UNIVERSITY OF TURKU

CHILDHOOD SOCIOECONOMIC STATUS AND CARDIOMETABOLIC HEALTH IN ADULTHOOD

The Cardiovascular Risk in Young Finns Study

Elina Puolakka

TURUN YLIOPISTON JULKAISUJA – ANNALES UNIVERSITATIS TURKUENSIS
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ABSTRACT

The association of conventional childhood and adulthood cardiometabolic risk factors and adulthood socioeconomic status with adulthood cardiometabolic health is well-established. However, the association between childhood socioeconomic status and adulthood cardiometabolic health is less studied. The aim of this thesis was to study the role of childhood socioeconomic status in determining adulthood cardiometabolic health by investigating the association of childhood socioeconomic status with adulthood health behaviors, risk of metabolic syndrome and glucose abnormalities, and with subclinical markers of cardiovascular disease.

The thesis is a part of the Cardiovascular Risk in Young Finns Study (Young Finns Study), which is a prospective population-based follow-up study. The first cross-sectional study was conducted in 1980 and included 3596 children aged 3 to 18 years. Since then, regular follow-ups have been performed and, in this thesis, data until the 2011 follow-up was used. Cardiometabolic risk factors of participants have been measured since childhood and subclinical markers of cardiovascular disease were examined using noninvasive measurements in adulthood.

Higher childhood socioeconomic status associated with healthier lifestyle in adulthood in terms of dietary intake, smoking and leisure-time physical activity. Childhood socioeconomic status was inversely associated with the risk of having metabolic syndrome and impaired fasting glucose or type 2 diabetes in adulthood. Higher childhood socioeconomic status was associated with lower arterial stiffness, lower left ventricular mass of the heart and its better diastolic performance in adulthood. These findings show that higher socioeconomic status in childhood predicts better cardiometabolic health in adulthood determined by several well-established markers of cardiometabolic risk. Moreover, the findings of this thesis highlight the role of low childhood socioeconomic status as a risk factor of cardiometabolic health, alongside other conventional risk factors.

KEYWORDS: Childhood, socioeconomic status, cardiometabolic health, health behaviors, arterial stiffness, left ventricular mass, diastolic function, longitudinal

TURUN YLIOPISTO
Lääketieteellinen tiedekunta
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TIIVISTELMÄ

Lapsuuden ja aikuisuuden perinteisten kardiometabolisten riskitekijöiden ja aikuisiän sosioekonomisen aseman yhteys kardiometaboliseen terveyteen aikuisiässä on laajalti tunnustettu. Yhteyttä lapsuuden sosioekonomisen aseman ja aikuisuuden kardiometabolisen terveyden välillä on tutkittu vähemmän. Tämän väitöskirjatutkimuksen tavoitteena oli selvittää lapsuuden sosioekonomisen aseman yhteyttä aikuisiän kardiometaboliseen terveyteen tutkimalla lapsuuden sosioekonomisen aseman yhteyttä aikuisiän terveyskäyttäytymiseen, metabolisen oireyhtymän ja sokeriaineenvaihdunnan häiriöiden riskiin sekä subkliinisiin sydänsairauksien markkereihin.

Väitöskirjatutkimus on osa Lasten Sepelvaltimotaudin Riskitekijät (LASERI) -tutkimusta, joka on prospektiivinen väestöpohjainen seurantatutkimus. Ensimmäinen poikkileikkaustutkimus suoritettiin vuonna 1980 ja siihen osallistui 3596 3–18-vuotiasta lasta ja nuorta. Tämän jälkeen seurantatutkimukset ovat toteutuneet säännöllisesti ja tässä väitöskirjassa käytettiin dataa aina vuoden 2011 seurantatutkimukseen saakka. Osallistujien kardiometabolisia riskitekijöitä on mitattu lapsuudesta alkaen ja lisäksi subkliinisia sydänsairauksien markkereita on määritetty aikuisuudessa käyttäen kajoamattomia mittausmenetelmiä.

Korkeampi sosioekonominen asema lapsuudessa oli yhteydessä terveellisempiin ruokavalio-, tupakointi-, ja vapaa-ajan liikuntatottumuksiin aikuisuudessa. Lapsuuden sosioekonominen asema oli käänteisesti yhteydessä metabolisen oireyhtymän ja sokeriaineenvaihdunnan häiriöiden riskiin aikuisiässä. Lisäksi korkeampi sosioekonominen asema lapsuudessa oli yhteydessä joustavampiin valtimoihin, pienempään sydämen vasemman kammion massaan ja sen parempaan diastoliseen toimintaan aikuisiässä. Löydökset osoittavat, että korkeampi sosioekonominen asema lapsuudessa ennustaa parempaa kardiometabolista terveyttä aikuisiässä määritettynä useiden vakiintuneiden kardiometabolista riskiä osoittavien markkereiden avulla. Lisäksi löydökset korostavat lapsuuden alhaisen sosioekonomisen aseman roolia kardiometabolisen terveyden riskitekijänä, muiden perinteisten riskitekijöiden rinnalla.

AVAINSANAT: Lapsuus, sosioekonominen asema, kardiometabolinen terveys, terveyskäyttäytyminen, valtimoiden joustavuus, vasemman kammion massa, diastolinen toiminta, pitkittäistutkimus

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Abbreviations

ACC	American College of Cardiology
ADA	American Diabetes Association
AHA	American Heart Association
ARIC	Atherosclerosis Risk in Communities
BMI	body mass index
BSA	body surface area
Cdist	carotid artery distensibility
CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
FHS	Framingham Heart Study
FFQ	food frequency questionnaire
HDL	high-density lipoprotein
HbA1c	glycated hemoglobin
IFG	impaired fasting glucose
IGT	impaired glucose tolerance
IMT	intima-media thickness
LDL	low-density lipoprotein
LVH	left ventricular hypertrophy
LVM	left ventricular mass
LV	left ventricle
MetS	metabolic syndrome
PAI	physical activity index
PWV	pulse wave velocity
SD	standard deviation
SE	standard error
SEM	standard error of mean
SES	socioeconomic status
T2DM	type 2 diabetes mellitus
WHO	World Health Organization
Young Finns Study	Cardiovascular Risk in Young Finns Study
2-D	2-dimensional

List of Original Publications

This dissertation is based on the following original publications, which are referred to in the text by their Roman numerals:

I Puolakka E, Pahkala K, Laitinen TT, Magnussen CG, Hutri-Kähönen N, Männistö S, Pälve KS, Tammelin T, Tossavainen P, Jokinen E, Smith KJ, Laitinen T, Elovainio M, Pulkki-Råback L, Viikari JS, Raitakari OT, Juonala M. Childhood socioeconomic status and lifetime health behaviors: The Young Finns Study. *International Journal of Cardiology*, 2018; 258: 289–294.

II Puolakka E, Pahkala K, Laitinen TT, Magnussen CG, Hutri-Kähönen N, Tossavainen P, Jokinen E, Sabin MA, Laitinen T, Elovainio M, Pulkki-Råback L, Viikari JS, Raitakari OT, Juonala M. Childhood socioeconomic status in predicting metabolic syndrome and glucose abnormalities in adulthood: The Cardiovascular Risk in Young Finns Study. *Diabetes Care*, 2016; 39(12): 2311–2317.

III Puolakka E, Pahkala K, Laitinen TT, Magnussen CG, Hutri-Kähönen N, Kähönen M, Lehtimäki T, Tossavainen P, Jokinen E, Sabin MA, Laitinen T, Elovainio M, Pulkki-Råback L, Viikari JS, Raitakari OT, Juonala M. Childhood socioeconomic status and arterial stiffness in adulthood: The Cardiovascular Risk in Young Finns Study. *Hypertension*, 2017; 70(4): 729–735.

IV Laitinen TT, Puolakka E, Ruohonen S, Magnussen CG, Smith KJ, Viikari JS, Heinonen OJ, Kartiosuo N, Hutri-Kähönen N, Kähönen M, Jokinen E, Laitinen T, Tossavainen P, Pulkki-Råback L, Elovainio M, Raitakari OT, Pahkala K, Juonala M. Association of socioeconomic status in childhood with left ventricular structure and diastolic function in adulthood: The Cardiovascular Risk in Young Finns Study. *JAMA Pediatrics, 2017;* 171(8): 781–787.

1 Introduction

The early-life beginnings of the atherosclerotic process has been known for over a century, with the first studies to detail early atherosclerotic disease among autopsy specimens from young male casualties in World War I and the Vietnam war (Mcnamara et al., 1971; Mönckeberg, 1915). Current evidence suggests risk factors affect the development of the disease process in childhood and that the influence of these childhood risk factors persists into adulthood (Li et al., 2004; Mikola et al., 2017; Raitakari et al., 2003). Furthermore, these risk factors tend to cluster and persist from childhood to adulthood with life-course risk factor trajectories shown to be strong determinants of adulthood metabolic indicators and markers of vascular health (Juhola et al., 2011; Pollock et al., 2019; Raitakari et al., 1994).

The gradual inverse association of socioeconomic status (SES) with cardiovascular disease (CVD) is established (Mackenbach et al., 2008). Importantly, despite declines in national mortality rates of CVD in developed countries, socioeconomic inequalities in cardiovascular risk factors and in mortality remain or have increased (Bajekal et al., 2013; Kanjilal et al., 2006; Palosuo et al., 2009). Although several public health programs have been initiated in Finland that aim to diminish the socioeconomic gradient in cardiovascular health, the results have been unimpressive (Rotko et al., 2016, 2012). Since CVD remains the most common cause of mortality and one of the main reasons for disability in the Finnish population, diminishing the socioeconomic gap and improving health among those of low SES is relevant also from the perspective of the national economy (Ministry of Social Affairs and Health, 2009; Statistics Finland, 2019a). Furthermore, since Finland is a welfare state with an affirmed intention to ensure the equality of all citizens, it is suggested that all health inequalities in Finland, which are on paper avoidable, can be considered unfair and unjust and be called as health inequities (Marmot et al., 2014; Palosuo et al., 2009).

The long-term impact of socioeconomic inequalities have been identified, with childhood SES shown to determine the future risk of CVD independent of adulthood SES (Galobardes et al., 2006c). The association between low SES and higher risk of CVD is largely mediated by biological, behavioral, psychological and social risk factors (Lynch et al., 1996). Since the detrimental effect of low childhood SES on

adulthood risk of CVD is at least partly determined by the differences in the risk factors across the lifetime, interventions focusing on families and children with low SES might be important not only to diminish the socioeconomic gap in CVD but also to reduce the overall morbidity and mortality due to CVD (Lynch et al., 1997). However, since previous studies have revealed conflicting results on the association of childhood SES with different health behaviors, the role of childhood SES on long-term health outcomes requires further study to reinforce, refine, and promote prevention and intervention programs.

Although the association of childhood SES with biological risk factor levels in adulthood has been shown, evidence of the long-term association between childhood SES and subclinical markers of CVD is limited (Kivimäki et al., 2006c; Nash et al., 2011). Subclinical changes in the function and structure of the cardiovascular system can be detected noninvasively several decades before clinical disease presents and are independent predictors of future cardiovascular events (Levy et al., 1990; Mitchell et al., 2010).

In addition to subclinical changes of CVD, metabolic disturbances, especially abnormalities of glucose metabolism, are important predictors of cardiovascular outcomes (Sarwar et al., 2010). Cardiometabolic disease is used to describe the clustering of disorders that together lead to CVD and type 2 diabetes mellitus (T2DM) and also the interface between these two diseases (Fisher, 2006; Pescatello, 1999). One interrelated concept is metabolic syndrome (MetS), a cluster of risk factors that can increase the risk of both CVD and T2DM more than its components alone (Dekker et al., 2005; Laaksonen et al., 2002). However, the literature on the association between childhood SES and MetS show inconclusive results (Chichlowska et al., 2009; Langenberg et al., 2006).

Therefore, novel, high-quality studies that examine the association of childhood SES with the conditions preceding clinical cardiometabolic disease will help determine the pathways leading from childhood SES to adulthood cardiometabolic health. The premise being that only by understanding these pathways can children of low SES families be most effectively averted from adverse trajectories of cardiometabolic health towards those that promote cardiometabolic well-being in adulthood (Pollock et al., 2019). The Cardiovascular Risk in Young Finns Study (Young Finns Study) is an on-going multicenter follow-up study of 3596 participants that was initiated to assess biological and lifestyle factors underlying CVD and its risk factors beginning from childhood. This thesis, as part of the Young Finns Study, will add evidence to the case concerning the association between childhood SES and adulthood cardiometabolic health.

2 Review of the Literature

2.1 Indicators of socioeconomic status

SES represents an individual's position in society, comprising resource-based elements (income, educational achievements, occupation, wealth) and prestige-based measures linked to views of status in social hierarchy (Krieger et al., 1997). Multiple different indicators are used to define SES, but income, education and occupation are those most often applied in epidemiological studies (Kaplan et al., 1993). These different indicators interact with each other and have shared effects in creating the socioeconomic differences in health, morbidity and mortality. Although correlated, these indicators cannot replace each other, as they measure different phenomena and represent distinct causal processes (Geyer et al., 2006).

In addition to these conventional indicators of SES, the material conditions under which an individual lives can be used. For example, does the individual own a refrigerator, car, or television (Kaplan et al., 1993), and among urban populations whether housing is owned or rented (Galobardes et al., 2006a). In recent years, an increasing number of studies have used area-based measures that consider characteristics of the area where an individual lives, in lieu of individual SES (Kivimäki et al., 2018; Roux et al., 2001). Area-based measures reflect differing psychosocial factors, physical environment, traditions and cultural norms across societies (Kivimäki et al., 2018; Roux et al., 2001), with disadvantaged neighborhoods shown to associate with increased risk factors and incidence of cardiometabolic disease, independent of individual SES (Kivimäki et al., 2018; Roux et al., 2001).

To take the multidimensionality of socioeconomic indicators into account, several different composite indices and scores have been formulated. The indices are mainly used in sociological studies to measure social stratification (Haug, 1977). Therefore, the indices are not systematically validated in public health studies and their role in research regarding the association of SES with cardiovascular health is not established. In addition, combining different indicators of SES into one index can obscure distinct effects of different indicators of SES on the outcome of interest (Krieger et al., 1997; Liberatos et al., 1988).

Despite having multiple ways to determine SES, occupation, education and income are valued above others in developed countries and are discussed further in the following sections.

2.1.1 Occupation

The mechanisms underlying the association between occupation and health have been well considered. Occupation takes both education and income into account as it reflects both the conventional income level of the occupation and the educational skills required for the job (Galobardes et al., 2006a). Furthermore, occupation can affect health by reflecting physical exposures related to work environments or physical demands related to certain work tasks. These exposures can have toxic effects or activate different endocrinal and immune pathways. Occupation can also affect psychosocial factors through the environment where the work is accomplished (Backé et al., 2012). These psychosocial factors include social networks, stress level, feeling of control and independence (Galobardes et al., 2006a). A substantial part of the association between lower occupation and worse cardiovascular health can be explained by lower job control and lower social support among lower grade workers (Bosma et al., 1998; Marmot et al., 1997). Findings from the Whitehall II prospective cohort study suggested that an imbalance between personal efforts and rewards increases the risk for coronary heart disease (CHD) 2-fold (Bosma et al., 1998). Similarly, according to the demand-control-support model, workers with jobs characterized by high demands, low decision liberty and low social support are predisposed to poor psychological welfare and to higher risk of CVD (Johnson et al., 1989). Occupational prestige might also impact the occupational gradient in health, through higher self-esteem and better opportunities of social support among individuals with high-prestige jobs compared with those with low-prestige jobs (Fujishiro et al., 2010).

Occupation can be classified on the basis of status, roles, power, prestige, job characteristics, income, education, traditions, beliefs or values (Kaplan et al., 1993). It can be assessed by asking individuals to record their current or longest held occupation or to indicate their occupational category from predefined categories (Galobardes et al., 2006a). To categorize occupations, different scales, conventionally based on prestige or social standing, have been formed (Galobardes et al., 2006b). A commonly used scale in health equity research is the British Registrar General's Scale, which includes 5 categories (I professional, II managerial and technical, IIIN skilled non-manual; IIIM skilled manual; IV partly skilled; V unskilled), based on occupational skills (Pevalin et al., 2002). Another widely used but more basic classification divides occupations to white-collar (professional, managerial and administrative) and blue-collar (manual) jobs. Occupation of the

head of household is commonly used as a measure of early-life SES but the definition varies across studies. Occupation of the father has been widely used in epidemiological studies but consideration of the occupation of the mother has gained attention (Boylan et al., 2018; Lamont et al., 2000; Leino et al., 1996; Osler et al., 2003).

The advantage of using occupation as an indicator of SES relates to its availability from many large and routine data sources such as from census data and death certificates (Galobardes et al., 2006a). Other important advantages are the willingness to report one's occupation and that it is easily remembered, which reduces the amount of missing data and misclassification in responses to questionnaires. A disadvantage is that comparisons between time points and nationalities are difficult (Galobardes et al., 2006a) and that the variety of occupations and the status of certain occupations continuously changes (Kaplan et al., 1993). In addition, health status of the individual can complicate the use of occupation as an indicator of SES in health studies (Brunner et al., 2006). For example, poor health could result in a decrease in occupational level or even in unemployment. When occupation is used as an indicator of SES there are also difficulties in categorizing those not currently employed, as the reasons for not working are diverse (e.g. studying, retired, home or carer responsibilities, unemployed) (Galobardes et al., 2006a). Therefore, a global measure that categorizes all of those not currently working as "unemployed" might misclassify current SES and diminish the socioeconomic differences observed in study populations.

2.1.2 Education

Of the three conventional indicators of SES, education is typically obtained first in the life-course and contributes both to occupation and, through occupation, to income (Lahelma et al., 2004). Higher education is associated with a better economic situation owing to a lower risk of under or unemployment and because education level moderately determines income (Saegert et al., 2007). The important role of education in setting-up an individual for long-term higher SES is exemplified by a Finnish study that has shown between 40% and 80% of educational inequalities in long standing illness and self-rated health are mediated through occupational attainment and income (Lahelma et al., 2004).

Education might influence health through attitudes towards health and health behaviors, and might associate with knowledge and skills that promote health (Zajacova et al., 2018). As poor health literacy skills might complicate adherence to health promotion and medication advice and be a barrier in access to health care and information, educational differences in 'health literacy' are suggested to partially explain the association between education and health (Van Der Heide et al., 2013).

Although, education might not explain all barriers to adherence with a Finnish study suggesting that there was no association between correct knowledge of national physical activity recommendations and physical activity behavior (Hankonen et al., 2017). Higher education is also associated with greater social resources including greater social support and better sense of control (Ross et al., 1995). These resources might help cope with problematic and stressful life-events and promote better cardiovascular health (Rozanski et al., 1999).

There are multiple ways to define educational attainment. A conventional way is to measure education as the number of years of education completed. This supposes that the time spent in education is more important than the educational achievements themselves and that every year spent in education is as important as the previous (Galobardes et al., 2006a). However, the effect of years of education completed on income might not be incremental, with benefits of education having bounds at time points when milestones in education are achieved (James et al., 2015). Because this might be reflected in the health outcomes, some studies have used ordinal variables coinciding with achievement of academic qualifications (primary, vocational, high school, university, etc.) to describe the level of education attained.

Parental or guardian education is widely used in epidemiological studies to describe early-life SES (Kestila et al., 2006; Smith et al., 2011). Although only few studies compare the association of mother's and father's education with adult cardiometabolic risk, the results tend to suggest that father's education has the most consistent associations with studied risk factors (Ball et al., 2006; Kvaavik et al., 2012). A weaker association between mother's education and adulthood health is suggested to relate to lower general educational level among women in research populations (Kvaavik et al., 2012). Therefore, in populations with growing gender equality, the significance of mother's education might be increasing.

Education has multiple advantages as an indicator of SES. The final educational attainment is typically reached in young adulthood and when compared with occupational status or income, education is less easily influenced by changeable conditions such as acute or chronic disease (Galobardes et al., 2006a). Furthermore, education can be appropriately used to describe SES among those not in paid employment, including home makers, the retired and the unemployed (Krieger et al., 1997). Education also tends to have better reliability and validity as an indicator of SES compared with occupation and income. When education is inquired by questionnaire, missing items tend to be lower than inquiry of income (Kaplan et al., 1993); and categorization of educational attainments tends to be easier than occupation. When a U.S. study investigated associations between different conventional socioeconomic indicators and cardiovascular risk factors, education appeared to have the strongest and most consistent associations with the cardiovascular risk factors (Winkleby et al., 1992). Of different causes of mortality,

the strongest association with education is seen for cardiovascular mortality (Smith et al., 1998). Therefore, education is proposed to be an especially good indicator of early life socioeconomic circumstances, which are thought to play a unique role in the risk of CVD.

Educational level is widely used in Finnish epidemiological studies to asses SES with the large Finnish population survey, the FinHealth Study, using educational attainment as an indicator of SES (Koponen et al., 2018). However, in studies with a wide age-range, like the FinHealth Study, it should be noted that, on average, educational levels among the younger generation are relevantly higher than among the older generation (Lahelma et al., 1994). Furthermore, despite education representing quite a stable measurement of SES, health selection (reverse causality between SES and health) can play a role in the association between education and health, especially during the first decades of life (Hoffmann et al., 2018). The stability of education can also be a drawback as it is not sensitive to varyiable socioeconomic resources related to, for example, changing employment status or income (Davey Smith et al., 1998; Kuhn et al., 1995; Liberatos et al., 1988). Moreover, education is suggested to be a less sensitive measure of socioeconomic inequality owing to the relatively small range of educational levels compared with income and occupation; and this variability is decreasing in younger-aged cohorts (Krieger et al., 1994; Liberatos et al., 1988). Because of these limitations, other markers of SES are suggested to be used alongside education (Krieger et al., 1994).

2.1.3 Income

Income operates mainly through a direct effect on material resources. Better income affords access to better food, housing, health services and sporting activities, which all affect an individual's health (Galobardes et al., 2006a). In several countries, income might affect health indirectly through educational level and social mobility by influencing access to education (Saegert et al., 2007). The opposite correlation between income and education also exists, but it has been shown to be weak to moderate (Geyer et al., 2006). Moreover, income is not equivalent to occupational class, as income can vary within the same occupation (Liberatos et al., 1988).

Income is presumed to affect psychological factors of self-confidence and the sense of security, which together can influence health (Galobardes et al., 2006a). Lack of money can create challenging events and conflicts in life that might affect mental and physical health (Saegert et al., 2007). Additionally, when confronting stressful or demanding life events, higher income might impact the ability to cope with the stress (Geyer et al., 2006).

Information on income can be gathered by asking people to report their absolute income or by placing them into predefined income categories (Galobardes et al.,

2006a). Income can be measured according to the individual's income, the income of the head of household or the income of the household. Household income is suggested to be an optimal economic indicator of SES in health research (Duncan et al., 2002) and it can be adjusted to the size of the family and its associated cost of living; referred to as an "equivalent income" (McClements, 1977). To compare households of different sizes and structures, Statistics Finland reports income statistics calculated per consumption unit (separate weights for adults and children of different ages) (Statistics Finland, 2018b). However, adjustment for family size does not substantially affect relative mortality rates (Duncan et al., 2002). Currently, the most commonly used way is to report the gross income of the family, but studies have suggested, that the ideal method would be to gather data on the income that persons can actually spend on living (Galobarde et al., 2006a).

Income is suggested to be the best measurement for material living standards and to have similar or even stronger effects on mortality when compared with occupation or education (Duncan et al., 2002; Geyer et al., 2006). When the association between education and mortality was studied, the results revealed that educational attainment was not associated with mortality after adjusting for health behaviors and income (Lantz et al., 2010). However, income was associated with mortality even after adjustment for conventional behavioral risk factors. Income is suggested to be superior to education in indicating progression of chronic conditions and physical limitations (Herd et al., 2007). Consistent with this were data from a longitudinal U.S. study that suggested of the conventional indicators of SES, economic indicators (family income and wealth) were the most optimal for health research (Duncan et al., 2002). Conversely, a Finnish study did not support the superiority of income in assessing socioeconomic health differences (Lahelma et al., 2004), with two thirds of differences in self-rated health by income explained by education and occupation.

Limitations of using income as an indicator of SES include higher non-response and reporting bias than for occupation or education, owing to the sensitive nature of income in many cultures (Galobardes et al., 2006a). Secondly, of the conventional indicators of SES, income can change most during a short time period (Elfassy et al., 2019). However, income volatility and drops have independent impacts on the risk of CVD mortality.

In conclusion, all three key indicators of SES (income, education, occupation) are widely used in epidemiological research to examine health inequalities and to describe childhood or early-life SES. A Finnish study has shown these three key indicators of SES are independent determinants of health, even though some of the effects of each indicator can be accounted for, or mediated through, other indicators (Lahelma et al., 2004). Therefore, it is recommended to refrain from seeking a single definitive socioeconomic indicator to use in health equity studies. Similarly, studies

from Sweden and Germany have suggested that in the case of myocardial infarction, all three conventional indicators of SES are independently associated with morbidity and mortality, even after taking into account the other two indicators (Geyer et al., 2006). The authors proposed that to understand the persistent pattern of socioeconomic differences in health, all dimensions of SES should be acknowledged. Pathways behind the socioeconomic differences are likely to be complex and all three conventional indicators tend both to illustrate general socioeconomic hierarchy and mirror specific effects of each of the indicators (Lahelma et al., 2004).

2.2 Association of socioeconomic status with health behaviors

The estimated contribution of health behaviors to cardiovascular health varies but their major role in prevention of CVD is well-established. According to the American College of Cardiology (ACC) / American Heart Association (AHA) Guideline on the Primary Prevention of Cardiovascular Disease, more than 80 percent of all cardiovascular events are preventable through modification of lifestyle factors (Arnett et al., 2019). Dietary behavior, smoking, physical activity and alcohol consumption are the four key health behaviors that substantially influence the risk of cardiometabolic disease and are among the main targets of primary prevention in clinical practice. These behaviors also tend to cluster early in life and associate with CVD risk factor profile (Raitakari et al., 1995).

Favorable modification of health behaviors is recognized to be a cornerstone of the reduction of the risk of CVD (Havranek et al., 2015; Maruthur et al., 2009). For instance, an intensive 6-month behavioral intervention focusing on loss of weight, increase in physical activity and favorable changes in diet reduced 10-year risk of CHD by 12% to 14% (Maruthur et al., 2009). Similar interventions toward more favorable health behaviors have proven successful also among children (Laitinen et al., 2018; Van Horn et al., 2003).

Virtually every health behavior is determined by SES and these socioeconomic differences play major roles in forming the socioeconomic gap in cardiovascular health (Petrovic et al., 2018). In Finland, health behaviors vary most between socioeconomic groups among those of working-age (Palosuo et al., 2009). However, the Young Finns Study has previously shown that the socioeconomic gap in health behaviors is evident in youth and affected by both individual and parental SES (Leino et al., 1999). A systematic review of 114 studies showed that the median contribution of four main health behaviors (diet, physical activity, smoking, alcohol consumption) to the association between SES and CVD varied between 18% and 30% depending on which indicator of SES was used (Petrovic et al., 2018). However,

in those studies from northern Europe, the contribution was higher varying from 24% to 36%. Nationwide Finnish survey data found that seven health behaviors explained 54% (men) and 22% (women) of the observed educational differences in cardiovascular mortality (Laaksonen et al., 2008). Smoking, consumption of vegetables and physical activity explained most of the differences, whereas the effects of type of fat used on bread, relative weight, coffee drinking and alcohol consumption were small.

Although people are considered to be largely responsible for their own health behaviors, current evidence suggests the socioeconomic gradient in health behaviors involves "more than freely chosen lifestyle" (Pampel et al., 2010, 350). Incentives and motives to follow healthful behaviors and means and possibilities to reach the goals according to the motives might differ across SES groups. Therefore, to diminish health inequalities, more understanding of the social determinants of health behaviors is needed (Marmot et al., 2014). Understanding and improving the social determinants and *the causes of causes* of health behaviors might enable the healthy choice to become the easiest one, even for those with low SES. An increasing knowledge in clinical practice of the importance of SES might help shift the focus of health professionals from intervening on individual behaviors to discovering and impacting on drivers of the behaviors.

In the subsequent chapters, the role of SES and social determinants on the four main aspects of cardiometabolic health behavior (diet, smoking, physical activity, alcohol consumption) are discussed.

2.2.1 Diet

There is broad evidence suggesting the association of dietary factors with the development of CVD (Micha et al., 2017). The U.S. Dietary Guidelines suggest that a beneficial diet for cardiometabolic health includes vegetables, fruits, whole grains, low-fat or non-fat dairy, seafood, legumes and nuts, but only moderately alcohol, low amounts of red and processed meat, sugar-sweetened foods and drinks and refined grains (U.S. Department of Health and Human Services et al., 2015). According to the Finnish nutrition recommendations people should eat at least 500 g or 5–6 portions a day of vegetables, berries, fruits and mushrooms (Ravitsemus-neuvottelukunta, 2014). Fish should be eaten two to three times a week and weekly intake of red meat and meat products should not exceed 500 g. Furthermore, the dietary guidelines include recommendations for replacing saturated fat with polyunsaturated and monounsaturated fat, which can be accomplished by following the overall healthful dietary pattern of these dietary guidelines. The AHA guidelines for heart healthy diet match the U.S. and Finnish dietary guidelines (Eckel et al.,

2014). Different components of diet can have either preventive or damaging effects on cardiovascular health and thus, the key dietary factors are also part of the goals of ideal cardiovascular health published by the AHA (Lloyd-Jones et al., 2010). In spite of the international and national recommendations, which are based on wide evidence, approximately 45% of cardiometabolic deaths in the U.S. are attributable to poor diet quality (Micha et al., 2017).

Because the effectiveness of multivitamin and mineral supplementation in prevention of CVD has not been shown, even if the protective effects of nutrients as part of dietary intake have been shown, the effects of particular foods in prevention of CVD are emphasized (Mozaffarian et al., 2011). The most important food groups shown to have protective effects against CVD include vegetables, fruits and fish (Bazzano et al., 2002; de Goede et al., 2010). The association between fruit and vegetable consumption and the risk of CVD is dose-dependent and the mechanisms might include antioxidant and anti-inflammatory properties of nutrients but also low glycemic load, low energy density and other functional properties of diets rich in fruits and vegetables (Bazzano et al., 2003; Wang et al., 2014). Furthermore, randomized controlled trials have shown that replacing saturated fats with polyunsaturated vegetable oil reduces CVD by one third (Sacks et al., 2017). Also health-promoting effects of fish mostly function through marine omega-3-fatty acids, which are shown to have anti-lipidemic, anti-inflammatory and anti-arrhythmic effects (Saravanan et al., 2010). Conversely, higher consumption of red and processed meat is associated with higher risk of T2DM and CVD (Micha et al., 2010). The mechanisms explaining this association include unfavorable effects of red meat on the lipid profile and detrimental effects of heme iron (Guasch-Ferré et al., 2019; Qi et al., 2007). A systematic review of the evidence for causally-linked dietary exposures with CVD found strong evidence for higher intake of vegetables and lower risk of CVD whereas the evidence for fruit and fish was moderate, and for meat insufficient (Mente et al., 2009).

Data from the Young Finns Study has shown that food behavior and dietary patterns are established in childhood, from which they tend to track into adulthood (Mikkilä et al., 2005). Key dietary components of cardiovascular health (fruit and vegetable consumption) are shown to associate with subclinical markers of CVD in adulthood, independent of other conventional childhood and adulthood risk factors (Aatola et al., 2010b; Juonala et al., 2010b). Additionally, the results of the Special Turku Coronary Risk Factor Intervention Project have shown that dietary counselling starting in early childhood has favorable effects on serum lipid profile and insulin sensitivity throughout the early years of life (Laitinen et al., 2018). Therefore, components of diet are a key modifiable risk factors, already in childhood, in terms of promoting future cardiometabolic health.

In addition to their strong contribution to CVD burden, some components of diet are suggested to explain a significant part of the socioeconomic differences in CVD mortality (Laaksonen et al., 2008; Roth et al., 2018). Individuals with higher SES are more likely to report healthy food habits and different indicators of SES have been suggested to have cumulative effects on consumption of different foods. (Galobardes et al., 2001; Lallukka et al., 2007). Longitudinal data from the Coronary Artery Risk Development in Young Adults Study has shown that the socioeconomic gap in diet quality was apparent during the whole 20 year study period, although the gap between SES groups was diminished over time (Sijtsma et al., 2012).

Several studies have investigated the association between SES and different foods shown to have strong associations with cardiovascular health. Some have suggested that of all food patterns, the consumption of fruit is most consistently associated with SES (Boylan et al., 2011). According to meta-analysis of data from eleven studies across seven countries, fruit consumption was about 24 g/day higher for men and 34 g/day higher for women among those with the highest education compared with those with the lowest education (Irala-Estévez et al., 2000). The direction of the association between SES and vegetable consumption has varied across different European countries (Boylan et al., 2011; Irala-Estévez et al., 2000; Prättälä et al., 2009). In northern Europe, with lower availability and affordability of vegetables and lack of cultural tradition to use them in cooking, higher SES is associated with higher consumption of vegetables (Prättälä et al., 2009). Finnish, Danish, Swedish and Norwegian cross-sectional data from three nationally representative surveys in the 2000s showed that socioeconomic differences in consumption of fruit and vegetables were apparent in all countries and that the differences persisted over time (Fismen et al., 2016). Studies from the U.S. have also shown similar results (Deshmukh-Taskar et al., 2007; Dubowitz et al., 2008). More traditional food pattern, characterized by higher consumption of rye, potatoes, milk, butter, sausages and coffee and lower consumption of fruit, berries and dairy products, is associated with lower educational status in data from the Young Finns Study (Mikkilä et al., 2005). In contrast, in southern Europe, where there are strong traditions to use fresh vegetables in everyday cooking, lower SES has been shown to associate with higher consumption of vegetables (Prättälä et al., 2009).

The FINDIET study has investigated dietary differences between education levels since 1982 (Valsta et al., 2018). According to the results of the FINDIET study, the most apparent dietary differences between socioeconomic groups have been in the consumption of fruits and vegetables – with higher consumption among those with higher SES (Raulio et al., 2016). Results from the Finnish national survey, FinHealth 2017, suggest highest educated women consume the highest amounts of fresh vegetables, fruits and berries (Koponen et al., 2018). Although a similar gradient was seen in consumption of fresh vegetables for men, consumption of fruits

and berries were not significantly different across the three educational groups. Higher intake of fruits and vegetables among people with higher SES follows the overall dietary pattern that has proven more health-conscious in the Young Finns Study (Mikkilä et al., 2005).

Health-consciousness in the diet is also reflected in the type of meat consumed since high SES is associated with higher intake of lean meat and fish and lower SES associated with consumption of more fatty and processed meats (Maguire et al., 2015; Raulio et al., 2016; Shimakawa et al., 1994). The inverse association between SES and consumption of red and processed meat has been shown using all three conventional indicators of SES; income, education and occupation (Maguire et al., 2015). Similarly, a cross-sectional study conducted in Denmark suggested that higher SES determined by educational level was associated with lower energy-adjusted intake of fat (Groth et al., 2001). In contrast, a Swedish cross-sectional study suggested no differences across occupational groups in energy-adjusted intake of fat, saturated, mono-unsaturated or poly-unsaturated fatty acids, or the ratio of polyunsaturated to saturated fatty acids (Lindstrom, 2000). An analysis of European studies on dairy consumption from 1985–1997 indicated the consumption of cheese was higher among people with higher SES whereas lower SES was associated with higher consumption of butter, but only in Nordic countries (Prättälä et al., 2003). Therefore, in addition to absolute differences in intake of fat, also the sources of different types of fats might differ across SES groups.

The association between parental SES and food pattern of children has also been shown in several countries (Emmett et al., 2015; Fernández-Alvira et al., 2014), with an English cohort finding maternal education affects dietary pattern of children already at the age of 3 years (North et al., 2000). According to a study on children aged 2–9 years from eight European countries, children with lower parental SES have a greater tendency to have a more highly "processed" dietary pattern, describing an unhealthy dietary behavior (Fernández-Alvira et al., 2014). In the same study, the direct association between SES and healthy food pattern, suggesting that children of parents with higher SES may have greater tendency to eat healthy, was also apparent but only in half of the studied populations. Cross-sectional data from Finnish children are consistent with these, showing low maternal education to be associated with low healthy eating index among six-year-old children (Kyttälä et al., 2014). As for adult diet, Finnish studies among children have consistently shown higher SES is associated with higher vegetable use, both in school lunches and at home (Haapalahti et al., 2003; Kaikkonen et al., 2012).

Additionally, low SES in childhood has been shown to associate with poorer diet quality in adulthood (Atkins et al., 2015; Mishra et al., 2004). However, the mutual role of child and adult SES is emphasized since the most favorable diet is associated with stable high SES from childhood to adulthood and upward intergenerational

mobility is shown to diminish the effects of low child SES on adult diet quality. Of the diet components related to cardiovascular health, the strongest association with childhood SES is observed for fruit and vegetable intake, independent of adult SES (Watt et al., 2009). Sex has been suggested to impact the temporal effect of SES on diet quality, with a Danish study finding childhood SES was more important in determining adult food patterns among women whereas adult SES was of greater importance in men (Hare-Bruun et al., 2011).

Previous studies in Finnish populations have shown conflicting results on the role of childhood SES on adult diet quality. Retrospectively assessed parental education and childhood economic difficulties showed no significant associations with adult index of healthy food habits (Lallukka et al., 2007). In contrast, the Young Finns Study has shown that low parental education predicts poorer diet quality in young adults, in terms of daily use of butter, even better than the participant's own education (Leino et al., 1999).

Mechanisms behind the association between child SES and adult diet might involve several factors. Low SES people can lack nutrition knowledge and therefore be more neglected from nutrition-related communication, which can shape the dietary behavior of parents in low SES families (Patterson et al., 2001; Wardle et al., 2000). For children, parents influence eating behavior by restricting and promoting exposures to different foods and are role models for different eating patterns (Scaglioni et al., 2018). Children model not just their parent's eating behaviors, but also attitudes regarding different dietary patterns and body image. The younger the child, the stronger the direct control that parents have on their child's food choices (Gibson et al., 2012). Low SES mothers tend to focus on infant weight as the best marker of their child's health and use food as reward for good behavior that can result in feeding patterns predisposing to weight gain (Baughcum et al., 1998).

The association between low SES and poor diet has been suggested to be mediated by the costs of different diets, as lowest-cost diets tend to be the least healthy (Andrieu et al., 2006). Higher SES associates with higher food expenditure, which associates with healthier food purchasing (Pechey et al., 2016). Data from three European countries suggested that middle-class mothers considered health more often, and costs less often, in their food choices than lower-class mothers (Hupkens et al., 2000). However, these differences did not explain the class patterns in food consumption. In contrast, an English study found that the association between SES and fiber intake was diminished after adjustment for motivational factors (Steptoe et al., 1999). These motivational factors were importance of price, and familiarity, mood control and sensory appeal in food selection. The socioeconomic differences in dietary intake might also be reinforced by environmental inequity. Neighborhoods in areas of low SES are often considered "food deserts" because people have only limited access to nutritious food (Walker et

al., 2010). In contrast, those in high SES areas tend to have easier access to supermarkets, which is shown to associate with higher intake of fruit and vegetable (Morland et al., 2002; Zenk et al., 2005).

In conclusion, both social and physical environmental factors influence the development of eating behavior from the early life through to adulthood. Since diet behavior and dietary patterns established in childhood tend to track into adulthood, the environmental factors related to childhood SES are of major importance in determining lifelong dietary behaviors (Mikkilä et al., 2005).

2.2.2 Smoking

Smoking accounts for a significant portion of cardiovascular morbidity and mortality globally (National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health., 2014). Among smokers aged over 60 years, the risk of cardiovascular mortality is 2 times higher than among non-smokers (Mons et al., 2015). The mechanisms explaining the association between smoking and atherothrombotic disease are multiple (Csordas et al., 2013). They include endothelial dysfunction and injury caused by oxidative stress and enhanced development of vulnerable plaques caused by activation of matrix metalloproteinases and enhanced inflammation. In addition, smoking operates synergistically with conventional CVD risk factors that tend to cluster in smokers (Kvaavik et al., 2004). The evidence supports that smoking cessation has beneficial effects on cardiovascular health and that the risk for CHD is reduced significantly within 5 years from cessation, even among heavy smokers (Duncan et al., 2019).

Of all health behaviors, smoking is one of the main contributors to socioeconomic differences in cardiometabolic disease, which might relate to the distinct gradient in smoking prevalence across socioeconomic groups (Harald et al., 2006; Petrovic et al., 2018). Data from the Whitehall Study found that of the best-practice interventions they examined, smoking cessation had the greatest effect on reducing socioeconomic differences in the risk of ischemic heart disease mortality (Kivimäki et al., 2008).

The higher prevalence of smoking among people with low SES is widely recognized in developed countries. For example, in the U.S. in 2016, 25% of people in poverty were daily smokers; higher than the 14% of those not in poverty (Jamal et al., 2018). A similar difference was also seen by education status. Results from a study in Finnish adults were comparable with structural, material and subjective measurements of SES all strongly associated with the prevalence of smoking (Laaksonen et al., 2005). Consistently, it has been shown that the direction of the association between SES and smoking differs between southern and northern European countries and that in southern European countries, especially among older

women, the association is direct (Cavelaars et al., 2000). However, a more recent study on youth populations of 13 European countries revealed that among younger generations this difference has declined (Kuipers et al., 2015), with lower SES associated with higher smoking frequency in all studied countries, except Portugal.

The Tobacco-free Finland 2030 network has succeeded in substantially decreasing the smoking prevalence in the general adult population (Patja et al., 2017). The prevalence has decreased in all educational groups, but the socioeconomic difference is still substantial. In 2017, the prevalence of daily-smokers among the lowest educated was 23% in men and 22% in women whereas among highest educated the proportions were 9% in men and 8% in women (Koponen et al., 2018). In line with other developed countries, the socioeconomic gap in smoking is even more apparent among young people and it has been suggested that of all adolescent behavioral factors, smoking is the strongest determinant of socioeconomic differences in adolescent health (Kestilä et al., 2009). In 2017, 23% of Finnish students in vocational schools reported to be a daily-smoker whereas the corresponding prevalence was only 3% in high schools (Halme et al., 2018).

Socioeconomic inequalities in tobacco use are most apparent at two stages of life (Kunst et al., 2004): adolescence, where people with lower family SES are at greater risk of starting smoking; and later in adulthood, when attempts to quit smoking tend to be more challenging for people with lower SES. Social determinants of smoking include different exposure and vulnerability to tobacco use across SES groups (David et al., 2010). Adolescents with lower family SES tend to have higher exposure to smoking as the probability of having a parent that smokes or has a more permissive attitude toward smoking is also higher. In 2007-2009 almost half of Finnish children with vocational (or less) educated mothers live in households in which at least one parent/guardian is a daily smoker, which is more than double of that among children with higher educated mothers (Kaikkonen et al., 2012). Smoking uptake among children is strongly influenced by parental smoking (Gilman et al., 2009) with smoking patterns transmitted even across three generations (El-Amin et al., 2015). When home smoking bans in Finnish homes were investigated, lower family SES was strongly associated with the lack of a total ban, which in turn associated with higher risk among adolescents for being a daily smoker (Rainio et al., 2008). Another varying exposure between adolescents with different family SES might be tobacco use of friends (David et al., 2010), which has been shown to be a strong predictor of adolescent smoking uptake (Lastunen et al., 2017; Molyneux et al., 2004). Furthermore, exposure to advertising might also differ between adolescents of low and high SES families since tobacco company documents indicate a key target of their market and advertising in developed countries are young people with low SES (Sorensen et al., 2004). However, direct advertising of tobacco products is unlikely to impact the socioeconomic differences in smoking among

Finnish adolescents because marketing has been prohibited since 1978 (Leppo et al., 2003).

It has been proposed that not only socioeconomic differences in exposure but also in vulnerability to tobacco use might be involved in creating the socioeconomic difference in smoking (David et al., 2010). The probability of adolescents from low SES families to intake smoking might be increased due to psychosocial strain, related to family conflicts (Brook et al., 2007). Vulnerability of adolescents might also be affected by their perceptions of smokers and non-smokers which might differ across SES groups and are suggested to play crucial roles in whether to start smoking in adolescence (Spijkerman et al., 2007). The different perceptions might be reflected in the smoking prevalence between Finnish students from vocational and high schools, with 61% of students in vocational schools considering smoking acceptable compared with only 39% in high schools (Halme et al., 2018). Social competence and self-esteem have been suggested to be weaker among adolescents from low SES families, meaning their ability to resist peer pressure might be difficult (Carvajal et al., 2000; Twenge et al., 2002).

Disparities in vulnerability and exposure might have significant impacts on socioeconomic differences for cessation of smoking (David et al., 2010). For example, those who continue to smoke despite increasing social, legislative and economic pressure are either resistant to this pressure or just unable to quit smoking (Clare et al., 2014; Hughes, 2011). These people are often referred to as “hardcore” smokers. A study based on a large Australian national survey showed that hardcore smoking was more prevalent among low SES smokers, and that the amount of hardcore smokers was decreasing with time only among those with high SES (Clare et al., 2014). In line, those with low SES are at higher risk for having no intention to quit and higher nicotine dependence compared with those with high SES (Siahpush et al., 2006). Additionally, it has been suggested that people with higher SES have more supportive social networks that can make it easier for them to cease smoking (Kunst et al., 2004). Furthermore, lower SES is associated with higher risk of several conditions, which might affect the burden of chronic stress and these stressors are associated with the persistence of smoking (Baum et al., 1999; Pisinger et al., 2011; Slopen et al., 2013). However, evidence on the importance of stress is not entirely consistent. For instance, a study on Danes and Swedes showed that psychosocial work environment had no effects on socioeconomic differences in smoking (Andersen et al., 2008).

The risk from tobacco smoke is not just from the effects of smoking by the individual but also from exposure to second-hand smoke (Steinberger et al., 2016). Children with at least one smoking parent have nearly twice the risk of atherosclerotic plaque in adulthood compared with children of non-smoking parents and for children with detectable levels of cotinine (indicator of exposure to second-

hand smoke) the risk of plaque is approximately 4 times higher (West et al., 2015). The socioeconomic differences in exposure to second-hand smoke are similar to active smoking. In the US., those living in poverty were more often exposed to secondhand smoke (43%) than those not living in poverty (21%) (Homa et al., 2015). The results were essentially similar when SES was assessed by education or by home ownership. In Finland, the restrictions imposed by smoking laws and the increasing consciousness of the hazards of second-hand smoke have been effective, with data from a national survey conducted in 2007-2009 showing that 99.9% of children below school-age had not been exposed to second-hand smoke (Kaikkonen et al., 2012), with no socioeconomic differences detected.

There is some evidence to suggest the inverse association between childhood SES and prevalence of smoking and nicotine dependence in adolescence and adulthood (Jefferis et al., 2004; Pedersen et al., 2017; Soteriades et al., 2003; Tjora et al., 2011). A birth cohort from the U.S. showed that in addition to lower SES in childhood being associated with higher risk for first cigarette use, both low child and adult SES were independently associated with progression to permanent smoking and with lower rates of quitting (Gilman et al., 2003). Therefore, it is postulated that the adverse effects of low SES on persistent smoking accumulate over the entire lifetime and that long-lasting socioeconomic disadvantage from childhood to adulthood might predispose to increased risk of persistent smoking in adulthood. However, a study using seven population-based surveys from six Western countries reported inconsistent results concerning the likelihood of quitting (Power et al., 2005). Manual occupation of father in childhood was associated with reduced likelihood of quitting in women in some countries, but in men (including in Finland), no association between parental occupation and the likelihood of quitting smoking later in life was found. Importantly, SES was determined by simple dichotomous manual/non-manual classification and all but one of the studies used retrospectively obtained information on childhood SES, which could have led to underestimation of the observed effects.

Results of a population-based prospective cohort from Norway suggested that the association between parental SES and smoking in young adulthood is mediated by several factors (Pedersen et al., 2017). These factors included parental smoking, poor school performance and welfare assistance. The impact of parental smoking is well-known (Jefferis et al., 2004; Tjora et al., 2011), with the effect of father's smoking tending to be strongest in male offspring and the effect of mother's smoking tending to be strongest in female offspring (Jefferis et al., 2004). However, this study suggested that the factors related to the individual's pathways into adulthood might be stronger mediators than parental smoking and in men, the association between child SES and adulthood smoking was no longer apparent when adult SES was taken into account. Similarly, results of a longitudinal Norwegian study indicated that

parental SES is associated with adolescent smoking both directly and indirectly through smoking status of parents, siblings and peers (Tjora et al., 2011).

A study that investigated the impact of price- and non-price-related population-wide tobacco control policies on socioeconomic differences in smoking in Europe between the 1990s and 2000s showed that tobacco control policies have helped reduce the prevalence of smoking, particularly in lower SES groups (Hu et al., 2017). Still, the smoking inequalities in European populations have continued to increase. Therefore, as growing inequalities are not dissolved by conventional population-wide policies, the need for more targeted interventions and policies exists. Because broad evidence suggests socioeconomic differences in the initiation, prevalence and quitting of smoking, social determinants should be considered in every actions of policies related to smoking (David et al., 2010) and might be a future challenge also for Finnish tobacco control policies.

2.2.3 Physical activity

Consistent evidence supports an inverse association between physical activity and the prevalence of CVD (Sattelmair et al., 2011). According to the Physical Activity Guidelines for Americans, to have substantial health benefits adults should do at least 150 minutes per week of moderate-intensity aerobic activity, 75 minutes per week of vigorous aerobic activity or a combination of both (U.S. Department of Health and Human Services, 2018). For children and adolescents, the guidelines recommend at least 60 minutes of moderate-to-vigorous activity daily. Estimates from a meta-analysis found that adults meeting the guidelines have a 14% lower risk of CHD compared to individuals having no leisure-time physical activity (Sattelmair et al., 2011), and that beneficial effects on cardiovascular risk are seen with lesser amounts of activity versus no activity and for higher amounts of physical activity versus meeting the guidelines. The Finnish guidelines of health promoting physical activity for children and adults are based on these American guidelines (Laakso et al., 2008; Working group appointed by the Finnish Medical Society Duodecim et al., 2016).

The mechanisms behind the association of higher levels of physical activity and decreased risk for CVD include positive effects of physical activity on inflammatory factors, blood pressure, lipids, body mass index (BMI) and glucose metabolism (Mora et al., 2007). Physical activity is shown to protect from subclinical changes of CVD in youth (Pahkala et al., 2011), and to be a major determinant of cardiorespiratory fitness, which has been shown to counteract adverse effects of other conventional cardiovascular risk factors (Blair et al., 1996).

Socioeconomic differences in physical activity have been studied across the life-course, but the strongest evidence is available for adults (O'Donoghue et al., 2018).

A large review showed that there is broad evidence that those with the highest levels of SES have the highest levels of leisure-time or moderate-to-vigorous physical activity in adulthood (Gidlow et al., 2006). However, evidence across socioeconomic groups remains inconsistent. Studies have also shown that the direction of socioeconomic inequalities in physical activity differs by domain of physical activity. A systematic review on European studies showed that when occupational physical activity was separately studied the association between SES and physical activity was not direct but inverse (Beenackers et al., 2012). Similarly, in a Finnish sample, total physical activity was higher among those with low SES, whereas both higher education and higher income were gradually associated with higher levels of leisure time activity (Lehto et al., 2013). According to the FinHealth 2017 Survey, 57% of men and 63% of women from the lowest educated report exercising during their leisure-time whereas among the highest educated the proportions were 80% among men and 74% among women (Koponen et al., 2018). Total 24-hour physical activity was measured using accelerometers from over 900 participants in the FinHealth Survey with levels seemingly higher among the lowest educated, even though no statistically significant differences were seen (Wennman et al., 2019). Importantly, people with lower education and higher obesity tend to under-report and those with higher education tend to over-report their physical activity in questionnaires when compared with accelerometers, which should be considered when interpreting results (Koolhaas et al., 2018; Slootmaker et al., 2009).

As with adults, a systematic review of studies on adolescents only cautiously suggested a positive association between SES and physical activity, since 42% of the studies reported no or an inverse association (Stalsberg et al., 2010). Similar inconsistency in the results of studies on child physical activity is apparent. Family income has been shown to associate positively and dose-dependently with attendance of British children to out-of-school structural physical activities (Voss et al., 2008). However, studies on Finnish and Australian children showed no association between maternal education and self-reported physical activity of children (Ball et al., 2009; Kaikkonen et al., 2012). In contrast, results of a recent study on German children with data from accelerometers indicated that both parental education and income were positively associated with daily moderate-to-vigorous physical activity (Mutz et al., 2017). However, other studies using accelerometer data have not indicated differences in physical activity of children across parental income or education (Ball et al., 2009; Voss et al., 2008).

Contrary to physical activity, sedentary behavior is shown to associate with increased risk of cardiovascular mortality (Ekelund et al., 2016). It has been suggested that high levels of physical activity might eliminate the increased risk of mortality associated with high levels of sitting, but only diminish the increased risk associated with high TV-viewing time. Sedentary behavior, and especially television

viewing time, has been shown to be higher among children and adolescents of low SES families (Drenowatz et al., 2010; Kaikkonen et al., 2012; Kantomaa et al., 2007). Importantly, of the different types of sedentary behaviors, television viewing has been shown to associate most strongly with adiposity markers (Heinonen et al., 2013). However, as with physical activity, the results concerning sedentary time are inconsistent. A Swedish study showed that family SES was positively associated with sedentary time and that the total physical activity of children with lower family SES was higher when compared to those with higher family SES (Henriksson et al., 2016). Still, the Swedish children with low family SES were more likely to be obese or overweight when compared to children with higher family SES.

Studies investigating the association between childhood SES and adulthood physical activity have revealed results that are congruent to those concerning the effects of current SES. Childhood SES has been shown to be positively associated with adult leisure time physical activity in several studies, though some studies have found an association only among men or women (Aarnio et al., 2002; Huurre et al., 2003; Juneau et al., 2014; Silverwood et al., 2012). In a Finnish study, the association of childhood SES with adult leisure time physical activity was distinguished in both sexes, but was independent of current SES only among men and only up to 22 years of age (Huurre et al., 2003). Furthermore, some studies from Scandinavian countries have failed to show any association between childhood SES and later leisure time physical activity (Osler et al., 2001; Oygard et al., 1998). Still, a systematic review suggested that there is consistent evidence of a positive association between childhood SES and adulthood leisure time physical activity, independent of adult SES (Juneau et al., 2015).

In contrast to leisure time physical activity, transport and work physical activity are presumed to be higher among adults with low childhood SES, refleced in higher walking levels, which is consistent with adult SES (Silverwood et al., 2012). A British study showed more inconsistent results concerning socioeconomic differences in adult occupation and transport physical activity by life course SES (Juneau et al., 2014). Using accumulation of risk model with additive effects lower SES at birth and at age 10 years was associated with higher adult transport physical activity in women but not in men. Using the same model for work physical activity, SES at birth was significant predictor of adult work physical activity only for the logistic portion of the model and only in men. Therefore, even though current evidence cautiously supports the association between higher early life SES and higher leisure time physical activity in adulthood, the influence of childhood SES on the overall physical activity remains inconclusive.

Mechanisms behind the pathway from childhood SES to physical activity in adulthood are not widely investigated but socioeconomic differences in physical activity in adults are suggested to relate to better social support, higher self-efficacy

and better availability of facilities enabling or promoting physical activity among higher SES individuals (Cerin et al., 2008; Powell et al., 2006). A Finnish study on vocational and high school students showed that the strongest mediators in the direct association between SES and physical activity were self-monitoring, physical activity identity and intention (Hankonen et al., 2017). Self-monitoring is suggested to be a key behavior change technique in physical activity interventions (Michie et al., 2009). Therefore, by better understanding the social determinants of physical activity and different phenomena related to them, more precise and effective interventions to promote physically active lifestyle might be possible in the future.

2.2.4 Alcohol consumption

The association between the use of alcohol and cardiometabolic disease is complex and strongly affected by the pattern and amount of alcohol consumption. Some evidence suggests the association between alcohol consumption and CVD to be U-shaped (Carlsson et al., 2003; Corrao et al., 2000; Mukamal et al., 2005). However, the evidence supporting the cardioprotective role of moderate alcohol consumption is not conclusive since it has been shown mainly in observational studies and the effects of moderate alcohol consumption are suggested to operate through social factors and other favorable health behaviors (Naimi et al., 2005). Furthermore, a Mendelian randomization analysis has shown that lower alcohol consumption associates with reduced risk of coronary heart disease at all levels of alcohol consumption (Holmes et al., 2014). According to the Finnish Current Care Guideline, the risk of alcohol use disorders in women is highly increased if the daily consumption is two or more drinks regularly or weekly consumption is 12–16 or more drinks (Working group appointed by the Finnish Medical Society Duodecim & the Finnish Society of Addiction Medicine, 2018). In men, the same risk levels are three or more drinks per day or 23–24 or more drinks per week.

Studies have concurred that drinking patterns and the effects of alcohol consumption tend to differ between socioeconomic groups, but as with the effects of alcohol consumption on cardiometabolic health, the evidence on the association between SES and alcohol consumption remains controversial. A study on European populations suggested that among women, those higher educated were more likely to drink above the high-risk level whereas among men, the association was inverse in most of the countries (Bloomfield et al., 2006). Similarly, results from the Whitehall studies indicate that the association of SES with heavy drinking (defined as more than 15 drinks per week) was positive among women but with moderate drinking (11–21 drinks per week in men and 8–14 drinks per week in women) the association was positive in both sexes (Marmot, 1997). A study from the U.S. showed that the highest prevalence of binge drinking (defined as four or more drinks

for women and five or more drinks for men on an occasion during the past 30 days) existed among those with the highest incomes but among those with lower incomes, the mean frequency and intensity of binge drinking was higher (Centers for Disease Control and Prevention (CDC), 2012). In Finland, according to the FinHealth 2017 Study, women with low education and men with mid-level education had higher prevalence of binge drinking (6 or more drinks on an occasion monthly) than individuals from other educational groups. Possibly relating to the harms of binge drinking, alcohol-related mortality and other negative consequences tend to be more frequent among people with low SES (Collins, 2016).

Available evidence suggests that lower child SES is associated with higher probability to engage in risky alcohol use later in life including consuming a high amount of drinks on a single occasion, alcohol abundance and alcohol-attributable death (Droomers et al., 2003; Pensola et al., 2003; Poulton et al., 2002). In line, adverse childhood experiences are shown to associate with higher risk of hazardous drinking in midlife, independent of adult SES (Leung et al., 2016). The association between low child SES and higher tendency for risky use of alcohol later in life might be explained by higher prevalence of familial alcohol problems, higher approval of alcohol consumption by friends and lower parental attachment among individuals with low child SES (Droomers et al., 2003). However, a study with 25-year follow-up found that low childhood SES was associated with alcohol and other substance use problems only indirectly, operating through fewer years of participant's own education (Fothergill et al., 2006), and some studies from Finland and Sweden have failed to show any association between childhood SES and risky or total use of alcohol later in life (Kumpulainen, 2000; Urban et al., 1992). Moreover, the Young Finns Study has previously shown a direct association between parental income and risk of frequent inebriation by alcoholic beverages (Leino et al., 2000). In line, a systematic review of 19 longitudinal studies investigating the association of childhood SES with alcohol consumption later in life concluded that evidence on the adverse effects of low childhood SES on later life alcohol consumption is sparse (Wiles et al., 2007).

In a Norwegian study the association between frequent consumption of alcohol and lower risk of CVD was more profound among those with high SES compared to those with middle or low SES whereas very frequent consumption of alcohol was associated with increased risk of CVD only among those with low SES (Degerud et al., 2018). The authors suggested that these differences might reflect differential confounding factors related to alcohol intake or differing effects of alcohol on cardiovascular health across SES groups. Therefore, possible socioeconomic differences in the relationship between alcohol consumption and cardiometabolic health should be taken into account, when alcohol consumption is studied from the perspective of cardiometabolic health.

2.3 Association of socioeconomic status with cardiometabolic risk factors and risk factor clustering

Biological risk factors account for a great part of the socioeconomic differences in the risk of CVD, even though they operate together with behavioral, psychologic and social risks. In a Finnish study, adjustment for biological risk factors reduced the relative risk of lowest income quintile for cardiovascular mortality in relation to highest income quintile from 2.66 to 1.24 (Lynch et al., 1996). Data from the Whitehall cohort showed that best-practice interventions (reduction of systolic blood-pressure by 10 mmHg, total cholesterol by 2 mmol/l, blood glucose by 1 mmol/l and cessation of cigarette smoking) would reduce the difference in CHD mortality between socioeconomic groups by 69% (Kivimäki et al., 2008). These different clinical risk factors are easy to assess and thus play a critical role in interventions to diminish the socioeconomic gap in CVD mortality.

The biological risk factors of CVD tend to cluster already in childhood and the risk of CVD increases considerably with each additional risk factor (Raitakari et al., 1994; Wilson et al., 1999; Yusuf et al., 1998). Higher education tends to protect against risk factor clustering while people with low SES tend to have more risk factors than those with higher SES (Kivimäki et al., 2007; Raitakari et al., 1995). However, *within-group clustering* is shown to be similar in all SES groups meaning that no socioeconomic differences in clustering exist on an individual level (Kivimäki et al., 2007; Ljung et al., 2007).

Several risk scores are based on clusters of risk factors, which are shown to predict future risk of CVD. In Finland, the FINRISK risk calculator is commonly used in health counselling or to assess the need for hypercholesterolemia or other treatments for CVD (Vartiainen et al., 2016). It was developed to predict 10-year risk for acute coronary event, stroke and their combination in the Finnish population. A widely adopted score in clinical practice and in research is the Framingham Risk Score, which evaluates the 10-year risk of general CVD and specific cardiovascular events (D'Agostino et al., 2008). The risk is calculated based on age, total cholesterol, smoking status, high-density lipoprotein (HDL) cholesterol and systolic blood pressure. When the Framingham Risk Score was applied to those of high and low SES, the calculated cardiovascular risks were 3.7% and 3.9%, respectively (Fiscella et al., 2009). However, the observed risks were 3.2% and 5.6%, suggesting a distinct difference in the risk of low SES people and a deficiency in the score. With socioeconomic position added to Framingham Risk Score, the predicted risks were more similar to the observed, 3.1% for high SES people and 5.2% for low SES people. Thus, SES is not only associated with risk factor clustering but also contributes an independent part of the risk factor cluster associated with increased cardiovascular risk.

Risk factor clusters are not just creating different risk scores but can also be the basis of diagnostic criteria of certain pathological conditions. One widely used condition is MetS, which might have prognostic value in predicting the risk of cardiovascular mortality above established risk factors (Sundström et al., 2006). The following sections discuss MetS and its association with SES. Furthermore, two important clinical risk factors, hypertension and obesity are both closely related to MetS. Both are important constituents of MetS and the pathophysiological bases of all three conditions are tightly linked (Yanai et al., 2008). However, both obesity and hypertension have individual roles in mediating the association between childhood adversity and cardiometabolic health (Suglia et al., 2018). Therefore, they are also separately discussed in the following sections.

2.3.1 High blood pressure

High blood pressure is one of the most important risk factors for CVD. In 2015, 55% of mortality related to ischemic heart disease was associated with elevated systolic blood pressure (Forouzanfar et al., 2017). Systolic blood pressure depends partially on arterial compliance and reflects underlying arterial disease better than diastolic blood pressure (Randall, 1982). For example, a positive correlation with total CVD has been shown to be stronger for systolic than for diastolic blood pressure (Rapsomaniki et al., 2014). Even though the continuous direct association between blood pressure and the risk of vascular mortality extends down to levels as low as 115/75 mmHg, blood pressure levels are categorized (conventionally to normal blood pressure, elevated blood pressure and hypertension) to simplify clinical and public health decision making (Lewington et al., 2002). The definition of hypertension differs between European and American guidelines. The ACC renewed the definition for hypertension in 2017 as an individual with blood pressure over 130/80 mmHg (Whelton et al., 2018). According to the European guidelines, hypertension is still defined as having blood pressure level above 140/90 mmHg at which level drug treatment is recommended for all patients (Williams et al., 2018). Finnish guidelines follow the European Guidelines and suggest drug treatment if blood pressure at the clinic is above 140/90 mmHg or the mean value of repeated home assessments is above 135/85 mmHg (Working group appointed by the Finnish Medical Society Duodecim et al., 2014).

95% of all cases of hypertension are called “essential” meaning that there are no secondary causes explaining the increased blood pressure levels (Carretero et al., 2000). Several hypertensinogenic factors, including obesity, insulin resistance, high salt and alcohol intake, physical inactivity, stress and dyslipidemia, are known to associate with essential hypertension in genetically susceptible people. Because these and other cardiometabolic risk factors tend to co-exist with hypertension, they

might modify the association between blood pressure and the risk of CVD. Therefore, risk evaluation of CVD mortality among hypertensive patients should take into account the other risk factors associated with hypertension (Thomas et al., 2001).

A meta-analysis of 51 studies indicated that low SES determined by all three conventional indicators (education, occupation and income) was associated with increased risk for hypertension (Leng et al., 2015). Of these three indicators, educational level had the strongest impact on risk of hypertension. However, a U.S. longitudinal study suggested the effects of educational attainment on systolic blood pressure operate through BMI, waist circumference and heart rate, whereas income associates inversely with systolic blood pressure independent of these potential mediators (Brummett et al., 2011).

A Swedish study has shown that from year 1994 to 2014 the socioeconomic gap in systolic blood pressure levels decreased, but the mean blood pressure level among people with primary education was still higher than among people with a university education (Eriksson et al., 2017). Similarly, the National FINRISK Study showed that when educational groups were compared, systolic blood pressure was lowest among the highest educated Finns in all study years from 1982 to 2012 (Vartiainen et al., 2016). In a nationwide sample of the Finnish adult population, the Health 2011 Study found that although hypertension is more prevalent among lower educated individuals there is no socioeconomic differences by education in awareness, treatment or control of hypertension (Sivén et al., 2015). The FinHealth 2017 Study found that socioeconomic differences in blood pressure levels have remained as the prevalence of increased blood pressure was lowest among the highest educated (Koponen et al., 2018).

Furthermore, studies have suggested that lower child SES predicts higher blood pressure in adulthood (Blane et al., 1996; Kivimäki et al., 2006b; Poulton et al., 2002; Power et al., 2007). A prospective British study showed that both child and adult SES contribute to blood pressure levels in mid-life, independently of each other (Power et al., 2007). Furthermore, results of the Young Finns Study suggested that child SES is a stronger predictor of adult blood pressure than current adult SES (Kivimäki et al., 2006b). Socioeconomic differences in blood pressure have been shown to begin in childhood where they tend to persist into adulthood (Kivimäki et al., 2006a). The Young Finns Study has shown that, in addition to childhood blood pressure, adult BMI is a strong mediator in the association between child SES and adult blood pressure. Likewise, a British birth cohort study showed that parental occupational status was associated with adult blood pressure in adulthood from age 36 to 53 years but that the association was attenuated after adjustment for adult BMI (Hardy et al., 2003). These studies suggest that an effective way to diminish the impact of child SES on adult differences in blood pressure levels includes successful

weight control across the life-course. Therefore, socioeconomic differences in blood pressure levels are tightly linked with the general socioeconomic gap in cardiometabolic health and might be avoidable with shared efforts.

2.3.2 Obesity

Obesity is a result of an imbalance between energy intake from food and energy expenditure due to physical activity and metabolic processes (Hill et al., 2012). The surplus energy leads to hypertrophy of fat cells and to accumulation of subcutaneous and visceral fat (Bays et al., 2013). Visceral fat is suggested to mediate the association between obesity and the increased risk of metabolic disturbances both in lipid and glucose metabolism. These metabolic disturbances play a major role in the pathophysiology of MetS, T2DM, hypertension and CHD (Kopelman, 2000). Despite the awareness of risks related to obesity, the trend in global age-standardized prevalence of obesity has long been increasing (Abarca-Gómez et al., 2017). Data on Finnish working-aged individuals showed that the prevalence of obesity has been increasing over recent years (Koponen et al., 2018). Among children and adolescents in Finland and in other high-income countries, the trend of mean BMI has plateaued since 2000, but still remains high (Abarca-Gómez et al., 2017; Lundqvist et al., 2019).

The most common way to evaluate the amount of body fat in clinical practice is to use BMI, which is calculated as weight in kilograms divided by the square of height in meters. People are considered to be overweight when BMI is between 25 and 30 kg/m^2 and obese when BMI is over 30 kg/m^2 (Working group appointed by the Finnish Medical Society Duodecim et al., 2011). Waist circumference is also suggested to be measured in clinical practice, especially if BMI is under 30 kg/m^2. The cut-points used for central obesity are 88 cm for women and 102 cm for men (National Cholesterol Education Program (NCEP) Expert Panel on Detection Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III), 2002).

Child obesity is a strong predictor of cardiometabolic disease in adulthood (Baker et al., 2007; Juonala et al., 2011b). Results of a cross-sectional study suggested that obese children have increased carotid intima-media thickness (IMT) and carotid stiffness compared with normal-weight children (Iannuzzi et al., 2004). It has also been shown that BMI in early childhood as young as 3 years of age predicts carotid IMT in adulthood (Juonala et al., 2010a). Data from a cohort of over 270 000 Danish participants suggested that child BMI associates directly with the risk of CHD events in adulthood, across the entire BMI distribution (Baker et al., 2007). Although childhood BMI is strongly correlated with adulthood BMI, a report of four longitudinal cohort studies suggested that those who were overweight in

childhood but who were non-obese in adulthood had similar risk of cardiometabolic outcomes to those who were normal-weight in childhood and non-obese in adulthood (Juhola et al., 2011; Juonala et al., 2011b).

A review published 3 decades ago was among the first to summarize the results of investigations on the association between SES and obesity (Sobal et al., 1989). The results suggested that in women the association between SES and adiposity was inverse, whereas among men a positive association was seen in 35% of the studies. Similar results were obtained in a more recent review comprising studies published between years 1988 and 2004 (McLaren, 2007). SES was inversely associated with body size in women, but among men the results were inconsistent. However, studies on Finnish adult populations have shown that the prevalence of obesity is highest among the least educated in both sexes (Lahti-Koski et al., 2010, 2000). Furthermore, according to these repeated cross-sectional population surveys from 1980s to the beginning of 2000s, socioeconomic gradient in obesity among Finnish adults has not widened but diminished, contrary to the results of WHO MONICA Project in 26 countries that indicated growing differences in the Finnish population (Lahti-Koski et al., 2010, 2000; Molarius et al., 2000). However, more recent results from the National FINRISK Study in 2012 showed that overweight was more prevalent among the least educated compared with other education levels but that trends in BMI from year 2007 to 2012 were mostly stable in all educational groups (Männistö et al., 2012). Yet, among the least educated and youngest women there was some evidence for decreasing BMI across study years.

In parallel with observations in cross-sectional studies, of which also the above-mentioned reviews were comprised of, similar associations have been observed in prospective studies (Ball et al., 2005). However, both cross-sectional and prospective studies have shown that there is inconsistency in the results likely due to the indicator of SES used. In developed countries, higher weight gain over time is consistently associated with lower occupational status but the results have been less consistent for income.

As with adulthood obesity, current evidence suggests that low child SES is associated with higher risk of child overweight and obesity. Results of a systematic review including 45 studies on children in western developed countries revealed that child SES was inversely associated with child adiposity in the major part of the studies (Shrewsbury et al., 2008). In addition, a study from the U.S. suggested that although the overall trend in childhood obesity has plateaued, the socioeconomic disparities in obesity among children have increased (Datar et al., 2015). Parallel to other developed countries, several studies in Finnish children and adolescents have shown that lower parental SES is associated with higher prevalence of obesity (Kaikkonen et al., 2012; Kautiainen et al., 2009). However, in contrast to the U.S., the findings showed no socioeconomic differences in the prevalence of obesity over

time (Kautiainen et al., 2009). Conversely, an international study of 35 countries suggested that in Finland, an inverse association between SES and the prevalence of overweight was seen only among girls whereas in boys an association between SES and the prevalence of overweight was direct (Due et al., 2009).

A systematic review consisting of longitudinal observational studies found that early life SES did not associate with child overweight and obesity, but had a strong association with these conditions in adulthood (Parsons et al., 1999). A British birth cohort study has shown childhood SES at the age of 2 years to be inversely associated with adult obesity at the age of 53 years in men and women, independent of SES in young adulthood and in midlife (Langenberg et al., 2003) In a younger British birth cohort, people were followed-up for 33 years from birth and the association of SES at five different time points with adult obesity was compared (Power et al., 2003). Among women, social class at the age 7 years was predictive of adult obesity while among men social class was predictive at birth and at the age of 23 years. The Young Finns Study has also shown that low child SES defined by annual family income is an independent risk factor of adulthood obesity (Juonala et al., 2011a). Results of the Helsinki Birth Cohort Study differed, showing the association between child SES and adult obesity existed only in men (Salonen et al., 2009). When the effect of intergenerational mobility on adulthood obesity was studied in a British cohort, upwardly mobile persons tended to be between the class they left and the class they joined in all obesity indices (Langenberg et al., 2003). Therefore, the current evidence suggests that the effects of SES on obesity accumulate over the lifetime and that childhood SES might have both independent and mutual influences with later life SES on the risk of obesity later in life.

Increasing socioeconomic disparities in obesity have been suggested to relate to growing differences in energy intake and in physical activity (Frederick et al., 2014). Data from the U.S. suggest that children of high-educated parents consume lower energy and are more physically active than a decade ago, whereas children with low parental SES have not increased physical activity or decreased energy intake. Additionally, children from low SES families might have more opportunities to be sedentary, which is associated with childhood obesity (Andersen et al., 1998; Kaikkonen et al., 2012; Tandon et al., 2012). Furthermore, childhood SES might affect adulthood obesity by accumulation of childhood psychosocial factors, which are shown to associate with the development of BMI over time (Elovainio et al., 2017). Moreover, one major childhood risk factor for obesity is parental obesity, which multiplies the risk of a child to be obese later in life (Eriksson et al., 2003; Jääskeläinen et al., 2011). However, a British birth cohort study showed that the effect of child SES on adulthood obesity was not entirely mediated by parental obesity, since the association remained after adjustment for parental BMI (Power et al., 2003).

CHD and the most important risk factors of CVD, like obesity, hypertension and glucose abnormalities, have strong genetic components (Russo et al., 2010b; Steptoe et al., 2002). Therefore, it could be that some of the socioeconomic gradient is determined by the higher genetic burden among low SES people. Since the changes in socioeconomic differences in CVD have been distinct and occurred with high speed, the explanatory role of genetic factors can be challenged (Steptoe et al., 2002), but a longitudinal study from the U.S. has shown that stable low SES and downwardly mobile SES enhance the influence of genetics on BMI whereas high SES and upwardly mobile SES compensate it (Liu et al., 2015). In addition to conventional genetics, epigenetics (variations in gene functions unrelated to DNA sequence) are suggested to be involved in mechanisms in the development of obesity and CVD (Lopomo et al., 2016; Zhang et al., 2018). It is suggested that nutrients and components of diet might cause epigenetic alterations leading to modifications in gene expression and that genes are most vulnerable to these changes in the early years (Milagro et al., 2013). Thus, the childhood environment, on which childhood SES has strong effects, might modulate the risk of adulthood obesity, and other cardiometabolic outcomes, also through the mechanisms of epigenetics.

2.3.3 Metabolic syndrome

Obesity is suggested to be an important determinant of risk factor clustering (Wilson et al., 1999). In addition, weight gain tends to increase clustering and weight reduction to decrease clustering. MetS is a cluster of cardiometabolic risk factors strongly related to obesity (Reaven, 1988). The pathophysiological basis of MetS includes insulin resistance and compensatory hyperinsulinemia, that predispose to clustering of hypertension, dyslipidemia and hyperglycemia. The first generally known definition with specific thresholds for components of MetS (insulin resistance, obesity, dyslipidemia, hypertension and microalbuminuria) was published by the World Health Organization (WHO) (Alberti et al., 1998). Since that time, there have been several attempts to define MetS, but the most recent generally accepted definition is the Joint Interim Societies definition of “harmonized MetS” (Alberti et al., 2009). According to the harmonized criteria, to have MetS a person requires any three of the five components of MetS (central obesity, hypertension, low HDL cholesterol, elevated triglycerides and impaired fasting glycemia or diagnosed T2DM).

Detection of MetS is important as it predisposes individuals to at least a five-fold increased risk of developing T2DM and a two-fold increased risk of CVD (Dekker et al., 2005; Laaksonen et al., 2002). A prospective cohort study on Swedish men found that diagnosis of MetS increases the prognostic value of established risk factors of cardiovascular mortality, suggesting the importance of clustering of risk

factors (Sundström et al., 2006). Large studies on the prevalence of MetS in European populations are lacking, but it is estimated to be at least 25% among adults and in parallel to the global increase in obesity and sedentary lifestyle, the prevalence of MetS is suggested to increase (Grundy, 2008). Among participants of the National FINRISK Study aged 24–75 years, the total prevalence of MetS in Finland was 36% (Sundvall et al., 2011). According to the Health 2011 survey, the prevalence of MetS was 58% among men and 49% among women in Finns aged more than 30 years (Koskinen et al., 2012). However, direct comparison of the results from different studies and cohorts is difficult due to the use of different criteria for MetS across studies.

The Young Finns Study has previously shown that the prevalence of components of MetS is increased in lower socioeconomic groups (Kivimäki et al., 2006c). Similarly, the Whitehall II study showed a strong inverse association of three different indicators of wealth with the prevalence of MetS (Perel et al., 2006). Additionally, a Finnish study showed an inverse association between adulthood education and the prevalence of MetS in both sexes (Silventoinen et al., 2005). However, studies from the U.S. and Europe suggest that association of SES with MetS differs by sex and that SES is inversely associated with the prevalence of MetS in women but not in men, similar to the association between SES and obesity (Chichlowska et al., 2008; Santos et al., 2008; Sobal et al., 1989; Vernay et al., 2013). The differences by sex might relate to the stigmatization of obesity and to the social pressure to be thin, which are both suggested to be stronger among women than men and predominantly among those with high SES (Sobal et al., 1989). Moreover, a Mendelian randomization study suggested that higher BMI is causally associated with having lower income, but only in women (Tyrrell et al., 2016). Another mechanisms for the sex difference might operate through psychosocial factors related to low SES; as, when compared to men with low SES, psychosocial factors have been shown to affect women with low SES more adversely (Thurston et al., 2005).

Only few studies have examined if child SES associates with the risk of MetS in adulthood (Langenberg et al., 2006; Lucove et al., 2007; Montez et al., 2016; Ramsay et al., 2008). The results have been inconsistent and only a British study assessed childhood SES prospectively (Langenberg et al., 2006). A study on an English cohort showed an inverse association between child and adult SES and MetS in both sexes but mutual adjustment attenuated the effect of child SES while the effect of adult SES remained (Ramsay et al., 2008). Furthermore, a study on African Americans suggested that child SES was not associated with the prevalence of MetS, whereas an inverse association between adult SES and MetS was evident (Lucove et al., 2007).

Parallel to data from adults, childhood SES has shown sex-specific differences in the association between SES and MetS. The Atherosclerosis Risk in Communities (ARIC) study investigated the association of life-course SES, including SES in childhood and adulthood, with MetS and showed that among women both low SES at one time-point from childhood to adulthood and cumulative low SES were associated with increased risk of MetS (Chichlowska et al., 2009). In contrast, among men, no association was seen between SES and MetS. Furthermore, it is suggested that pathways and mechanisms behind the socioeconomic differences in MetS might differ by sex (Lee et al., 2018). A national longitudinal study from the U.S. showed an inverse association between SES and MetS for both sexes but among men the association between childhood SES and adulthood MetS was largely explained by unhealthy diet and lack of leisure-time physical activity, whereas among women the association remained significant after adjustment for diet, physical activity and other potential mediators and confounders.

MetS is strongly linked to obesity and lifestyle behaviors related to it, which are all shown to associate with low SES (Galobardes et al., 2001; McLaren, 2007; O'Donoghue et al., 2018). A Finnish study suggested that the risk of MetS was higher among adults whose obesity continued from childhood to adulthood compared with those who became obese in adulthood (Vanhala et al., 1998). This emphasizes the importance of the childhood environment, including childhood SES, in determining future risk of MetS. Along with biological risk factors, the mechanisms underlying the relation of childhood SES with adult MetS might include psychosocial factors of childhood environment, similar to adulthood obesity (Lehman et al., 2005).

In conclusion, individuals with low childhood SES are at higher risk for weight gain and related clustering of cardiometabolic risk factors across the lifetime. These alterations predispose to increased risk of developing MetS in adulthood. This seems to be particularly evident among women; possibly due to the higher vulnerability of women to SES-related adverse environmental risk factors.

2.4 Association of socioeconomic status with measurements of subclinical cardiovascular disease

CVD is a continuum from subclinical disease to clinical symptoms. Still, about 50% of men and 60% of women who die suddenly from CHD have no prior diagnosis or manifestation of the disease (Hayashi et al., 2015). A pronounced part of the pathophysiologic basis of CVD relates to atherosclerosis, which is a chronic process starting in utero (Barker, 1995; Herrington et al., 2016). Therefore the asymptomatic

phase of CVD is long but there are several subclinical changes that can be detected by noninvasive methods long before clinical outcomes present (de Simone et al., 2008; Hamilton et al., 2007).

In the human body, the cardiovascular system can be considered as the transport system of the body. The heart is the pump in the system and blood vessels are the routes on which the blood is delivered throughout the body. During every cardiac cycle, the left ventricle (LV) ejects blood into the large elastic arteries, which store the ejected blood during systole and drain it to the peripheral vessels during diastole (Shirwany et al., 2010). Thus, the perfusion in the peripheral vessels is guaranteed during the whole cardiac cycle. Due to normal aging and as the consequence of atherosclerosis and other cardiometabolic manifestations, the distensibility of arteries decreases, a process known as arterial stiffening (Zieman et al., 2005). Arterial stiffening increases the workload of the heart, which leads to cardiac adaptation and to increases in left ventricular mass (LVM) (Grossman et al., 1975). An increasing LVM results in a decrease in the compliance of a heart and finally in impairment of diastolic function (Wan et al., 2014). Therefore, by measuring stiffness of arteries, LVM and LV diastolic function of the heart, it is possible to encompass the structure and function of both vessels and the heart. As SES has been shown to associate with subclinical atherosclerosis in previous studies (Kestilä et al., 2012; Nash et al., 2011), it can be assumed that this association would extend to arterial stiffness, LVM and LV diastolic function. In the following sections these markers of subclinical CVD and their associations with SES are further discussed.

2.4.1 Arterial stiffness

According to the model of Windkessel, the buffering role of large arteries secures the constant perfusion of the periphery during both periods of the cardiac cycle (Frank, 1990). The elasticity of the arteries depends on the regulated balance in synthesis and degradation of two framework proteins: elastin and collagen (Zieman et al., 2005). Ageing and different pathological processes accelerate the degradation of elastin, which results in arterial stiffening (Johnson et al., 2001; Xu et al., 2000). In addition to passive mechanical structures, functional changes in smooth muscle tone and in integrity of extracellular matrix might modulate arterial stiffness (Townsend et al., 2015). In line with the arterial changes of atherosclerosis, studies have reported that arterial stiffening with age starts in early childhood (Berenson et al., 1998; Hauser et al., 2013; Mikola et al., 2015). Additionally, the conventional cardiometabolic risk factors in childhood associate with arterial elasticity in childhood, adolescence and in mid-adulthood (Juonala et al., 2005; Mikola et al., 2017).

Arterial stiffness can be measured by different methods non-invasively or invasively, and regionally or locally (Pereira et al., 2015). Conventional measures include pressure transducer, applanation tonometry, ultrasound, magnetic resonance imaging and impedance meter. The gold standard to assess arterial stiffening is to measure carotid-femoral pulse wave velocity (PWV) (Kööbi et al., 2003; Laurent et al., 2006). PWV is the speed of the pulse wave travelling the distance between two sites of the arterial system. The speed of the pulse wave is increased with rising arterial stiffness and leads to an increased load on the heart (Nichols et al., 2011). Typically, PWV is measured between the carotid and femoral artery, but another practice is to use measurements between the carotid and popliteal artery, which describes vascular health more globally (Figure 1) (Aatola et al., 2010a).

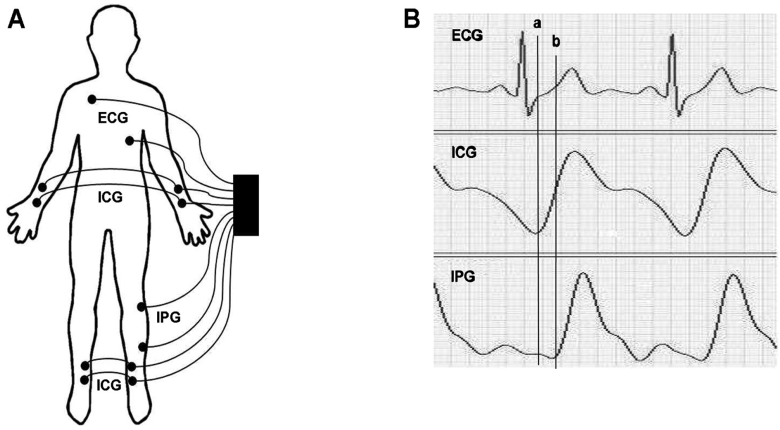

Figure 1. **A**, Placement of electrodes in whole-body impedance cardiography with an additional voltage-sensing channel on the left calf for pulse wave velocity (PWV) measurement. **B**, Synchronous recording of electrocardiography (ECG), whole-body impedance cardiography channel (ICG), and impedance plethysmogram channel (IPG). Time difference between the feet of the ICG and IPG indicates the pulse transit time from aortic arch to popliteal artery. Reproduced from Hypertension (Aatola et al., 2010a) with permission of Wolters Kluwer Health, Inc.

Reference values of PWV are established for healthy people and for people with CVD risk factors (Mattace-Raso et al., 2010). Current guidelines of the European Society of Hypertension have proposed that PWV larger than 10 m/s is an independent marker of significant changes in aortic function in the middle-aged hypertensive patient (Williams et al., 2018). Despite the additive value of PWV on traditional risk factors and conventional risk scores, the European Society of Hypertension does not suggest the measurement of PWV in routine clinical practice.

More local arterial stiffness can be determined by measuring the change in arterial diameter related to the distending pressure (Godia et al., 2007). Of superficial

arteries, atherosclerosis is fairly frequent in the carotid artery and therefore, the general interest is on the elasticity of the carotid artery (Laurent et al., 2006). The change in diameter of the carotid artery during the cardiac cycle by a given change in pressure is called carotid artery distensibility (Cdist) (Figure 2) (Godia et al., 2007). Distensibility of large conduit arteries describes the ability of the artery to expand during cardiac contraction, and therefore, Cdist is increased when arterial stiffness is decreased. Age and sex-specific reference values of Cdist have been established for the healthy population (Engelen et al., 2015).

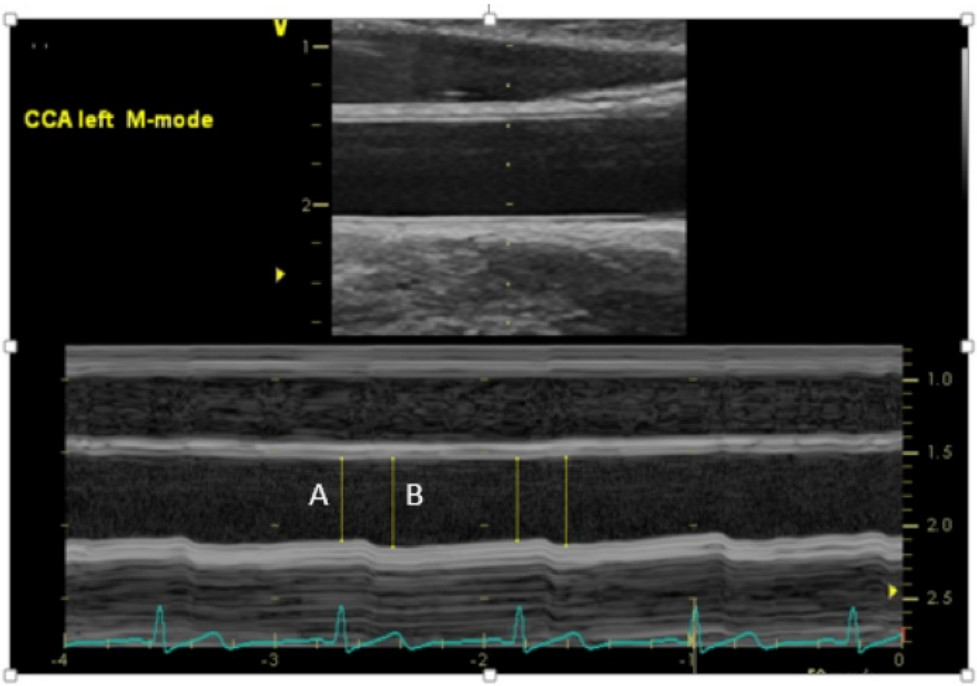

Figure 2. Ultrasound image obtained during measurement of carotid artery distensibility. The change in arterial diameter during diastole (A) and systole (B) related to pulse pressure is used to describe arterial distensibility. Reproduced with permission from the thesis by Mikola, (2018).

Arterial stiffness relates to atherosclerosis at multiple sites of the vascular tree, with several hypotheses for the mechanism proposed (Van Popele et al., 2001). One theory suggests that decreased elasticity is an early pathophysiological change of atherosclerosis (Oliver et al., 2003). This theory is supported by a study on monkeys that found the PWV of monkeys fed a cholesterol progression diet increased whereas the PWV of monkeys fed a cholesterol regression diet decreased (Farrar et al., 1991). Alternatively, arteries with increased arterial stiffness lack shock-absorbing capabilities making them more susceptible to increased pulse pressure, which might

lead to arterial wall damage associated with progression of atherosclerosis (Demer, 1991). One of the stressors, which might induce remodeling in the arterial wall and accelerate arterial stiffening, is increased blood pressure (Intengan et al., 2001). Equally, increased systolic blood pressure and pulse pressure are important clinical manifestations of arterial stiffening (Dart et al., 2001). However, a study on patients with end-stage renal failure showed that decreased PWV enhanced cardiovascular survival independent of decreased blood pressure (Guerin et al., 2001).

In addition to increased systolic blood pressure and pulse pressure, cross-sectional studies have shown an association between many other cardiovascular risk factors (lipid abnormalities, obesity, MetS, diabetes) and lower arterial elasticity in childhood and later in life (Aggoun et al., 2000; Ferreira et al., 2005; Mitchell et al., 2007; Tounian et al., 2001). Vascular stiffening is reversible with healthy behaviors (body mass reduction, exercise) suggested to reduce arterial stiffening (Balkestein et al., 1999; Madden et al., 2009). However, results from the Young Finns Study suggest sustained deleterious effects of childhood risk factors on arterial stiffness in adulthood (Juonala et al., 2005).

Arterial stiffness was suggested to be a new potential risk factor for CVD in 1994 (Arnett et al., 1994). The authors proposed that elevated arterial stiffness could serve as an early marker of atherosclerotic disease or a marker of structural arterial changes caused by hypertension. Since then, evidence for the predictive role of arterial stiffness on CVD has grown (Ben-Shlomo et al., 2014; Vlachopoulos et al., 2010). Results from the ARIC study showed no association between carotid arterial stiffness and incident CVD events, but found an association between arterial elasticity and incident ischemic stroke (Yang et al., 2012). However, a meta-analysis of data from 11 longitudinal studies showed Cdist to be predictive of both cardiovascular events and mortality (Yuan et al., 2016). Furthermore, according to a meta-analysis on more than 17 000 participants, PWV is predictive for stroke, cardiovascular events and CHD, even after adjustment for conventional risk factors (Ben-Shlomo et al., 2014). Therefore, it is suggested that arterial stiffness integrates the harm of cardiovascular risk factors on the arterial wall and is a marker of individuals in which these risk factors are converted into real risk (Laurent et al., 2006). After adjustment, a 1 m/s increase in PWV is associated with a 7% increase in the risk for a cardiovascular event for a 60-year old man with a favorable risk factor profile (Ben-Shlomo et al., 2014). However, it is still unclear whether a decline in arterial stiffness is associated with a reduced risk of cardiovascular events independent of the related decline in conventional cardiovascular risk factors (Laurent et al., 2006).

Only one study has investigated the association between SES and Cdist (among other markers of carotid stiffness) (Climie et al., 2019). The study on a French population showed that more adverse individual-level SES (using composite index of individual deprivation and education) was associated with lower Cdist, which remained after adjustment for possible confounders (BMI, mean blood pressure, heart rate, glucose, HDL, triglycerides, alcohol, smoking, history of CVD and physical

activity). For men, the similar association was seen also using occupation level as individual-level SES. Additionally, the ARIC study has demonstrated a direct cross-sectional association between adult SES and pulsatile arterial diameter change, another marker of carotid artery elasticity (Din-Dzietham et al., 2000). Furthermore, the inverse association between SES and PWV has been distinguished in some studies. A Dutch prospective cohort study showed that educational attainment of the mother was negatively associated with PWV of their children, but the association was attenuated after adjustment for potential confounders (Bouthoorn et al., 2014). A study of African American and Caucasian adolescents showed that low or medium family income, low parental education and low neighborhood SES were all associated with higher PWV (Thurston et al., 2009). In line, results from the Whitehall study showed that the mean difference in 5-year PWV change in adulthood was higher among participants with lower adult SES assessed by several indicators including income, education and employment grade (Trudel et al., 2016). However, no association between adult SES and baseline PWV (at the mean age of 65.5 years) was observed. The same study also investigated the association of father's social class with baseline PWV and with the 5-year change in PWV and in contrast to adult SES, parental SES was inversely associated with baseline PWV but had no effect on 5-year change in PWV.

Previous studies have shown the unfavorable effects of early and long-term socioeconomic disadvantage on subclinical CVD using ultrasound measurements of carotid plaques and IMT (Kestilä et al., 2012; Nash et al., 2011; Thurston et al., 2014). Additionally, the severity of plaques and carotid IMT are shown to associate with arterial stiffness (Van Popele et al., 2001), and the predictive value of conventional cardiovascular risk factors in childhood is established both for Cdist and for PWV (Aatola et al., 2010a; Juonala et al., 2005). Therefore, the association of childhood SES with these two markers of arterial stiffness is plausible, although current data are limited.

2.4.2 Left ventricular mass

When the heart is predisposed to hemodynamic stress, it is able to compensate the load by increasing its mass (Kehat et al., 2010). As myocytes are only able to proliferate for a short time after birth, increased pressure or volume load leads to hypertrophy of existing myocytes. LVM is also strongly associated with somatic growth with LVM increasing 10-fold from childhood to adulthood (Lorell et al., 2000). Physiological and exercise-induced growth of the LV is mainly mediated through growth factors while the pathological pathway is mostly stimulated by neurohormones (Dorn et al., 2005). In obese adolescents, the increase in LVM is suggested to relate not only to the increase in hemodynamic load, but also to neurohormonal alterations due to metabolic disturbances of obesity (Chinali et al., 2006).

The first studies investigating the association between LVM and CHD were performed using the electrocardiography (ECG) method. Using this method, the Framingham Heart Study (FHS) was the first to show the association between higher LVM and increased risk of CHD (Kannel et al., 1970). Later, the association was replicated by another study of the FHS that used echocardiography as an indicator of LVM (Levy et al., 1989). The study found that LVM of elderly people free of clinical CHD was prognostic for CHD events over a 4-year follow-up. The prognostic value remained significant after adjustment for age, systolic blood pressure, smoking and the ratio of total/HDL cholesterol. Similar results were later shown in a cohort of middle-aged participants (Levy et al., 1990). Higher LVM was associated with higher risk of cardiovascular events and deaths after adjustment for several conventional cardiovascular risk factors. Moreover, results of the Cardiovascular Health Study's elderly cohort showed that those in the highest LVM quarter had over 3-fold higher risk for coronary disease in comparison to the lowest quarter (Gardin et al., 2001). Furthermore, LVM is an independent predictor of heart failure not related to previous myocardial infarction in elderly population (de Simone et al., 2008).

LVM is also shown to associate with increased risk of sudden cardiac death, possibly due to higher incidence of ventricular arrhythmias in patients with left ventricular hypertrophy (LVH), describing markedly increased LVM (Haider et al., 1998; Levy et al., 1987a). A study comparing the utility of LVH, LV ejection fraction and the number of stenosed vessels to predict survival in cardiac patients found that LVH was a stronger predictor of death than the other two more traditional markers of CHD (Liao et al., 1995). Furthermore, regression of LVH due to antihypertensive treatment is suggested to reduce risk for sudden cardiac death, independent of blood pressure and other cardiovascular risk factors (Wachtell et al., 2007).

Strong independent clinical determinants of LVM include age, height, systolic blood pressure and BMI (Savage et al., 1990). Among men, there was also a weak association between higher physical activity and higher LVM, but hypertrophy due to physical exercise is considered to be physiological, since it is associated with normal systolic and diastolic function (Colan, 1997). Of the clinical risk factors, BMI has been shown to have the strongest effect on LVM in childhood (Daniels et al., 1995). However, the major part of the variance in childhood was explained by lean body mass and the role of fat mass was minor.

To measure LVM, the most commonly used methods are ECG, echocardiography and magnetic resonance imaging. The sensitivity of ECG is depending on the assessment standard and varies between different populations. However, when ECG was compared to echocardiogram in the FHS cohort, the prevalence of hypertrophy was markedly lower (<1%) than using echocardiography (16–21%) (Levy et al., 1990). The accuracy of echocardiography in detecting LVH is shown also in a necropsy study (Devereux et al., 1986). Magnetic resonance imaging is suggested to have even better

accuracy and reproducibility, but echocardiography has the advantage of lower costs and better availability making it the most practical method clinically (Armstrong et al., 2012). The most commonly used method to estimate LVM by echocardiography is to measure the interventricular septum, LV inferolateral wall thickness and LV internal diameter from 2D-guided M-mode or from direct 2D echocardiography (Figure 3) (Lang et al., 2015). The measurements are used to calculate the myocardial volume, which is converted to mass by multiplying by myocardial density. When increased LVM reaches a certain cut-off-level, it is denoted as LVH. Although this term has particular importance in the clinical setting, several different criteria have been proposed to define LVH. According to the most commonly used criterion in echocardiography, to have LVH, LVM should be two standard deviations (SD) above the mean value of the healthy population sample of the FHS (Levy et al., 1987b).

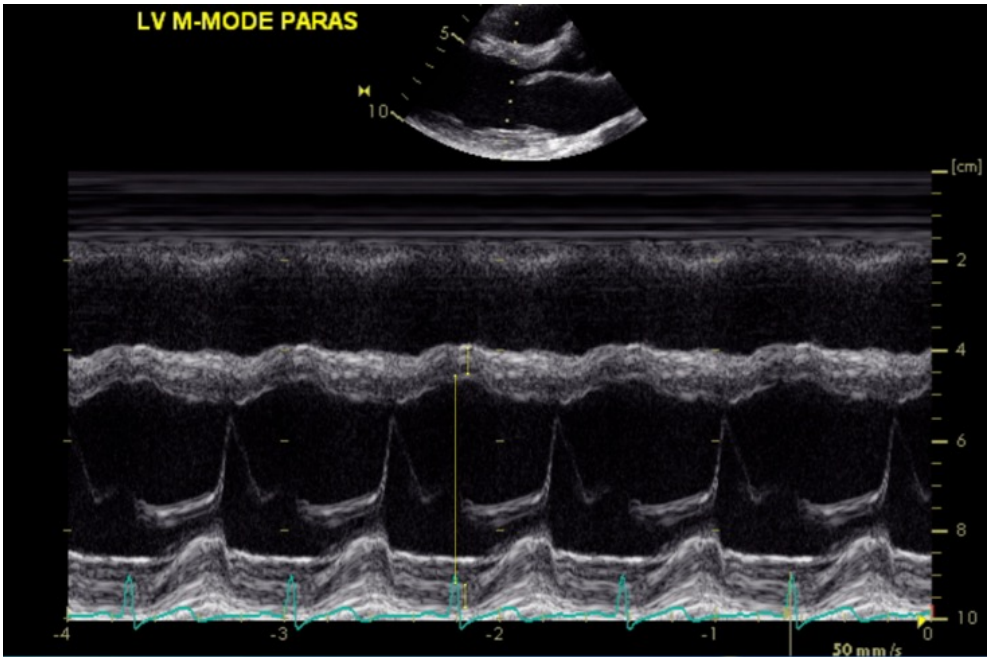

Figure 3. M-mode ultrasound image obtained during measurement of cardiac left ventricular mass. Reproduced with permission from the thesis by Mikola, (2018).

Inverse associations of SES with LVM (Medenwald et al., 2016; Murray et al., 2016; Rodriguez et al., 2004) and the prevalence of LVH (Christensen et al., 2011; Kubota et al., 2017) have been shown. A study from the U.S. suggested that low SES measured as years of education was associated with higher LVM that remained after adjustment for age, sex, systolic blood pressure, diabetes, physical activity and BMI (Rodriguez et al., 2004). Race-ethnic comparisons of the study revealed that SES

was inversely associated with LVM among blacks but not among whites or Hispanics. A British cohort study investigating the association between occupational SES and LVM at multiple time points from childhood to adulthood found that time spent in manual socioeconomic position in any of the 3 studied time points (childhood, young adulthood, later adulthood) increased LVM index by 2.5 $g/m^{2.7}$ (Murray et al., 2016). Adjustment for current BMI attenuated the association by 32%, suggesting the mediating role of adult BMI. In contrast, adjustment for several other cardiometabolic risk factors had only small effects on estimates. Similarly, a German study showed that more than 50% of the association between educational SES and LVM was explained by BMI but that other potential mediators had no significant effects on the association (Medenwald et al., 2016).

Prior results have suggested a potential pathway from low childhood SES to increased LVM and finally to LVH in adulthood, but the evidence is limited. This pathway might be strongly mediated by adulthood obesity, a well-established risk factor for LVH. However, not all of the association is explained by higher BMI among low SES individuals and studies have shown that other conventional risk factors of cardiometabolic health have no significant effects on the association between SES and LVM (Medenwald et al., 2016; Murray et al., 2016). Researchers propose the possible role of sympathetic stimulation due to higher chronic stress among low SES people, but studies are lacking. Regardless of the detailed mechanisms, the possible pathways between low child SES and increased LVM are complex and intertwined with the pathways that are explaining the associations between child SES and other outcomes of cardiometabolic disease.

2.4.3 Left ventricle diastolic function

Diastolic dysfunction means abnormal diastolic function of the heart resulting from a decrease in LV compliance and relaxation (Zile, 2002). When compliance of the LV is decreased, higher pressure during diastole is needed to fill the LV to the same volume. The mechanisms which cause impairment of diastolic function include alterations in structures and processes within the cardiomyocytes and within the extracellular matrix and different autocrine and paracrine neurohormonal systems related to them. Ischemia, pressure-overload hypertrophy and restrictive and hypertrophic cardiomyopathy can activate any of these mechanisms and affect the development of diastolic dysfunction.

Impaired diastolic performance can gradually lead to diastolic heart failure, which is characterized as having the traditional symptoms of heart failure, but preserved ejection fracture (Kane et al., 2011; Zile et al., 2004). Of all heart failures, 50% are suggested to relate to preserved LV systolic function (Jessup et al., 2003). Furthermore, mild diastolic dysfunction defined as impaired relaxation without signs

of increased filling pressure is associated with an 8-fold increase in risk of all-cause mortality, while more severe diastolic dysfunction with impaired filling pressures or pseudonormal filling is associated with a 10-fold increase in risk (Redfield et al., 2003).

Diastolic dysfunction is thought to be a link between hypertension and heart failure. Hypertension causes an increase in afterload which is reflected in thickening of the ventricular wall in response to wall stress during systole that can lead to concentric hypertrophy (Zile et al., 2002). Therefore, numerous people with diastolic heart failure have concentric hypertrophy on echocardiography and concentric hypertrophy in hypertensives is associated with impairment of diastolic function even when systolic function of the LV is normal (Hunt et al., 2005; Ren et al., 1994). Among middle-aged participants in the Young Finns Study, systolic blood pressure, waist circumference and smoking were identified as modifiable determinants of LV diastolic performance (Heiskanen et al., 2019).

The cardiac cycle consists of two periods: systole and diastole. During systole the LV is contracted and elastic elements of the ventricle wall become twisted and compressed (Paulus et al., 2007). In the beginning of diastole, the compression of the LV is reduced, and energy stored in compression is released (Little et al., 2009). This causes LV pressure to fall rapidly, which produces a gradient between the atrial and ventricle and initiates passive filling of the LV. If this passive filling due to ventricle relaxation fails, the pressure in the left atrial increases (Flachskampf et al., 2015). To measure the left atrial pressure, the golden standard is to use cardiac catheterization (Galderisi, 2005). However, catheterization is an invasive method with several associated risks that does not make it the primary choice for clinical practice. Therefore, non-invasive Doppler echocardiography is used to measure diastolic function and diagnose diastolic dysfunction.

There are several methods to measure the filling pressure by ultrasound. One commonly used technique is to measure characteristics of the flow pattern through the mitral valve in diastole (Galderisi, 2005). The peak velocity of blood flow through the mitral valve during early diastole (E) correlates with LV filling pressure, but is also affected by myocardial relaxation (Park et al., 2011). Thus, to distinguish increasing filling pressure due to LV diastolic dysfunction, mitral annular early diastolic velocity (e'), describing LV relaxation, needs to be taken into account. According to the American Society of Echocardiography and the European Association of Cardiovascular Imaging, diastolic dysfunction is categorized into four grades (Nagueh et al., 2016). E/e' is increased in grade II (impaired relaxation), grade III (pseudonormal) and grade IV (fixed restricted). In grade II, average E/e' ratio is 10–14 and in grade III over 14. In contrast, a comparative study between Doppler echocardiography and catheterization found that E/e' predicts normal filling pressure with values under 8 and increased filling pressure with values over 15 (Ommen et

al., 2000). However, a study on hypertensive patients showed that even though most of the E/e' values were within the "normal range", each one unit rise in the E/e' ratio associated with a 17% increase in the risk of a cardiac event (Sharp et al., 2010).

Morbidity and mortality related to heart failure are shown to be patterned by SES. A higher incidence and worse survival of heart failure is associated with both child and adult SES (Christensen et al., 2011; Ingelsson et al., 2006; Roberts et al., 2010). In addition, using education as an indicator of SES an inverse association with severe diastolic dysfunction was shown in Danish adults (Christensen et al., 2011). However, no association between SES and mild dysfunction was observed. In addition, a British study showed that lower SES in childhood and young adulthood associated with worse diastolic function at the age of 60-64 years (Murray et al., 2016). Consistent with findings for LVM, the association of diastolic function with SES was largely explained by BMI but adjustment for other cardiometabolic risk factors had only minor effects on the association. Contrary to diastolic function, SES was not associated with markers related to systolic dysfunction. Thus, in the pathway linking low SES, obesity, arterial stiffening and increased LVM, LV diastolic dysfunction might be the next link in the chain of risky cardiometabolic conditions, and one step closer to clinical outcomes of CVD.

2.5 Association of socioeconomic status with the risk of cardiometabolic outcomes

The interface between CVD and T2DM is well established and to describe their shared effects on clinical outcomes, the term cardiometabolic disease is often used (Fisher, 2006; Pescatello, 1999). Cardiometabolic disease accounts for a great part of non-communicable disease mortality (World Health Organization, 2013). Although the socioeconomic gap in cardiometabolic health is broadly recognized (Agardh et al., 2011; Schultz et al., 2018), SES was not included the WHO's 25x25 initiative that targets seven major risk factors of non-communicable disease with the aim to reduce the prevalence of premature mortality of non-communicable disease by 25% by 2025 (World Health Organization, 2013). This is strange as low SES has the third highest population attributable fraction (only preceded by smoking and physical activity) (Stringhini et al., 2017), underlying that SES needs consideration alongside the well-established risk factors of cardiometabolic disease. Therefore, to minimize cardiometabolic disease morbidity and mortality, interventions might need to take into account socioeconomic inequalities together with conventional cardiovascular risk factors.

2.5.1 Type 2 diabetes

According to the International Diabetes Federation, diabetes affects 425 million individuals globally, with 90% of these having T2DM (*IDF Diabetes Atlas Eighth Edition*, 2017). The prevalence of T2DM is increasing, and is predicted to be 629 million by 2045. The epidemic is principally related to lifestyle changes and the increasing prevalence of overweight and obesity. According to the FinHealth 2017 Study, 15% of men and 9% of women in Finland aged over 30 years are estimated to have diagnosed or undiagnosed diabetes (Koponen et al., 2018).

Glucose metabolism is strictly regulated in the human body by a feedback loop between pancreatic β-cells and insulin-sensitive tissues (Kahn et al., 2014). Obesity is strongly associated with increased insulin resistance which induces β-cells to increase insulin output to maintain normal glucose levels. However, when β-cells become unable to perform their duty, glucose concentrations in plasma increases, leading to clinical hyperglycemia. The diagnostic criteria of T2DM stated in the Finnish Current Care Guidelines are consistent with the American Diabetes Association (ADA) and the WHO (Table 1) (Working group appointed by the Finnish Medical Society Duodecim, the Finnish society of Internal Medicine, et al., 2018).

Table 1. Diagnosis of IGT, IFG and T2DM.

	Normal	IGT	IFG	T2DM
Fasting PG (mmol/l)	≤ 6,0 (WHO) ≤ 5,5 (ADA)		6,1–6,9 (WHO) 5,6–6,9 (ADA)	> 7,0
2-h PG during 75-g OGTT (mmol/l)	< 7,8	7,8–11,0		> 11,0
Random PG + symptoms (mmol/l)				> 11,0
HbA1c (mmol/l)	< 42			≥ 48

ADA= American Diabetes Association, HbA1c=glycated hemoglobin,
IFG= impaired fasting glucose, IGT= impaired glucose tolerance,
OGTT= oral glucose tolerance test, PG= plasma glucose,
T2DM= type 2 diabetes, WHO= World Health Organization
Adapted and modified from *Type 2 diabetes: Current Care Guidelines* (Working group appointed by the Finnish Medical Society Duodecim, the Finnish society of Internal Medicine, et al., 2018).

Chronic T2DM characterized by hyperglycemia, insulin resistance and the clustering of other risk factors (e.g. hypertension, dyslipidemia) leads to microvascular and macrovascular complications (Beckman et al., 2016). Macrovascular complications include CHD, peripheral arterial disease and cerebrovascular disease. Hyperglycemia and insulin resistance both contribute to the pathogenesis of macrovascular complications through modifications to normal physiology, but still,

the effects of intensive glycemic control on the risk of macrovascular end-points are warranted compared with the well-proven risk reduction in microvascular complications (Fox et al., 2015).

Individuals with T2DM have a 2-fold increased risk of CHD compared to those without diabetes (Sarwar et al., 2010). In the Finnish East-West study, the risk for myocardial infarction among persons with T2DM but without a previous myocardial infarction did not differ from those with prior myocardial infarction (Haffner et al., 1998). However, a more recent meta-analysis has shown that diabetic patients without prior myocardial infarction have 43% lower risk of developing cardiovascular events compared with patients without diabetes but with previous myocardial infarction (Bulugahapitiya et al., 2009). Nevertheless, a prospective observational study has indicated that for every 1% reduction in glycated hemoglobin (HbA1c) there is a 14% risk reduction in myocardial infarction with the lowest risk among those having HbA1c in the normal range (Stratton et al., 2000).

Clinical T2DM is preceded by impaired fasting glucose (IFG) and impaired glucose tolerance (IGT), which are early abnormalities of glucose metabolism. The ADA and the WHO have distinct diagnostic criteria for IFG and IGT, which are both presented in the Finnish Current Care Guidelines (Table 1). It is suggested that about 70% of individuals having these impairments of glucose metabolism, also called prediabetic states, will develop T2DM because of progressive decline in β-cell function (Nathan et al., 2007; Weyer et al., 1999). Furthermore, the prediabetic values of fasting glucose and 2-h glucose are shown to associate with about 20% increased risk of CVD (Ford et al., 2010).

Low SES has been linked to higher prevalence of T2DM in multiple developed countries including the U.S. (Maty et al., 2005), UK (Kumari et al., 2004), Netherlands (Mackenbach et al., 1996), Germany (Rathmann et al., 2005), Sweden (Agardh et al., 2007) and Finland (Wikström et al., 2011). Furthermore, results from a systematic review and meta-analysis of 23 studies showed a higher prevalence of T2DM among those of low SES (Agardh et al., 2011). Low levels of occupation, education and income were associated with 31%, 41% and 40% increased risks of T2DM, respectively, compared with high levels of SES. As individuals with low education more often underreport the prevalence of T2DM, socioeconomic differences reported in studies that collect data on T2DM by questionnaires or interviews likely underestimate the true difference (Mackenbach et al., 1996).

The available evidence suggests that the association between SES and T2DM tends to be stronger in women compared with men (Agardh et al., 2011; Rathmann et al., 2005; Wikström et al., 2011). The reasons for sex-related differences in the association between SES and T2DM are postulated but remain unclear. Obesity and MetS are important mediators in the association between SES and T2DM and, as discussed earlier in this thesis, SES associates inversely with the prevalence of

obesity and MetS, particularly in women (Chichlowska et al., 2009; Sobal et al., 1989).

Results of studies investigating the association between childhood SES and adult T2DM have varied. Some studies have suggested that childhood SES is an independent predictor of insulin resistance and prevalence of T2DM in adulthood, especially among women (Kivimäki et al., 2006c; Lawlor, 2002; Maty et al., 2008; Pikhartova et al., 2014; Wray et al., 2006). In contrast, a previous study in the Young Finns Study found the association between child SES and adult insulin resistance to be stronger in men (Kivimäki et al., 2006c). In some studies, childhood SES is not independently associated with adult T2DM (Agardh et al., 2007; Smith et al., 2011). The differing results might be due to differences in adjustments for risk factors, in years of follow-up and in the indicators of childhood SES.

A study which failed to show the independent association between childhood SES and adult T2DM showed that there was an inverse association between cumulative life-course SES and T2DM, but only among women (Smith et al., 2011). Results of a study in which childhood SES was determined retrospectively suggested that women with decreasing SES from childhood to adulthood had higher risk of T2DM than women with stable high SES (Lidfeldt et al., 2007). However, women, who improved their SES from childhood to adulthood did not differ in their risk of T2DM from those with stable high SES. Therefore, timing of socioeconomic disadvantage might be important in the association with risk for T2DM and studies should take into account life-course SES instead of SES from a single timepoint.

Overweight or obesity accounts for up to 90% of T2DM (Hossain et al., 2007). A study in the U.S. suggested that although childhood SES was not significantly associated with adult prediabetes, low childhood SES predicted higher waist circumference and lower physical activity independent of adult SES (Tsenkova et al., 2014). The authors suggested that while child SES had no independent effect on glucose tolerance, abdominal obesity might work as a strong mediator in the pathway from low child SES to increased risk of T2DM in adulthood. Furthermore, studies supporting the independent association of childhood SES with the prevalence of T2DM have distinguished the modifying effect of BMI. A prospective study with 34-years of follow-up showed that after adjustment for health behaviors, the association of childhood SES with risk of T2DM was seen only among those with obesity (Maty et al., 2008). Therefore, those who were disadvantaged in childhood but managed to be normal weight in adulthood, were not at higher risk for T2DM, suggesting a combined effect of child SES and adult obesity.

In addition to BMI, there are several lifestyle and environmental factors that associate with the risk of T2DM and are also patterned by SES (Dendup et al., 2018; Galobardes et al., 2001; O'Donoghue et al., 2018). When an Australian study investigated the mediating role of different health behaviors on the association

between low education and risk for T2DM, health behaviors explained 27% of the relationship (Williams et al., 2010). Of different health behaviors, smoking and exercise were the strongest mediators. Similarly, the results of the Whitehall II study indicated that health behaviors and BMI explained over 30% of the socioeconomic differences in T2DM incidence (Stringhini et al., 2012). However, obesity was again the strongest contributor, explaining approximately 20% of the differences.

Results of the Finnish Diabetes Prevention Study have shown that lifestyle changes in terms of weight reduction, regular exercise and modification of diet can reduce the incidence of T2DM by 58% during a 3-year follow-up among overweight participants with impaired glucose tolerance (Tuomilehto et al., 2001). However, results from an evidence-based lifestyle intervention study to prevent diabetes on American Indian and Alaska Native participants showed that those with lower household income had a smaller reduction in BMI after 16 sessions (Jiang et al., 2015). In contrast, the effect of lifestyle intervention of the Finnish national diabetes prevention program on anthropometric measurements was similar in all socioeconomic groups, suggesting the effectiveness of lifestyle interventions also among people with low SES (Rautio et al., 2011). Similarly, another Finnish intervention study to prevent T2DM in women showed that lifestyle changes can be equally effective irrespective of the participant's education level (Hankonen et al., 2012). Taken together, these results suggest that identifying individuals with increased risk for T2DM at early stages of life, and the implementation of timely and effective interventions, could reduce the incidence of T2DM across all SES groups of the Finnish population.

2.5.2 Cardiovascular outcomes

In the late 1960s, Finnish men had the highest CHD mortality in the world (Thom et al., 1994). However, favorable changes in risk factor levels in the Finnish population led to over 80% decline in coronary mortality, similar to the U.S. and other developed countries (Benjamin et al., 2019; Jousilahti et al., 2016). A policy statement from the American Heart Association in 2011 forecasted that from year 2010 to 2030 the prevalence of CVD would rise due to the increase in prevalence of obesity, hypertension, T2DM and physical inactivity and aging of the population (Havranek et al., 2015; Heidenreich et al., 2011). Data from the U.S. indicate that although the crude prevalence of CVD had increased between years 2006 and 2016, the age-adjusted prevalence of CVD had decreased despite the increasing age-standardized prevalence of adult obesity across the same period (Benjamin et al., 2019; Roth et al., 2018b). Likewise, in the Finnish population the prevalence of obesity among those of working-age has increased but trends of other cardiometabolic risk factors have improved, including the downward trend in daily smoking and cholesterol, and an increasing trend in those meetig sufficient physical activity levels (Koponen et al., 2018).

Furthermore, increasing trends in the prevalence of T2DM and raised blood glucose levels seem to have leveled off (Abouzeid et al., 2015; Koponen et al., 2018). Still, CVD remains the current leading cause of death in Finland (Statistics Finland, 2019a). As growing evidence suggests that prevention and treatment of CVD are not equally shared between socioeconomic groups, a better understanding of the social determinants of cardiovascular morbidity and mortality is needed to improve cardiovascular health across the whole population (Havranek et al., 2015). It is evaluated that if the prevalence of public health problems were the same in the general Finnish population as it is among the higher educated, the mortality caused by CHD would diminish by 45% (Ministry of Social Affairs and Health, 2014).

Pioneers in the association between SES and cardiovascular mortality, the Whitehall studies found an inverse social gradient in cardiovascular mortality at all levels of the occupational hierarchy (Kaplan et al., 1993; Marmot et al., 1984, 1991). Additionally, the association between SES and cardiovascular mortality was not totally explained by conventional cardiovascular risk factors (Marmot et al., 1991). Research on the association between SES and CVD continues today, with results suggesting the socioeconomic gap in CVD has not narrowed but prevailed or even widened (Bajekal et al., 2013; Palosuo et al., 2009). A report of the European Commission suggested that CVD accounts for 40% of the socioeconomic difference in mortality rates among men and 60% among women in Europe (Mackenbach, 2006). As with all-cause mortality, socioeconomic inequalities in ischemic heart disease have been proposed to have a North-South gradient, since relative and absolute inequalities are suggested to be larger in Northern than in Southern European countries.

All three conventional indicators of individual SES; income, education and occupation, have a strong association with CVD in high-income countries (Schultz et al., 2018). The roles of the different indicators vary between geographical locations and different societies but also within one society and across the life-course of an individual (Mackenbach et al., 2000; Mosquera et al., 2016). Different indicators of SES are suggested to measure different causal pathways, but also have combined effects in creating the socioeconomic differences in the incidence of CHD (Geyer et al., 2006; Lewis et al., 2015).

A Swedish study suggested that inequalities in income explained a substantial portion of socioeconomic inequality in CVD in all time periods from mid-life to old age (Mosquera et al., 2016). Level of income is shown to associate with the incidence of sudden and non-sudden cardiac death, non-fatal myocardial infarction and the risk of hospitalization from CVD in high income countries (Kucharska-Newton et al., 2011; Mosquera et al., 2016). A Finnish study suggested that about 40-50% of socioeconomic differences in cardiovascular mortality are explained by higher case-fatality among low SES people, whereas the rest was explained by differences in incidence of myocardial events (Salomaa et al., 2001). Those in the high income

group were more frequently treated in specialist hospitals and the delay from the onset of symptoms to hospitalization was shorter when compared with those in the low income group. Furthermore, people with low income are less likely to get prescription medications for secondary prevention and men with high income have higher incidence of coronary angiography and revascularization after the event. Finally, despite increased resources, the relative socioeconomic differences by income in standards of care, in terms of access to coronary revascularization, are still seen in Finland (Lumme et al., 2017).

While the explanatory role of income to socioeconomic inequalities in CVD is stable from midlife to older age, the role of education varies across the lifetime (Mosquera et al., 2016). A follow-up study from the U.S. found that at middle age, educational attainment was inversely associated with lifetime risk of CVD independent of current income, occupation or parent education (Kubota et al., 2017). Similar results for income are seen in the risk of cardiovascular events and mortality, especially among those of working-age (Gerber et al., 2008; Kelly et al., 2010; Woodward et al., 2015), but also in standards of care (Alter et al., 2004). A Canadian study showed that lower educated people were less likely to undergo coronary angiography, receive cardiac rehabilitation or be followed-up by a cardiologist after an acute myocardial infarction (Alter et al., 2004).

Despite the Whitehall studies using occupational level as indicator of SES, the current evidence on the association between occupation and CVD is weaker than for income and education. Data from 11 European countries showed that lower occupational class associated with higher mortality from ischemic heart disease in the UK and northern European countries (Kunst et al., 1998), but in France, Switzerland, and Mediterranean countries no association was seen. Additionally, a U.S. study has shown that higher education is associated with lower risk of cardiovascular events regardless of occupation (Kubota et al., 2017).

Conventionally, studies have investigated socioeconomic differences in cardiovascular health by focusing on adulthood SES but the importance of social determinants of early life has augmented the focus to socioeconomic differences in childhood and to chains of risks, which tend to persist over the life-course (Lynch et al., 2005). The Barker hypothesis suggests that the intrauterine environment and especially malnutrition of the fetus results in adaption to a limited supply of nutrition, which can lead to reduced insulin sensitivity, hypercholesterolemia, T2DM and CHD later in life (Barker, 1998). Inspired by the Barker hypothesis, sociologists suggested social programming theory to better explain socioeconomic differences (Vågerö et al., 1995). According to social programming theory, social conditions in childhood, in addition to biological factors such as malnutrition, directly and indirectly influence adulthood health.

A systematic review of 40 studies found worse socioeconomic circumstances in childhood to associate with higher risk for developing CVD in adulthood (Galobardes et al., 2006c). Lower SES in childhood was associated with greater risk for CHD and associations were consistent both in prospective and cross-sectional studies. However, only few studies investigating the association between childhood SES and cardiovascular mortality used prospective data and conventional indicators of SES (Table 2). The majority of these studies suggested an inverse association between childhood SES and cardiovascular mortality but one Swedish study included adjustment for adult SES. Of note was that all studies used occupational status as an indicator of parental SES.

Table 2. Childhood SES and cardiovascular mortality in prospective studies.

Study	N and sex	Country	Baseline and mortality follow-up	Age at baseline	Indicator of child SES	Outcome
Glasgow cohort (Smith et al., 2001)	8396, men	the UK	baseline at 1948–1968, follow-up until 1998	20 years (average)	father's occupation	CVD mortality: inverse association
Boyd Orr cohort (Frankel et al., 1999)	3750, men	the UK	baseline at 1937–39, follow-up 1948–1997/1998	6–9 years	father's occupation	CHD mortality: no association
Finnish cohort (Pensola et al., 2003)	112735, men	Finland	baseline at 1970, follow-up 1990–1998	10–14 years	household head's occupation	CVD mortality: inverse association
Finnish cohort (Pensola et al., 2003)	1414221 (person-years), women	Finland	baseline at 1970, follow-up 1990–1998	10–14 years	household head's occupation	CVD mortality: inverse association
Swedish cohort (Vågerö et al., 1994)	404450, men	Sweden	baseline at 1960, follow-up 1980–1986	5–14 years	household head's occupation	CHD mortality: inverse association
Metropolit Study (Osler et al., 2003)	7493, men	Denmark	baseline at 1965, follow-up 1986–1998	12 years	father's occupation	CVD mortality: inverse association

CHD=coronary heart disease, CVD=cardiovascular disease, SES=socioeconomic status

Using data from the 1950 Finnish census linked to registry data collected from 1988-2010, a professional or administrative background was associated with lower incidence of myocardial infarction compared with a manual worker background (Kilpi et al., 2017). However, other indicators of child SES showed no association

with myocardial infarction and the shown association was partially attenuated after adjustment for adult education and income. Additionally, of the broad array of indicators for childhood SES, only rented childhood home background in women was associated with short-term fatality after myocardial infarction following adjustment for adult SES. In line, results of a Swedish study indicated that SES at birth was not associated with 28 day case fatality following myocardial infarction (Rajaleid et al., 2009). Additionally, a study from the Netherlands showed that retrospectively determined childhood SES was associated with cardiovascular mortality only among men and that 88% of the association was explained by material, behavioral and psychosocial risk factors in adulthood and by adult SES (Kamphuis et al., 2013).

In conclusion, data from developed countries with comprehensive welfare policies suggest that childhood SES has a shared role with adulthood SES in determining the risk of cardiovascular morbidity and mortality in adulthood. However, evidence is limited and on-going prospective studies will offer further understanding of the role of child SES in determining the risk of cardiovascular outcomes.

2.6 Summary of the literature review

Evidence of the socioeconomic gradient in health behaviors is extensive. Cross-sectional studies have shown that those with lower SES have more adverse diet, lower leisure-time physical activity and higher smoking prevalence both in childhood and adulthood. The associations between child SES and adult health behaviors are suggested to be parallel but data from prospective studies is more limited.

Higher SES is shown to be protective of risk factor clustering but evidence for an inverse association between SES and MetS exists only in women. Evidence on the association between child SES and risk of adult MetS is mainly limited to studies that use retrospectively defined childhood SES. Similarly, the results concerning the association between childhood SES and T2DM have been inconsistent, possibly reflecting different measures of exposure, adjustments, study populations and follow-up times across studies.

Evidence of the influence of SES, and especially childhood SES, on subclinical markers of cardiovascular health is weak. An inverse association between SES and PWV has been shown in some cross-sectional studies but evidence of the association between child SES and adult PWV is deficient. For Cdist, the evidence is even scarcer especially from prospective studies. One prospective birth cohort has suggested the association between lower child SES and both higher LVM and worse LV diastolic function in adulthood, but the study did not measure other cardiometabolic risk factors in childhood (Murray et al., 2016).

In light of previous literature, one of the major advantages of this thesis is the prospective design of the Young Finns Study with long follow-up spanning childhood to middle-age. Participants included both men and women who were well phenotyped both in childhood and adulthood. This allows conventional childhood cardiometabolic risk factors, along with child SES, to be taken into account. The availability of adulthood SES allows the independent and mediated effects of childhood SES to be examined. Finally, since evidence of the association between childhood SES and subclinical markers of cardiovascular health (PWV, Cdist, LVM and LV dysfunction) has been limited, having them assessed in adulthood follow-ups of the Young Finns Study provides the ability to examine novel associations and potential pathways.

3 Aims

This thesis is based on findings from the Cardiovascular Risk in Young Finns Study. The aim was to study the role of childhood SES in determining adulthood cardiometabolic health by investigating the association of childhood SES with health behaviors, MetS and glucose abnormalities, and subclinical markers of CVD (Figure 4).

The specific aims were:

1. to study the association of childhood SES with dietary intake, smoking, alcohol consumption and leisure-time physical activity in adulthood, and to examine differences in these health behaviors between participants with low and high childhood SES at multiple time-points from childhood to adulthood (Study I);
2. to investigate the association of childhood SES with MetS and glucose abnormalities (IFG and T2DM) in adulthood (Study II);
3. to determine the association of childhood SES with subclinical markers of CVD in adulthood (Studies III, V); and
4. to examine whether childhood SES is an independent risk factor of cardiometabolic health alongside other conventional childhood cardiometabolic risk factors (lipids, systolic blood pressure, insulin, BMI, physical activity, frequency of fruit and vegetable consumption and leisure time physical activity) and adult SES (Studies I, II, III, IV).

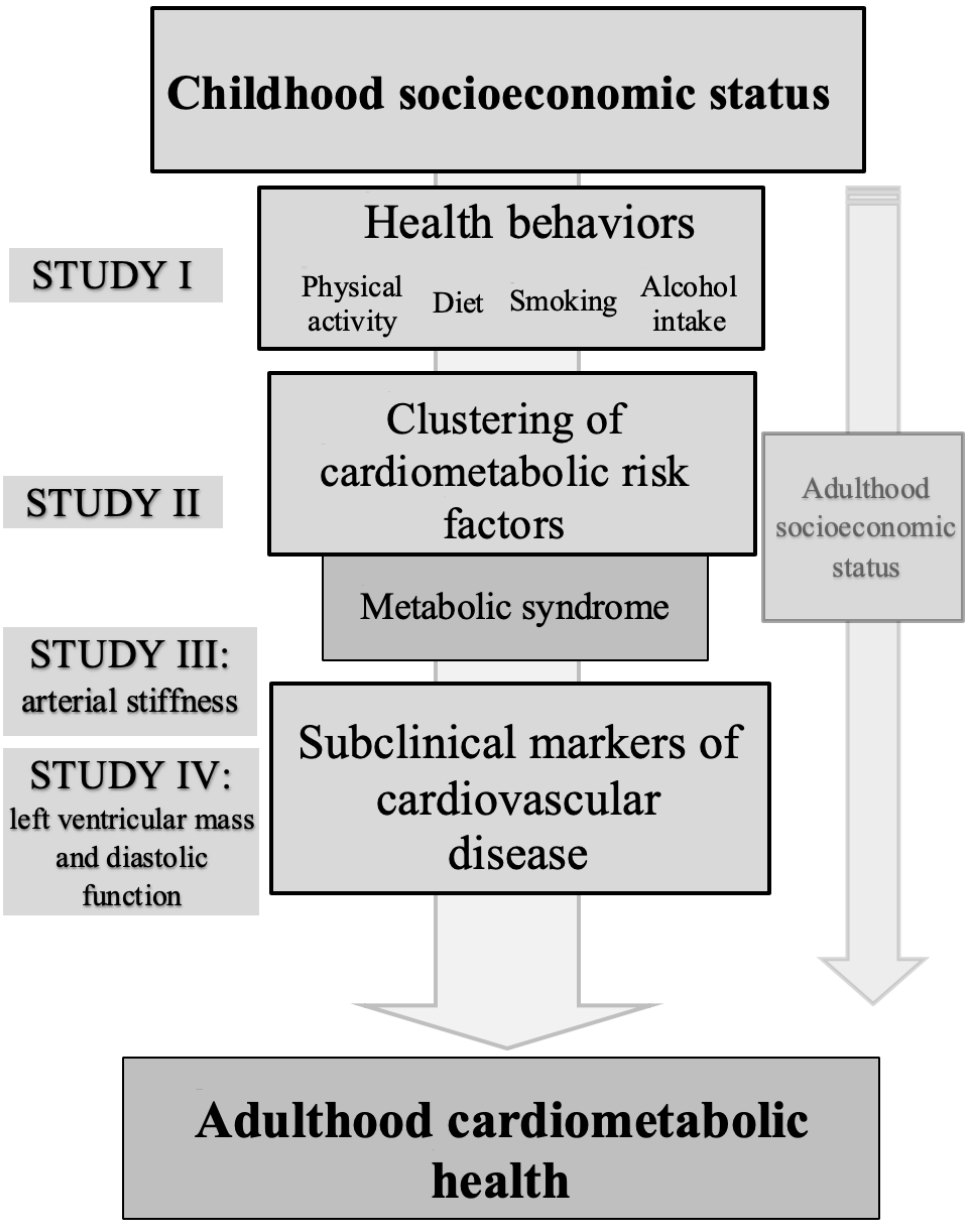

Figure 4. Overview of the thesis aims.

4 Materials and Methods

4.1 The Cardiovascular Risk in Young Finns Study

The study population comprised participants of the Young Finns Study. The Young Finns Study is an on-going population-based follow-up study carried out in all five Finnish cities with university hospitals and their rural surroundings. The aim of the Young Finns Study was to assess risk factors underlying CVD. The first cross-sectional study was conducted in 1980 and included 3596 (49% males, 83% of those invited) children and adolescents aged 3, 6, 9, 12, 15 and 18 years. The participants were randomly recruited from the national population register of these areas to produce representative sample of Finnish children. Since then, regular follow-ups have been performed. The participation rates in the follow-up studies are shown in Figure 5. In this thesis, data from 1980, 1983, 1986, 2001, 2007 and 2011 follow-ups were used. The Young Finns Study has been approved by the local ethics committees and it has been conducted according to the guidelines of the Declaration of Helsinki. Written informed consent was obtained from the participants or their parents.

Year	N	%	Age cohorts															
1980	**3596**	**100**	**3**	**6**	**9**	**12**	**15**	**18**										
1983	**2991**	**83**		**6**	**9**	**12**	**15**	**18**	**21**									
1986	**2779**	**77**			**9**	**12**	**15**	**18**	**21**	**24**								
1989	2737	76				12	15	18	21	24	27							
1992	2730	76					15	18	21	24	27	30						
2001	**2283**	**63**								**24**	**27**	**30**	**33**	**36**	**39**			
2007	**2204**	**61**										**30**	**33**	**36**	**39**	**42**	**45**	
2011/12	**2063**	**57**												**34**	**37**	**40**	**43**	**46**	**49**

Figure 5. Overview of the study design at each stage of the Cardiovascular Risk in Young Finns Study. Bold font represents the years from which data were used in this thesis. Rates (%) indicate the proportion of participants from the original sample measured at baseline.

4.2 Study design and participants

In **Study I,** the aim was to examine the association of childhood SES with health behaviors including diet, smoking, alcohol consumption and physical activity in adulthood. Differences in these health behaviors between participants with low and high childhood SES were also investigated at multiple time-points from childhood to adulthood. The study sample comprised 3453 participants aged 3 to 18 years at baseline (1980) who had data on SES and lifestyle factors. Sample sizes for the analyses varied from 1604 to 3432 (45–95% of the original sample) depending on the outcome variable.

Study II examined the association of childhood family SES at age 3 to 18 years on adult MetS and T2DM 27 to 31 years later. The sample comprised 2250 participants (63% of the original sample) aged 3 to 18 years at baseline who had data on SES and cardiometabolic risk factors in childhood (1980) and adulthood. Those who were pregnant or had type 1 diabetes in adulthood were excluded from the analyses. In adulthood, data from the 2011 survey were primarily used in the analysis. Complete data from 2011 were not available for 323 participants (14%) and for them, data from the 2007 survey were used.

Study III investigated whether family SES of children aged 3 to 18 years was associated with adult arterial stiffness measured 27 years later. Arterial stiffness was determined by two different indicators of arterial stiffness, PWV and Cdist. The sample comprised 2566 participants (71% of the original sample) aged 3 to 18 years at baseline who were followed up as adults 21 and 27 years later and who had data on family SES in childhood and PWV and Cdist at the 2001 and 2007 follow-ups.

Study IV examined the association of childhood SES in participants aged 3 to 18 years on LVM and LV diastolic function 31 years later in adulthood. The 31-year follow-up survey included 2063 of the original participants and of these, 97% (1994) had echocardiographic examination. The final sample comprised 1871 (52% of the original sample) participants who had data on family SES in childhood and echocardiographic data at the 2011 follow-up.

4.3 Data acquisition in the Cardiovascular Risk in Young Finns Study

At the baseline study in 1980, physical measurements including blood sampling were performed by specially trained nurses, one in each of the five study areas, supervised by local investigators. Physical examinations were held across the five study areas at the outpatient departments of the Departments of Pediatrics, and in the rural communes at Public Health Centers. Data on the socioeconomic background of the family, the environment where the children were living, the children's general health and development and the parents' health habits were collected by a general questionnaire sent by mail two to three weeks in advance of the physical examination. At the same

time, questionnaires on daily physical activities and dietary habits were sent to participants. Data on smoking was collected at the physical examination in a solitary room where participants were able to respond confidentially and undisturbed. At subsequent follow-up studies, the protocols of the physical examinations and questionnaire material were essentially similar to those at the baseline study. However, in 2011 participants were able to complete web-based or paper questionnaires. In 2001 and 2007, vascular structure and function were measured by ultrasonographers following a standardized protocol in the five study centers. In 2011, echocardiographic measurements of the heart were performed in all study centers by sonographers who were trained by a cardiac imaging specialist. In 2011, cognitive function was also tested for the first time using a computerized cognitive testing battery. Table 3 indicates when assessments of key variables were performed in the Young Finns Study and when particular variables were used in this thesis.

Table 3. Variables of the Young Finn Study used in this thesis according to the assessment years

Variables:	**1980**	**1983**	**1986**	**2001**	**2007**	**2011**
	Childhood			**Adulthood**		
Anthropometric measurements						
Height and weight	**U**	**x**	**x**	**x**	**U**	**U**
Waist and hip circumferences				**x**	**U**	**U**
Blood pressure	**U**	**x**	**x**	**x**	**U**	**U**
Biochemical markers and clinical diagnosis						
Diabetes type 2 (diagnosis)				**x**	**U**	**U**
Cholesterol (total, LDL- and HDL-)	**U**	**x**	**x**	**x**	**U**	**U**
Triglycerides	**U**	**x**	**x**	**x**	**U**	**U**
Insulin	**U**	**x**	**x**	**x**	**x**	**x**
Glucose			**x**	**x**	**U**	**U**
Questionnaire data						
Physical activity	**U**	**U**	**U**	**x**	**U**	**U**
Dietary intake	**U**	**U**	**U**	**x**	**U**	**U**
Smoking	**U**	**U**	**U**	**x**	**U**	**U**
Alcohol intake	**x**	**x**	**x**	**x**	**x**	**U**
Parental income	**U**					
Parental education and occupation	**U**	**x**	**x**			
Participant's own SES*				**x**	**U**	**U**
Vascular and cardiac structure and function						
Carotid artery distensibility				**U**	**U**	
Pulse wave velocity					**U**	
Cardiac structure and function						**U**

x = variable measured, U = variable measured and used in this thesis. HDL = high-density lipoprotein; LDL = low-density lipoprotein; YFS = Young Finns Study. *SES = income, education and occupation

4.3.1 Physical examination

The physical examination included measurements of height, weight, waist and hip circumferences, and systolic and diastolic blood pressure. Weight was measured in light clothes without shoes using a digital scale to the nearest 0.1 kg. Height was measured by a wall-mounted stadiometer to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by the square of height in meters. Waist circumference was first collected in 2001, measured midway between the iliac crest and the lowest rib. In 1980, the blood pressure of 3-year-old children was measured with an ultrasound device (Arteriosonde 1020, Roche). For those aged 6-18 years, a standard mercury sphygmomanometer was used. Since 1986 blood pressure was measured using a random zero sphygmomanometer from participants right arm from the supine position following 5 minutes rest. Korotkoff's first sound was used to assign systolic blood pressure and Korotkoff's fifth sound was used to assign diastolic blood pressure. Readings to the nearest even number of millimeters of mercury was performed three times on each participant with the average of three measurements used in the analyses.

4.3.2 Questionnaires

Information on eating habits was obtained using a food frequency questionnaire (FFQ). In 1980, 1983 and 1986, information on dietary habits was obtained with a non-quantitative FFQ on the consumption of selected foods relevant to the development of CVD (e.g. vegetables, fruits, fish and meat). For participants aged 3 to 9 years, the FFQ was completed by a parent. Older participants answered the questions themselves, assisted by a parent when necessary. Consumption of these foods during the last month was assessed on a 6-point scale from 1 (daily) to 6 (only occasionally or never). The response categories were converted into times of consumption per week (Aatola et al., 2010). In 2007 and 2011, a more detailed quantitative FFQ was used that provided an estimate of food consumption during the last 12 months in grams per day. Data on vegetable, fruit, fish and meat consumption that were available at both baseline and subsequent follow-ups was used, which provided longitudinal data from childhood to adulthood. Using the quantitative FFQ in 2007 and 2011, total energy intake per day was calculated. The FFQ in 2011 also provided an estimate of the consumption of beer, wine, spirits and other alcohol beverages (g/day), from which the mean amount of drinks per day was calculated.
To complement the longitudinal dietary data, a diet score was calculated from the FFQ data obtained in 2007. The score describes the diet more extensively, defined based on the intakes of 9 food groups. In the score, whole grains, fish, fruits, vegetables, and nuts/seeds were designated as favorable, healthy foods, whereas red and processed meats, sweets, sugar-sweetened beverages, and fried potatoes were

designated as unfavorable. Intake of each food group was categorized into quartiles and assigned ascending values (0, 1, 2, 3) for favorable foods and descending values (3, 2, 1, 0) for unfavorable foods. These values were summed to generate a diet score (range: 0–27 points), with higher scores representing healthier diets (Nettleton et al., 2013).

Data on cigarette smoking was reported among participants aged 12 years or older at the 1980, 1983, 1986, 2007, and 2011 studies. Individuals who reported daily smoking were defined as smokers. The number of pack years of smoking was calculated from current and former smokers as the number of cigarette packs smoked per day multiplied by the duration of daily smoking in years. Data on parental smoking was assessed by questionnaires at baseline. Parental smoking was defined to be present if either of the parents had smoked regularly at least for one year.

Physical activity was assessed using separate questionnaires for younger children (3–6 years of age), older children (9–18 years of age) and adults. A physical activity index (PAI) indicating mainly leisure-time physical activity was calculated based on responses to the questionnaires. Physical activity of 3- and 6-year-old children was measured using responses from the participant's mother. Mothers were asked questions concerning their child's outdoor play time in summer and winter, the amount of physical activity in play as compared with other children, the intensity of physical activity, the child's enjoyment of indoor/outdoor play, the child's general level of activity as compared with other children, the encouragement given to participate in sports, and the patterns of physical activity. Each item was coded from 1 to 3, except for encouragement to engage in sport that was coded from 1 to 2. PAI was determined by summing the variables, with the PAI scores of preschool children ranging from 8 to 23. The physical activity of 9- to 18-year-old participants was measured using a short self-report questionnaire with the help of parents if needed. The questions concerned the frequency and intensity of leisure-time physical activity, participation in sports club training, participation in sport competitions, and habitual use of leisure time. The items were coded from 1 to 3, except for participation in sport competitions that was coded from 1 to 2. PAI was calculated by summing the items and ranged from 5 to 14. In adults, the physical activity questionnaire included items on intensity of physical activity, frequency of vigorous physical activity, hours spent on vigorous physical activity, average duration of a physical activity session, and participation in organized physical activity. The items were coded from 1 to 3 and PAI was calculated by summing the items and had a range from 5 to 15. Due to the different scaling of the PAI across the study period, PAI values were age-standardized for analysis.

Data on favorable emotional family environment at baseline in 1980 was assessed using questionnaires. The favorable emotional family environment score consisted of four components: absence of previously diagnosed parental mental

disorder; high parental caregiving nurturance; high parental life satisfaction; and reasonable parental alcohol use (Pulkki-Råback et al., 2015). Each component contributed 1 point to the score thus had a range from 0 to 4. In addition, a questionnaire was used to gather data on whether a female participant was pregnant or not.

4.3.3 Biochemical analyses

Venous blood samples were drawn from the right antecubital vein of recumbent participants after a 12-hour fast. In 1980, an aliquot for serum lipid analyses was stored at -25°C until analysis. All lipid determinations were done in duplicate and in the same laboratory. In 1980, total cholesterol concentrations were measured using a fully enzymatic CHOD-PAP method (Boehringer Mannheim, Mannheim, Germany) with OLLI 3000 and Kone CD analyzers (Kone Co., Espoo, Finland). In 1980, serum HDL-C concentrations were measured from the supernatant after precipitation of very low density lipoprotein, intermediate-density lipoprotein, and low-density lipoprotein (LDL) particles with dextran sulphate-MgCl2 (Pharmacia, Uppsala, Sweden) (Kostner, 1976). All analyses were performed as simultaneously as possible in the laboratory of Rehabilitation Centre of the Social Insurance Institution, Turku, Finland. The concentration of LDL-C was calculated using the Friedewald formula (Friedewald et al., 1972).

In adult surveys conducted in 2001, 2007 and 2011, serum or plasma was separated and stored at -70 °C until analysis. In 2001, all analyses were performed in the laboratory of the Research and Development Unit of the Social Insurance Institution, and in 2007 and in 2011 in the laboratory for the Population Research of the National Institute for Health and Welfare, Turku. Standard enzymatic methods (Olympus System Reagent; Germany) were used for serum cholesterol and triglycerides and HDL cholesterol was measured from the serum supernatant after precipitation of very-low-density lipoprotein and LDL with dextrane suplhate-MgCl2. LDL cholesterol concentration was calculated using the Friedewald formula for participants whose triglyceride levels were below 4 mmol/l (Friedewald et al., 1972). Because of changes in determination methods and kits across the study years, lipid levels from 1980 and triglycerides from 2007 were corrected by using correction factor equations to correspond with the samples taken in 2001 (Juonala et al., 2004). No correction equations were needed for the 2007 total cholesterol, LDL cholesterol and HDL cholesterol values and for the 2011 values.

In 2001, 2007 and 2011, serum glucose concentration was determined by the enzymatic hexokinase method (Glocose reagent, Beckman Coulter Biomedical). Due to changes in methods or reagents from 2001 to 2007, glucose levels in 2007 were corrected to 2001 levels: (glucose (2007)-0.0235)/0.9471). The concentration

of HbA1c was assayed with an immunoturbidimetric method (Hemoglobin A1c assay, Abbot, USA) on an Architect ci8200 analyzer (Abbott). In 1980, serum insulin was measured using a modification of the immunoassay method of Herbert et al. (Herbert et al., 1965).

4.4 Assessment of socioeconomic status

Annual gross income was considered as an indicator of SES in both childhood and adulthood (Liu et al., 2016). Parents of the participants reported the annual income of the family in childhood at the baseline examination in 1980. The questionnaire included income categories from 1 (lowest) to 8 (highest). Annual family income strata at the time of enrollment were determined as follows: category 1, <15,000 Finnish marks (FIM); category 2, 15,001–25,000 FIM; category 3, 25,001–35,000 FIM; category 4, 35,001–45,000 FIM; category 5, 45,001–55,000 FIM; category 6, 55,001–75,000 FIM; category 7, 75,001–100,000 FIM and category 8>100,000 FIM. Likewise, participant's own income in adulthood was classified on an 8-point scale, ranging from 1 (<10,000 €) to 8 (>70,000 €) in 2007 and on a 13-point scale, ranging from 1 (<5000 €) to 13 (>60,000 €) in 2011. To harmonize the scale in 2011 to the corresponding scale in 2007, the harmonized scale, ranging from 1 (<10,000 €) to 7 (>60 000 €), was formulated and used in two of the studies (I, II). In one of the studies (study IV), family income in 1980 was converted into its present-day value and 3 income groups were formed: low (response options 1–2, ≤16 000 €); medium (response options 3–5, >16 000 € to ≤35 000 €); and high (response options 6–8, >35 000 €).

In sensitivity analyses, parental educational years and parental occupation were used to define childhood SES. Parental education and occupational status of both parents in 1980 were obtained from self-report questionnaires completed by one or both parents. Total years of education completed in 1980 by each parent was used as the measure of parental education. Parental occupational status was coded using the scale presented by the Central Statistical Office of Finland in 1979. In this thesis, the 14 original subgroups were combined to form five categories: farmers, lower manual, upper manual, lower nonmanual, upper nonmanual and then coded from 1 to 5 according to the categories (1, indicating farmers; 2, lower manual; 3, upper manual; 4, lower nonmanual; and 5, upper nonmanual) (Laitinen et al., 2013; Leino et al., 1996). In the analyses, parental education was defined according to the parent with the most years of education and parental occupation defined according to the parent with the highest level of occupation on the above scale. Participant's own education in adulthood (in 2007 and 2011) was assessed as total years of education completed.

4.5 Definition and assessment of metabolic syndrome, impaired fasting glucose and type 2 diabetes

In 2007 and 2011, MetS was defined using the harmonized criteria (Alberti et al., 2009). Data from the follow-up conducted in 2011 were primarily used. If the participant did not attend the 2011 follow-up, data from the 2007 follow-up were used. MetS was diagnosed if the participant had at least three of the following five components: 1) waist circumference ≥102 cm for men and ≥88 cm for women; 2) triglycerides ≥1.7 mmol/L or specific treatment for this lipid abnormality; 3) HDL cholesterol <1.0 mmol/L in men or <1.3 mmol/L in women or specific treatment for this lipid abnormality; 4) blood pressure ≥130/85 mmHg or treatment of previously diagnosed hypertension; and 5) fasting plasma glucose ≥5.6 mmol/L or specific drug treatment of elevated glucose.

A fasting plasma glucose ≥5.6 mmol/L was defined as IFG (American Diabetes Association, 2010). Participants were classified as having T2DM if they had a fasting plasma glucose ≥7.0 mmol/L (126 mg/dL) in accordance with ADA criteria, had an HbA1c level of ≥6.5% (≥48 mmol/mol), reported receiving oral hypoglycemic agents and/or insulin injections, and did not have type 1 diabetes or reported a diagnosis made by a physician (American Diabetes Association, 2010) Data on having type 1 diabetes were assessed by a self-report questionnaire. As was done for MetS, data from the 2011 follow-up were primarily used and in the case of missing data, data from year the 2007 follow-up was used. Separate analyses were performed for the group of participants with T2DM and for a combined group of participants with either IFG or T2DM.

4.6 Assessment of arterial stiffness, left ventricular mass and diastolic function

4.6.1 Carotid artery distensibility

A high-resolution ultrasound system (Sequoia 512, Acuson, CA) with 13.0 MHz linear array transducer was used to study Cdist. Cdist describes the elasticity of the artery with a lower distensibility value indicating increased arterial stiffness. To assess Cdist, the best quality cardiac cycle was selected from 5-second clip images. The common carotid diameter was measured 10 mm proximal to the carotid bifurcation at least twice at end-diastole and end-systole from B-mode images. To calculate Cdist, ultrasound and concomitant brachial blood pressure was used. Cdist was calculated as [(Ds-Dd)/Dd]/(Ps-Pd), where Ds is systolic diameter, Dd is diastolic diameter, Ps is systolic blood pressure and Pd is diastolic blood pressure.

The between-visit coefficient of variation was 16.3% for Cdist (Juonala et al., 2005). In this study, the mean of Cdist values examined at the 2001 and 2007 follow-ups was calculated and used in the analyses. To minimize the variation between different age groups across the two time points, the analyses were performed using the mean Cdist z-score from the 2001 and 2007 follow-ups.

4.6.2 Pulse wave velocity

A whole-body impedance cardiography device (CircMon; JR Medical, Ltd) was used to determine PWV in 2007. When the pulse pressure wave enters the aortic arch and the diameter of the aorta changes, the whole-body impedance decreases. The CircMon software measures the time difference between the onset of the decrease in the whole-body impedance signal and the distal plethysmographic signal from a popliteal artery at knee-joint level. PWV can be determined from the distance and time difference between the 2 recording sites. PWV values calculated using the whole-body impedance cardiogram (PWV_{ICG}) systematically overestimated the results of the Doppler method, but the PWV using the impedance technique with selective electrode configuration (PWV_{IS}) agreed well with the Doppler ultrasound method. The following regression equation was used to derive PWV_{IS} from PWV_{ICG}: PWV_{IS} =0.696xPWV_{ICG}+0.864. The repeatability values between two time periods separated by a 1-minute time interval were 0.88 ms^{-1} for PWV_{ICG} and 0.54 ms^{-1} for PWV_{IS}. Reproducibility values, calculated using measurements on different days, were 2.98 ms^{-1} for PWV_{ICG} and 2.42 ms^{-1} for PWV_{IS}; this represents the true physiological variability of the parameter (Kööbi et al., 2003). In this thesis, PWV_{ICG} is primarily used in the analyses and henceforth is called PWV.

4.6.3 Echocardiographic assessments of the heart: left ventricular mass and diastolic function

Echocardiographic examinations of cardiac structure were conducted according to American and European guidelines in 2011 (Lang et al., 2015; Maragiannis et al., 2015). Transthoracic echocardiography was performed with a high-resolution ultrasound system (Sequoia 512, Acuson, CA) using 3.5 MHz scanning frequency phased-array transducer. Both the sonographer and the observer were blinded to participant details. Standard echocardiographic examinations were produced from the parasternal long and short axis in 2-dimensional (2-D) and M-mode and apical 4-chamber views (Ruohonen et al., 2016). LVM in grams was calculated as: 0.8{1.04[([lef ventricular end-diastolic diameter + posterior wall thickness + septal wall thickness]^3 – left ventricular (LV) end-diastolic diameter^3)]} + 0.6 (Ruohonen et al., 2016). LVM was indexed to height at the allometric power of 2.7 (indexed

LVM = LVM/ height$^{2.7}$) (de Simone et al., 1992). Transmitral flow and tissue velocities were measured using continuous and pulsed-wave Doppler (Ruohonen et al., 2016). These measurements were used to define LV diastolic performance index, E/e′ ratio, where E stands for peak velocity of early diastolic transmitral flow and e' stands for peak velocity of early diastolic mitral annular motion.

4.7 Statistical methods

Continuous variables are expressed as mean ± SD and categorical variables as percentages unless stated otherwise. The values of serum TG were $\log_{10}$ transformed before analyses due to skewed distributions. The statistical tests were performed using SAS version 9.4 (SAS Institute) with statistical significance inferred at a 2-tailed P value < 0.05.

Study I

Differences between baseline characteristics of men and women were studied using age-adjusted linear regression and logistic regression. Associations of family SES in childhood with diet, alcohol consumption, physical activity and pack years of smoking in adulthood were studied using linear regression. To examine the association of childhood SES and current smoking status (smoker/non-smoker) in adulthood the risk ratios (RR) were examined using Poisson logistic regression. The analyses were adjusted for age and sex and additionally for participant's own SES in adulthood. Additional analyses were performed for diet variables adjusting for daily energy intake. To study the association between childhood SES and life-course levels of consumption of fruit, vegetable, fish, meat, and PAI, the study population was classified into 2 groups according to their SES status in childhood: Group 1; SES below median in childhood and Group 2; SES above median in childhood. The differences of mean values in these groups in 1980, 1983, 1986, 2007 and 2011, were studied using pairwise comparisons adjusted for age and sex. The consumption of fruit, vegetable, fish and meat was defined as mean frequency per week in 1980, 1983 and 1986, and amount per day (g/day) in 2007 and 2011. The prevalence of smoking across the life-course in childhood SES groups was examined using cross-tabulation. The differences in prevalence of smoking between SES-groups were analyzed adjusting for age and sex.

Study II

To examine differences between characteristics of men and women, age-adjusted linear regression for continuous outcomes and logistic regression for binary

outcomes was used. No significant sex differences, using interaction term of sex×SES, were detected indicating that the effect of childhood SES on MetS and T2DM was similar between men and women participants. Thus, sexes were analyzed combined. To study associations of family SES in childhood with MetS, IFG, T2DM, and components of MetS (categorical variable) in adulthood, the RRs were examined using Poisson regression. The analyses were adjusted successively for age and sex only; then by adding conventional childhood cardiometabolic risk factors (lipids, systolic blood pressure, insulin, and BMI, physical activity, and fruit and vegetable consumption); and then by adding adult SES. To examine the association of intergenerational mobility of SES on the prevalence of MetS in adulthood, the study population was classified into four groups according to SES status in childhood and adulthood as follows: group 1, SES under the median in both childhood and adulthood; group 2, SES above the median in childhood and below the median in adulthood; group 3, SES below the median in childhood and above the median in adulthood; and group 4, SES above the median in both childhood and adulthood. The prevalence of MetS in these SES groups as well as the RRs between the groups were studied using a log-binomial regression adjusted for age and sex. Multiple comparisons using Tukey-Kramer method adjusted for age and sex were used for every risk variable to compare mean values of risk factor levels in childhood and adulthood among these SES groups.

Study III

To examine differences between characteristics of men and women, age-adjusted linear regression was used. Linear regression was used to study associations of family SES in childhood with PWV and Cdist in adulthood. No significant sex differences, using interaction term of sex×SES, were detected indicating that the effect of childhood SES on PWV and Cdist was similar between men and women participants. Thus, sexes were analyzed combined. The models were adjusted successively for age and sex only, then by additionally including independent conventional cardiometabolic risk factors in childhood, and further for adult SES. The independent childhood predictors of PWV and Cdist in adulthood were determined using stepwise modeling. Variables in the initial stepwise multivariable models included childhood SES, LDL cholesterol, HDL cholesterol, triglycerides, insulin, systolic blood pressure, resting heart rate, BMI, frequency of vegetable and fruit consumption per week, and physical activity. Age and sex were forced into the models. In addition, the analyses were adjusted for conventional risk factors in adulthood. The effect of each risk factor on diluting the β coefficients for the association between childhood SES and Cdist and PWV was determined. The association between childhood SES and Cdist and PWV was also examined after

including parental smoking in the model. Furthermore, associations between SES and Cdist and PWV were studied among the subsample of adolescents aged 12 to 18 years at baseline. These analyses were adjusted for age, sex, childhood cardiovascular risk factors, and additionally for adolescent smoking status.

To study the association of intergenerational mobility of SES on PWV and Cdist in adulthood, the study population was classified into 4 groups according to their SES status in childhood and adulthood: group 1 (stable low), SES below median in childhood and in adulthood; group 2 (downwardly mobile), SES above median in childhood but below median in adulthood; group 3 (upwardly mobile), SES below median in childhood and above median in adulthood; and group 4 (stable high), SES above median in childhood and adulthood. The differences in means of PWV and Cdist in these SES groups were examined using multiple comparisons adjusted for age and sex. *P* values were controlled for multiple comparison using the Tukey–Kramer method.

Study IV

To study the associations of childhood cardiovascular risk factors with childhood family SES, age- and sex- adjusted linear regression and age- and sex-adjusted logistic regression was used. Associations of family SES in childhood with cardiac structure and function in adulthood were examined using linear regression. No significant age or sex differences were detected indicating that the effect of SES on LVM and E/e′ ratio was similar between different age groups and between men and women participants. Thus, age groups and sexes were analyzed combined. To examine age and sex differences in the association of SES with LVM and E/e′ ratio, interaction terms of age×SES and sex×SES were used. Pairwise comparisons of the SES groups were adjusted with the Tukey-Kramer method. The analyses were performed both unadjusted and adjusted with age, sex, and conventional cardiovascular risk factors (BMI, systolic blood pressure, smoking, LDL cholesterol, HDL cholesterol, and triglycerides) in childhood and adulthood.

5 Results

5.1 Characteristics of the participants

Table 4 shows characteristics of the study participants at baseline in 1980 according to the childhood family SES status. Low family SES indicates that the annual family income (converted into its present-day value) was ≤16 000 €, medium income >16 000 € to ≤ 35 000 € and high income >35 000 €. The mean age of participants was 10.8 years and participants with higher family SES tended to be younger at baseline. There were no significant differences in sex distribution between SES groups. The prevalence of parental smoking was higher among participants with low family SES. Systolic and diastolic blood pressure were both lowest among participants with high childhood SES. HDL cholesterol was lower and triglycerides higher among participants with low family SES compared with participants who had high family SES. No differences between participants across childhood SES groups were observed for smoking prevalence among adolescents, BMI, LDL cholesterol, or favorable emotional family environment score.

Table 5 shows characteristics of study participants in 2011 by sex. The mean age of participants was 41.9 years. The annual income was higher in men. Women had lower BMI and smaller waist circumference and the current smoking prevalence was lower in women. Additionally, both systolic and diastolic blood pressure were lower in women. Men had higher LDL cholesterol, triglyceride and fasting plasma glucose levels and lower HDL cholesterol.

Table 4. Characteristics of the Cardiovascular Risk in Young Finns Study participants at the baseline by childhood family socioeconomic status (SES).

	Childhood family SES, Mean (SD)			
Characteristics	Low (N=262)	Medium (N=779)	High (N=830)	P-value
Age at baseline	11.7 (5.0)	10.5 (5.0)	10.7(4.9)	0.003
Boys, No. (%)	115 (43.9)	362 (46.5)	379 (45.7)	0.83
Smokers, No. (%)*	42 (27.6)	76 (20.1)	95 (23.6)	0.93
Parental smoking, No (%)	175 (79.2)	540 (74.3)	571 (69.6)	0.002
BMI (kg/m^2)	18.4 (3.3)	17.9 (3.1)	17.8 (2.9)	0.32
Systolic blood pressure (mmHg)	115.3 (12.7)	112.5 (12.1)	112.0 (11.5)	0.03
Diastolic blood pressure (mmHg)	68.3 (10.0)	69.3 (9.3)	67.8 (9.3)	0.008
HDL cholesterol (mmol/l)	1.50 (0.30)	1.60 (0.30)	1.60 (0.30)	0.01
LDL cholesterol (mmol/l)	3.50 (0.80)	3.50 (0.90)	3.40 (0.80)	0.15
Triglycerides (mmol/l)	1.60 (0.69)	1.60 (0.69)	1.38 (0.69)	0.002
Favorable emotional family environment score$^{\pi}$	2.6 (1.0)	2.5 (0.9)	2.4 (0.9)	0.5

*Data collected only on participants aged 12 to 18 years (n=934). BMI=body mass index, HDL=high-density lipoprotein, LDL=low-density lipoprotein, SD=standard deviation, SES=socioeconomic status. P-values for differences between SES groups are adjusted for age and sex. $^{\pi}$Scale ranged from 1 to 8 (8 is highest).

Table 5. Characteristics of the Cardiovascular Risk in Young Finns Study participants in 2011 by sex.

	Sex, Mean (SD)			
Characteristics	Women (N=1001)	Men (N=804)	Both (N=1805)	P-value
Age	42.1 (5.0)	41.8 (5.1)	41.9 (5.0)	0.22
Annual income (€)*	6.6 (2.8)	8.4 (3.1)	7.4 (3.1)	<0.001
Smokers, No. (%)	130 (13.0)	136 (16.9)	266 (14.7)	0.02
BMI (kg/m2)	26.2 (5.5)	26.8 (4.4)	26.5 (5.1)	0.004
Waist (cm)	87.8 (14.0)	96.4 (12.7)	91.6 (14.1)	<0.001
Systolic blood pressure (mmHg)	115.5(13.6)	122.6(13.1)	119(14)	<0.001
Diastolic blood pressure (mmHg)	72.5 (9.5)	77.4 (10.9)	74.7(10.4)	<0.001
HDL cholesterol (mmol/l)	1.43(0.32)	1.22 (0.29)	1.34 (0.33)	<0.001
LDL cholesterol (mmol/l)	3.15 (0.75)	3.43 (0.90)	3.27 (0.83)	<0.001
Triglycerides (mmol/l)	1.08 (0.53)	1.40 (0.69)	1.22 (1.23)	<0.001
Glucose (mmol/l)	5.24 (0.86)	5.52 (0.72)	5.36 (0.81)	<0.001

*Ordinal variable of 2011 annual gross income ranging from 1 (<5000€) to 13 (>60,000€). P-values for differences between sexes are adjusted for age. BMI=body mass index, HDL=high-density lipoprotein, LDL=low-density lipoprotein, SD=standard deviation.

5.2 Childhood socioeconomic status and life-course health behaviors

In this thesis, health behaviors comprised diet, smoking, physical activity and alcohol consumption. The associations of life-course and adulthood behaviors with childhood SES were studied.

5.2.1 Diet

The associations of childhood family SES defined by annual family income with consumption of vegetable, fruit, meat and fish in adulthood in 2011 were studied (Model A and B, Table 6). Higher SES in childhood was associated with higher consumption of fish and lower consumption of meat in adulthood, after adjustment for age and sex. The association between higher childhood SES and lower meat consumption in adulthood remained even after adjustment for own adulthood SES. Childhood SES was not associated with fruit or vegetable consumption in adulthood. However, higher childhood SES was associated with higher diet score in 2007 indicative of healthier diet after adjustment for age, sex and participant's own SES in adulthood (β-value±standard error(SE), 0.10 ± 0.046; P=0.03).

The association of childhood SES with health behaviors in 2011 was studied using parental years of education as the marker of childhood SES (Model a and b, Table 6). In line with family income, higher parental years of education was associated with higher consumption of fish and lower consumption of meat after adjustment for age and sex. Similar to family income, no significant associations between childhood SES defined by parental education and vegetable and fruit consumption were observed. Although the association remained significant for fish consumption, associations between parental years of education and meat consumption was attenuated after adjustment for participant's own educational years in adulthood. In line with childhood family income, higher childhood SES defined by parental years of education was associated with higher diet score in 2007 and the association remained significant after adjustment for age, sex and participant's own years of education in adulthood (β-value±SE, 0.10±0.029; P=0.001).

Table 6. Associations of childhood SES (annual family income and parental years of education) on vegetable, fruit, meat and fish consumption in adulthood in 2011.

Diet (g/day)	β±SE	P-value	β±SE	P-value
	Childhood SES = annual family income			
	Model A (N=1675)		Model B (N=1514)	
Vegetable	2.9±2.3	0.21	-0.13±2.5	0.96
Fruit	-2.05±2.0	0.30	-3.8±2.0	0.06
Meat	-3.6 ±0.99	<0.001	-2.8±1.0	0.007
Fish	1.1±0.5	0.04	0.73±0.54	0.18
	Childhood SES = parental educational years			
	Model a (N=1710)		Model b (N=1420)	
Vegetable	2.1±1.3	0.11	-0.11±1.5	0.94
Fruit	0.77±1.1	0.48	-0.60±1.2	0.61
Meat	-2.5±0.55	<0.001	-1.1±0.64	0.09
Fish	1.2±0.28	<0.001	1.2±0.32	<0.001

Model A/a=adjusted for age and sex, Model B=additionally adjusted for participant's own income in adulthood, Model b=additionally adjusted for participant's own years of education. SE=standard error, SES= socioeconomic status. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

In addition, we analyzed the life-course levels of fruit, vegetable, fish and meat consumption in the childhood SES groups (above or below the median) (Figure 6). The consumption of meat differed between the groups throughout follow-up; in 1980, 1983 and 1986, meat consumption was higher among those in the high child SES group (above the median), but in 2007 and 2011 the consumption was higher among those in the low child SES group (below the median). The consumption of fruit was higher among those with high SES in 1980, 1983 and 1986 after which the difference disappeared. For vegetable consumption the differences between childhood SES groups were seen still in 2007, after which the difference was reduced and no longer statistically significant. No significant differences in consumption of fish across the life-course were observed between child SES groups (Figure 6).

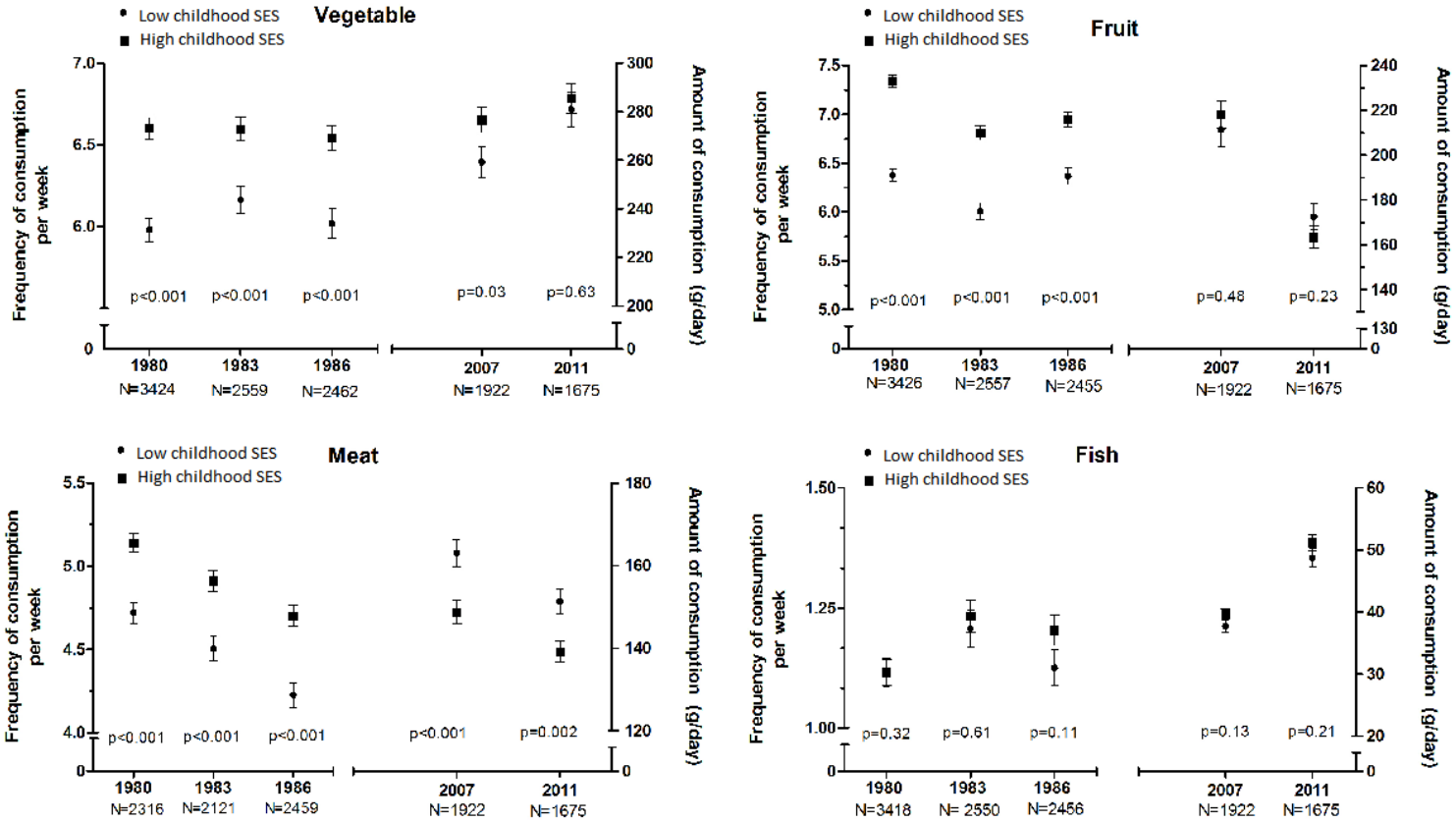

Figure 6. Life-course levels of consumption of vegetable (N=1675–3426), fruit (N=1675–3424), meat (N=1675–3418) and fish (N=1675–3432) according to child socioeconomic status (SES) groups (above and below median family income). P-values are for pairwise comparisons adjusted for age and sex. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

5.2.2 Smoking

Higher childhood SES defined by annual family income was associated with lower risk of being a current smoker in adulthood (Model A, Table 7). The association remained significant after additional adjustment for participant's own income in adulthood (Model B, Table 7). Among current and former smokers, childhood SES was inversely associated with pack years of smoking in adulthood but the association attenuated after additional adjustment for own SES in adulthood (Model A and B, Table 7).

When childhood SES was determined by parental years of education, the results were essentially similar to the results using annual family income. A higher number of parental educational years was associated with lower risk of being a current smoker, even after adjustment for participant's own educational years (Model a and b, Table 7). Additionally, childhood SES defined by parental years of education was inversely associated with pack years, but the difference was reduced and no longer statistically significant after adjustment for participant's own SES (Model a and b, Table 7).

In addition, when the frequency of daily smoking was compared between the high and low childhood SES groups, with the exception of smoking at baseline, there was a higher proportion of smokers in the low SES group throughout the life-course (Figure 7).

Table 7. Associations of childhood SES (annual family income and parental years of education) on risk of being a current smoker in adulthood and on pack years in adulthood.

	Childhood SES (= annual family income)			
	Model A		Model B	
Being a current smoker	RR, 95%CI	P-value	RR, 95%CI	P-value
	0.90, 0.85–0.95	<0.001	0.92, 0.87–0.98	0.006
Pack years	β±SE	P-value	β±SE	P-value
	-0.47±0.18	0.01	-0.31±0.18	0.09

	Childhood SES (= parental educational years)			
	Model a		Model b	
Being a current smoker	RR, 95%CI	P-value	RR, 95%CI	P-value
	0.92, 0.89–0.95	<0.001	0.96, 0.92–0.99	0.03
Pack years	β±SE	P-value	β±SE	P-value
	-0.32±0.10	0.002	-0.10±0.10	0.38

Model A/a=adjusted for age and sex, Model B=additionally adjusted for participant's own income in adulthood, Model b=additionally adjusted for participant's own educational years. N for being a current smoker in Model A: 1930, in Model B: 1897, in Model a: 1972, in Model b: 1952. N for pack years in Model A: 817, in Model B: 799, in Model a: 844, in Model b: 834. CI=confidence interval, SE=standard error, SES= socioeconomic status. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

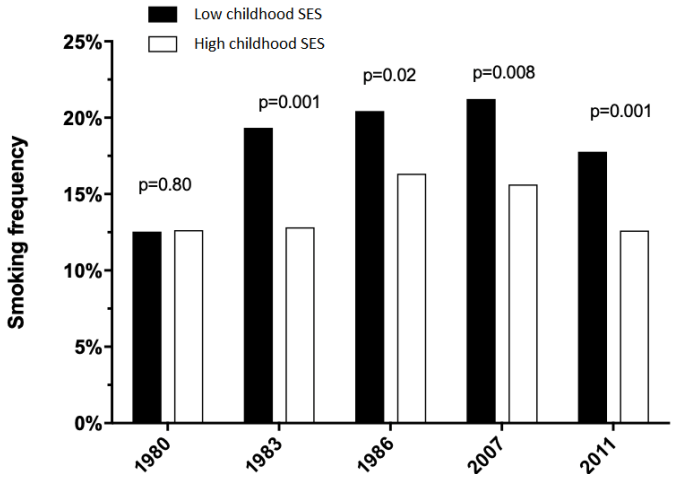

Figure 7. Smoking frequency throughout the life-course according to child socioeconomic status (SES) groups (above and below median) (N(1980) = 1604, N(1983) = 1720, N(1986) = 1923, N(2007) = 2143, N(2011) = 1930). P-values are adjusted for age and sex. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

5.2.3 Physical activity

Higher SES defined by family income in childhood was associated with higher PAI in adulthood in 2011 (Model A, Table 8). The association attenuated after additional adjustment for own annual income in adulthood (Model B, Table 8). The results were essentially similar when parental years of education was used as an indicator of childhood SES (Model a and b, Table 8). A higher number of parental years of education was associated with higher PAI, but the association was attenuated after additional adjustment for participant's own educational years.

When the life-course levels of PAI were compared between the high and low SES groups, the differences were significant, with higher childhood SES associated with higher PAI in 1980, 1983, 1986, 2007 and 2011 (Figure 8).

Table 8. Associations of childhood SES (annual family income and parental years of education) on physical activity index in adulthood.

	Childhood SES = annual family income	
	Model A (N=1847)	Model B (N=1820)
β±SE, P-value	0.059±0.023, 0.009	0.039±0.023, 0.09
	Childhood SES = parental educational years	
	Model a (N=1886)	Model b (N=1865)
β±SE, P-value	0.034±0.01, 0.006	0.017±0.013, 0.20

Model A/a= adjusted for age and sex, Model B=additionally adjusted for participant's own income in adulthood, Model b= additionally adjusted for participant's own educational years. SE= standard error, SES= socioeconomic status. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

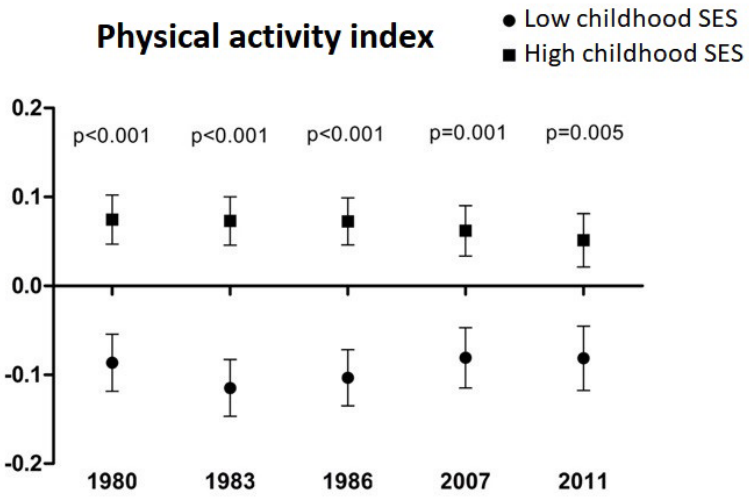

Figure 8. Life-course levels of physical activity index (PAI) (z-scored for age) according to child SES groups (above and below median) (N(1980)=2213, N(1983)=2261, N(1986)=2395, N(2007)=2088, N(2011)=1847). P-values are for pairwise comparisons adjusted for age and sex. SES= socioeconomic status. Reproduced from International Journal of Cardiology with permission of Elsevier (Study I).

5.2.4 Alcohol consumption

Childhood SES was not associated with alcohol consumption in adulthood defined by the number of drinks per day (β-value±SE, -0.002 ± 0.01; P=0.99).

5.3 Childhood socioeconomic status and clustering of risk factors

5.3.1 Components of metabolic syndrome

Childhood SES was shown to have an inverse association with all five components of MetS (Model 1, Table 9). The RR for a 1-unit increase in childhood SES varied from 0.94 to 0.96. Except for blood pressure and fasting plasma glucose, these associations persisted after adjustment for cardiovascular risk factors in childhood (Model 2, Table 9). When the associations were additionally adjusted for own SES in adulthood, only the associations between childhood SES and waist circumference and triglyceride levels remained statistically significant (Model 3, Table 9). After full adjustment for covariates, a 1-unit increase in childhood SES was associated with a 4% decrease in the risk of having a high waist circumference (at least 88cm for women and 102 cm for men) and a high triglyceride level (≥1.7mmol/l) in adulthood.

Table 9. Relative risk for components of MetS in adulthood according to family SES in childhood (N = 2250).

Adulthood variable	%	Model 1	P-value	Model 2	P-value	Model 3	P-value
High waist	34.8	0.96 (0.93-0.98)	0.002	0.95 (0.93-0.98)	0.001	0.96 (0.93-0.99)	0.003
High blood pressure	21.7	0.96 (0.92-1.00)	0.04	0.97 (0.94-1.01)	0.19	0.97 (0.94-1.01)	0.20
Low HDL cholesterol	34.0	0.95 (0.93-0.98)	0.0007	0.97 (0.93-1.00)	0.03	0.97 (0.94-1.00)	0.06
High triglycerides	20.6	0.94 (0.90-0.98)	0.002	0.95 (0.92-1.00)	0.03	0.96 (0.92-1.00)	0.04
High fasting plasma glucose	25.1	0.96. (0.92-0.99)	0.01	0.96 (0.93-1.00)	0.05	0.97 (0.94-1.01)	0.15

Components of MetS are defined according to the harmonized criteria (Alberti et al., 2009). Data are risk ratios (RR) (95% confidence intervals (CI)). Relative risks are for a 1-unit increase in family socioeconomic status (SES) in childhood. Model 1 was adjusted for age and sex. Model 2 included model 1 covariates plus childhood low low-density lipoprotein cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, systolic blood pressure, body mass index, insulin, frequency of fruit and vegetable consumption, and physical activity index. Model 3 included model 2 covariates plus participant's own SES in adulthood. Reproduced from Diabetes Care with permission of American Diabetes Association (Study II).

5.3.2 The risk of metabolic syndrome in adulthood

When the prevalence of adult MetS across the child SES groups was compared, the lowest prevalence was in group 8 (12.4%) and highest in group 2 (29.2%) (Figure 9). A 1-unit increase in childhood SES was associated with a 7% decrease in the risk of having MetS in adulthood (Model A, Table 10). The association was similar when childhood SES was defined by parental years of education, with a 3% decrease in risk for each additional year spent in education (Model a, Table 10). The association between childhood SES and risk of MetS remained after adjustment for cardiovascular risk factors in childhood (Model B, Table 10), and adult SES (Model C, Table 10). In the final model with full adjustment for covariates, the relative risk of having MetS in adulthood decreased by 5% for each 1-unit increase in childhood SES.

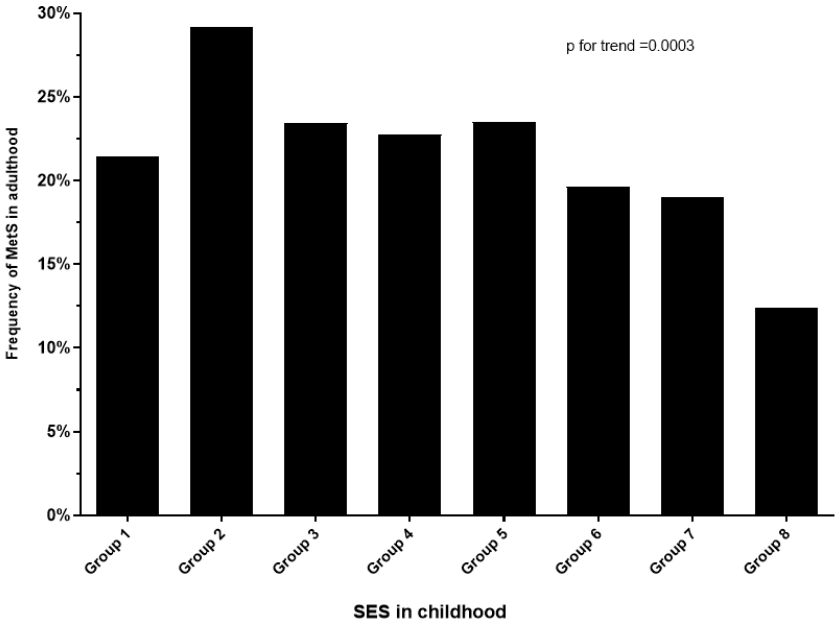

Figure 9. Prevalence of MetS according to child socioeconomic status (SES) groups (N = 2250). Groups of annual family income are ordered as 1 (low) to 8 (high). Reproduced from Diabetes Care with permission of American Diabetes Association (Study II).

Table 10. Relative risk of having MetS in adulthood according to the childhood SES.

Model A		Model a	
RR, 95%CI	P-value	RR, 95%CI	P-value
0.93, 0.90–0.97	0.0003	0.97, 0.95–0.99	0.01
Model B		Model C	
RR, 95%CI	P-value	RR, 95%CI	P-value
0.94, 0.91–0.99	0.003	0.95, 0.91–0.99	0.005

Childhood socioeconomic status (SES) is defined by annual family income in Model A, B and C and by parental years of education in Model a. Model A =adjusted for age and sex. Model a = similar adjustment as model A, but childhood SES is defined by parental years of education. Model B=additionally adjusted for low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, insulin, frequency of fruit and vegetable consumption, and physical activity index. Model C=additionally adjusted for participants' own SES in adulthood. RR=risk ratio, CI=confidence interval. (N=2250/N=2311(education)). Reproduced from Diabetes Care with permission of American Diabetes Association (Study II).

5.3.3 Intergenerational mobility in socioeconomic status and metabolic syndrome

To study the effect of intergenerational mobility on the prevalence of MetS, participants were divided into four groups according to their SES in childhood and

adulthood. When the prevalence of adult MetS was compared among these childhood-adulthood SES groups, the prevalence was lowest (mean (SE) 16.73%(1.28%)) in group 4 (high SES in both childhood and adulthood) compared with the other groups (Figure 10). The prevalence of group 4 was significantly lower compared with the other groups after adjustment for age and sex. Differences in risk factor levels among those in group 4 were apparent in both childhood and in adulthood (Table 11). Those in group 4 had lower BMI compared with those in group 2, and lower systolic blood pressure when compared with those in group 3. Those in group 4 also had higher HDL cholesterol compared with those in group 1 and lower triglycerides when compared with those in groups 1 and 3. Differences were not statistically significant for insulin levels. In adulthood, those in group 4 had lower BMI, lower waist circumference and lower systolic blood pressure than those in the other three groups. Additionally, group 4 had higher HDL cholesterol than groups 1 and 3 and lower triglyceride levels than group 1. Fasting plasma glucose levels were lower in group 4 compared with groups 1 and 2.

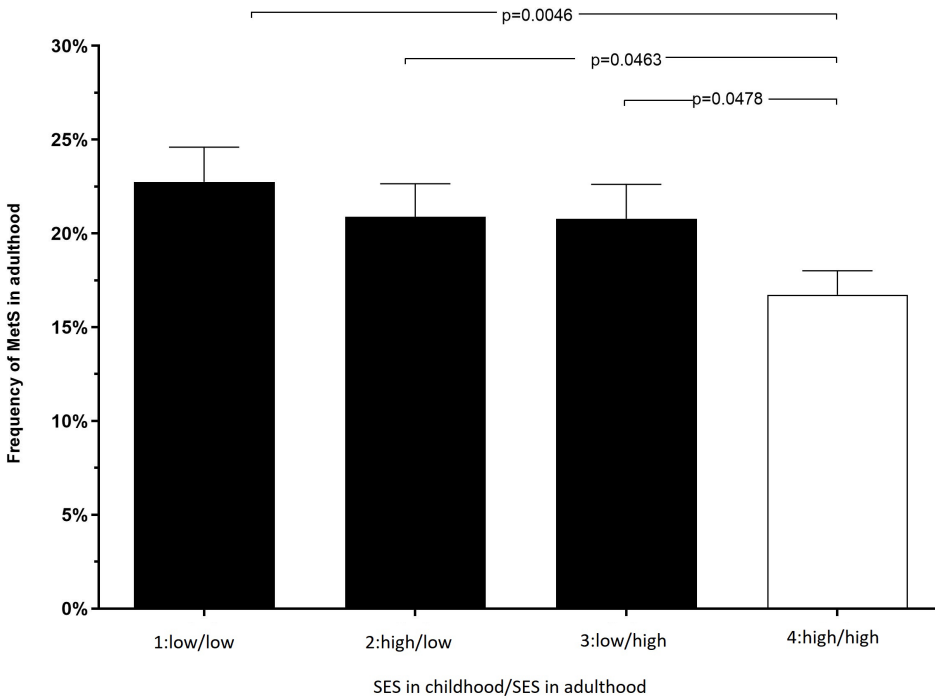

Figure 10. Prevalence of MetS according to childhood and adulthood socioeconomic status (SES) groups (N = 2250). Data are mean ± standard error (SE) adjusted for age and sex. Group 1, stable low (SES below median in childhood and adulthood); group 2, downwardly mobile (SES above median in childhood and below median in adulthood); group 3, upwardly mobile (SES below median in childhood and above median in adulthood); and group 4, stable high (SES above median in childhood and adulthood).

Table 11. Risk factors in childhood and in adulthood in various SES groups compared with group 4.

	Group 1: -/-	Group 2: +/-	Group 3: -/+	Group 4: +/+	P (compared with group 1)	P (compared with group 2)	P (compared with group 3)
Risk factors in childhood, mean (SE)							
BMI,kg/m2 (N=2233)	17.8(0.10)	18.0 (0.09)	17.8 (0.11)	17.6 (0.08)	0.46	0.007	0.62
Systolic BP, mmHg (N=2231)	112.2 (0.46)	112.52 (0.44)	113.35(0.50)	111.70 (0.37)	0.84	0.49	0.04
HDL cholesterol, mmol/L (N=2223)	1.53 (0.01)	1.57 (0.01)	1.54 (0.01)	1.58 (0.01)	0.03	0.89	0.13
Triglycerides, mmol/L (N=2225)	0.69 (0.01)	0.66 (0.01)	0.69 (0.01)	0.63 (0.01)	0.0008	0.34	0.006
Insulin, mU/L (N=2212)	9.61(0.22)	9.69 (0.22)	9.61 (0.24)	9.26 (0.18)	1.00	1.00	0.63
Risk factors in adulthood, mean (SE)							
BMI,kg/m2 (N=2248)	27.1 (0.22)	26.6 (0.22)	26.8 (0.24)	25.7 (0.18)	<0.0001	0.005	0.0007
Waist circumference, cm (N=2250)	93.0 (0.60)	92.4 (0.58)	92.2 (0.65)	89.9 (0.47)	0.0004	0.004	0.02
Systolic BP, mmHg (N=2250)	121,1 (0.60)	119.8 (0.59)	121.1 (0.65)	117.6 (0.48)	<0.0001	0.02	<0.0001
HDL cholesterol, mmol/L (N=2250)	1.28 (0.01)	1.31 (0.01)	1.30 (0.02)	1.35 (0.01)	0.0009	0.14	0.02
Triglycerides, mmol/L (N=2250)	1.43 (0.05)	1.41 (0.05)	1.43 (0.06)	1.25 (0.04)	0.04	0.07	0.06
Fasting plasma glucose, mmol/L (N=2250)	5.48 (0.03)	5.41 (0.03)	5.32 (0.04)	5.29 (0.03)	<0.0001	0.03	0.94

Group 1, stable low (SES below median in childhood and adulthood); group 2, downwardly mobile (SES above median in childhood and below median in adulthood); group 3, upwardly mobile (SES below median in childhood and above median in adulthood); and group 4, stable high (SES above median in childhood and adulthood). Means and P-values are adjusted for age and sex. BMI=body mass index, BP=blood pressure, HDL=high-density lipoprotein, SE=standard error, SES=socioeconomic status.

5.3.4 The risk of impaired fasting glucose and type 2 diabetes in adulthood

Childhood SES was inversely associated with the combined variable of glucose abnormalities (i.e., having either IFG or T2DM) (RR 0.96 [95% CI 0.92–0.99]; P=0.01, adjusted for age and sex). The association was attenuated after adjustment for conventional cardiometabolic risk factors in childhood (RR 0.97 [95% CI 0.93–1.00]; P=0.08). When T2DM was analyzed separately, no association was found between childhood SES and T2DM in adulthood (RR 0.92 [95% CI 0.82–1.02; P=0.10, adjusted for age and sex] and 0.95 [95% CI 0.85–1.06; P=0.34, adjusted for age, sex, and conventional cardiometabolic risk factors in childhood], respectively).

5.4 Childhood socioeconomic status and subclinical markers of cardiovascular disease in adulthood

5.4.1 Arterial stiffness

SES in childhood defined by annual income was directly associated with Cdist (Model A, Table 12) and inversely with PWV (Model a, Table 12) in adulthood, when adjusted for age and sex. The associations remained statistically significant after further adjustment for child cardiometabolic risk factors that were independently associated with Cdist or PWV (Model B and b, Table 12). Associations also remained after additional adjustment for participant SES in adulthood (Model C and c, Table 12). When parental years of education was used in place of family income as the indicator of childhood SES, the results were essentially similar for Cdist (*β*-value±SE, 0.015±0.0050; *P*=0.003, adjusted for age and sex, N=2542) and PWV *(β*-value±SE, -0.046±0.012 m/s; *P*<0.001, adjusted for age and sex, N=1792).

Table 12. Association of childhood SES with carotid artery distensibility and pulse wave velocity.

Carotid artery distensibility (z-scored)

Model	β±SE	P-value
A (N=2562)	0.033±0.0090	0.004
B (N=2526)	0.029±0.0089	0.001
C (N=2026)	0.026±0.010	0.01
D (N=2526)	0.031±0.010	0.01

Pulse wave velocity (m/s)

Model	β ±SE	P-value
a (N=1803)	-0.069±0.022	0.002
b (N=1771)	-0.062±0.022	0.006
c (N=1704)	-0.048±0.023	0.04
d (N=1771)	-0.068±0.025	0.006

β-values are for a 1-unit increase in family socioeconomic status (SES) in childhood. Model A/a: adjusted for age (3–18 y at baseline) and sex. Results for model B/b are from stepwise multivariable analysis, including childhood risk factors. Final model B included age, sex, systolic blood pressure and resting heart rate. Final model b included age, sex, body mass index, systolic blood pressure, and vegetable consumption. Initial model also included low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, insulin, frequency of fruit consumption per week and physical activity. Model C/c included model B/b covariates plus participants' own SES in adulthood. Model D/d included Model B/b covariates plus parental smoking in childhood. SE= standard error. Reproduced from Hypertension with permission of Wolters Kluwer Health, Inc. (Study III).

When the association was adjusted for conventional cardiometabolic risk factors in adulthood instead of those in childhood, the association was attenuated both for Cdist (β-value±SE, 0.014±0.008; P=0.10) and for PWV (β-value±SE, −0.019±0.02 m/s; P=0.38). Of these adulthood risk factors, systolic blood pressure had the strongest diluting effect on associations between childhood SES and both markers of arterial stiffness, attenuating the β-value of the association between childhood SES and Cdist by 44% (from 0.032 to 0.018) and the β-value of association between childhood SES and PWV by 54% (from -0.069 to -0.032 m/s). For the association of childhood SES with Cdist, two other risk factors with strong diluting effects were HDL cholesterol (β-value attenuated by 22%) and triglycerides (β-value attenuated by 11%). For the association between childhood SES and PWV, triglycerides (β-value attenuated by 18%) and plasma glucose (β-value attenuated by 10%) had the strongest diluting effects after systolic blood pressure.

Data on smoking was available only from adolescents aged 12 to 18 years at baseline (N=1282). In this subsample, the association between child SES and Cdist was not statistically significant when the analysis was adjusted for conventional cardiometabolic risk factors in childhood used in model B (β-value±SE, 0.020±0.013; P=0.13) or when smoking in adulthood was added to the model (β value±SE, 0.017±0.013; P=0.21). When PWV was used as the outcome, the observed effect for childhood SES remained in this subsample after adjustment for conventional cardiometabolic risk factors (β value±SE, −0.078±0.035 m/s; P=0.02) and when additionally adjusted for smoking (β value±SE, −0.079±0.036 m/s; P=0.03). When parental smoking was included in model B (which included several cardiometabolic risk factors in childhood) the results remained essentially similar for the association between childhood SES and Cdist (Model D, Table 12) and between childhood SES and PWV (Model d, Table 12).

Mean values of Cdist and PWV in adulthood were compared between the 4 childhood–adulthood SES groups (Figure 11, 12 and 13). Participants with high SES both in childhood and adulthood (stable high, group 4) had higher Cdist (mean±standard error of mean (SEM), 2.10 ±0.02 %/10mmHg) compared with participants in all other childhood–adulthood SES groups (Figure 11, other pairwise comparisons were not statistically significant). Participants with stable high SES (group 4) had significantly lower PWV (mean±SEM, 10.21±0.07 m/s) compared with those with stable low SES and those with downwardly mobile SES. Although participants with stable high SES had lower PWV than those in the upwardly mobile SES group, this difference was not statistically significant (Figure 12). The results concerning PWV are also presented using $PWV_{IS,}$ to simplify comparison to other studies using Doppler ultrasound methods in determining PWV (Figure 13).

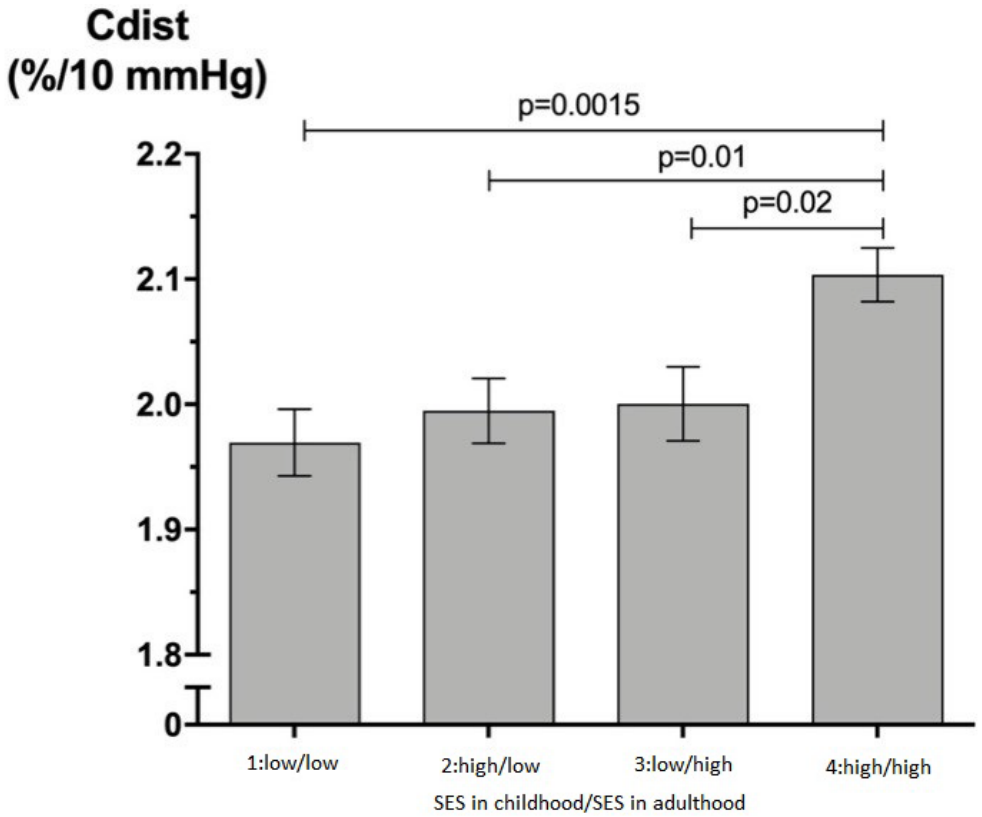

Figure 11. Mean ± standard error of mean (SEM) for carotid artery distensibility (Cdist) according to different childhood/adulthood socioeconomic status (SES) groups (N=2223). Group 1, stable low (N=502; SES below median in childhood and adulthood); group 2, downwardly mobile (N=535; SES above median in childhood and below median in adulthood); group 3, upwardly mobile (N=409; SES below median in childhood and above median in adulthood); and group 4, stable high (N=777; SES above median in childhood and adulthood). *P* values are shown for significant differences between the groups using multiple comparisons adjusted for age and sex. Reproduced from Hypertension with permission of Wolters Kluwer Health, Inc. (Study III).

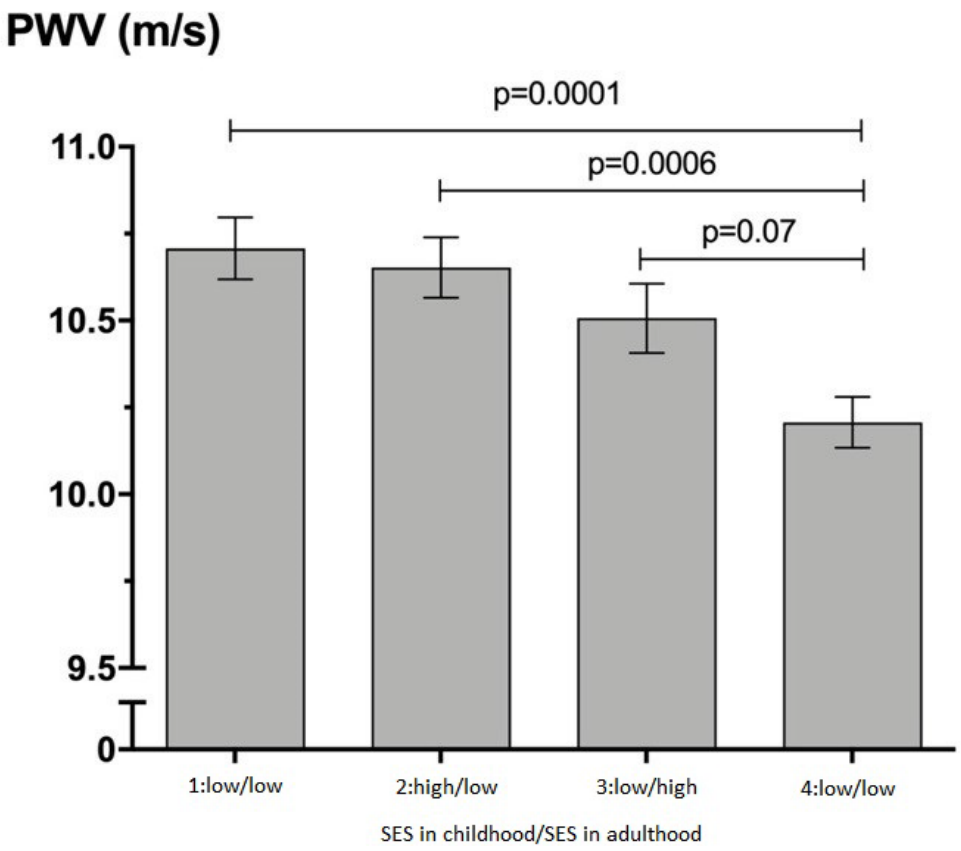

Figure 12. Mean ± standard error of mean (SEM) for pulse wave velocity (PWV) according to different childhood/adulthood socioeconomic status (SES) groups (N=1759). Group 1, stable low (N=405; SES below median in childhood and adulthood); group 2, downwardly mobile (N=430; SES above median in childhood and below median in adulthood); group 3, upwardly mobile (N=326; SES below median in childhood and above median in adulthood); and group 4, stable high (N=598; SES above median in childhood and adulthood). *P* values are shown for significant differences between the groups and additionally between difference of groups 3 and 4 using multiple comparisons adjusted for age and sex. Reproduced from Hypertension with permission of Wolters Kluwer Health, Inc. (Study III).

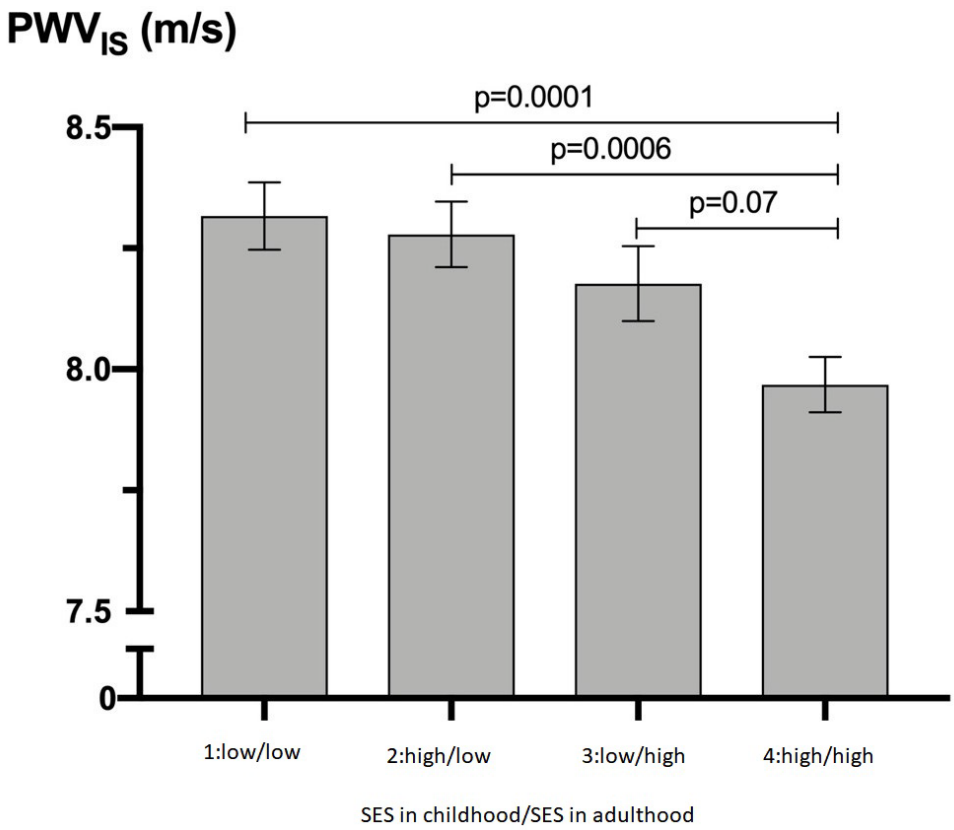

Figure 13. Mean ± standard error of mean (SEM) for pulse wave velocity with selective electrode configuration (PVW_{IS}) according to different childhood/adulthood socioeconomic status (SES) groups (N=1759). Group 1, stable low (N=405; SES under median in childhood and adulthood); group 2, downwardly mobile (N=430; SES above median in childhood and below median in adulthood); group 3, upwardly mobile (N=326; SES below median in childhood and above median in adulthood); and group 4, stable high (N=598; SES above median in childhood and adulthood). *P* values are shown for significant differences between the groups and additionally between difference of groups 3 and 4 using multiple comparisons adjusted for age and sex.

5.4.2 Echocardiographic measurements of the heart

Childhood SES defined by annual family income was inversely associated with LVM (Figure 14) and E/e' ratio (Figure 15). In analyses that adjusted for childhood risk factors, the associations between childhood SES and both LVM and E/e' ratio remained statistically significant and for LVM, the difference between low and high SES group increased (Model 2, Table 13). When the analyses were additionally adjusted for childhood emotional environment score, adulthood conventional risk factors and for SES in adulthood, associations remained significant but the difference between low and high SES group was slightly diminished, especially for LVM (Model 3, Table 13). In the subsample aged 12 to 18 years at baseline who had data on child smoking status, the results remained similar for LVM after adjustment for child smoking but the effect of childhood SES was no longer significant for E/e' (Model 4, Table 13).

In age- and sex-adjusted multivariable analysis, child BMI and LDL cholesterol were directly associated, and child favorable emotional family environment score and triglycerides inversely associated, with adult LVM (Table 14). Of childhood risk factors, only SES was associated with E/e' ratio in adulthood. Of adulthood risk factors, BMI was directly, and LDL cholesterol inversely, associated with adult LVM. Adult BMI and systolic blood pressure were directly associated with adult E/e' ratio. Adult SES was not associated with adult LVM index or E/e' ratio.

When parental occupation was used as an indicator of childhood SES the associations between childhood SES and LVM and E/e' ratio were similar to those using annual income as the indicator of SES (Table 15). Childhood SES was inversely associated with both LVM and E/e' after adjustment for age, sex, childhood and adulthood risk factors, and adulthood SES (Model 3, Table 15).

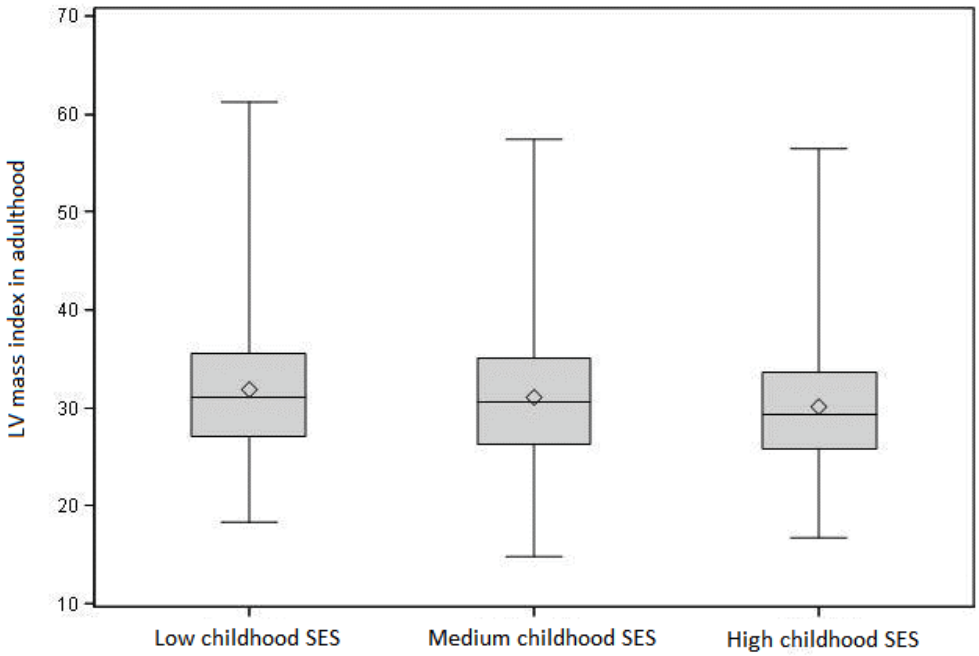

Figure 14. Adult left ventricular mass index (LVM/height$^{2.7}$) according to childhood family socioeconomic status (SES) groups (N = 1845). Age- and sex-adjusted *P* <0.001. The lowest point of the whiskers is the minimum and the highest point is the maximum value among each childhood SES group. The box is drawn from first quartile to third quartile with the horizontal line denoting the median and a diamond denoting the mean. Reproduced from JAMA Pediatrics with permission of the American Medical Association (Study IV).

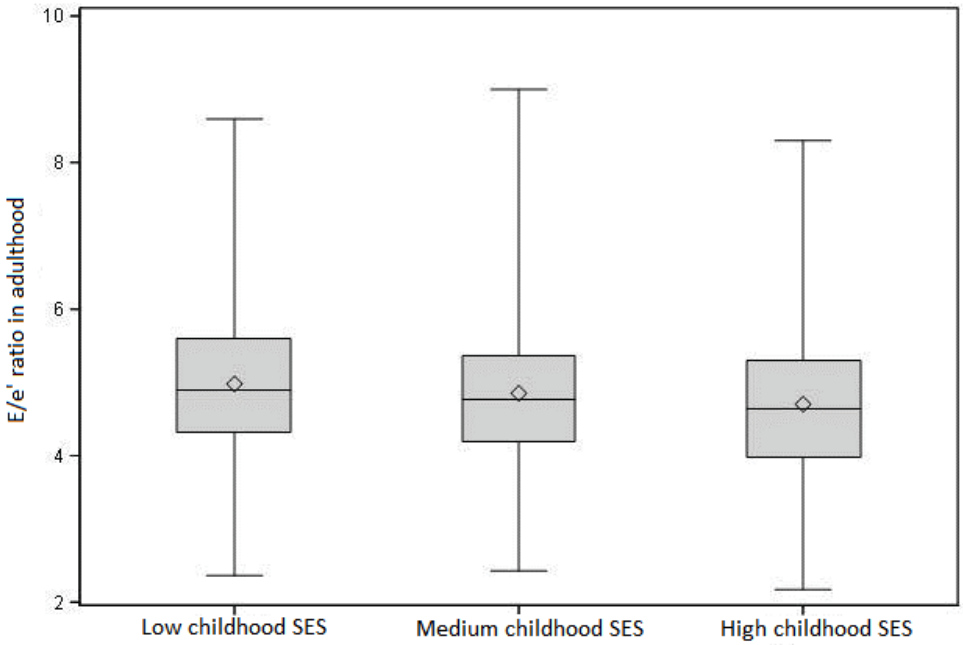

Figure 15. Adult E/e′ ratio according to childhood family socioeconomic status (SES) groups (N = 1871)**.** Age- and sex-adjusted *P* <0.001. The lowest point of the whiskers is the minimum and the highest point is the maximum value among each childhood SES group. The box is drawn from first quartile to third quartile with the horizontal line denoting the median and a diamond denoting the mean. Reproduced from JAMA Pediatrics with permission of the American Medical Association (Study IV).

Table 13. Association between family socioeconomic status (SES) in childhood with left ventricular mass (LVM) and diastolic function (E/e′ ratio) 31 years later in adulthood.

LVM (g/m$^{2.7}$)

	SES			SES difference (95% CI)			
Childhood SES Model	Low SES	Medium SES	High SES	Low vs High SES	Low vs Medium SES	Medium vs High SES	P-value
Model 1, mean (SD) (N=1845)	31.8 (6.7)	31.0 (6.6)	30.1 (6.4)	1.7 (0.6–2.8)	0.8 (-0.3–1.9)	0.9 (0.1–1.6)	0.001
Model 2, mean (N=1712)	32.4	31.1.	30.3	2.1 (1.0–3.3)	1.3 (0.2–2.4)	0.8 (0.1–1.6)	<0.001
Model 3, mean (N=1214)	32.1	30.8	30.6.	1.5 (0.2–2.8)	1.3 (0–2.6)	0.2 (-0.6–1.0)	0.03
Model 4, mean (N=588)	34.5	31.8	31.6	2.8 (0.8–4.9)	2.7 (0.6–4.7)	0.2 (−1.1–1.6)	0.004

E/e' Ratio

	SES			SES difference (95% CI)			
Childhood SES Model	Low SES	Medium SES	High SES	Low vs High SES	Low vs Medium SES	Medium vs High SES	P-value
Model 1, mean (SD) (N=1871)	5.0 (1.0)	4.9 (1.0)	4.7 (1.0)	0.3 (0.1–0.4)	0.1 (0–0.3)	0.2 (0–0.3)	<0.001
Model 2, mean (N=1733)	4.9	4.8	4.7	0.2 (0.1–0.4)	0.1 (-0.1–0.3)	0.1 (0–0.03)	0.001
Model 3, mean (N=1222)	4.9	4.8	4.7	0.2 (0–0.5)	0.1 (-0.1–0.4)	0.2 (0–0.3)	0.02
Model 4, mean (N=593)	5.1	4.9	4.8	0.3 (-0.1–0.6)	0.2 (-0.2–0.5)	0.1 (-0.1–0.3)	0.18

Model 1 was unadjusted. Model 2 included age, sex, and childhood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, and parental smoking). Model 3 included age, sex, childhood risk factors as in adjusted model 2, childhood favorable emotional family environment score and adulthood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, smoking, glucose, and participants' own SES [annual income] in adulthood). Model 4 additionally included participants' own smoking (aged 12–18 years). SD= standard deviation, CI= confidence interval. Reproduced from JAMA Pediatrics with permission of the American Medical Association (Study IV).

Table 14. Multivariable age- and sex-adjusted associations between cardiovascular risk factors in childhood and adulthood and adulthood left ventricular mass (LVM) and diastolic function (E/e′ ratio).

	LV mass (g/m2.7) (N=1214)		E/e′ ratio (N=1222)	
Childhood risk factors	β (95% CI)	P-value	β (95% CI)	P-value
Family SES in childhood *	-0.5 (-1.0 to -0.1)	0.03	-0.12 (-0.20 to -0.03)	0.02
Childhood BMI (kg/m2)	0.2 (0.0 to 0.4)	0.03	0.00 (-0.03 to 0.03)	0.92
Childhood systolic BP (mmHg)	-0.0 (-0.0 to 0.0)	0.39	-0.00 (-0.01 to 0.01)	0.92
Childhood HDL cholesterol (mmol/L)	-1.0 (-2.3 to 0.2)	0.11	0.09 (-0.13 to 0.30)	0.43
Childhood LDL cholesterol (mmol/L)	0.7 (0.3 to 1.2)	0.002	-0.01 (-0.08 to 0.07)	0.88
Childhood triglycerides (mmol/L)	-1.4 (-2.6 to -0.2)	0.02	0.05 (-0.16 to 0.25)	0.65
Parental smoking in childhood (no vs. yes)	-0.1 (-0.8 to 0.6)	0.78	0.02 (-0.10 to 0.14)	0.72
Favorable emotional family environment score ‡	-0.4 (-0.8 to -0.1)	0.02	0.02 (-0.04 to 0.08)	0.51
Adulthood risk factors				
Adulthood BMI (kg/m2)	0.6 (0.5 to 0.7)	<0.001	0.02 (0.00 to 0.03)	0.01
Adulthood systolic BP (mmHg)	0.0 (-0.0 to 0.1)	0.05	0.01 (0.01 to 0.02)	<0.001
Adulthood HDL cholesterol (mmol/L)	0.5 (-0.7 to 1.8)	0.79	0.03 (-0.18 to 0.24)	0.79
Adulthood LDL cholesterol (mmol/L)	-0.6 (-1.1 to -0.2)	0.01	0.01 (-0.07 to 0.09)	0.77
Adulthood triglycerides (mmol/L)	-0.3 (-0.9 to 0.4)	0.41	-0.01 (-0.12 to 0.10)	0.85
Adulthood plasma glucose (mmol/L)	-0.4 (-0.8 to 0.1)	0.09	-0.02 (-0.10 to 0.05)	0.54
Adulthood smoking (yes vs. no)	-0.7 (-1.5 to 0.1)	0.09	-0.10 (-0.23 to 0.03)	0.14
Adulthood SES ¥	0.0 (-0.1 to 0.1)	0.44	-0.01 (-0.03 to 0.01)	0.23
Age (years)	0.1 (0.0 to 0.2)	0.004	0.02 (0.01 to 0.04)	0.006
Sex (female vs. male)	3.0 (2.2 to 3.7)	<0.001	-0.49 (-0.63 to -0.36)	<0.001

Estimates (β) and 95% confidence intervals (CI) are for 1-unit change in the covariate. *Indicator of family socioeconomic status (SES) was family annual income: low=1, medium=2, high=3.‡ Scale range was 0 to 4 (4 is highest). ¥ Scale range was 1 to 8 (8 is highest). Reproduced from JAMA Pediatrics with permission of the American Medical Association (Study IV).

Table 15. Association between family socioeconomic status (SES; parental occupational status) in childhood with left ventricular mass (LVM) and diastolic function (E/e′ Ratio) 31 years later in adulthood.

	LVM ($g/m^{2.7}$)		E/e' ratio	
	β (95% CI)	P-value	β (95% CI)	P-value
Model 1 (N=1878/1906)	-0.6 (-0.9 to -0.4)	<0.001	-0.10 (-0.14 to -0.06)	<0.001
Model 2 (N=1739/1763)	-0.6(-0.8 to -0.3)	<0.001	-0.08 (-0.11 to -0.04)	<0.001
Model 3 (N=1235/1245)	-0.3 (-0.6 to 0.0)	0.03	-0.05 (-0.10 to -0.01)	0.03

Estimates (β) and 95% confidence intervals (CI) are for a 1-unit change in the covariate. Indicator of family SES was parental occupational status, classified from 1 to 5 (5 is highest). Model 1 was unadjusted. Model 2 included age (3–18 years at baseline), sex, and childhood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, and parental smoking). Model 3 included age (3-18 years at baseline), sex, childhood risk factors as in adjusted model 2, childhood favorable emotional family environment score and adulthood risk factors (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides, systolic blood pressure, body mass index, smoking, glucose and participants own SES (annual income) in adulthood). Reproduced from JAMA Pediatrics with permission of the American Medical Association (Study IV).

6 Discussion

The research performed as part of this thesis showed that higher childhood SES was associated with more healthy adulthood lifestyle in terms of diet, physical activity and smoking (Study I) and that childhood SES groups associated with differences in lifestyle factors across the life-course. Additionally, higher childhood SES was associated with lower risk for MetS and glucose abnormalities over 30 years later in adulthood (Study II). Higher SES in childhood was associated with lower arterial stiffness, better diastolic function and lower LVM in adulthood, indicating a more favorable CVD profile (Study III, Study IV). The effect of child SES was partly independent of conventional risk factors and of adulthood SES, but the results also indicated the importance of stable high SES from childhood to adulthood to minimize the risk of MetS and arterial stiffness in adulthood. Figure 16 provides a pictorial overview of the main findings from this thesis.

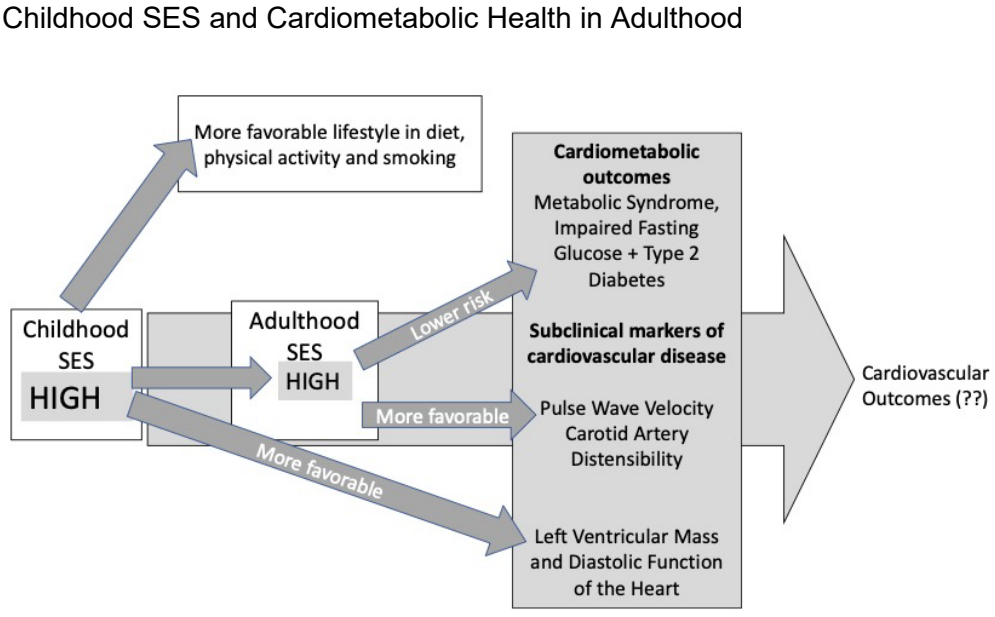

Figure 16. Overview of the main findings from this thesis.

6.1 Participants

The participants in this thesis were from the on-going epidemiological study of CVD risk factors, the Young Finns Study. The original sampling in 1980 consisted of 4320 participants aged 3, 6, 9, 12, 15 and 18 years who were invited to the cross-sectional survey. Children and adolescents were randomly selected from different parts of the country, equally representing both sexes. In addition, participants were selected equally from eastern and western parts and from rural and urban areas of Finland. The aim was to select a cohort representing Finnish children and adolescents. A total of 3596 individuals (49% boys/51% girls, 83% of those invited) participated in the study in 1980 and the sample was concluded to be representative of the total random sample (Åkerblom et al., 1985).

Of those that participated at baseline in 1980, 2283 (63%) also participated in 2001, 2204 (61%) participated in 2007, and 2063 (57%, 45% men) participated in 2011. In longitudinal studies with extensive study periods, like the Young Finns Study, it is inevitable that loss to follow-up occurs. However, participation of those in the study across the follow-ups has been dynamic (non-participants at one follow-up tend to re-participate at subsequent follow-ups) and it has been shown that baseline risk factor levels were similar among participants and non-participants (Nuotio et al., 2014). Finally, as the study group was racially homogenous, the results of the study may not be generalizable to other populations.

6.2 Methods

6.2.1 Assessing health metrics

The methods used in this study to measure health factors; weight, height, serum lipids, plasma glucose and blood pressure, are well-standardized and can be generalized from study to study. Questionnaires were used to measure dietary intake, alcohol consumption, leisure-time physical activity, smoking and favorable emotional family environment, which is a suitable method to obtain data about health behaviors and environmental factors (Aday et al., 2006). Although self-report questionnaires are linked with recall bias, this tends to be more of a problem in questionnaires that ask participants to retrospectively report behaviors or exposures (Althubaiti, 2016). Additionally, answers might be influenced by external factors related to social desirability. The social desirability bias means that people might have the tendency to overstate “healthy habits” like exercising and understate “detrimental habits” like smoking. Self-report bias is also recognized in questionnaires of alcohol consumption and the uneven distribution of self-report bias by SES is suggested to lead to misestimating socioeconomic differences in alcohol

consumption (Devaux et al., 2016). In the smoking data of the Young Finns Study, a further limitation is that data on participant's smoking habits was reported beginning from age 12 years. Nevertheless, questionnaires are widely used in epidemiological studies as they provide a feasible and cost-effective means to measure health behaviors.

Information on dietary habits was obtained with the FFQ. In 1980, 1983 and 1986, information on dietary habits was obtained with the non-quantitative FFQ, which meant a precise estimate of food consumption, energy and nutrient intake could not be estimated at these time-points. In 2007 and 2011, the more detailed quantitative FFQ was used providing an estimate of food consumption during the last 12 months in grams per day. The quantitative FFQ also provided an estimate of energy intake, which allowed adjustment of food behaviors by energy intake to separate e.g. those just generally eating more from those having higher intake of particular food components.

Another limitation is that the food selection of FFQs was limited, especially in childhood years 1980,1983 and 1986. However, the validity of this FFQ is shown in epidemiological studies on Finnish population (Paalanen et al., 2006). To complement the longitudinal dietary data, we also used a diet score calculated from the FFQ data obtained in 2007, higher scores representing healthier diets (Nettleton et al., 2013).

Physical activity was assessed as PAI using participant (or for young children, parent) responses to a self-report questionnaire. The criterion and predictive validity of the PAI has been tested. The PAI significantly correlated with indicators of exercise capacity in a cycle ergometer test among a subsample of 102 Young Finns Study participants (Telama et al., 2005). The correlations of PAI in 2011 with pedometer step counts in a subsample of the Young Finns Study were shown to be moderate (Kari et al., 2015). The possible explanation is that the estimated physical activity by PAI includes also cycling, swimming, skiing and fitness club activities and reflects mainly leisure-time physical activity, whereas pedometer detects the overall daily physical activity but is not sensitive to non-ambulatory activities. Additionally, an inverse correlation between PAI and waist circumference has been shown in both sexes (Telama et al., 2005; Yang et al., 2006).

Furthermore, to measure favorable emotional environment, a score consisting of 4 components was used: absence of previously diagnosed parental mental disorder, high parental caregiving nurturance, high parental life satisfaction, and reasonable alcohol use. The emotional environment is proposed to be an important part of the childhood psychosocial environment and associate with the risk of future chronic disease (Pulkki-Råback et al., 2015; Repetti et al., 2002).

6.2.2 Indicators of socioeconomic status

In the present thesis, childhood SES was mainly determined by annual family income. Similarly, participants' own SES in adulthood was defined by gross annual income. Of the conventionally used indicators of SES, the economic indicators (family income and wealth) are shown to be the most optimal ones for health research (Duncan et al., 2002). However, some limitations in the classifications of SES by income are noteworthy. For example, unemployment, poor health, and other life circumstances may have affected the family's income and subsequent classification of SES. Additionally, the data on SES was assessed by self-completed questionnaires, which might relate to certain biases (discussed in more details in previous chapter). However, we additionally made sensitivity analyses using parental educational or occupational levels as indicators of childhood SES and the results were in line with the main results. Since these three different perspectives of SES are the most commonly used indicators in epidemiological studies, using them as measurements of SES in this thesis provides a wide and reliable interpretation of the role of childhood SES (Kaplan et al., 1993).

To describe parental SES and, especially parental occupation, epidemiological studies have conventionally used status of the father as a surrogate for parental SES (Osler et al., 2003; Smith et al., 2001). Although there is no consensus on the superiority of using either mother's or father's SES, some studies have shown stronger associations for father's SES compared with mother's SES on the adulthood cardiovascular risk factor profile (Ball et al., 2006; Kvaavik et al., 2012). However, researchers of a Norwegian study proposed that the weaker association observed for mother's SES on adulthood cardiovascular risk factors might relate to a lower general level of education among women in the sample (Kvaavik et al., 2012). With the contemporary push toward gender equality, the importance of mother's SES in predicting offspring's adulthood health might therefore be increasing. Consequently, a growing number of studies have used higher SES of the two parents to define parental SES (Boylan et al., 2018; Poulton et al., 2002; Thurston et al., 2009). Finland has been a pioneer in gender equality, exemplified by the proportion of Finnish women passing the matriculation examination exceeding that of Finnish men already in 1947 (Statistics Finland, 2007). Therefore, in this thesis, to define parental occupation and education, the higher status of the two parents was used in the analyses.

In Finland, family income is shown to be important in shaping the future income of children (Pekkala et al., 2007). Therefore, we also used childhood-adulthood-SES groups to investigate the association of intergenerational mobility in SES with studied outcomes. The Finnish 1980-1982 birth cohorts showed, on average, a 10 percentile increase in parents' income rank was associated with a 2.2 percentile increase in a child's income rank, which is lower than in the USA but slightly higher

than in the Scandinavian countries, suggesting the effect of parent income on the income of their offspring might be lower in Finland than in the USA (Chetty et al., 2014; Suoniemi, 2017). However, the rates of SES mobility depend on how mobility is defined and how the various groups of mobility are formed and therefore the rates of mobility in the present study cannot be fully compared with those from other cohorts.

6.2.3 Arterial stiffness and echocardiographic assessments of the heart

To assess arterial stiffness, two different indicators of arterial stiffness, Cdist and PWV were used. Cdist measures the ability of the arteries to expand as a response to pulse pressure. In this thesis, pulse pressure was determined by measuring the difference between systolic and diastolic blood pressure from the brachial artery. Measuring pulse pressure from the brachial artery, and not from the artery in question (central artery), is a possible limitation, since the use of brachial pulse pressure can overestimate the pulse pressure in central arteries due to physiological amplification of pulse pressure (Karamanoglu et al., 1993). However, a previous study has shown an excellent correlation between blood pressures measured invasively from the ascending aorta and noninvasively from the brachial artery (Borow et al., 1982). Additionally, a study using methods, which were similar to those used in this thesis to determine Cdist, showed that when estimated central pulse pressures were used in lieu of brachial blood pressure measurements to determine modified Cdist, the results remained essentially similar (Mikola et al., 2015). Furthermore, the techniques used in this thesis to determine pulse pressure and Cdist are similar to techniques used in conventional cardiovascular research (Aggoun et al., 2000; Tounian et al., 2001; Van Popele et al., 2001).

Because accurate measurement of Cdist requires the assessment of several variables, the between-visit coefficient of variation of Cdist was somewhat high, but was comparable to others (Arnett et al., 1999). However, only small variations in carotid artery diameter was observed, which suggests that much of the long-term variation in Cdist is caused by physiological fluctuation and not by measurement error (Juonala et al., 2005).

Measurements of PWV were made using CircMon software, which uses the time difference between the decrease in the whole-body impedance signal and the distal plethysmographic signal from a popliteal artery at the level of the knee-joint. Therefore, the majority of the path includes muscular arteries, which are modifiable by neurogenic and geometric factors, which might affect the interpretation of the results. Additionally, aortic PWV is considered to associate more strongly with cardiovascular risk than peripheral PWV (Mitchell et al., 2010). However, some

studies have suggested that the deleterious effects of cardiovascular risk factors on local arterial stiffness are more pronounced in the peripheral than in the central arteries (Ferreira et al., 2005; Schram et al., 2004). Furthermore, the longer pathway from the aorta to the popliteal artery could provide a more global index of vascular health.

Transthoracic 2-D echocardiography was used in this thesis to measure ventricular mass and volumes. When compared to the ECG, the sensitivity of echocardiography is considerably higher (Levy et al., 1990). However, compared with 2-D echocardiography, magnetic resonance imaging and real-time 3-dimensional echocardiography are more accurate and reliable for measuring LVM (Grothues et al., 2002; Kühl et al., 2000). Despite this, current guidelines of the American Society of Echocardiography and European Association of Cardiovascular Imaging Chamber Quantification Guidelines recommend 2-D guided M-mode or direct 2D to be the best methods to screen for LVH (Lang et al., 2015; Marwick et al., 2015). In addition, most of the studies concerning the predictive value of LVM for the risk of CVD are made using 2-D techniques (Gardin et al., 2001; Levy et al., 1989).

Since body size is a strong contributor to LVM, to allow the comparison between individuals with different body sizes, different allometric variables are used to index LVM. These include body surface area (BSA), height and body weight (de Simone et al., 1992). However, the use of BSA is suggested to diminish the effect of obesity on LVM and underestimate the prevalence of LVM related to obesity (Cuspidi et al., 2009). In addition, height raised to the allometric power of 2.7 is shown to be superior in predicting cardiovascular outcomes in comparison with BSA or height alone (de Simone et al., 1992, 2005). Therefore, in this thesis, linear measurements from M-mode images were used to determine LVM that was indexed to height at the allometric power of 2.7.

To determine LV diastolic performance, echocardiography with doppler was used to measure the E/e'-ratio that describes the peak velocity of blood flow through the mitral valve during early diastole (E) standardized by mitral annular early diastolic velocity (e'). There are several studies showing correlation of E/e' ratio to invasively measured increase in LV filling pressure (Ommen et al., 2000; Ritzema et al., 2011). Furthermore, when compared to several other ultrasound markers conventionally used to distinguish diastolic dysfunction, E/e' is a better predictor of cardiac outcomes in hypertensive patients without recognized cardiac disease (Sharp et al., 2010).

6.3 Results

6.3.1 Health behaviors

Generally, health behaviors are patterned by SES and they account for a substantial portion of the socioeconomic gradient in cardiovascular health (Petrovic et al., 2018). In this thesis, health behaviors comprised diet, smoking, alcohol consumption and physical activity. These health behaviors are shown to mediate the pathway between childhood SES and adult health (Cohen et al., 2010; Petrovic et al., 2018; Van De Mheen et al., 1998). Because health behaviors are modifiable risk factors of CVD, and account for more than 90% of the risk of myocardial infarction (Yusuf et al., 2004), identifying and investigating associations between childhood SES and health behaviors is important to promote effective prevention of CVD.

Diet

Dietary behaviors are an important determinant of cardiometabolic disease. When the AHA released its seven health metrics concerning cardiovascular health, diet was one metric and four other metrics were closely related to diet (Lloyd-Jones et al., 2010). In this thesis, the association of childhood SES with diet was examined using consumption of meat, fish, fruit and vegetables. The dietary guidelines recommend that a cardiometabolic healthy diet be rich in vegetables, fruit and fish and that red and processed meat be consumed in low amounts (U.S. Department of Health and Human Services et al., 2015; Valtion ravitsemusneuvottelukunta, 2018). SES is an important determinant of dietary choices. Longitudinal data have shown that the socioeconomic gap in diet quality was apparent over a 20-year period, starting from the 1980s (Sijtsma et al., 2012). Additionally, socioeconomic differences exist at the food group level and in index foods, which are highly correlated with cardiovascular health (Darmon et al., 2008).

Previous studies have shown that lower SES is associated with higher consumption of fatty, red and processed meats whereas those with higher SES consume more lean meats, fish and other seafood (Maguire et al., 2015; Shimakawa et al., 1994; Van Rossum et al., 2000). In line with these findings, data from the FINDIET study has shown the intake of red and processed meat has been highest among those with the lowest levels of education during the whole 21st century in Finland (Raulio et al., 2016). In contrast, another Finnish study using data collected as part of nationally representative surveys from 1985 to 2012 indicated that meat consumption of the highest income group had diminished, but was still higher in comparison with lower income groups in 2012 (Lindblom, 2017). However, in the Lindblom study the category of meat included all fresh and processed meat

excluding fish, whereas in the FINDIET study only red and processed meats were included in their meat category. In line with the FINDIET study, poultry was excluded from the meat category in this thesis. In the 1980, 1983 and 1986 study years, poultry consumption was not separately assessed, but since the consumption of poultry was only marginal in Finland in the 1980s, this is unlikely to influence the results (Statistics Finland, 2018a).

Participants with SES above the median in childhood had higher consumption of meat across the three 1980s timepoints but lower consumption in 2007 and 2011 compared with those having SES below the median. In line with these findings, higher childhood SES, defined by annual family income, was associated with lower consumption of meat in adulthood in 2011, after adjustment for age and sex. The association remained after further adjustment for adult SES. However, in sensitivity analyses that used child SES defined by parental years of education, the inverse association between childhood SES and meat consumption was significant after adjustment for age and sex, but the association was attenuated after adjustment for SES in adulthood.

According to Statistics Finland, the overall consumption of meat in the country has increased from 58.9 kg per person in 1980 to 80.8 kg per person in 2017 (Statistics Finland, 2018a). Although the consumption of beef and pork in whole meat, sausages and cold cuts has decreased, consumption of processed meat has increased (Aalto, 2018). During this same time period, consumption of fish has nearly doubled and the consumption of poultry has increased from 3.0 kg per person in 1980 to 24.9 kg per person in 2017 (Statistics Finland, 2018a). These trends in consumption of meat have occurred with the real prices of both red meat and fish increasing during the last decades whereas the price of chicken has decreased (Statistics Finland, 2019b). Trends of socioeconomic differences in consumption of red meat, poultry and fish do not directly parallel with the price development, indicating it is not just the price but also cultural attitudes and values that have strong effects on food consumption patterns in different SES groups. In line, an analysis of the National FINRISK Study 2007 indicated that these values tend to differ between different SES groups, since families with low SES valued price and familiarity in their diet choices, whereas healthiness was more valued in the diet choices among people of high SES (Konttinen et al., 2013).

Results from this thesis indicate that childhood SES has a direct effect on meat consumption in adulthood, independent of adult SES. At the time when the Young Finns Study participants were children, red meat was potentially inaccessible to families with lower incomes and might have symbolized luxury and wealth among those of higher SES. Data from 120 countries collected from 1970–2007 has shown an inverted U-shaped relationship between income and meat consumption (Vranken et al., 2014). These data would suggest that as the mean level of gross income among

Finnish families has grown, meat consumption among those of higher SES might have diminished. Furthermore, it can be assumed that as red meat has become more accessible to the wider population, those having higher SES and accessibility to red meat in childhood might not consider it a luxury item in adulthood, which together with growing general knowledge on the harms of red meat consumption, might affect their meat consumption in adulthood. Contrary, people with lower childhood SES who went without meat in adulthood and who now, with improved availability, have better opportunities to consume meat, might be at higher risk to ignore the national diet recommendations and information on the harms related to consumption of red and processed meats, independent of their current (adulthood) SES.

On SES differences in fish consumption, the results presented in this thesis parallel those of other studies that together suggest that those who have higher SES consume higher amounts of fish (Galobardes et al., 2001; Maguire et al., 2015; Raulio et al., 2016; Shimakawa et al., 1994). In contrast to meat consumption, the association for fish consumption observed in this thesis was not significant after adjustment for adulthood SES, indicating that the effects of child SES might operate through adult SES. However, when parental years of education was used as an indicator of adult SES instead of family income, the effect of childhood SES on fish consumption in 2011 remained after adjustment for adult SES. Thus, the results indicate that having a higher childhood SES might positively impact on consumption of fish and meat, and that there are potentially unique pathways linking different types of child SES to long-term dietary choice. Similarly, a Norwegian study showed that education was directly associated with the frequency of seafood consumption whereas income was not (Myrland et al., 2000). Interest in healthy eating and knowledge of the healthiness of oily fish are shown to positively affect the frequency of fish consumption and are at least partly determining socioeconomic differences in fish consumption related to educational attainment (Pieniak et al., 2010). Furthermore, regular fish consumption in childhood is shown to positively affect adulthood fish consumption (Trondsen et al., 2003). Taken together, these factors might partly explain the independent effect of parental education on adulthood fish consumption in our cohort.

According to the FINDIET study, socioeconomic differences in diet have been most evident in consumption of fruit and vegetables for several decades (Raulio et al., 2016). The positive association between SES and consumption of fruit and vegetables appears in childhood, including Finnish children (Haapalahti et al., 2003; Kaikkonen et al., 2012; Rasmussen et al., 2006). Additionally, vegetable consumption has been shown to explain a considerable part of the socioeconomic differences in CVD mortality in Finland (Laaksonen et al., 2008). Consistent with these findings, when life-course levels between two SES groups were compared in this study, the consumption of fruit was higher among those having SES above

median levels in 1980, 1983 and 1986. However, the difference was no longer significant in subsequent follow-ups. For vegetable consumption, the differences between childhood SES groups were seen still in 2007, after which the difference was not significant. Likewise, in longitudinal analysis, childhood SES was not associated with consumption of fruit or vegetables in 2011, irrespective of how childhood SES was defined (family income or parental years of education). These results indicate that childhood SES might affect the consumption of fruit and vegetables in childhood, adolescence and young adulthood, but not into middle-age. A study on U.S. men followed from ages 7 to 32 supports these findings, showing that childhood SES was not directly associated with fruit or vegetable consumption in adulthood but that the effects were mediated through adulthood SES (Boylan et al., 2018). These findings are in contrast to those from a cohort of elderly British women, where although an association was observed for current SES on consumption of fruit, vegetables, fish and meat, higher childhood SES associated with higher consumption of vegetables and fruit independent of adult SES (Watt et al., 2009). The potential independent role of childhood SES on consumption of fruit and vegetable was also shown in a Danish cohort, but only among women (Hare-Bruun et al., 2011).

Of the different indicators and components of childhood diet, vegetable and fruit consumption are among the best predictors of overall healthier diet in childhood, midlife and older age (Maynard et al., 2006; Mikkilä et al., 2005). In the Young Finns Study, consumption of fruit and vegetables in childhood has been shown to associate with other favorable health behaviors (Mikkilä et al., 2005). At the time of baseline and first follow-ups of the Young Finns Study in the 1980s, the selection of fruit and vegetables in Finland was limited. Therefore, higher consumption of fruit and vegetables might have reflected the overall health-promoting attitude of the family. Data from the Young Finns Study has shown that consumption of fruit in childhood is associated with adult IMT independent of conventional adulthood risk factors (Juonala al., 2010b), and child vegetable consumption to inversely associate with PWV in adulthood, independent of other conventional cardiovascular risk factors in childhood and adult vegetable consumption (Aatola et al., 2010b). Therefore, the favorable effects of higher childhood SES on health-promoting lifestyle, reflected in the higher consumption of fruit and vegetable in childhood, are likely to be echoed in subclinical markers of CVD still in adulthood.

The results of this thesis emphasize the role of childhood SES on diet, independent of current SES in adulthood, with a higher (healthier) diet score among participants with higher childhood SES. These findings are supported by others that have described the role of childhood SES in determining different dietary patterns and diet quality in adulthood (Atkins et al., 2015; Hare-Bruun et al., 2011; Mishra et al., 2004; Watt et al., 2009). Data from two British studies suggested that those who

transition to a higher social class adopted more favorable dietary patterns related to their new social class (Mishra et al., 2004), and that diet quality was highest among men with high SES in childhood and adulthood and lowest among men with stable low SES (Atkins et al., 2015). Furthermore, a Finnish study that collected retrospective recall data on childhood SES (parental education and economic difficulties) showed that childhood SES was not associated with adulthood diet, measured as an index of healthy food habits (Lallukka et al., 2007). However, data from the Young Finns Study has shown that parental education was a stronger predictor of diet of young adults aged 18–24 years than their own education, when the healthiness of diet was determined by daily use of butter (Leino et al., 1999). Taken together, the differences observed in these studies might be explained by different determinants used to describe healthy food habits and SES in childhood and to cultural, political and economic differences between study populations.

In conclusion, there is growing evidence that the socioeconomic differences in diet begin in childhood, which in this study cohort was reflected in higher consumption of fruit and vegetables in the 1980s among participants with higher childhood SES. Attitudes toward a healthier diet might be preserved until adulthood but are, at that time, reflected in lower consumption of meat and higher consumption of fish. This change might relate to the temporal development or change in attitudes and knowledge about the health effects of components of diet among different socioeconomic groups and the transformation in availability of certain foods. However, the childhood SES might have impact on overall healthiness of adult dietary pattern since longitudinal analysis performed as part of this thesis showed the association between higher childhood SES and higher adult multidimensional diet score, independent of adulthood SES. Possibly, children in high SES families adopt the more health-conscious attitudes of their parents or families towards dietary choices that equip them for long-lasting healthy dietary patterns and better cardiovascular health.

Smoking

Smoking multiplies the risk of cardiovascular mortality and is shown to account for a third of all cardiovascular deaths (Banks et al., 2019; National Center for Chronic Disease Prevention and Health Promotion (US) Office on Smoking and Health., 2014). Of different health behaviors, smoking has been claimed to be the strongest contributor to socioeconomic differences in cardiometabolic health (Petrovic et al., 2018). In this thesis, lower childhood SES defined by annual family income was associated with higher risk of being a smoker in adulthood and with higher number of pack years, which remained after adjustment for income in adulthood. The results were essentially similar when parental years of education was used to determine

childhood SES, with only the association of childhood SES with pack years attenuating after adjustment for adult SES. In line with our results, previous studies have shown the inverse association between SES and prevalence of smoking using both educational and monetary indicators (Jamal et al., 2018; Laaksonen et al., 2005).

In this thesis, the difference in prevalence of smoking between low and high childhood SES groups was observed already in 1983 when participants were aged 12 to 21 years and it remained significant during the 1986, 2007 and 2011 follow-ups. In line with the findings from this thesis, others have indicated the socioeconomic gap occurs already in adolescence and young adulthood and that people with lower family SES are at greater risk to start smoking in adolescence (Kuipers et al., 2015; Pedersen et al., 2017; Tjora et al., 2011). The social determinants of smoking are suggested to include both differential exposure and vulnerability to tobacco use between SES groups (David et al., 2010). The association between childhood family SES and initiation and development of smoking behavior is strongly mediated by smoking behaviors of parents and older siblings (Tjora et al., 2011). Therefore, the association shown in this thesis between childhood SES and adulthood smoking is possibly influenced by smoking of other family members. However, a previous study also suggested that the association between childhood SES and adulthood smoking is not entirely mediated by smoking behavior of close relatives (Tjora et al., 2011). Another varying exposure between adolescents with different family SES is possibly related to smoking by peers, which strongly affects the uptake of smoking in adolescence (Lastunen et al., 2017; Molyneux et al., 2004). Also differential vulnerability between adolescents with different SES might influence the socioeconomic differences in initiation of smoking, also in this thesis (David et al., 2010). These mechanisms might include socioeconomic differences in adolescents' perceptions of smokers, social competence, self-esteem and psychosocial strain (Brook et al., 2007; Carvajal et al., 2000; Halme et al., 2018; Twenge et al., 2002).

In this thesis, those having lower SES in childhood had higher number of pack years in adulthood, suggesting that of all ever-smokers, people with lower SES in childhood had longer smoking history and/or had smoked more per day. Childhood SES defined by annual family income was associated with pack years, even after adjustment for own income in adulthood suggesting that it is not just the current SES in adulthood, but also childhood SES, which affects the length and amount of smoking. This is in line with a previous study showing the accumulating effect of SES on start and continuation of smoking across the whole lifetime (Gilman et al., 2003). Therefore, when concerning the effects of SES on persistent smoking, which is particularly harmful in terms of cardiovascular health, the effects of both childhood and adulthood SES should be taken into account. In lieu of this, the results

of a U.S. study are alarming as they indicated that health-care providers were less likely to provide assistance for quitting smoking for low SES people (Browning et al., 2008). Additionally, lower SES is associated with higher risk of having no intention to quit smoking and with higher levels of nicotine dependence determined by the heaviness of smoking (Siahpush et al., 2006). Therefore, to diminish the socioeconomic gradient in smoking, public health campaigns could be targeted to areas of low SES and health care professionals should give equal or more attention to smoking behaviors among those people with lower SES or from areas of low SES.

An international comparison which mostly included studies with retrospectively assessed childhood SES, showed that manual childhood origins lowered the chance of smoking cessation in adulthood (Power et al., 2005). Contrary to other countries, the association between SES and adult cessation of smoking was not observed in Finnish population neither for childhood nor for adulthood SES. Since smoking cessation has beneficial effects on coronary heart mortality even among heavy smokers, to encourage a diminished socioeconomic gradient in cardiometabolic health, future studies should investigate the association of SES in different time-points of life with attempts and success in smoking cessation (Duncan et al., 2019).

Furthermore, better understanding of the mechanisms behind the conceivable socioeconomic differences in maintained cessation might help with future interventions and public policies aimed at reducing the socioeconomic differences in smoking. The Tobacco-free Finland 2030 network has succeeded in substantially decreasing the smoking prevalence in the general adult population but the socioeconomic difference remains (Koponen et al., 2018; Patja et al., 2017). A study, that investigated the impact of price- and non-price-related population-wide tobacco control policies on socioeconomic differences in smoking in Europe between the 1990s and 2000s, showed that tobacco control policies have helped reduce the prevalence of smoking, particularly in lower SES groups (Hu et al., 2017). Therefore, the simultaneous increasing trend in smoking inequalities might be explained by other factors and these factors should be disentangled in future studies.

Physical activity

Although the association between high SES and high levels of leisure-time activity in adulthood appear consistent, the evidence across socioeconomic groups remains unclear (Gidlow et al., 2006). In this thesis, childhood SES, defined by both annual family income and parental educational years, was positively associated with PAI 31 years later in adulthood. The associations were attenuated after adjustments for current SES in adulthood. Likewise, higher levels of PAI were observed throughout the life-course in those above versus those below the median annual family income in childhood.

Previous studies investigating childhood SES on physical activity in youth and adulthood have revealed inconsistent results. These results tend to differ depending on the measurements of SES and physical activity used and whether the results are for men, women or for both sexes combined (Aarnio et al., 2002; Huurre et al., 2003; Juneau et al., 2014; Osler et al., 2001; Silverwood et al., 2012). However, in studies with prospectively determined childhood SES, positive association between childhood SES and adulthood physical activity is observed only concerning leisure time activity (Juneau et al., 2015). Similarly, adulthood PAI in this study is taking account the frequency, intensity and duration of leisure-time physical activity.

The importance of leisure-time activity on cardiometabolic health has been identified. The PAI applied in this study has been shown to correlate with the indicators of exercise capacity and with waist circumference (Telama et al., 2005; Yang et al., 2006), while, the positive effect of occupational physical activity on BMI is shown to be only minor in Finnish population (Böckerman et al., 2008). Additionally, a study from the USA showed that even though individuals with lower SES were more likely to be physically active during work days or chores, the work-related physical activity was not significantly associated with the risk of T2DM (Tsenkova et al., 2017). As physically demanding jobs are typically characterized by high demands and low control, the adverse psychosocial factors might counterbalance the health effects of physical activity (Johnson et al., 1989).

Although systematic review found a positive association between child SES and adult leisure time physical activity independent of adulthood SES, in this thesis, the association of child SES with adulthood PAI was attenuated after adjustment for adult SES (Juneau et al., 2015). Similarly, findings from the Young Finns Study showed that parental educational attainment was not associated with life-course trajectories of physical activity (Rovio et al., 2018). Still, children's success at school and higher educational level in adulthood associated with persistently active and increasingly active trajectories of physical activity and worse school performance and lower educational level in adulthood associated with persistently inactive and decreasingly active trajectories. Similar results were seen in the Health 2000 Study among Finnish young adults (Kestilä et al., 2015) In men, low parental education was associated with higher risk of being physically inactive in leisure time but the association was mediated by participant's own current educational attainment. Furthermore, a systematic review has suggested that those with the highest levels of leisure-time physical activity in adulthood are those with stable high SES from childhood to adulthood (Elhakeem et al., 2015). Therefore, results of this thesis support the previous evidence that the effects of childhood SES on adulthood physical activity might at least partly operate through the participant's own academic performance and adulthood SES.

However, differences in leisure-time physical activity between low and high childhood SES groups were evident in this study already in childhood and adolescence in the 1980s. This supports more independent effect of childhood SES at least on childhood and adolescence physical activity, and is in line with a study on 7-to-8-year-old children from the UK that reported a dose-dependent positive association between family income and attendance to out-of-school structural activities (Voss et al., 2008). Similarly, a review consisting of 150 studies suggested that of different environmental factors, both mother's education and family income showed especially strong associations with physical activity of children and adolescents (Ferreira et al., 2007). Parents who are physically active tend to have more physically active children, but the effect of parental physical activity diminishes from young adulthood onwards (Rovio et al., 2018). Therefore, because parents in families with high SES are more physically active, they might promote physically active lifestyle for their children. Parents might also provide better and more possibilities for their children to be physically active in childhood such as through sports clubs and other organized activities. The data suggest that as the child transitions into adulthood the effects of parental support and role modeling diminish, and physical activity might be more strongly determined by current SES and other prevailing environmental factors.

Although adult SES might play a more important role in adult physical activity, it does not invalidate the important role that high child SES plays in physical activity in early life. For example, higher levels of childhood physical activity associates with several favorable health outcomes, including weight control and lower blood pressure (Leary et al., 2008; Yang et al., 2006), increases the odds of being physically active in adulthood, and is an independent predictor of better vascular health (Juonala et al., 2010; Pälve et al., 2014; Telama et al., 2014). Therefore, even if childhood SES does not directly impact adult physical activity, it associates with child levels of physical activity and is likely to play an important indirect role in adult physical activity and its related outcomes.

Alcohol consumption

The current evidence suggests that association between consumption of alcohol and cardiometabolic disease is patterned by the amount of alcohol consumed but that reduction of alcohol consumption might be beneficial for cardiovascular health at all levels of alcohol consumption (Corrao et al., 2000; Holmes et al., 2014; Mukamal et al., 2005). However, there is no consensus on the association of adult SES with patterns and amounts of alcohol consumption (Bloomfield et al., 2006; Collins, 2016; Mäkelä et al., 2017; Marmot, 1997). Evidence is even weaker on the role of childhood SES, with most studies that have shown a relationship between

child SES and adult alcohol consumption using variables that describe the risk-use of alcohol, such as alcohol abuse, heavy drinking or alcohol dependence (Droomers et al., 2003; Leino et al., 2000; Poulton et al., 2002).

In this study, no association was observed for childhood SES on the amount of alcohol consumption (drinks/day) in adulthood. Similarly, a study from the UK found no association between parental SES and alcohol consumption measured as a frequency of consumption in adolescence (Shucksmith et al., 1997). Furthermore, a Swedish follow-up study that measured alcohol consumption in a cohort of school leavers, showed that paternal working class background was not correlated with alcohol consumption (centiliters of absolute alcohol per year) at the age of 21 years (Urban et al., 1992). Therefore, the results of this thesis are in line with these findings, indicating that childhood SES is not strongly associated with the total amount of alcohol consumption later in life. However, childhood SES might impact on the risky use of alcohol, possibly as a reflection of general disadvantage (Droomers et al., 2003; Leino et al., 2000; Poulton et al., 2002). Still, a systematic review of 19 longitudinal studies concluded that the overall evidence on the association between childhood SES and alcohol use later in life is sparse (Wiles et al., 2007). Therefore, more studies are needed to understand the influence of childhood SES on alcohol consumption later in life, especially concerning its harmful effects on cardiometabolic health.

In conclusion, this thesis showed that higher childhood SES is associated with more favorable health behaviors in childhood, in adolescence and in adulthood. As long as health behaviors are patterned by SES, disappearance of the socioeconomic gap in cardiovascular morbidity and mortality is implausible (Van De Mheen et al., 1998). Therefore, public strategies that aim to reduce risky health behaviors from childhood are warranted, and low family SES should be emphasized as a long-lasting risk factor of unhealthy behaviors.

6.3.2 Clustering of risk factors

Clustering of biological risk factors of CVD is evident already from childhood (Raitakari et al., 1994). Additionally, the risk of CVD is increased with every additional risk factor (Yusuf et al., 1998). The main determinants of risk factor clustering include obesity and weight gain, whereas higher SES is shown to have a protective role (Raitakari et al., 1995; Wilson et al., 1999). MetS is a cluster of cardiometabolic risk factors, strongly associated with obesity, which predisposes to markedly increased risk for CVD and T2DM (Dekker et al., 2005; Laaksonen et al., 2002; Reaven, 1988).

According to the harmonized criteria, to have MetS a person should have any three of the five components (central obesity, hypertension, low HDL cholesterol, elevated triglycerides and elevated fasting plasma glucose) (Alberti et al., 2009). In this study, childhood SES had an inverse association with all the MetS components and, except for blood pressure and fasting plasma glucose, these associations persisted after adjustment for cardiovascular risk factors in childhood. When the associations were additionally adjusted for the adult SES, only the associations of childhood SES with waist circumference and triglyceride levels remained statistically significant.

Since waist circumference is inherently related to obesity and the inverse association of childhood annual family income with adulthood BMI has been shown in this cohort, the result concerning the independent association between childhood annual family income and waist circumference in adulthood was expected (Juonala et al., 2011a). Additionally, data from the Young Finns Study have shown that lower parent occupational class is associated with increased risk of having high waist circumference in adulthood (Kivimäki et al., 2006c). These data from the Young Finns Study suggest the inverse association of childhood SES with the risk of abdominal obesity in adulthood, which is shown to be an independent risk factor for CVD and to explain a large part of the increased mortality risk associated with obesity (Bigaard et al., 2005; Després, 2012).

Abdominal obesity is also strongly associated with other components of MetS (hyperlipidemia, hypertension, insulin resistance) and predisposes afflicted individuals with an increased risk of T2DM later in life (Després et al., 2006). In this thesis, low childhood SES associated with a more adverse lipid profile and fasting plasma glucose levels in adulthood, however only the association with adult triglycerides remained significant after adjustment for adult SES. Socioeconomic differences in lipid concentrations between occupational groups have been shown in the Whitehall II study, where higher employment level was associated with lower triglyceride levels and with higher concentrations of HDL cholesterol (Brunner et al., 1999). However, evidence for an inverse association between childhood SES and lipid profile, including triglycerides, later in adulthood, is sparse (Kvaavik et al., 2012). The association between SES and triglycerides might be mediated by weight gain, which is likely to be the mechanism also in this cohort (Shohaimi et al., 2014).

In this thesis, an association between childhood SES and risk of having MetS in adulthood was also observed. A 1-unit increase in child SES was associated with a 7% decrease in the risk of having MetS in adulthood. The association was similar when childhood SES was defined by parental years of education, suggesting a 3% decrease in risk of MetS for adult offspring with every additional year spent in education by the parents. This association remained after further adjustment for adulthood SES. These data are consistent with those from the ARIC cohort, where

women with low childhood, early adulthood, mature adulthood, and cumulative SES had higher risk of MetS than those with high SES (Chichlowska et al., 2009). In contrast with these findings are data from a UK cohort that showed the association of childhood SES with risk of having adult MetS attenuated when adulthood SES was included in the model, but the effect of adult SES remained (Ramsay et al., 2008). Moreover, the association between SES and MetS was observed only among women.

Sex-differences in the association between child SES and MetS have been observed in other studies and are hypothesized to be explained by sex-differences in the effects of psychosocial factors among those of low SES (Thurston et al., 2005). A study based on a population-based health survey in the USA has shown that depression doubles the risk of MetS in women, but not in men (Kinder et al., 2004). Additionally, the inverse association between SES and BMI is suggested to be stronger among women, possibly due to higher social pressure to be thin among women than men, especially among women with high SES (Sobal et al., 1989). Stigma of obesity is also stronger among women and therefore, among women the association between SES and obesity is more likely to be bidirectional (Sobal et al., 1989; Tyrrell et al., 2016). No significant sex differences were detected in interaction analyses performed in this thesis, which suggests that the effects of annual family income on the risk of MetS were similar between men and women participants. Although similar associations were observed in both sexes in this study, the pathways behind the associations of childhood SES and risk of MetS might differ by sex (Lee et al., 2018).

Increased risk for MetS is strongly associated with abdominal obesity and lifestyle behaviors related to it, which are both shown to associate with low childhood SES (Frederick et al., 2014; Juonala et al., 2011). Therefore, the association between childhood SES and adulthood MetS shown in this study might be partly mediated by other adulthood cardiometabolic risk factors, including health behaviors, not included in the MetS. Additionally, a Finnish study has suggested that risk of MetS is higher among adults whose obesity continued from childhood to adulthood compared with people who became obese in adulthood, possibly related to prolonged insulin resistance (Vanhala et al., 1998). However, the results of this thesis indicated that the association of child SES with adult MetS was independent of conventional childhood risk factors, including BMI. In addition to conventional health behaviors, other related risk factors such as psychosocial environment and functioning in childhood and adulthood have been shown to associate with increased risk of having MetS in adulthood and might explain some of the pathway linking low childhood SES to MetS later in life (Lehman et al., 2005; Pulkki-Raback, 2012). Poorer psychosocial environment and stressful life-events in childhood and adolescence predispose individuals to higher risk of weight gain and associated risk

factor clustering (Elovainio et al., 2017). These psychosocial factors of the early environment are possible mediators of the observed associations in this study.

To study the effect of intergenerational SES mobility on the prevalence of MetS, participants were divided into four groups according to their SES in childhood and adulthood. When the prevalence of MetS in adulthood was compared among these childhood-adulthood SES groups, the prevalence was lowest among the "stable high" group (high SES in both childhood and adulthood) compared with all other groups. In comparison to the groups who had low SES in childhood, people in the "stable high" group had lower blood pressure and more favorable lipid levels in childhood, possibly reflecting childhood socioeconomic differences in diet and physical activity. In adulthood, the most favorable risk factor profile was seen among people from the "stable high" group, as they had lower BMI, lower waist circumference and lower systolic blood pressure in comparison to the other three groups. These data are suggestive of the cardiometabolic benefit of maintaining high SES. Interestingly, when compared to the "downwardly mobile" group, who also had SES above median in childhood, the BMI in the "stable high" group was lower already in childhood. This difference might relate to reverse causality between BMI and SES, meaning that people with higher BMI already in childhood might have encountered stigmatization and labor-market discrimination due to a higher BMI that has adversely affected their socioeconomic attainment and their adulthood income (Tyrrell et al., 2016).

Data from the Young Finns Study have shown that 33% of the association between childhood SES and the AHA's ideal cardiovascular health index in adulthood is mediated by adulthood SES but that childhood SES is also independently associated with adult ideal cardiovascular health (Savelieva et al., 2017). Reinforcing the direct contribution of adult SES, results from this thesis showed individuals in the "stable high" group had lower risk of MetS and more favorable adult risk factor levels than those from the "downwardly mobile" group. Similarly, a study using register data from Sweden showed that individuals in the "downwardly mobile" group had higher risk for cardiovascular mortality than the "stable high" group (Tiikkaja et al., 2008). On the other hand, the risk of MetS was higher among the "upwardly mobile" group when compared with those in the "stable high" group. This is in line with a cohort study of 1000 children born in New Zealand that showed that upward mobility did not mitigate the adverse effects of low SES in childhood on poorer adulthood cardiovascular health (Poulton et al., 2002). In conclusion, results from this thesis suggest that childhood SES is independently associated with the risk of MetS in adulthood, but that the favorable role of childhood SES is most pronounced if high SES is maintained from childhood to adulthood.

In this thesis, child SES was also inversely associated with the combined variable of glucose abnormalities (i.e., having either IFG or T2DM). This is consistent with

other data from the Young Finns Study that showed that in men, childhood social class was inversely associated with adult insulin resistance, independent of adulthood social class (Kivimäki et al., 2006c). However, in this thesis, the association was attenuated after adjustment for conventional cardiometabolic risk factors in childhood. When analyzed separately, no association was found between childhood SES and adult T2DM. Similarly, some studies have failed to show any association between childhood SES and the prevalence of T2DM in both men and women (Agardh et al., 2007; Smith et al., 2011). Few studies have managed to show an association of childhood SES with incident T2DM in adulthood and some of them only among women (Maty et al., 2008; Pikhartova et al., 2014; Wray et al., 2006). However, these studies used retrospective recall of childhood SES, which might have influenced the results. Furthermore, a study which failed to show an independent association between child SES and adult T2DM showed, among women, an inverse association between cumulative life-course SES and prevalence of T2DM in adulthood (Smith et al., 2011). This supports the beneficial effects of sustained high SES, as was observed for the risk of MetS in this thesis. In the case of glucose abnormalities, the results of this thesis supported the importance of adult SES showing no statistically significant difference between adult fasting plasma glucose levels in the "stable high" and "upwardly mobile" group. The non-significant association between childhood SES and adult T2DM might relate to the relatively low prevalence of T2DM owing to the rather young age of the study cohort at last follow-up. Given that 90% of T2DM is related to overweight or obesity and MetS is associated with a five-fold increase in the risk of developing T2DM later in life, the association of child SES with adult T2DM might be seen in later follow-ups of the Young Finns Study cohort (Hossain et al., 2007; Reaven, 1988).

The results of this thesis are in line with evidence that higher child SES is protective of risk factor clustering, and adult MetS (Kivimäki et al., 2007; Raitakari et al., 1995). These results support the importance of the early life environment in determining future risk of cardiometabolic disease and could form part of future policy making. Although the results showed that the association of childhood SES with adult MetS was independent of childhood risk factors, those having the lowest risk of MetS had not only "stable high" SES from childhood to adulthood but also the most favorable risk factor profile both in childhood and adulthood. Importantly, these risk factors included obesity and several associated risk factors. Since as little as 5% weight loss can reduce insulin resistance, improve glycemic control and prevent development of T2DM, distinguishing at risk individuals early and supporting their weight loss attempts should be prioritized (Klein et al., 2004). Supporting the importance of identyifying and intervening in MetS as early as possible, the resolution of youth MetS by adulthood has been suggested to normalize the risk of T2DM to levels observed amongst those who have never had MetS

(Magnussen et al., 2012). Therefore, the socioeconomic differences in the risk of MetS and T2DM could be partially diminished by early interventions focused on obesity and related risk factors.

6.3.3 Arterial stiffness

Although CVD is a chronic process with a long asymptomatic phase, subclinical changes can be detected noninvasively by several methods long before overt symptoms appear (de Simone et al., 2008; Hamilton et al., 2007). One subclinical marker of CVD is arterial stiffness, which has been shown to predict cardiovascular events and CHD, independent of conventional risk factors (Ben-Shlomo et al., 2014). Arterial stiffness is shown to relate to atherosclerosis at multiple sites of the vascular tree (Van Popele et al., 2001). Moreover, studies have reported that arterial stiffening starts in early childhood and is affected by conventional childhood cardiometabolic risk factors (Berenson et al., 1998; Juonala et al., 2005; Mikola et al., 2017, 2015). However, evidence on the association of child SES on arterial stiffening has been sparse.

In this study, childhood SES was inversely associated with two markers of arterial stiffness, Cdist and PWV, after adjustment for age and sex. The results were similar whether child SES was defined as annual family income or parental years of education. The Young Finns Study has previously shown that cardiovascular risk factors (elevated blood pressure, obesity) and health behaviors (consumption of fruit and vegetables, physical activity) in childhood have long-lasting associations on arterial elasticity to adulthood (Aatola et al., 2010a; Aatola et al., 2010b; Juonala et al., 2005; Pälve et al., 2014). As such, the models were adjusted for conventional cardiovascular risk factors in childhood. The associations between child SES and Cdist and PWV remained significant after adjustment for childhood cardiometabolic risk factors that were independently associated with Cdist or PWV.

Smoking is suggested to be an independent risk factor for increased arterial stiffness, possibly through acute and chronic effects of tobacco smoke on arterial structure and function (Jatoi et al., 2007). Therefore, the role of early life smoking behavior on the association between child SES and arterial stiffness in adulthood among participants aged 12 to 18 years at baseline was examined. In this sub-sample, the results were similar whether the association was adjusted for the participant's own smoking status or not, indicating that the observed association was not modified by higher levels of smoking among the socioeconomic disadvantaged. Because tobacco smoke exposure is greater in families with low SES and we have shown childhood exposure to tobacco smoke to associate with arterial function in adulthood, we hypothesized that parental smoking habits in childhood might affect arterial stiffness in adulthood (Juonala et al., 2012; Kuntz et al., 2016). However,

when the models were adjusted for parental smoking, results were similar indicating that childhood exposure to secondhand smoke did not affect the association between child SES and adult arterial stiffness. Therefore, in this study, the association between child SES and arterial stiffness was not explained by childhood exposure to tobacco smoke.

Only one previous study has investigated the association between SES and Cdist (Climie et al., 2019). The study showed that higher individual deprivation, meaning lower individual SES, was associated with lower Cdist which persisted after adjustment for a wide spectrum of known or suspected contributors of arterial stiffness. Furthermore, a few studies have investigated the association of child SES with adult PWV. A small study among adolescents showed that low family income, low parental education and low neighborhood SES associated with higher PWV compared with those in higher SES categories (Thurston et al., 2009). Additionally, the Whitehall study investigated the association between father's social class and PWV at old age (Trudel et al., 2016). Parental SES was inversely associated with baseline PWV, but not with 5-year progression of arterial stiffness. In contrast to child SES, lower adult SES was associated with a larger 5-year-change in PWV during old ages, but not with baseline PWV. These results suggest that childhood SES has long-lasting effects on arterial stiffness, but that the changes influenced by disadvantage in childhood might occur earlier in life. Unfortunately, investigating whether the association of child SES with arterial stiffness was evident already in childhood or in adolescence is not possible in the Young Finns cohort, as measurements of arterials stiffness were not performed in earlier study years. Nevertheless, the results in this thesis support the limited available evidence of an association between childhood SES and arterial stiffness later in life, and suggests the association of childhood SES is independent of conventional childhood risk factors. Therefore, programs that aim to address only conventional risk factors will leave a number of children at higher risk for future arterial stiffening. Instead, low childhood SES should be considered as an independent childhood risk factor, alongside more conventional risk factors, for increased arterial stiffening later in life.

However, when the associations of child SES with PWV and Cdist were adjusted for independent conventional cardiometabolic risk factors in adulthood, the associations were attenuated. Of the adult risk factors, adjustment for systolic blood pressure had the strongest diluting effect on the associations between childhood SES and both markers of arterial stiffness, suggesting that blood pressure may be the main mediating factor. In line, data from the Young Finns Study have shown child SES is a stronger predictor of adulthood blood pressure than adult SES (Kivimäki et al., 2006b). The results presented in this thesis indicate that the association of child SES on adult blood pressure might be reflected in arterial stiffness. The arterial stress caused by increased systolic blood pressure might have led to increased arterial

stiffness, but arterial stiffening could also precede elevated blood pressure (Dart et al., 2001; Intengan et al., 2001). Therefore, the association between blood pressure and arterial stiffness may be bidirectional.

Apart from systolic blood pressure, the other two risk factors with strong diluting effects on the associations of childhood SES with Cdist were HDL cholesterol and triglycerides and PWV were triglycerides and plasma glucose. We have previously shown that in addition to blood pressure, adulthood triglycerides and insulin are directly associated with adult PWV (Aatola et al., 2010a). Similarly, in cross-sectional analyses of the FHS, people, who were obese, suffering from glucose or lipid abnormalities or had higher mean arterial pressure, had stiffer arteries in all age groups (Mitchell et al., 2007). However, a large systematic review concluded that the contribution of conventional cardiovascular risk factors to PWV, other than age and blood pressure, is small or insignificant (Cecelja et al., 2009).

No previous study has investigated the effect of intergenerational social mobility on adult arterial stiffening. However, the positive effect of upward mobility and the negative effect of downward mobility have been shown on the risk of hypertension (Högberg et al., 2012; Matthews et al., 2002). When mean values of Cdist and PWV in adulthood between childhood–adulthood SES groups were compared in this thesis, participants with high SES in childhood and adulthood had higher Cdist compared with participants in all other child–adult SES groups. However, for PWV, the difference between those whose SES remained high and those whose SES increased from childhood to adulthood was not statistically significant. This might indicate that the risk of greater arterial stiffening is at least partially diminished among those who improved their SES from childhood to adulthood. However, no similar compensating effect of upward mobility was seen for Cdist and additionally, in linear regression analyses, associations between child SES and both Cdist and PWV were independent of adult SES. Therefore, the results emphasize the role of child SES to be partly independent of adult SES.

In conclusion, results from this thesis show an inverse association between childhood SES and arterial stiffness that was independent of conventional childhood risk factors. As increased arterial stiffness predicts higher risk for cardiovascular events, independent of conventional risk factors, these results underscore evidence of the independent role of childhood SES on future risk of cardiovascular morbidity (Ben-Shlomo et al., 2014). However, since the association between childhood SES and adulthood arterial stiffness was largely mediated by adulthood blood pressure, it might be possible to decrease arterial stiffness and risk of cardiovascular morbidity by promoting healthy blood pressure levels in all SES groups, and across the lifetime.

6.3.4 Cardiac structure and function

When the heart is stressed with hemodynamic strain due to pressure or volume load, it results in hypertrophy of existing myocytes and a resultant increase in LVM (Kehat et al., 2010). Increased LVM is shown to be prognostic for cardiovascular events and deaths, even after adjustment for conventional cardiovascular risk factors (Gardin et al., 2001; Levy et al., 1990). Systolic blood pressure and BMI are shown to be strong independent predictors of LVM in both sexes (Savage et al., 1990). As childhood SES has been shown to associate with both adulthood obesity and blood pressure in previous Young Finns studies (Juonala et al., 2011a; Kivimäki et al., 2006b), it was hypothesized that the association of childhood SES would extend to LVM in adulthood in this cohort.

As hypothesized, childhood SES, defined by annual family income, was inversely associated with LVM index. This is the first study to prospectively investigate the association between child SES and LVM and to take into account other childhood risk factors. However, cross-sectional studies in adults have shown the relationship between SES and LVM to be a gradual inverse association (Medenwald et al., 2016; Rodriguez et al., 2004), while prospective adult studies have suggested lower SES increases the risk for LVH (Christensen et al., 2011; Kubota et al., 2017). Most previous studies have used educational attainment as the marker of SES, but data from this thesis expands available evidence to parental income. Furthermore, sensitivity analyses showed that the results for parental occupational class were essentially similar with results from the main analyses. Therefore, results from the sensitivity analyses consolidate previous evidence on the association between occupational SES and LVM (Murray et al., 2016); where occupational SES measured at 3 different time points from childhood to adulthood was associated with LVM in late adulthood.

Concerning risk variables other than childhood SES; both adulthood and childhood BMI were directly associated with adulthood LVM in multivariable models. Similarly, others have shown that BMI is a strong independent predictor of LVM in childhood, adolescence, and adulthood (Daniels et al., 1995; Savage et al., 1990; Toprak et al., 2008). Moreover, there is evidence that BMI might mediate the association between childhood SES and adulthood LVM (Murray et al., 2016). In this thesis, when analyses were adjusted for childhood risk factors, the associations between childhood SES and LVM index remained significant and the difference between low and high SES group became stronger. After adjustment for adulthood risk factors, the association between childhood SES and LVM remained significant but was slightly attenuated. Since the Young Finns Study has previously shown lower childhood SES is associated with higher adulthood BMI, and adult BMI was directly associated with LVM in this study, our results suggest a mediating role of adult BMI (Juonala et al., 2011a).

In addition to maintenance of ideal body weight, current evidence suggests an important role of normal blood pressure in primary and secondary prevention of increased LVM (Savage et al., 1990). Even though no causative pathway between increased arterial stiffness, blood pressure and LVM has been shown, the biological basis is sound. For example, decreased arterial elasticity is reflected by an increase in pulse pressure, which leads to growth in LV afterload and decreased perfusion in the coronary arteries (Vlachopoulos et al., 2006). Thus, the workload on the heart is increased, which induces cardiac hypertrophy and remodeling (Boutouyrie et al., 1995). In this thesis, the association between child SES and arterial stiffness was strongly mediated by adult blood pressure. This was not surprising given previous data from this cohort of an association between child SES and adult systolic blood pressure (Kivimäki et al., 2006b). However, no associations were observed between child or adult systolic blood pressure with adult LVM. Additionally, the association between child SES and LVM remained significant after adjustment for adult risk factors. Therefore, these findings suggest that the pathway from low child SES to increased LVM in adulthood is not totally accounted by conventional risk factors for increased LVM, such as BMI and blood pressure, but also includes other mediators and confounders.

In addition to increased LVM, lower childhood SES was associated with poorer LV diastolic performance. Importantly, of all child risk factors, only SES was associated with diastolic function in adulthood. Decline in LV diastolic function typically results from the decrease in LV compliance and relaxation, due to increased LVM (Zile, 2002). Impaired diastolic function of the heart is shown to progress over time and it is associated with development of heart failure (Kane et al., 2011). Although lower SES in childhood and adulthood associates with higher incidence of heart failure and lower survival in patients with heart failure, only few studies have investigated the association of SES on diastolic function (Christensen et al., 2011; Ingelsson et al., 2006; Murray et al., 2016; Roberts et al., 2010). In a Danish study, adulthood education was associated with diastolic function in relation to severe but not mild dysfunction (Christensen et al., 2011). To measure diastolic function, researchers used E/A ratio and deceleration time of the E-wave, which was different to what was used in this thesis. In a British study that used E/e' ratio, similarly to this thesis, SES in childhood and early adulthood were inversely associated with E/e' in adulthood (Murray et al., 2016). Consistent with data from the British study that used occupational class of parents to define childhood SES, sensitivity analyses in this thesis that used parental occupational as the marker of SES showed similar results with the main analyses for family income.

A previous study showed that the associations between SES and markers of diastolic function were largely diminished after adjustment for BMI, as was observed for LVM (Murray et al., 2016). Similarly, in multivariable analysis of this thesis

adulthood BMI was directly associated with E/e' ratio in adulthood. Even though some of the pathway between increased BMI and impaired diastolic function might operate through increased LVM, studies have shown that the association is not totally diminished after taking into account LVM and other conventional risk factors of diastolic dysfunction (Russo et al., 2011). Other possible mechanisms include lipotoxicity of free fatty acids leading to cardiomyocyte apoptosis and fat infiltration of the myocardium altering cardiac structure and function (Scheerder et al., 1987; Zhou et al., 2000). Another adulthood risk factor that associated with diastolic function in adulthood was systolic blood pressure. Since diastolic function is suggested to be a link between hypertension and heart failure and hypertension is an independent risk factor of diastolic dysfunction, this result was also expected (Russo et al., 2010a; Zile et al., 2002). However, adjustment for adulthood risk factors including systolic blood pressure and BMI in this thesis did not attenuate the association between childhood SES and diastolic function, indicating other potential mechanisms behind the association.

Previous studies have identified smoking to be a subtle determinant of both LVM and LV diastolic performance (Heiskanen et al., 2019; Nadruz et al., 2016). Furthermore, chronic passive smoking in childhood associates with impaired LV diastolic function in adulthood. (Peña et al., 2018) In this thesis, parental smoking in childhood was included as a risk factor and associations between child SES with both LVM and LV diastolic performance remained significant after adjustment for parental smoking. Indeed, parental smoking was not associated with LVM or diastolic function in multivariable analyses. In addition, the association of child SES on LVM and LV diastolic function remained significant in analyses that adjusted for the participant's smoking status in adolescence and adulthood. Nevertheless, the association of child SES with LV diastolic performance was attenuated after adjustment for child smoking status in a sub-sample of participants. However, this might be attributed to the small number of participants that had information available on child smoking status. In contrast, the association for LVM remained even after adjustment for child smoking status.

A previous study has suggested that the association between life-course SES and LV diastolic function is mostly attributed to an early life accumulation model whereas the whole of life accumulation model fits better the association between SES and LVM (Murray et al., 2016). Similarly, in this thesis, no association between adulthood SES and diastolic function was observed in multivariable analysis and the association of child SES with diastolic function remained significant after adjustment for adult SES. Similar results were observed for LVM, suggesting the importance of child SES above adulthood SES. Furthermore, the association between childhood SES and cardiac structure and function in adulthood in this thesis was independent of not only adult SES but also of conventional cardiometabolic risk

factors in childhood and adulthood. Therefore, the association might involve other potential moderators, confounders and mechanisms, which were not included in the analyses.

One potential mechanism might relate to exposure to chronic stress and other psychosocial factors across the lifetime. Data from the Young Finns Study has shown that a favorable psychosocial environment, together with high parental SES, is positively associated with ideal cardiovascular health, defined by the AHA (Pulkki-Råback et al., 2015). This might relate to favorable effects of positive psychosocial experiences, which are suggested to increase restorative health behaviors and biological processes and decrease deteriorative processes (Boehm et al., 2012). Furthermore, low SES associates with adverse psychosocial factors, including higher levels of stress, lower feeling of control and poorer social networks (Galobardes et al., 2006c; Ruberman et al., 1984). Psychosocial stressors predispose to clustering of behavioral and clinical cardiovascular risk factors but they might also contribute by direct pathophysiological mechanisms (Rozanski et al., 1999). For example, people with low SES were shown to have a heightened systolic blood pressure reaction to stress, which simultaneously was associated with increased progression of atherosclerosis (Lynch et al., 1998).

Therefore, to perceive the possible moderating effects of psychosocial childhood environment, we utilized a score measuring emotional environment in the family of children. The four favorable components included absence of previously diagnosed parental mental disorder, high parental caregiving nurturance, high parental life satisfaction, and reasonable alcohol use (Pulkki-Råback et al., 2015). In the multivariable analysis of this study, the favorable emotional family environment score in childhood was inversely associated with LVM but not with diastolic function in adulthood. However, between three childhood SES groups, no significant differences in the score were seen and the associations of childhood SES with both LVM and diastolic function remained significant after adjustment for the score. Still, it is possible that the psychological stressors related to childhood environment were not entirely comprised and these missing factors might at least partially mediate the association between childhood SES and poorer cardiovascular health in adulthood.

This thesis is among the first to show the role of childhood SES in predicting cardiac structure and function later in life in terms of LVM and LV diastolic function. Altogether, the results of this thesis suggest a conceivable pathway beginning from low childhood SES, which might lead to more adverse health behaviors and then travel through clustering of clinical risk factors to arterial stiffening and finish in alterations of cardiac structure and function. Therefore, the association of low childhood SES with higher LVM and poorer diastolic function still in adulthood confirms the evidence from previous studies and from other results of this thesis for long-lasting effects of childhood SES on cardiometabolic health.

6.3.5 Clinical and future research perspectives

When compared to the 1980s, the Finnish standards of living have improved considerably alongside decreasing cardiovascular mortality (Vartiainen et al., 2010). Still, according to the data from the national Finnish statistics agency, 11% of all children in 2017 lived in families, which were at risk of poverty, meaning that the disposable family income was smaller than 60% of the national median income (Statistics Finland, 2018b). Furthermore, as the socioeconomic gap in cardiovascular morbidity and mortality in Finland was observed multiple decades ago, there have been several public programs and strategies that have aimed to diminish the gap (*Kansallinen terveyserojen kaventamisen toimintaohjelma 2008-2011*, 2008; Ministry of Social Affairs and Health, 2001). Nevertheless, differences between socioeconomic groups have remained or even widened (Rotko et al., 2016, 2012).

Not all children from low-income families live in an unfavorable childhood environment or will have poor cardiovascular health in adulthood. Previous studies have suggested that socioeconomic differences in health might reflect the differences in intellectual, practical, financial and power-related resources (Sihto et al., 2013). The present thesis describes the association between childhood SES and cardiometabolic health in adulthood but does not offer causal explanations. Therefore, further understanding of mechanisms behind the unfavorable effects of low childhood SES is needed to direct interventions in order to diminish the socioeconomic disparities in cardiometabolic health. Evidence has shown, that generally, socioeconomic health differences are influenced by material, behavioral and psychosocial factors, which are interwoven with one another (Palosuo et al., 2009). Therefore, future studies should better consider the role of psychosocial and material environment.

Further, childhood SES comprises much more than annual family income. Even though the results were confirmed in sensitivity analyses using parental years of education and occupational class, future studies using a wider spectrum of different aspects of individual-level childhood SES and area-level SES should be considered. Since SES, and especially income, is unstable condition and affected by several life circumstances, including unemployment and poor health, using SES measured at multiple timepoints in childhood, in adolescence and in adulthood could lead to more accurate analysis of the effects of SES across the life-course.

Although MetS, arterial stiffness, LVM and impaired LV diastolic function are well established markers of increased cardiovascular risk, future studies that use cardiovascular events as the outcome would provide better understanding of the long-standing effects of childhood SES on cardiovascular health. Accumulation of cardiovascular events is expected as Young Finns Study participants age, which will enable investigations with cardiovascular events in this cohort. Even though this study provides evidence on the role of child SES, modern society is very different to

the childhood environment in which participants in the Young Finns Study experienced. Future studies should examine contemporary childhood SES with outcomes of interest – though prospective cohorts would likely encounter this same limitation in the future.

This thesis illustrates that at least part of the influence of child SES on adult cardiometabolic health might be conveyed through socioeconomic differences in life-course health behaviors. While socioeconomic differences (income, occupation, education) are unable to be totally avoided, socioeconomic differences in cardiometabolic health can be diminished by targeting detrimental health behaviors. Because interventions towards more favorable health behaviors have proven effective among children, and the results from this thesis and elsewhere suggest both direct and indirect roles of child SES on life-course health behaviors, low SES families should be emphasized when favorable health behavior is promoted to reduce the socioeconomic gap in health (Laitinen et al., 2018; Nader et al., 1999).

Two different strategies of public health policy are often distinguished in terms of reducing health inequalities: targeted and population-wide interventions (Vilhelmsson et al., 2018). Population-wide interventions are most effective if the mechanisms are apparent across the whole socioeconomic spectrum. These interventions include offering low-cost and public places to exercise and directing behavior (e.g. smoking and diet) towards healthy choices by taxation policies and legislation. Attempts to support favorable health behaviors from early-life are already a part of the principles in family health clinics, early childhood education and schools, and before birth, in maternity clinics. However, some of the nationwide policies to promote healthy behavior have led to increases in socioeconomic inequalities as higher SES people have benefitted more from the interventions (Schaap et al., 2008). Therefore, to diminish socioeconomic differences, targeted interventions might be most effective at reaching those with the highest need (Vilhelmsson et al., 2018). Nevertheless, these interventions might also have adverse effects including stigmatization and interpretation by the target groups of paternalism. Notably, whether targeted or not, most effective interventions tend to make the healthiest option the easiest or default that would impact the entire socioeconomic spectrum (Frieden, 2010).

By affecting the environment in which children of low SES families live, it might be possible to remodel developing habits and attitudes, which might affect lifestyle and therefore health status throughout life. This is exacerbated by lacking evidence on the efficient policies which can actually help reduce health inequalities in the Finnish population (Palosuo et al., 2009). Current evidence is also insufficient to determine the degree to which interventions should include downstream factors directly influencing health and upstream factors that generate social inequalities. In Finnish society, family health clinics, high-quality basic education and income

transfers to families have been major routes to fight growing inequalities (European Anti Poverty Network - Finland, 2018). In addition to the basic structures of the Finnish society, different programs of governmental, non-governmental and private agencies (such as the Finnish Schools on the Move by the Finnish National Agency of Education and the Ministry of Education and Culture and the Smart Family Project by the Finnish Heart Association) have been essential in advising and supporting professionals working with children to encourage favorable health-behaviors in the everyday life of children. In the future, these existing factors and structures of Finnish society should be even better used to recognize and support at-risk families and children, including those with low family SES. In the final evaluation of the Health 2015 public health program, the authors concluded that to succeed in decreasing socioeconomic differences, there is the need to better understood the phenomena behind the differences and their determinants (Rotko et al., 2016). With better knowledge of the root causes of socioeconomic inequalities in health, it would be possible to focus on specific phenomena and influence them by concrete targeted work and cross-sectoral collaboration.

Planning to start a family and the early stages of pregnancy provide an excellent platform for favorably influencing the health behaviors of the family. In Finland, almost all pregnant mothers attend antenatal care since it is provided free of charge and is easily accessible (Sihto et al., 2013). Despite this, Finnish data have shown that under- and non-attendance to antenatal care were associated with lower education of the mother (Raatikainen et al., 2007). These mothers should be perceived and given extra support. Among those attending the prenatal care, those in low SES families could be distinguished, and favorable health behaviors of the family be reinforced by health professionals. However, facilitators and barriers for uptake and maintaining healthy behaviors might differ between families with different SES. A qualitative study showed that among multi-problem families (including socioeconomic, psychosocial and child care problems) the main reasons for not engaging in healthy behavior were costs, higher stress and lack of time (Nagelhout et al., 2012). These barriers among low SES families should be recognized and taken into account in health prevention work of family health clinics such as providing free or low-cost options for physical activity or by intervening in causes of stress.

A public health physician and epidemiologist Martin Tobias asked a question as part of his comment on social rank in health in the Lancet in 2017: “Does this mean that it is no longer enough for us, as doctors, to know about clinical medicine and human biology? Must we in the health professionals also become adept at macroeconomics and sociology?” (Tobias, 2017, 1173) Martin answered the question: “Let us hope so”, and I could not agree more. However, diminishing the socioeconomic gap in health does not succeed only by measures of health

professionals and health policies but health considerations should be integrated also to economical, industrial, educational and employment policies (Ståhl et al., 2006). The main goals of the current governmental program include reducing inequality in health and decreasing poverty especially in families with children (Finnish Government, 2019). These goals are familiar to the programs of previous governments, but this does not invalidate the importance of them as a part of the current governmental program. If anything, the importance has increased due to the unsuccessful efforts during previous governments' terms of office.

In conclusion, this thesis emphasizes the role of early childhood environment and childhood SES in determining future cardiometabolic health. Therefore, the results support the wise words of the former Finnish ombudsman for children, Tuomas Kurttila: "It is the society that pays the high cost of poverty. Child poverty especially is an expensive social problem. We simply cannot afford it "(European Anti Poverty Network - Finland, 2018, 11).

7 Summary/Conclusions

The importance of this thesis is based on the prospective study with long follow-up time and well-phenotyped participants both in childhood and adulthood showing associations of childhood SES with health behaviors, risk factor clustering and subclinical markers of cardiovascular health still decades later in adulthood.

1. Low childhood SES predicts less healthy lifestyle in adulthood in terms of dietary intake, smoking and leisure-time physical activity. Socioeconomic differences are evident already in childhood, where from they appear to persist into adulthood. (Study I)
2. Low childhood SES is associated with higher risk for MetS 27–31 years later in adulthood. Additionally, low childhood SES is associated with higher risk of having IFG or T2DM in adulthood. (Study II)
3. Low childhood SES is associated with increased arterial stiffness over two decades later in adulthood, defined by two well-established markers, PWV and Cdist. In addition, low childhood SES is associated with increased LVM and poorer diastolic function of the heart 31 years later in adulthood. (Study III, V)
4. Lower SES in childhood predicts more adverse cardiometabolic health in adulthood determined by several well-established markers of increased cardiometabolic risk suggesting the role of low childhood SES as an important risk factor of cardiometabolic health, alongside other conventional childhood risk factors (lipids, systolic blood pressure, insulin, BMI, physical activity, frequency of fruit and vegetable consumption and leisure time physical activity) and adulthood SES. (Study I, II, III, IV)

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