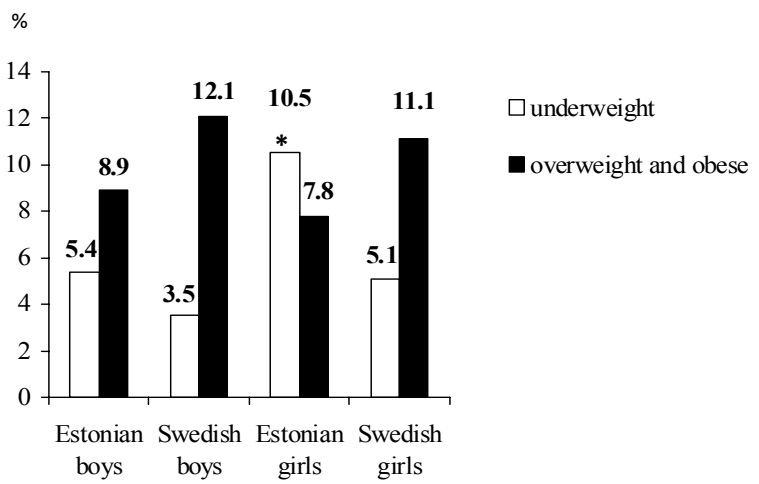

Figure 4. Distribution (%) of 15-year-old Estonian and Swedish adolescents into groups according to BMI (data for normal weight adolescents not shown).
*Significant difference from underweight Swedish girls (p=0.018)

Obesity was more prevalent in younger Swedish boys if compared to Estonian boys (3.7 vs. 0.4%, p=0.003). Overweight 9-year-old children from both countries were significantly taller than their under- and normal weight peers (Table 5). In adolescents the same result was found only as a trend (Table 6). Overweight children of both countries and adolescents in Sweden also had a significantly higher pubertal maturation score if compared to peers with a lower BMI. Parental BMI was significantly higher in overweight children of both countries if compared to participants with a lower BMI (Tables 5 and 6).

The association between diet and BMI in schoolchildren was found to be country-specific. Significant differences in nutrient intake between BMI groups were found only in younger Estonians. Estonian overweight children consumed more energy from fat and less as carbohydrates compared with their underweight peers. Swedish overweight adolescents tended to consume more energy from protein compared to peers with a lower BMI (Table 6). Country-specific differences in the current study can be explained by the difference in the consumption of larger amounts of certain foods or food groups, by the preference for more energy-dense foods inside the same food groups by overweight participants, or by the difference in the fat and energy content of the same foods in Estonia and Sweden. Several authors (Gazzaniga et al., 1993; Maffeis et al., 1996; Tucker et al., 1997; Rocandio et al., 2001) have shown that the macronutrient composition of children's diets, particularly higher dietary fat and lower carbohydrate intakes, may play a role in adiposity, independent of the influence of total energy intake, gender, physical fitness, and parental BMI. Similar results have been shown in animal studies (Oscai et al., 1987; Jen et al., 1988); macronutrient intake, particularly elevated dietary fat consumption, causes obesity, even without excessive energy intake. Other authors have proposed that dietary protein intake may modulate body fat content. Kim et al. (1991) found that the percentage of body fat in rats increased if they consumed an increasing proportion of protein. Hauner et al. (1989) has suggested that high protein intake in excess of metabolic requirements may enhance the secretion of insulin and insulin-like growth factor-1, which can stimulate adipogenic activity. Findings of our population-based study are consistent with these preclinical and clinical data.

We have described an association between the amount of food and BMI, although Janssen et al. (2004) who studied Canadian 11- to 16-year-old adolescents found no clear association between dietary habits (food frequency) and overweight. The amount of milk and milk products consumed by Estonian children and adolescents was almost two times smaller than in their Swedish peers. Estonian overweight children consumed more milk (grams per day) and tended to consume more meat than their normal and underweight peers, and Swedish overweight adolescents tended to consume more milk products. Nevertheless, the milk consumed by Estonian children yielded to higher energy consumption than from fats or fatty acids but in Swedish adolescents to higher energy intake from proteins. Such a finding can be explained by the fact that in Estonia the most common type of milk contains 2.5% of fat, whereas in Sweden

the most popular milk contains 0.5 or 1.5% of fat. Higher intake of energy as protein in overweight Swedish adolescents can also be explained by their higher intake of energy from meat.

In our study no difference was found in energy intake between different BMI groups. However, in Swedish children we found a positive association between absolute BMI and energy intake in regression analysis. Several authors have also described the association between total energy intake and overweight. Rocandio et al. (2001) have shown that overweight children consume less energy (kJ/day) than non-overweight children, and suggested that the positive energy balance causing overweight is possibly due to a low energy output. Several other authors (Maffeis et al. 1996; Gazzaniga et al. 1993; Bandini et al. 1999) have obtained similar results when adjusting energy intake per resting metabolic rate, per kilogram of body weight or as the ratio of reported energy intake to measured energy expenditure. The differences in BMI in our study could thus be explained by differences in energy output. This suggests that development of overweight can differ by country regarding the contribution of distinct components in energy balance.

Stepwise multiple linear regression analysis showed that absolute BMI values were positively associated with parental BMI and Tanner score of children and adolescents in both countries (Tables 5 and 6 of Paper IV). Earlier pubertal onset or menarche has been found to be associated with greater BMI values in several previous studies (Biro et al., 2001; Styne et al. 2004). Pubertal maturation score is strongly associated with age during the maturation, thus older children in the group are usually more mature and their BMI is higher. The risk of being overweight was significantly associated with sexual maturation only in Swedish participants. The lack of association between pubertal score and the risk of being overweight in Estonian participants can be explained by the low prevalence of overweight in the Estonian sample.

When the dietary data were analysed in a logistic regression analysis together with age, gender, Tanner score, and parental BMI, the risk of being overweight was associated with parental BMI in both countries. Danielzik et al. (2002) has shown that the nutritional state of pre-pubertal children is influenced by parental BMI, and parental overweight and obesity are risk factors of childhood overweight. Vogler et al. (1995) has proposed that most of the familial risk for childhood obesity is likely to be explained by genetic factors. Perusse & Bouchard (1999) resumed that in children the maximal heritability of obesity phenotypes ranges from about 30% to 50%. This finding can be interpreted as indicating that besides genetic factors, there is space left for environmental factors, such as nutrition. Wardle et al. (2001) described that children of overweight parents had a higher taste preference for fatty foods, a lower liking for vegetables and a more “overeating-type” eating style. In our study, the absolute BMI of both the father and the mother was positively associated with a participant´s absolute BMI and with the risk of being overweight in both countries, Estonia and Sweden. The associations found were stronger than between BMI and diet.

Table 5. Mean ± standard deviation (SD) of anthropometrical measurements, pubertal score, energy and macronutrients intake for 9-y-old male (M) and female (F) Estonian and Swedish children

	RDA[1]	9-year-old Estonian boys and girls				9-year-old Swedish boys and girls			
		Underweight (n=44: 23M, 21F)	Normal-weight (n=444: 206M, 238F)	Overweight (n=54: 29M, 25F)	p-value	Under-weight (n=23: 13M, 10F)	Normal-weight (n=441: 214M, 227F)	Overweight (n=80: 32M, 48F)	p-value
Height (cm)		135.2±5.7	136.9±6.3	141.8±6.6	b ($p<0.001$)	136.7±4.6	138.6±6.0	142.2±6.7	b, c ($p<0.001$)
Weight (kg)		25.4±2.4	30.6±4.3	42.5±5.8	a, b, c ($p<0.001$)	25.8±2.0	32.1±4.3	44.2±6.2	a, b, c ($p<0.001$)
BMI (kg/m^2)		13.9±0.6	16.3±1.3	21.0±1.7	a, b, c	13.8±0.5	16.6±1.3	21.8±1.9	a, b, c
Mother BMI (kg/m^2)		23.5±4.9	23.4±4.0	25.0±5.1	($p<0.001$)	22.3±3.2	23.3±3.6	25.6±4.7	($p<0.001$)
Father BMI (kg/m^2)		24.2±3.3	25.9±3.5	27.3±4.1	c ($p<0.01$)	24.0±2.1	25.5±3.2	26.6±3.6	b, c ($p<0.001$)
					a, b, c ($p<0.01$)				a, b, c ($p<0.001$)
Tanner score		2.2±0.4	2.3±0.7	2.8±0.9	b, c ($p<0.001$)	2.1±0.3	2.2±0.5	2.6±0.7	b, c ($p<0.001$)
Energy intake (MJ/day)	7.6 (F), 8.8 (M)	7.5±2.8	8.1±2.7	8.5±2.6		8.9±2.6	8.6±2.1	9.1±1.9	
Protein (%)	10–20	12.2±2.9	12.4±2.9	12.8±2.8		15.0±3.3	15.6±3.2	15.6±3.4	
Carbohydrate (%)	50–60	56.1±8.0	53.1±8.9	50.3±9.7	b, c ($p<0.01$)	52.7±7.5	52.1±7.0	51.0±8.1	
Fat (%)	25–35	31.7±7.4	34.5±8.2	36.8±9.4	b ($p<0.01$)	32.5±5.6	32.4±6.2	33.3±6.9	
SFA (%)	10	12.2±3.6	13.0±3.9	14.1±3.7	b ($p<0.05$)	15.5±3.9	15.1±3.3	15.4±3.6	
MUFA (%)	10–15	10.2±2.9	11.4±3.5	12.4±34.3	b ($p<0.01$)	11.7±2.0	11.8±2.7	12.1±2.9	
PUFA (%)	5–10	6.9±3.0	7.4±3.4	7.8±4.4		3.6±1.2	3.7±1.5	3.9±2.4	
Fibre (g/day)	14[2]	17.1±9.0	18.0±9.5	16.9±6.7		14.3±5.9	14.8±5.8	15.2±5.2	

[1] Recommended Dietary Allowance (Nordic Council of Ministers, 2004)

BMI – body mass index; SFA - saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids

p-values for post-hoc tests: a – difference between under- and normal weight; b – difference between under- and overweight; c – difference between normal- and overweight. In all other cases presented p-values are p-values for omnibus tests

[2] Age plus 5g as a reasonable minimum recommendation for dietary fibre intake was used (American Academy of Pediatrics, 1995; Dwyer, 1995)

Table 6. Mean ± standard deviation (SD) of anthropometrical measurements, pubertal score, energy and macronutrients intake for 15-y-old male (M) and female (F) Estonian and Swedish adolescents

	RDA[1]	15-year-old Estonian boys and girls				15-year-old Swedish boys and girls			
		Under-weight (n=48: 13M, 35F)	Normal-weight (n=467: 215M, 252F)	Over-weight (n=41: 21M, 20F)	p-value	Under-weight (n=25: 9M, 16F)	Normal-weight (n=460: 211M, 249F)	Over-weight (n=55: 28M, 27F)	p-value
Height (cm)		166.8±9.5	169.3±7.9	170.5±8.4	p=0.057	168.2±9.7	170.0±8.4	171.9±8.9	p=0.076
Weight (kg)		45.9±5.4	57.7±7.5	77.0±11.8	a, b, c (p<0.001)	47.2±5.3	59.1±7.2	77.9±10.2	a, b, c (p<0.001)
BMI (kg/m^2)		16.4±0.7	20.1±1.7	26.5±3.4	a, b, c (p<0.001)	16.6±0.7	20.4±1.7	26.3±1.9	a, b, c (p<0.001)
Mother BMI (kg/m^2)		23.0±3.0	24.6±4.1	27.6±5.7		23.1±2.7	24.1±3.8	26.9±5.4	
Father BMI (kg/m^2)		24.8±3.7	25.8±3.7	27.4±3.8	a, b, c (p<0.001) b, c (p<0.05)	24.7±3.6	25.8±3.1	27.0±3.1	b, c (p<0.001) b, c (p<0.01)
Tanner score		7.8±1.7	8.6±1.3	8.8±1.3	a, b (p<0.001)	7.8±2.1	9.2±1.1	9.6±0.9	a, b, c (p<0.001)
Energy intake (MJ/day)	9.6 (F), 11.3 (M)	9.5±3.2	10.1±3.6	10.3±4.7		9.3±2.5	10.7±3.6	10.3±2.8	
Protein (%)	10–20	13.1±3.0	12.7±3.1	13.1±3.4		14.4±3.9	15.1±3.5	16.1±3.2	p=0.053
Carbohydrate (%)	50–60	51.4±8.2	51.0±9.4	50.3±9.9		55.9±7.2	53.7±7.7	52.8±7.7	
Fat (%)	25–35	35.5±7.6	36.3±8.7	36.6±8.6		29.7±6.7	31.2±6.7	31.2±7.1	
SFA (%)	10	12.6±3.4	13.2±4.1	13.7±3.9		13.4±3.1	14.3±3.8	13.9±2.9	
MUFA (%)	10–15	12.2±3.6	12.3±3.6	12.6±3.6		10.9±2.5	11.5±3.1	11.1±3.1	
PUFA (%)	5–10	7.2±3.2	7.7±3.5	7.5±3.4		3.7±2.5	3.9±2.0	4.2±3.7	
Fibre (g/day)	20[2]	21.8±10.3	23.9±13.6	23.5±13.4		16.2±6.1	18.9±8.8	15.9±6.6	c (p<0.05)

[1]Recommended Dietary Allowance (Nordic Council of Ministers, 2004)

BMI – body mass index; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids

p-values for post-hoc tests: a – difference between under- and normal weight; b – difference between under- and overweight; c – difference between normal- and overweight. In all other cases presented p-values are p-values for omnibus tests

[2] Age plus 5g as a reasonable minimum recommendation for dietary fibre intake was used (American Academy of Pediatrics, 1995; Dwyer, 1995)

5.5. Associations of cardiovascular fitness with clustering of metabolic risk factors in schoolchildren (Paper V)

The analyses of this study were performed with Estonian and Swedish samples merged to have more statistical power. Valid CVF data were obtained in 85% of the studied subjects. Pubertal development status was obtained from 96% of the children; 97% had blood pressure measurements and 98% had clinical biochemistry data. The results clearly indicated that CVF is associated with clustering of metabolic risk factors in children. Figure 5 presents how a lower metabolic risk score is associated with higher levels of CVF in both boys and girls.

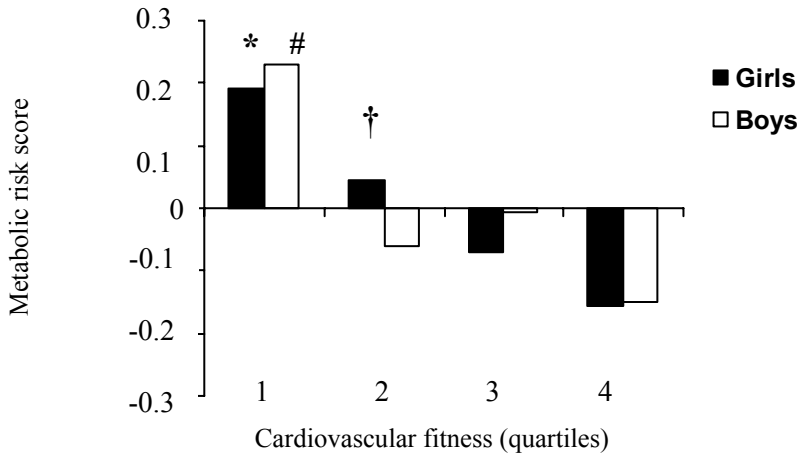

Figure 5. Associations of CVF (quartiles) with metabolic risk score in girls (black bars) and boys (white bars). Data shown as mean and SD. Girls in the first quartile had a higher metabolic risk score than those in the second, third and fourth quartiles ($p=0.006$, $p=0.002$, $p<0.001$, respectively), and girls in the second quartile had a higher metabolic risk score than those in the fourth quartile ($p=0.018$). Boys in the first quartile had a higher metabolic risk score than those in the second, third and fourth quartiles ($p=0.007$).

From different variables that were used to compute the clustering of metabolic risk factors, skinfold thickness and insulin values in both genders ($p<0.001$) and triglycerides in girls ($p=0.026$) decreased across CVF quartiles (Table 2 of Paper V). Many previous studies have shown associations between CVF and cardiovascular risk factors (Twisk et al., 2002; Reed et al., 2005; Mesa et al., 2006; Ruiz et al., 2006) in children and adolescents. The association between CVF and clustering of metabolic factors in children found here is similar to another study in Danish children of the same age (Brage et al., 2004).

The results also suggest a hypothetical CVF level for having a low metabolic risk. Receiver operating characteristic curve (ROC) analysis showed a significant discriminatory accuracy of CVF in identifying the low/high metabolic risk in girls (AUC=0.68, 95% CI 0.62–0.73, $p<0.001$), and in boys (AUC=0.67, 95% CI 0.61–0.73; $p<0.001$) (Figure 2 of Paper V). In girls, the optimal pair of true-high and false-high rates was 0.65 and 0.33, repectively, and 0.65 and 0.39 in boys. The CVF values at these points were 37.0 and 42.1 mL/kg/min in girls and boys, respectively. These levels found in this study are similar to the cut-off points suggested by the Cooper Institute: ≥38 and ≥42 mL/kg/min for girls and boys, respectively (The Cooper Institute for Aerobics Research, 1999). Logistic regression analysis showed that girls with CVF levels above 37.0 and boys above 42.1 mL/kg/min were 3.09 and 2.42 times, respectively, more likely to have a low metabolic risk when compared to those with CVF levels below this value. Among girls, 44% did not reach the required level of CVF, and neither did 40% of the boys. In the AVENA study, receiver operating characteristic curve (ROC) analysis showed a significant discriminatory accuracy of age- and sex-normalized CVF to identify either the presence or absence of a favourable plasma lipid profile in males but not in females (Mesa et al., 2006).

The data on CVF from an early age could be useful to identify the target population for health promotion policies. As the roots of cardiovascular diseases have been found in childhood (Berenson et al., 1998), lifestyle modification during this period may be effective in lowering CVD risk in adulthood. CVF has a large genetic component (up to 40%) (Wolfarth et al., 2005), but it is mainly determined by a person´s activity level (Ruiz et al., 2006; Ignico et al., 1995; Gutin et al., 2005). Variation in CVF has been significantly explained by at least moderate to vigorous [3–6 metabolic equivalents (MET)] physical activity (Ruiz et al., 2006). Further analysis revealed that children who engaged in at least 26 min/day of vigorous (>6 MET) PA had significantly higher CVF than those who accumulate 10–18 min/day of vigorous PA. These results suggest that children with a CVF level below that required to have a low metabolic risk may be able to reach the desirable CVF level with adequate aerobic PA.

Nevertheless, longitudinal studies are needed to reveal whether those children having a CVF above the suggested values have a lower incidence of cardiovascular diseases later in life than those having a CVF below the suggested value.

5.6. Effect of the *ADRA2A* C-1291G polymorphism on consumption of sweet food products (Paper VI)

The intake of sweet food products (e.g., chocolate, candies, nougat) and sweet sour milk products was higher in subjects with GG genotype, while fasting glucose was lower (Table 1 of Paper VI). No other food products were consumed differently among three genotypes (data not shown). These results indicate that C-1291G genotype had a significant effect on the consumption of ready-made sweet food products, of the type for which the subjects may show their own preference. Consumption of sugar added to food prepared at home was not different between genotypes, but this depends more on the dietary habits of the family. Daily energy intake was not significantly different, but a tendency for higher energy intake was found in the GG group. Subjects with the GG genotype have been previously found to be more susceptible to weight gain under clozapine and olanzapine treatment (Wang et al., 2005; Park et al., 2006). This study did not reveal any differences in body compositional parameters and the physical activity level among the three genotypes in a population-representative sample of children. However, higher consumption of sweet food products of GG genotype in childhood may result in a different body composition in adulthood.

6. CONCLUSIONS

The main conclusions of the present study are listed as follows:

1. Although healthy and positive tendencies in dietary intake occurred in schoolchildren in Estonia, still an overconsumption of fats and SFA was observed over the time period from 1984 to 1999. Consumption of fibre and some vitamins and minerals in schoolchildren was in 1998/99 below suggested recommendations.
2. The socio-economic changes during the transition period have had a clear impact on the nutrition and nutrition-related health status of people and these health changes can be observed during the following decades.
3. The percentage of adults involved in PA was remarkably lower in the Baltic countries than in Finland, but no clear country difference was observed in the participation in leisure time PA among schoolchildren between the Baltic countries and Finland. Both overweight and obese adults and schoolchildren tended to be less physically active than normal weight persons. Adults and young people from more affluent families were found to be more physically active than those in less affluent families. These data suggest that in the Baltic population, interventions to increase physical activity should be targeted in particular to overweight and less affluent groups. In order to better compare the trends and the determinants of PA in European countries, a regular monitoring system and identical methodology is needed.
4. The finding that differences in dietary intake between under-, normal- and overweight schoolchildren are country-specific suggests that local dietary habits should be considered in intervention projects addressing overweight. In both Estonia and Sweden the association between BMI and biological factors, such as pubertal maturation and parental BMI, was stronger than between BMI and diet. This suggests other factors rather than diet remain important in the development of overweight in children.
5. CVF was associated with clustering of metabolic risk factors in children. A lower metabolic risk score was associated with higher levels of CVF in both boys and girls. The CVF levels for having a low metabolic risk have been suggested for both genders. Longitudinal and /or intervention studies are needed to examine the impact of having low CVF in childhood on the likelihood of having CVD later in life.
6. α_{2A}-Adrenoceptor gene C-1291G polymorphism had a significant effect on the consumption of sweet food products. Further research should address the issue whether or not this genotype-dependent dietary preference can be tracked to adulthood, and bring about consequences to health.

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SUMMARY IN ESTONIAN

Kardiovaskulaarset tervist mõjutav toitumine, kehaline aktiivsus ja kehaline võimekus Eestis

Sissejuhatus

Kroonilised mittenakkushaigused, sealhulgas südame-veresoonkonna haigused (SVH), diabeet ja vähk, moodustasid 2001 a. 59% kogu maailma suremusest (Lopez et al., 2006). Prognooside kohaselt suureneb suremus nendesse haigustesse kokku ajavahemikul 2006–2015 17% võrra, mis moodustab aastaks 2030 ligi 70% globaalsest suremusest (Mathers et al., 2006).

SVH on jätkuvalt peamine surmapõhjus – peaaegu 17,5 miljonit surmajuhtumit kogu maailmas aastal 2005 (Smith et al., 2006). Euroopas sureb SVH tagajärjel igal aastal üle 4,3 miljoni ja Euroopa Liidus üle 1,9 miljoni inimese (Petersen et al., 2005). SVH on ka haiguskoormuse peamine põhjus Euroopas (23% kogu haiguskoormusest). Tõestust on leidnud, et sellised eluviisi eripärad nagu ebatervislik toitumine ja vähene kehaline aktiivsus on paljude haiguste, sealhulgas ka SVH, riskitegurid (Stampfer et al., 2000; Hu et al., 2001; Thompson et al., 2003; Hung et al., 2004; Dontas & Yiannakopoulos, 2007). *World Health Report 2002* andmetel põhjustab hinnanguliselt umbes 4% kogu haiguskoormusest arenenud riikides vähene puu- ja köögiviljade tarbimine (30% SVH ja peaaegu 20% insuldist on põhjustatud puu- ja köögiviljade tarbimisest alla 600 g/ööpäevas) ja 3% kogu haiguskoormusest põhjustab vähene kehaline aktiivsus (20% SVH ja 10% insuldist arenenud riikides on tingitud vähesest kehalisest aktiivsusest – vähem kui 2,5 tundi nädalas mõõdukat või 1 tund nädalas suurt koormust) (WHO, 2002). Mõned uuringud on näidanud, et kuni 80% SVH-st, kuni 90% II tüüpi diabeedi ja umbes üks kolmandik vähi juhtudest oleks võimalik vältida elustiili muutmisega (tervislik toitumine, piisav kehaline aktiivsus ja normaalne kehakaal) (Stampfer et al., 2000; Hu et al., 2001; Key et al., 2002). Mitmed kliinilised uuringud on samuti näidanud elustiili (toitumise ja kehalise aktiivsuse) muutmise tõhusust riskitegurite vähendamisel individuaalsel tasandil (Sacks et al., 2001; Vollmer et al., 2001; Knowler et al., 2002; Elmer et al., 2006).

Kuna on leitud, et SVH saavad alguse juba varases lapsepõlves (Strong et al., 1992; Berenson et al., 1998, McGill et al., 2000), siis on tervislik elustiil selles eaperioodis eriti vajalik ja efektiivne vähendamaks SVH riski täiskasvanueas.

Lisaks sellele pole ebatervislik toitumine ega vähene kehaline aktiivsus mitte ainult SVH riskitegurid, vaid need võivad süvendada teisi (patofüsioloogilisi) riskitegureid (nt ülekaalulisus/rasvumine). Ülekaalulisuse ja rasvumise levimus suureneb nii täiskasvanute kui ka laste seas kogu maailmas ja seda peetakse globaalseks epideemiaks (WHO, 1998; Koletzko et al., 2002; Frye & Heinrich, 2003; Lobstein & Frelut, 2003; Petersen et al., 2003; Hedley et al., 2004;

Matsushita et al., 2004; Ogden et al., 2004; Sundquist et al., 2004). Ülekaalulisus/rasvumine on omakorda tavaliselt seotud insuliiniresistentsuse, hüpertensiooni, südame isheemiatõve ja kolesterooli kõrvalekalletega ning võib olla metaboolse sündroomi üks komponente.

Käesoleva töö eesmärk oli hinnata SVH riskitegureid, nagu toitumine ja kehaline aktiivsus, lastel ja noorukitel, samuti uurida nende riskitegurite seoseid ülekaalulisuse/rasvumisega ning kardiovaskulaarse võimekuse seoseid metaboolse riskiga.

Töö eesmärgid

Käesoleva doktoritöö eesmärgid olid järgmised:

1) hinnata toitumisnäitajate vastavust soovitustele 9- ja 15-aastastel Eesti lastel ning kirjeldada 15-aastaste noorukite toitumistrende ajaperioodil 1984/85 kuni 1998/99;
2) kirjeldada toitumisega seotud terviseindikaatoreid ja neid mõjutavaid tegureid Eestis ning võrrelda neid teiste Euroopa riikide näitajatega;
3) kirjeldada kehalise aktiivsuse trende Baltimaades ja Soomes, samuti kehalise aktiivsuse, KMI (kehamassiindeksi) ja sotsiaal-majanduslike tegurite omavahelisi seoseid;
4) hinnata ala-, norm- ja ülekaaluliste Eesti ja Rootsi kooliõpilaste erinevusi toitumisnäitajates, samuti toitumise ja KMI omavahelisi seoseid;
5) uurida, kas kardiovaskulaarse võimekuse hindamine lastel võimaldab ennustada kõrget või madalat metaboolset riski ja sel juhul määrata kardiovaskulaarse võimekuse tase, mis vastaks madalale metaboolsele riskile;
6) uurida, kas C-1291G polümorfism adrenergilise α_{2A} retseptori geeni (*ADRA2A*) promootorpiirkonnas on seotud glükoosi metabolismi ja toitumisharjumustega.

Uuritavad ja metoodika

Epidemioloogilise ülevaate jaoks võrreldi toitumisega seotud Eesti terviseindikaatoreid vastavate Euroopa keskmiste näitajatega, kasutades selleks asjakohaseid rahvusvahelisi uuringuid (Artiklid II ja III). Ülekaalulisuse/rasvumuse levimust täiskasvanutel ja toidu tarbimisharjumusi Baltimaades ja Soomes võrreldi, kasutades *Baltic Nutrition and Health Survey* andmeid täiskasvanutel vanuses 19–64 a. (Pomerleau et al., 2000a) ja *FINBALT Health Monitoring Project* andmeid inimestel vanuses 16–64 a. (Helakorpi et al., 2002; Grabauskas et al., 2003; Kasmel et al., 2003; Pudule et al., 2003). Teatud toiduainete tarbimise sageduse hindamiseks kasutati *NORBAGREEN 2002* uuringu andmeid isikutel vanuses 15–74 a. (2003). HBSC (*Health Behaviour in School-aged Children*) uuringut 11-, 13- ja 15- aasta vanustel koolilastel kasutati

ülekaalulisuse levimuse võrdlemiseks lastel ja noorukitel (Currie et al., 2004). *FINBALT Health Monitoring Project* täiskasvanutel ja HBSC uuring lastel ja noorukitel võimaldasid ainsatena teha asjakohaseid rahvusvahelisi kehalise aktiivsuse trendide võrdlusi Baltimaades ja Soomes (Currie et al. 2000; Currie et al. 2004).

Uuringus käsitleti Eesti ja Rootsi lapsi ja noorukeid, kes osalesid Euroopa Noorte Südameuuringus (ENSU) (Artiklid I, IV, V, VI). ENSU valimi moodustamise põhiüksuseks oli kool, kus õppisid 9- ja 15-aastased lapsed. Planeeritud uuritavate arv mõlemas riigis oli 1000 õpilast, neist 500 9-aastast ja 500 15-aastast. Uuringus osalemiseks tehti ettepanek Eestis Tartu linna ja maakonna ning Kesk-Rootsis kahe piirkonna (Södertörni ja Örebro) koolidele. Osalema nõustunud koolid jagati klastritesse (linna-ja maapiirkondade koolid, eesti ja vene õppekeelega koolid Eestis). Kasutades eraldi igas klastris suurusega võrdelise tõenäosusega valikumeetodit, valiti välja 25 kooli Eestist ja 42 Rootsist. Uuringus kutsuti osalema kõik 3. klassi (keskmiselt 9-aastased) ja 9. klassi (keskmiselt 15-aastased) lapsed. Eestist andis nõusoleku osalemiseks 1176 (osalemismäär 79%) ja Rootsist 1132 (osalemismäär 50%) last ja noorukit ning nende vanemad. Andmeid koguti 1998. a. septembrist 1999. a. juuni lõpuni. Uuringu tegemiseks saadi ka luba Örebro linnavalitsuse, Huddinge Ülikooli haigla ja Tartu Ülikooli teadusuuringute eetikakomiteedelt.

Toitumise hindamiseks kasutati 24-tunni-toiduintervjuu meetodit. Toitumisandmete sisestamiseks ja esialgseks analüüsiks kasutati programmi *Micro-Nutrica* 2.0 (Artiklid I, IV, VI). Antropomeetrilistest näitajatest mõõdeti kehapikkus ja kehakaal ning nende näitajate põhjal arvutati kehamassiindeks. Mõõdeti uuritavate vererõhuväärtused. Kardiovaskulaarset võimekust (Artikkel V) hinnati maksimaalse koormustestiga (Hansen et al., 1989). Vere biokeemilistest näitajatest määrati TG, HDL-C, glükoos ja insuliin (Artiklid V ja VI). Metaboolsete riskitegurite klasterdamine (Artikkel V) toimus järgmiste näitajate põhjal: TG, HDL-C, insuliin, glükoos, nahavoltide paksus ja vererõhk. Metaboolse riski skoor arvutati nende kuue standardiseeritud näitaja keskmisena eraldi poistele ja tüdrukutele. Need uuritavad, kellel see skoor jäi alla 75 protsentiili, defineeriti kui madala metaboolse riskiga, ja need, kelle skoor oli üle 75 protsentiili, kõrge metaboolse riskiga lasteks. DNA ekstraheeriti verest ja C-1291G polümorfism adrenergilise α_{2A} retseptorgeeni (*ADRA2A*) promootorpiirkonnas määrati polümeraasi ahelreaktsiooni (PCR) teel vastavalt Lario et al. (1997) kirjeldusele (Artikkel VI). Andmete statistiliseks analüüsiks kasutati pakette StatView (versioon 4.0) ja SPSS (versioonid 11.0 ja 13.0) (Artiklid IV, V ja VI).

Tulemused

(I) Aastatel 1984–1999 koolilaste hulgas läbiviidud toitumisuuringute tulemused on küllaltki sarnased. Toitained, mida tarbitakse alla või üle soovitatu, on uuringutes jäänud samaks. Selgus, et Eesti koolilaste toitumises võib täheldada tendentsi tervislikumate valikute poole.

Üksikasjalikum uuring ENSU valimil näitas, et Eesti laste toidus oli perioodil 1998/99 siiski veel liigselt lipiide ja küllastatud rasvhappeid ning vähe kaltsiumi, vitamiine D ja B_2, 9-aastastel kiudaineid ning 15-aastastel magneesiumi ja tsinki.

(II) Sotsiaalmajandusliku ülemineku perioodil 1990ndatel aastatel võis Eestis täheldada ülekaalulisuse ja rasvumuse levimuse vähenemist nii täiskasvanud meeste kui ka naiste puhul. Võrreldes teiste Balti riikide ja Soomega oli ülekaalulisuse levimus meeste hulgas kõrgem Leedu ja Soome meestel; naiste puhul jäid näitajad riigiti üsna sarnaseks. Rasvumuse levimus täiskasvanutel on näidanud väikest tõusutrendi peaaegu kõikides riikides 2000ndate alguses, kuid samas oli ülekaalulisuse ja rasvumuse levimus Baltimaade lastel oluliselt madalam võrreldes teiste Euroopa riikide näitajatega. Vastavalt *FINBALT Health Monitoring Project´*i tulemustele olid positiivseteks muutusteks Eesti täiskasvanute toitumisharjumustes loomse rasva asendamine taimeõliga ning puu- ja köögiviljade tarbimise kasv. Sellest hoolimata näitasid *NORBAGREEN* uuringu andmed, et igapäevaste köögivilja tarbijate osakaal oli oluliselt madalam Eestis (35%) kui Rootsis (78%) ja Soomes (61%). *Baltic Nutrition and Health Survey* aastal 1997 näitas, et lipiidide osakaal päevasest energiatarbimisest oli meestel ja naistel Eestis madalam kui Lätis ja Leedus, kuid seda tarbiti siiski üle soovitatud taseme. Samas oli aastatel 1986–2001 keskmine päevane energiatarbimine (kcal) inimese kohta Eestis märkimisväärselt madalam kui teistes Euroopa riikides.

(III) FINBALT uuringu andmed näitasid, et kehaline aktiivsus (vähemalt kaks korda nädalas) oli täiskasvanute hulgas Baltimaades oluliselt madalam kui Soomes. Kui Läti ja Leedu mehed olid kehaliselt aktiivsemad kui naised, siis Soomes oli olukord vastupidine ja Eestis olulist vahet ei ilmnenud (alates 2002. a.). Kui võrrelda HBSC 1997/98 ja 2001/02 uuringute andmeid, siis kehaliselt aktiivsete laste arv suurenes kõikides riikides, välja arvatud Eestis. Baltimaade ülekaalulised/rasvunud täiskasvanud ja koolilapsed kaldusid olema kehaliselt vähem aktiivsed kui nende normkaalus eakaaslased. Majanduslikult kindlustatud peredest pärit koolilapsed ja täiskasvanud olid kehaliselt aktiivsemad võrreldes vähekindlustatutega.

(IV) Ülekaalulisus oli rohkem levinud nooremate Rootsi tüdrukute (17,0% vs 8,9% Eesti tüdrukutel) ja alakaalulisus Eesti mõlema vanuserühma tüdrukute hulgas (7,9% vs 3,5% Rootsi noorema ja 10,5% vs 5,1% Rootsi vanema vanuserühma tüdrukutel). Ülekaaluliste Eesti laste toit sisaldas norm- ja alakaaluliste eakaaslastega võrreldes rohkem lipiide (36,8% vs 31,7% päevasest toiduenergiast, %E), kuid vähem süsivesikuid ning nad tarbisid ka rohkem piima- ja lihatooteid. Eesti uuritavate absoluutne KMI seostus positiivselt munade (%E) ja negatiivselt maiustuste ja suhkru tarbimisega (%E). Rootsi ülekaalulisused noorukid tarbisid pigem rohkem valke (%E) ja piimatooteid. Risk ülekaalulisuseks seostus positiivselt päevase energiatarbimise ning kala- ja lihatoodete tarbimisega

(%E). Mõlemas riigis osutus ülekaalulisuse seos bioloogiliste teguritega (suguküpsus, vanemate KMI) tugevamaks kui seos toitumisega.

(V) Saadud tulemused kinnitasid kardiovaskulaarse võimekuse (CVF) pöördvõrdelist seost metaboolsete riskiteguritega lastel – kõrgema kardiovaskulaarse võimekusega lastel oli metaboolse riski skoor madalam. Tüdrukutel, kellel CVF tase oli üle 37,0 ml/kg/min, ja poistel, kellel see oli üle 42,1 ml/kg/min, on metaboolne risk vastavalt 3,09 ja 2,42 korda madalam kui nendel, kelle CVF jäi allapoole seda väärtust.

(VI) Tulemustest selgus, et α_{2A}-adrenoretseptori genotüüp omab märkimisväärset mõju magusate toiduainete tarbimisele. Magusate toiduainete (näit. shokolaad, kommid, magusad piimatooted) tarbimine oli oluliselt kõrgem ja vere glükoositase madalam GG genotüübiga isikutel võrreldes ülejäänud 2 genotüübiga. Teiste toiduainete tarbimise osas erinevusi genotüüpide vahel ei ilmnenud.

Järeldused

1. Kuigi Eesti koolilaste toitumises võis täheldada tendentsi tervislikumate valikute poole, esines ajaperioodil 1984 kuni 1989 siiski lipiidide ja küllastunud rasvhapete liigtarbimist. Kooliõpilaste toit sisaldas 1998/99. õppeaastal võrreldes toitumissoovitustega vähe kiudaineid ja mõningaid vitamiine ja mineraale.
2. Üleminekuperioodi sotsiaalmajanduslikud muutused on avaldanud mõju inimeste toitumisele ja toitumisega seotud terviseseisundile ning neid muutusi selles võib täheldada veel järgnevate aastakümnete jooksul.
3. Ülevaade kehalise aktiivsuse olukorrast Baltimaades ja Soomes näitas, et kehaline aktiivsus oli täiskasvanute hulgas Baltimaades oluliselt madalam kui Soomes. Koolilaste vaba aja kehalises aktiivsuses selget erinevust Baltimaade ja Soome vahel ei ilmnenud. Baltimaade ülekaalulised/rasvunud täiskasvanud ja koolilapsed kaldusid olema kehaliselt vähem aktiivsed kui nende normkaalulised eakaaslased. Jõukamatest peredest pärit koolilapsed ja täiskasvanud olid samas kehaliselt aktiivsemad võrreldes vähekindlustatutega. Saadud tulemused näitavad, et Baltimaade ülekaaluliste või majanduslikult vähekindlustatute seas on tõhusam sekkumistegevus tõstmaks kehalise aktiivsuse taset eriti vajalik. Selleks, et paremini võrrelda kehalise aktiivsuse trende ja seda mõjutavaid tegureid Euroopa riikides, on vajalik korrapärase järelevalvesüsteemi ja identse metodoloogia olemasolu.
4. Toitumise erinevused ala-, norm- ja ülekaaluliste kooliõpilaste vahel on riigiomased, mis viitab sellele, et ülekaalulistele isikutele suunatud sekkumisprojektide kavandamisel tuleb arvesse võtta kohalikke toitumisharjumusi. Laste KMI ning suguküpsuse ja vanemate KMI omavaheline seos osutus tugevamaks kui seos toitumisega nii Eesti kui ka Rootsi laste puhul. See viitab asjaolule, et lisaks toitumisele on mitmeid teisi olulisi tegureid, mis mängivad oma osa laste ülekaalulisuse kujunemisel.

5. Kardiovaskulaarne võimekus seostus metaboolsete riskitegurite klastriga lastel. Madalam metaboolse riski skoor seostus kõrgema kardiovaskulaarse võimekusega nii poistel kui ka tüdrukutel. Madala metaboolse riski jaoks said antud soovitused kardiovaskulaarse võimekuse tasemete kohta eraldi poistele ja tüdrukutele. Tulevikus on vajalikud longituud- ja/või sekkumis-uuringud, et käsitleda madala kardiovaskulaarse võimekuse mõju lapse-põlves haigestumusele südame-veresoonkonna haigustesse hilisemas eas.
6. α_{2A}-adrenoretseptori genotüüp omab märkimisväärset mõju magusate toidu-ainete tarbimisele. Täiendavad uuringud peaksid välja selgitama, kas see genotüübist sõltuv toidueelistus püsib täiskasvanuikka jõudmiseni ja kas see võib avaldada mõju tervisele.

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PUBLICATIONS

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Teadustöö

Peamised uurimisvaldkonnad on seotud kooliõpilaste toitumistavade, kehalise aktiivsuse ning tervislikku seisundit ja keha koostist mõjutavate tegurite, samuti nende omavahelise koostoime hindamise ja kirjeldamisega.

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