TEMPERAMENT, SOCIOECONOMIC STATUS AND CARDIOVASCULAR RISK.

The longitudinal Cardiovascular Risk in Young Finns study.

Laura Pulkki-Råback

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Supervisors:

Professor Liisa Keltikangas-Järvinen
Department of Psychology, University of Helsinki, Finland

Professor Mika Kivimäki
Department of Psychology, University of Helsinki, Finland and
Finnish Institute of Occupational Health

Reviewers:

Professor Jaakko Kaprio
Department of Public Health, University of Helsinki, Finland

Docent Jussi Vahtera
Finnish Institute of Occupational Health

Opponent:

Neil Schneiderman
James L. Knight Professor of Psychology
Departments of Psychology, Medicine, Psychiatry, and Behavioral Sciences, and Biomedical engineering
Director, Behavioral Medicine Research Center
University of Miami, USA

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ABSTRACT

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The present study examined the contribution of socioeconomic status and temperament-related behaviors in childhood and adolescence to cardiovascular disease (CVD) risk factors in adulthood, including health-risk behaviors, metabolic syndrome, and body fat. The participants were derived from the population-based Cardiovascular Risk in Young Finns data (N=3,596). Based on the theoretical framework presented by Gallo and Matthews (2003), it was hypothesized that: a) adverse socioeconomic circumstances may give rise to negative emotions and CVD risk factors, b) temperament-related factors may cause individuals to drift down in the socioeconomic hierarchy and to adopt unhealthy lifestyles, and c) psychological and socioeconomic factors may interact in such a way that psychological factors may have different impacts on CVD depending on the socioeconomic environment in which they are manifested.

The findings supported the hypotheses. Firstly, it was found that exposure to low socioeconomic status in childhood and in adulthood was associated with high levels of cynical hostility and health-compromising behaviors, such as smoking and unhealthy diet, in adulthood. Secondly, child's temperament-related personality characteristics indicating impatience, low responsibility, and negative emotionality (manifested as aggression) predicted adverse socioeconomic trajectories and increased CVD risk in adulthood. Finally, parenting styles characterized as hostile and neglectful were associated with higher levels of metabolic syndrome in children who came from families with low socioeconomic status compared to children who came from families with higher socioeconomic status. These findings suggest that temperament-related characteristics that are manifested as aggressiveness and poor self-control in children may be early indicators of adverse cardiovascular profiles in adulthood. Adverse socioeconomic conditions in early life may increase cardiovascular risk in later life by giving rise to poor health habits and by making children vulnerable to psychosocial stress. As the foundations for cardiovascular risk are laid early in life, the challenge of prevention is to detect the children at high risk in good time and to support these children in order to prevent them from drifting into adverse life trajectories.

Key words:
Temperament, socioeconomic status, cardiovascular risk, health behavior, childhood, longitudinal study

TIIVISTELMÄ

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Temperamentti, sosioekonominen asema ja sydän- ja verisuonitautien riskitekijät. Lasten ja Nuorten Sepelvaltimotautiriski –pitkittäistutkimus.

Tutkimuksessa tarkasteltiin varhaisen sosioekonomisen aseman ja temperamentin yhteyttä myöhempiin sydän- ja verisuonitautien (SVT) riskitekijöihin. SVT-riskin osoittimena käytettiin epäterveitä elämäntapoja, metabolista oireyhtymää ja lihavuutta. Tutkimusaineistona käytettiin kansallisesti edustavaa Lasten ja Nuorten Sepelvaltimotautiriski –aineistoa (N = 3 596), jossa samoja henkilöitä on seurattu lapsuudesta aikuisuuteen. Gallon ja Matthews'in (2003) viitekehyksen mukaan tutkimuksessa oletettiin, että: a) matala varhainen sosioekonomisen asema ennustaa taipumusta kokea negatiivisia tunteita ja korkeaa SVT-riskitekijöiden tasoa aikuisuudessa, b) varhainen temperamentti ennustaa heikkoa sosioekonomista asemaa ja korkeaa SVT- riskitekijöiden tasoa aikuisuudessa, sekä c) psykologiset ja sosioekonomiset tekijät toimivat vuorovaikutuksessa siten, että psykologisten tekijöiden vaikutus riippuu siitä sosioekonomisesta ympäristöstä, jossa ne ilmenevät.

Tulokset tukivat oletuksia. Altistuminen matalalle sosioekonomiselle asemalle elämän varrella oli yhteydessä korkeaan SVT:n riskitekijöiden tasoon kuten kyyniseen vihamielisyyteen, tupakointiin ja epäterveellisiin ruokailutottumuksiin aikuisuudessa. Lapsen temperamentti-piirteistä kärsimättömyys, matala vastuuntunto, ja negatiivinen emotionaalisuus (aggressiivinen käytös) ennustivat heikkoa sosioekonomista asemaa ja korkeaa SVT-riskitekijöiden tasoa aikuisuudessa. Vanhempien vihamielinen kasvatustyyli oli voimakkaammin yhteydessä lapsen metaboliseen oireyhtymään matalan sosioekonomisen taustan kuin korkean sosioekonomisen taustan omaavilla lapsilla. Tulokset viittaavat siihen, että temperamentti-pohjainen käyttäytyminen joka ilmenee aggressiivisuutena ja heikkona itsesäätelynä voi olla merkki myöhemmästä kohonneesta SVT-riskistä. Myös varhain koettu heikko sosioekonominen asema altistaa myöhemmälle SVT-riskille. Lapset, jotka kasvavat matalan sosioekonomisen aseman perheissä perheissä omaksuvat epäterveitä elämäntapoja ja ovat erityisen haavoittuvia psykososiaaliselle stressille. Ennaltaehkäisyn haasteena on tunnistaa nämä riski-lapset hyvissä ajoin ja tukea heitä siten, että heidän ajautumisensa epäsuotuisille elämänurille voidaan katkaista.

Avainsanat:
Temperamentti, sosioekonominen asema, sydän- ja verisuonitaudit, terveyskäyttäytyminen, lapsuus, pitkittäistutkimus

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1. INTRODUCTION

1.1. CARDIOVASCULAR DISEASE (CVD): CALL FOR LONGITUDINAL STUDIES

Cardiovascular disease (CVD) is a major cause of mortality in industrialized countries, accounting for 20,000 annual deaths of which more than 2,000 occur in working-age people in Finland (Statistics Finland, 2001). The prevalence of cardiovascular risk factors such as obesity and non-insulin dependent diabetes mellitus is increasing rapidly (American Diabetes Association, 2000; Mohdad et al., 1999; Ogden, Flegal, Carroll, & Johnson, 2002), and it has even been argued that they represent a potential crisis in terms of health care resources in the future. Therefore, efforts to prevent the development and progression of coronary risk factors are of great public health relevance.

The most common form of CVD is coronary artery disease (CAD), which is almost always due to significant narrowing of coronary arteries caused by atherosclerosis. The most common clinical syndromes are acute myocardial infarction, angina pectoris, and sudden cardiac death (Braunwald, 1992). The atherosclerotic process is known to begin already in childhood (Pathobiological Determinants of Atherosclerosis in Youth (PDAY) Research Group, Berenson et al., 1992; Newman et al., 1986; 1990), and the biological and behavioral risk factors of CVD, such as adverse serum lipid levels, hypertension, obesity, hyperinsulinemia, smoking, and physical inactivity, show significant tracking from childhood to adulthood (Bao, Srinivasan, Wattigney, & Berenson, 1994; Mulder, Ranchor, Sanderman, Bouma, & van den Heuvel, 1998; Porkka & Viikari, 1995; Porkka, Viikari, Taimela, Dahl, & Åkerblom, 1994; Telama, Yang, Laakso, & Viikari, 1997). Taken these facts, childhood seems to be a critical time for the development of later cardiovascular disease.

It is known that CVD is socioeconomically patterned. Individuals who belong to the lowest socioeconomic classes have a high prevalence of cardiovascular diseases and they die from them at an earlier age than individuals in higher socioeconomic classes (G A Kaplan & Keil, 1993; Kunst et al., 1999; Kunst, Groenhof, Machenbach, & EU Working Group on Socioeconomic Inequalities in health, 1998; Marmot, Shipley, & Rose, 1984). Low socioeconomic status is also associated with cardiovascular risk behaviors such as smoking, heavy alcohol use, unhealthy diet, and a sedentary lifestyle (Adler, Marmot, Ewen, & Stewart, 1999; G A Kaplan & Keil, 1993). Despite this well-known fact, attempts to reduce socioeconomic differences in cardiovascular health have not been successful. One reason for this may be the lack of understanding of the complex relationships between socioeconomic factors, behavioral factors, and adverse cardiovascular outcomes.

A substantial body of evidence shows that stress, depression, negative emotions, hostile attitudes,

and poor interpersonal relations are among the psychological factors that contribute to the development of CVD (Kivimäki et al., 2002; Krantz & McCeney, 2003; Kubzansky & Kawachi, 2000; Rozanski, Blumenthal, & Kaplan, 1999; Schneiderman, Antoni, Saab, & Ironson, 2001; Schneiderman, Chesney, & Krantz, 1989; Vahtera et al., 2004). Recently it has been shown that the association between psychosocial factors and cardiovascular disease is universal across different geographic regions and ethnic groups in the world (Rosengren et al., 2004; Yusuf et al., 2004). Much of the research on psychosocial factors and CVD has been conducted in adults, and much less is known about the childhood origins of CVD. Therefore, longitudinal, prospective, designs that include measurements of psychosocial and biological risk factors over different developmental stages are warranted.

While separate lines of research have examined the associations between socioeconomic circumstances and CVD, on the one hand, and psychological factors and CVD, on the other hand, very little attention has been paid to these three elements together. Psychological and socioeconomic factors may be intertwined in several possible ways. Firstly, adverse socioeconomic circumstances may give rise to stress and negative emotions, thereby increasing susceptibility to CVD. Secondly, personality-related factors may cause individuals to drift down in the socioeconomic hierarchy and to adopt unhealthy lifestyles. Thirdly, psychological and socioeconomic factors may interact in such a way that psychological factors may have different impacts on CVD depending on the socioeconomic environment in which they are manifested (Gallo & Matthews, 2003; Taylor & Repetti, 1997; Taylor & Seeman, 1999).

A better understanding of early predictors that affect the etiology of CVD may be critical for prevention of CVD. In the present study, we examined the role of socioeconomic and psychological risk factors in a lifecourse perspective. The study was conducted in the ongoing, population-based, prospective Cardiovascular Risk in Young Finns study that has followed cardiovascular risk factors of the same children and adolescents from childhood into adulthood.

1.2. THEORETICAL FRAMEWORK OF THE STUDY: PERSON AND CONTEXT IN HEALTH OUTCOMES

A common approach in cardiovascular disease etiology has aimed at identifying individual and environmental factors that independently predict the development of cardiovascular disease. This approach largely ignores potential moderated and mediated effects between the individual and environmental factors. Recently, several researchers have suggested that integrating the individual and environmental factors may contribute to understanding of the CVD etiology. The *social ecological theory* (Bronfenbrenner & Morris, 1998; Grzywacz & Fuqua, 2000) contends that health is the result of the interplay between the physical environment, social factors, and individual characteristics that all are intertwined with each other in nested, interacting systems. Instead of seeing the person or the environment as a primary contributor to disease, the social ecological theory sees them as being associated with disease to the extent that they are related to each other. The social ecological theory sees a joint, equal focus on both the person and the environment. Thus, multidisciplinary interventions that consider both the individual and his or her environment are considered most effective in improving public health (Grzywacz & Fuqua, 2000).

Among the most notable models in accordance with the social ecological theory are Taylor and Repetti's (1997) theory of psychosocial resources affecting the socioeconomic gradient in health, and Gallo and Matthew's (2003) dynamic model of socioeconomic and cognitive-emotional factors contributing to cardiovascular health. A common feature in both models is their emphasis on transactions between the person and the environment as an essential element in the disease process. In the model by Taylor and Repetti (1997), a theoretical model of the possible pathways between environments, individual, and health is presented. They suggest that psychosocial characteristics are nested within social environments, and that the larger-level social environment may have an effect on health through mediation or moderation by psychosocial factors. Taylor and Repetti (1997) suggest several hypothetical pathways through which the environments "get under the skin". These pathways include the experience of chronic stress, increased levels of negative emotions, poor coping, and poor health habits. These pathways are not independent or even discrete, but all represent potiential causal chains involving multiple processes.

In theoretizing the pathways leading to CVD, Gallo and Matthews (2003) present a dynamic model of the socioeconomic and cognitive-emotional factors that predict cardiovascular disease and cardiovascular mortality. According to this model, socioeconomic and psychosocial factors are inter-

twined in several possible ways. First, exposure to adverse socioeconomic conditions may influence exposure to stressful events, which, in turn, has a negative impact on emotion and cognition. Negative emotions and cognitions are hypothesized to affect health outcomes through a variety of behavioral and physiological pathways. Secondly, an opposite condition may be true, that is, an individual's dispositions (negative emotions and cognitions) may affect the individual's ability to achieve high social status, which, in turn, has adverse effects on health. Thirdly, adverse environmental conditions may deplete an individual's resources to cope with stress, thereby making the individual susceptible to stress and stress-related health outcomes. To sum up, this model hypothesizes that a) socioeconomic factors may affect health outcomes through personality-related factors, b) personality-related factors may affect socioeconomic position and health, and c) the environment and the individual may have combined, synergistic effects on cardiovascular health.

The theoretical models presented here, that is, the ecological model (Bronfenbrenner & Morris, 1998; Grzywacz & Fuqua, 2000) and the more specific models by Taylor and Repetti (1997), and Gallo & Matthews (2003), have a common feature: they emphasize the reciprocal associations between the person and the environment in the etiology of disease. The present study adopted this approach. The dynamic model by Gallo and Matthews (2003) was adopted as a framework because it attempts to incorporate the literature on socioeconomic factors and personality, and it presents several possible pathways that may be tested only in a longitudinal design. Even though several of the pathways in the model have received empirical support, most of the research has been conducted in cross-sectional designs using middle-aged samples. Far less is known about the childhood roots of socioeconomic inequalities (Chen, Matthews, & Boyce, 2002). In line with the model by Gallo and Matthews, we studied the role of environmental factors (socioeconomic status and a stressful family environment) and temperament-related factors, and their inter-relations, as predictors of CVD risk factors in a longitudinal design by following the same individuals from childhood into early adulthood.

1.3. SOCIOECONOMIC AND ENVIRONMENTAL DETERMINANTS OF CVD

A large body of literature has shown that currently in the Western world, low socioeconomic status is associated with a high prevalence of CVD and its risk factors (Adler et al., 1999; G A Kaplan & Keil, 1993). During the past decade, several studies have adopted a lifecourse approach in an effort to show that the accumulation of socioeconomic exposures at several stages of life has implications for cardiac risk. In the following, we summarize research on the association of socioeconomic status in childhood and adulthood on CVD and its risk factors.

1.3.1. Measures of socioeconomic status

Socioeconomic status (SES) reflects an individual's position in the society with respect to material wealth, social capital, and prestige. SES, sometimes referred to as "socioeconomic class", "socioeconomic position", or "socioeconomic circumstances", covers a wide range of measures. The most common indicators at the individual level are income, education and occupation. Although SES can also be assessed at the level of the neighbourhood, community, or geographic unit, in the present study we concentrate on SES at the individual and at the family level.

Education is a widely used measure of SES in epidemiological studies because it is rather easy to assess, and it is usually fixed after young adulthood and is unlikely to be influenced by poor health later in life. In addition, availability of educational data makes it possible to classify non-working people into socioeconomic groups (Adler & Ostrove, 1999; G A Kaplan & Keil, 1993; Liberatos, Link, & Kelsey, 1988). *Occupational status* ranks people in terms of prestige and social status and is therefore thought to reflect one's position in the social hierarchy. However, occupational status is sensitive to health selection; that is, it may reflect the impact of poor health. Classifications of unemployed persons into occupational categories may also be problematic (Adler & Ostrove, 1999; G A Kaplan & Keil, 1993; Liberatos et al., 1988). *Income* seems to be a valid indicator of access to goods and services, but it is also sensitive to health selection. Measurement of income level is complex due to different family sizes and definitions, and the non-response rate may be high because reporting income is a rather delicate matter (Adler & Ostrove, 1999; G A Kaplan & Keil, 1993; Liberatos et al., 1988). Furthermore, high taxation and a high level of social benefits in a welfare state such as

Finland reduces differences in disposable income between social groups.

In the lifecourse approach, socioeconomic status is considered both in childhood and adulthood. *Intergenerational social mobility* refers to an individual's position in the socioeconomic hierarchy in relation to that of his/her caregivers. Individuals who have achieved a higher socioeconomic position than their caregivers are usually called "upwardly mobile", while those who have drifted down in the socioeconomic hierarchy are called "downwardly mobile". Individuals who have achieved a similar socioeconomic status than their family of origin are usually called "socioeconomically stable".

1.3.2. Lifecourse socioeconomic status and CVD

In the beginning of the 20th century, CVD was a disease of affluent Western people. Between 1940 and 1960, there was a reversal in this pattern, and since then cardiovascular morbidity and mortality have been significantly more common in the lower than in the higher socioeconomic classes (G A Kaplan & Keil, 1993). Socioeconomic differentials in health have persisted during the past decade and in some aspects they may even be widening (Ferrie, Shipley, Davey Smith, Stansfeld, & Marmot, 2002; Osler et al., 2000; Pappas, Queen, Hadden, & Fisher, 1993). In a comparison of 11 European countries, socioeconomic status had a stronger impact on cardiovascular mortality in Finland than in most other countries studied (Kunst et al., 1999; Kunst et al., 1998).

A cornerstone has been the understanding that health-effects of low SES are not only limited to those in extreme poverty, but are relevant at higher levels of SES as well. The seminal work by Marmot and others (1984; 1991), conducted on 17,530 British civil servants, showed that health improved and mortality decreased at each successive step of occupational grade up to the very top. Since then, several studies conducted in countries such as the United States, Britain, and Finland have shown a corresponding graded association between indicators of SES and cardiovascular mortality and morbidity (Davey Smith, Hart, Blane, Gillis, & Hawthorne, 1997; G A Kaplan & Keil, 1993; Lynch et al., 1994; Rogot, Sorlie, Johnson, & Schmitt, 1992; Salomaa et al., 2000; Salonen, 1982).

The lifecourse approach to socioeconomic differences in health suggests that the socioeconomic conditions to which one has previously been exposed have consequences for an individual's health later in life. Anders Forsdahl (1977) was the first to show that poverty in childhood was a risk factor for atherosclerotic disease, especially if it was followed by prosperity. Research done in connection with the Seven Countries Study showed that those born landless in eastern Finland had systematically higher risks for coronary death and coronary heart disease later in life (Notkola, Punsar, Karvonen, & Haapakoski, 1985). Since then, an association between childhood SES and adulthood cardiovascular disease or mortality has been shown in several (Barker & Osmond, 1986; Davey Smith et al., 1997; Davey Smith, Hart, Blane, & Hole, 1998; G A Kaplan & Salonen, 1990; Kuh, Hardy, Langenberg, Richards, & Wadsworth, 2002; Poulton et al., 2002; Wannamathee, Whincup, Shaper, & Walker, 1996), but not all (Elford, Whincup, & Shaper, 1991; Kannisto, Christensen, & Vaupel, 1997; Lynch et al., 1994), studies. In conclusion, the lifecourse approach suggests that instead of examining differences in health status on the basis of an individual's current social position, they should be followed on the basis of socioeconomic exposures at several stages over the lifecourse (Davey Smith, Ben-Shlomo, & Lynch, 2002; Davey Smith et al., 1997; Power, Matthews, & Manor, 1998). Recently, proponents of the lifecourse approach have begun to emphasize the role of prenatal factors in adulthood disease. The lifecourse approach suggests that socioeconomic exposures already at the fetal stage may play a role in an individual's neuroendocrine programming and subsequent risk for disease (Barker, 1995; Ben-Shlomo & Kuh, 2002; Shanks, 2002).

In addition to being associated with CVD events, low SES is associated with lifestyle and biological risk factors for CVD (Lynch, Kaplan, & Salonen, 1997; Poulton et al., 2002; Power & Matthews, 1997; van de Mheen, Stronks, Looman, & Mackenbach, 1998). In the Whitehall study, civil servants who had a lower grade of employment smoked more, exercised less, and had unhealthier dietary practices than those with a higher employment grade (Brunner, Shipley, Blane, Smith, & Marmot, 1999; Marmot et al., 1984; Marmot et al., 1991). Low SES in adulthood has been associated with obesity (Blane et al., 1996; Ferrie et al., 2002; Leino, Raitakari, Porkka, Taimela, & Viikari, 1999; Matthews, Kelsey, Meilahn, Kuller, & Wing, 1989; Räikkönen, Matthews, & Kuller, 1999; Rosmond & Björntorp, 1999; Rosmond, Lapidius, & Björntorp, 1996; Wamala, Mittleman, Schenck-Gustafsson, & Orth-Gomér, 1999; Wing, Matthews, Kuller, Meilahn, & Platinga, 1991), adverse cholesterol levels (Leino et al., 1999; Marmot et al., 1984; Matthews et al., 1989; Wamala et al., 1999), systolic blood pressure (Brunner et al., 1999; Marmot et al., 1984; Marmot et al., 1991; Matthews et al., 1989; Wamala, Wolk, Schenck-Gustafsson, & Orth-Gomér, 1997), and the metabolic syndrome (Brunner et al., 1997). In the Whitehall studies, it has been shown that the traditional coronary-risk factors (biological and lifestyle) partly explain the socioeconomic differences in CVD (Marmot et al., 1984; Marmot et al., 1991). Likewise, Lynch and Kaplan have shown that the association between SES and cardiovascular and all-cause mortality was partly, but not totally, mediated by biological CHD risk factors (Lynch, Kaplan, Cohen, Tuomilehto, & Salonen, 1996).

Importantly, several studies have shown that socioeconomic position in childhood is associated with adult risk factors independently of socioeconomic position in adulthood (Blane et al., 1996; Brunner et al., 1999; Davey Smith et al., 1997; Lawlor, Ebrahim, & Davey Smith, 2002; Poulton et al., 2002). Low childhood SES has been associated with excessive alcohol intake, smoking, unhealthy diet, and lack of physical activity in adulthood (Jefferis, Power, Graham, & Manor, 2004; Leino et al., 1996; Leino et al., 1999; Leino, Raitakari, Porkka, Helenius, & Viikari, 2000; Lynch, Kaplan, & Salonen, 1997; Power & Matthews, 1997; van de Mheen et al., 1998). It has also been shown that exposure to low SES in early life are associated with adult levels of obesity (Brunner et al., 1999; Lawlor et al., 2002; Leino et al., 1999; Leino et al., 2000; Poulton et al., 2002; Power & Matthews, 1997), cholesterol (Blane et al., 1996; Leino et al., 1999; Leino et al., 2000), blood pressure (Blane et al., 1996; Poulton et al., 2002), and the metabolic syndrome (Lawlor et al., 2002). These findings suggest that childhood may be a critical phase during which the foundations for adult risk are laid. Recent findings suggested that the mother's low social class may predispose the fetus to an unfavorable growing environment, thus programming the child's health even before birth (Barker, 1997; Shanks, 2002).

1.3.3. Early-life stressful family environment

Psychosocial stress is probably the most widely acknowledged psychosocial risk factor for CVD (Hemingway & Marmot, 1999; Kivimäki et al., 2002; Mc Ewen & Seeman, 1999; Räikkönen, Keltikangas-Järvinen, Adlercreutz, & Hautanen, 1996; Rosengren et al., 2004; Rozanski et al., 1999; Schneiderman, 1987; Schneiderman & Skyler, 1996; Shepherd et al., 1987; Vahtera et al., 2004). Research consistently suggests that families characterized by certain qualities have damaging outcomes for children's mental and physical health. Repetti, Taylor and Seeman (2002) characterize a "risky family" as having frequent conflicts, recurrent episodes of anger and aggression, and a lack of warmth and support. These families are risky because they leave their children vulnerable to a wide array of mental and physical disorders. Children growing up in risky families are at heightened risk for disease not only because of immediate threats (such as physical abuse), but also because their

environment does not provide the necessary self-regulatory skills to manage with stress. This is manifested as deficits in emotion control and expression, as well as disruptions in physiological and neuroendocrine systems (Repetti et al., 2002).

Parental behaviors and attitudes toward the child generally reflect the family's emotional atmosphere and may create a risky environment to the child (Keltikangas-Järvinen, 2000). It has been shown that the parents' own psychopathology and parental stress contributes to compromised quality of parenting (Tebes, Kaufman, Adnopoz, & Racusin, 2001; Troxel & Matthews, 2004). Parenting may also be affected by adverse socioeconomic conditions; it has been shown that unemployment and economic stress negatively affect parents' well-being and thereby impair the quality of parenting (Kinnunen & Pulkkinen, 1998; Sallinen, Kinnunen, & Rönkä, 2004). A recent Finnish study showed that financial difficulties in the family predicted parents' mental health problems and poor quality of parenting which, in turn, was reflected in their children's internalising and externalising problems (Solantaus, Leinonen, & Punamäki, 2004). Thus, parental attitudes toward their children may be a key factor in how risky the environment of the child is.

While there is a body of literature linking parental behaviors and children's mental health, much less is known about the role of parenting for children's levels of CVD risk factors. Lissau and Sorensen (1994) have shown that parental neglect in childhood predicted obesity in adulthood. The Thousand Families Study has shown that "unsatisfactory" maternal care was associated with health problems in children (Parker et al., 1999). Weidner and others (1992) have shown that the presence of family conflicts predicted adverse serum lipid levels in boys aged 6 to 18. Ravaja and others (2000) found that mothering labelled as "rejecting" was associated with high initial levels of and increases in metabolic syndrome in girls between ages 9 and 12.

However, not all children living in stressful family environments develop health problems, and some children may suffer less from stressful family environments than others. It has been suggested that protective features, such as a resilient temperament, or environmental characteristics, such as high socioeconomic status, may buffer against stressful family interactions (Gallo & Matthews, 2003; Repetti et al., 2002; Taylor & Repetti, 1997). Recently, it has been shown that warm mothering protected children from the adverse effects of SES deprivation (Kim-Cohen, Moffitt, Caspi, & Taylor, 2004). The protective role of genes has also been shown in studies where maltreated children were less likely to develop antisocial behavior if they had a particular monoamine oxidase A enzyme genotype (Caspi et al., 2002), and were less likely to develop depression if they had a particular genotype that confers efficient transport of serotonin (Caspi et al., 2003).

Likewise, it has been suggested that an environmental factor or a personality trait as such is not always sufficient to produce adverse outcomes, but often certain triggers are needed before they turn into risk factors. This individual-environment interaction is seen for instance in a classic animal study which showed that social dominance in cynomolgus monkeys was associated with increased coronary artery atherosclerosis, but only in monkeys living under unstable and stressful social conditions (J R Kaplan, Manuck, Clarkson, Lusso, & Taub, 1982). It has been suggested that CVD results from the synergistic effects of several personality-related and environmental attributes working together and acting as moderators on each other (Schneiderman, 1987; Williams, Barefoot, & Schneiderman, 2003). All in all, it may be hypothesized that family stress alone may not be a determinant of health, but if it is combined with other risk factors, child health may be endangered. Relatively little attention has been paid to the potential buffering effects of third factors against the adverse health consequences of stressful family environments.

1.4. TEMPERAMENT-RELATED PERSONALITY CHARACTERISTICS AS RISK FACTORS FOR CHD

Evidence is accumulating that psychosocial factors contribute to CVD morbidity and mortality, as well as to metabolic and lifestyle risk factors of CVD (Kivimäki et al., 2002; Krantz & McCeney, 2003; Kubzansky & Kawachi, 2000; Rozanski et al., 1999; Schneiderman et al., 2001; Schneiderman et al., 1989; Vahtera et al., 2004). Psychosocial factors start operating already during early years of life, first in the form of caretaker-child interactions and the child's unique temperament characteristics. As the individual grows older, personality in a wider perspective starts to play a role in the individual's health and adaptation. In the following, we summarize research on the role of individual differences in temperament to CVD.

1.4.1. Definition of temperament

From the first day of life, and presumably already before birth, each child has unique behavioral features. This uniqueness can be seen, for instance, by the frequency and intensity of crying, laughter, attention, and sleeping and eating rhythms. These behaviors belong to the concept of temperament. Although temperament is an ancient concept having its roots in ancient Greek philosophy (see Kagan, 1998) scientific research in temperament did not begin until the 20th century. Temperament may be defined as "early emerging personality traits", consisting of biologically rooted individual differences in behavior tendencies that are present early in life and that constitute the basis for later personality (Bates, 1989; Buss & Plomin, 1984; Rothbart & Bates, 1998; Taylor & Seeman, 1999). Temperament is seen as the observed behavioral pattern, or stylistic differences, in behaviors that differentiate individuals from one another (Goldsmith et al., 1987; Thomas & Chess, 1977).

Among the influential pioneers on research on temperament are Thomas, Chess, Birch, Hertzig, and Korn (1963) with their New York Longitudinal study which examined individual differences in what they called the primary reaction patterns of infants 3 to 6 months of age. Since then, several models of childhood temperament have emerged. Some models of temperament focus more on environmental influences and environment-individual transactions in the formation of temperament, while some models focus more on the genetic and neurological basis of behavior. Despite subtle differences between the different models of temperament, there exists agreement over several factors:

a) temperament has a genetic basis, b) temperament reflects individual differences in the autonomic nervous system function and the brain's neuroendocrinological functions, c) emotional experience and emotional regulation are intrinsic to temperament, d) temperament has significant stability and it is the core of later personality (Bates, 1989; Buss & Plomin, 1984; Caspi, 1998; Costa & McRae, 2001; Goldsmith et al., 1987; Rothbart & Bates, 1998).

1.4.2. Theoretical framework of temperament

In the following, we present three approaches to temperament that we have adopted in the present study. These are the temperament theory by Buss and Plomin (1975; 1984), the concept of difficult temperament (Thomas, Chess, & Korn, 1968), and the Type A behavior pattern as a temperament-related personality construct (M Friedman & Rosenman, 1974). The theory put forth by Buss and Plomin (1975; 1984) represents a widely accepted view of temperament which encompasses the most salient temperament dimensions that exist almost in every model of temperament. In this model, the three dimensions that constitute temperament are emotionality, activity, and sociability. These dimensions are seen as inherited early personality traits that appear already in infancy. *Emotionality* comprises of negative emotions and a tendency to become easily upset, distressed, and aroused in the face of threatening, annoying, or frustrating situations. *Activity* comprises of the frequency and intensity of motor responses. Active individuals have a high energy level and they engage in activities with high tempo and vigor. Individuals high in *sociability* prefer being with people and are highly responsive to others. Sociable individuals approach new situations with excitement, and they tend to be high in positive emotionality (Buss & Plomin, 1975, 1984). The model excludes personality differences that are not shown to have a strong genetic component. Thus, skills, habits, interests, values, moral, attitudes, and related characteristics do not belong to their concept of temperament. Nor does the model include different forms of psychopathology, because it is regarded to measure *variations in normal behavior* (Buss & Plomin, 1984).

The concept of *difficult temperament* (Thomas et al., 1968) may be useful in explaining why some children seem to be in constant problems with the environment, while others seem to be easy and well-adjusted. Difficult temperament may be characterized as the child's behavioral tendencies that are challenging for the caretakers. A difficult child may be described as having frequent negative mood, adapting slowly to new situations, expressing anger intensively, and having irregular rhythms. Using the Buss and Plomin's (1975; 1984) theory, temperamental difficulty is the combination of high emotionality, extreme activity (hyperactivity), and low sociability (Katainen, 1999). The difficult temperament constellation has been criticized for putting too much weight on the child and not taking into account that ultimately the environment defines which behaviors are seen as difficult, and which are seen as adaptive (Goldsmith et al., 1987). While acknowledging this limitation, the difficult temperament constellation may be helpful in integrating different research findings on several temperament dimensions under one construct. Another advantage of the difficult temperament construct is the fact that it has been shown to be clinically relevant. The components of difficult temperament have been associated with adjustment problems, such as externalizing/internalizing problems, attention deficit hyperactivity disorder, and depression (reviewed in Caspi, 1998; Rothbart & Bates, 1998). Even though temperament may assess early subclinical manifestations of later behavioral disorders, the present study has adopted the traditional view of temperament traits as variations in normal behavior (Buss & Plomin, 1984; Caspi, 1998).

Type A behavior pattern is conceptually close to temperament and has its origins in childhood temperament (Keltikangas-Järvinen & Räikkönen, 1990a; MacEvoy et al., 1988; Räikkönen & Keltikan-

gas-Järvinen, 1992; Steinberg, 1985). This much studied personality construct in cardiovascular research was introduced in the 1950's by the cardiologists Meyer Friedman and Ray Rosenman (1959) who noted that their cardiac patients had common behavioral characteristics such as high aggressiveness, impatience, time urgency, and excessive competitive achievement-striving. They described the Type A behavior pattern as "an action-emotion complex in persons who are aggressively involved in chronic, incessant struggle to achieve more and more in less and less time, and if required to do so against the opposing efforts of other things or other persons" (M Friedman & Rosenman, 1974). The first prospective evidence for the significance of the Type A behavior in cardiovascular disease came from two studies: the Western Collaborative Group Study (Rosenman et al., 1975) and the Framingham Heart Study (Haynes, Feinleib, & Kannel, 1980). During the 1960's and 1970's the evidence linking Type A behavior to CHD became so convincing that the distinguished Review Panel on Coronary-Prone Behavior and Coronary Heart Disease listed Type A as a risk factor for heart disease of equal magnitude to the traditional risk factors such as smoking, elevated serum cholesterol, and hypertension (Review Panel on Coronary-Prone Behavior and coronary heart disease, 1981). However, in the 1980's, a body of research emerged showing that Type A behavior was *not* related to cardiac disease (Dembroski & Williams, 1989; H S Friedman & Booth-Kewley, 1987; Matthews & Haynes, 1986). Perhaps the most important reason for the null-findings, however, was the long-ignored fact that, instead of being a single construct, Type A behavior is comprised of several subcomponents that may work in opposite directions. Some components may be even cardioprotective, while other components may be more "toxic" (Dembroski & Williams, 1989; Matthews, 1988; Matthews & Haynes, 1986). Current research favors the use of multidimensional Type A behavior measures instead of using Type A as a global construct (Matthews, 1988; Matthews & Haynes, 1986).

Even though it has been noted that there are considerable similarities between Type A behavior and temperament (Matthews, Rosenman, Dembroski, Harris, & MacDougall, 1984; Steinberg, 1985), these two behavioral constructs have been studied rather separately from each other in two different lines of research. Type A behavior has been a research topic in behavioral medicine and medical research tradition, while temperament is a much-studied construct in developmental psychology. It has been ignored that ultimately they may both represent the same phenomenon because, by definition, both are reaction patterns that differentiate people from each other and that are manifested in overt behaviors (Bates, 1989; M Friedman & Rosenman, 1974). The dimensions of Type A behavior and temperament have significant overlap, and the most central components of the Type A behavior (eg., impatience, irritability, aggression) pattern are included into almost every temperament theory. Furthermore, Type A behaviors are known to have their origins in early temperament (Carmelli, Rosenman, Chesney et al., 1988; MacEvoy et al., 1988; Räikkönen & Keltikangas-Järvinen, 1992). In addition, both temperament and Type A behaviors are known to have significant heritability (Buss & Plomin, 1984; Matthews et al., 1984; Pedersen et al., 1989; Rothbart & Bates, 1998) and stability (Bates, 1989; Bergman & Magnusson, 1986; Carmelli, Rosenman, & Chesney, 1987; Keltikangas-Järvinen, 1989; Rothbart, Ahadi, & Evans, 2000) over different age periods. In the present study, we concentrate on childhood and adolescent temperament-related behaviors (including Type A behaviors) as predictors of socioeconomic status and cardiovascular risk factors.

1.4.3. Childhood temperament and CVD risk factors

The model by Gallo and Matthews (2003) suggests that negative emotions may lead to physiological alterations which are responsible for the ultimate cardiovascular endpoints. Temperament seems to be an appropriate concept for studying the childhood origins of cardiovascular disease for at least two reasons.

Firstly, negative emotions in adulthood have been associated with cardiovascular disease (Kubzansky & Kawachi, 2000; Rozanski et al., 1999). As emotions are a central component of the concept of temperament, it may be a useful concept when studying the early-life origins of cardiovascular disease. Secondly, the fact that temperament reflects individual differences in autonomic nervous system and the brain's neuroendocrinological functions makes it especially interesting for studying the etiology of cardiovascular disease. It has been suggested that sympathetic nervous system activation and neuroendocrinological functions play central mediating roles in the associations of psychosocial factors with the development of CVD (Manuck, Marsland, Kaplan, & Williams, 1995; Schneiderman & Skyler, 1996). Thus, temperament and cardiovascular disease may have their origins in similar physiological functions, and understanding their relations may enhance our understanding of the mechanisms between behavioral factors and cardiovascular disease.

Much of the research on the relationship between childhood temperament and cardiovascular risk has been conducted in the Cardiovascular Risk in Young Finns data. To summarize these findings, it seems that child difficulty is a coronary-prone temperament trait. Ravaja and Keltikangas-Järvinen (1995) have shown that negative emotionality (aggression and anger) and hyperactivity predicted a high level of the metabolic syndrome in a 3-year follow-up in children and adolescents, while Ravaja, Katainen, and Keltikangas-Järvinen (2000) have shown that the difficult temperament cluster (consisting of high negative emotionality, hyperactivity, and low sociability) predicted the metabolic syndrome in a 3-year follow-up. Furthermore, hyperactivity in childhood has been found to be associated with the apoE phenotypes E3/4 and E4/4 which are known to be the most coronary-prone apoE phenotypes (Ravaja et al., 2000). On the other hand, such temperament characteristics as sociability and positive emotionality have been shown to be protective temperament characteristics against coronary risk factors (Ravaja & Keltikangas-Järvinen, 1995).

There is also evidence suggesting that temperament may have significance to obesity which, in turn, is a significant CVD risk factor (Parsons, Power, Logan, & Summerbell, 1999). Negative mood has been found to predict increased weight gain in infants between 6 and 12 months of age (Carey, 1985), and easily distressed infants who are difficult to soothe have been found to have higher skinfold thickness than others (J C K Wells et al., 1997). Maternally-rated hyperactivity, unpredictability and low attention span have been associated with excess weight gain in middle childhood (Carey, Hegvik, & McDevitt, 1988). A high level of negative emotionality and a lack of energy have been found to correlate with body mass in boys aged 9 to 15 (Ravaja & Keltikangas-Järvinen, 1995), and a high level of hostile affect and aggressiveness have been shown to predict increased body mass index in adolescents in a 3-year follow-up (Räikkönen, Matthews, & Salomon, 2003). These findings suggest that childhood temperament might be an early marker of subsequent overweight and obesity. However, to our best knowledge, no previous study has prospectively related childhood temperament to *adulthood* body mass or body fat distribution. The present study examined this question.

Using the Type A behavior construct in children and adolescents, it has been found that high scores in global Type A behavior are associated with a high level of physiological coronary risk factors and increased cardiovascular reactivity (Hunter et al., 1982; Matthews & Jennings, 1984; J M Siegel & Leitch, 1981), but there also is evidence showing no associations between Type A behavior and physiological coronary risk factors in children (Lee, Gomez-Marin, & Prineas, 1996; Weidner, McLellarn, Sexton, Istvan, & Connor, 1986). In the Cardiovascular Risk in Young Finns data, it has been found that some dimensions were associated with adverse levels of CVD risk factors, while other dimensions seemed to be protective against CVD risk factors. Type A components of impatience and aggressiveness have been shown to be related to a multitude of CVD risk factors such as poor school achievement, adverse lipid profiles, high systolic blood pressure, and high levels of the parameters of the metabolic syndrome (Keltikangas-Järvinen, 1992; Keltikangas-Järvinen & Räikkönen, 1989, 1990a, 1990b; Ravaja, Keltikangas-Järvinen, & Keskivaara, 1996). In

contrast, Type A components indicating leadership and responsibility have been associated with positive outcomes such as high school grades, low level of the metabolic syndrome parameters, and benign levels of serum lipids (Keltikangas-Järvinen, 1992; Keltikangas-Järvinen & Räikkönen, 1989, 1990a, 1990b; Ravaja et al., 1996). Inspired by these findings, the Type A components indicating impatience and aggression were named as "pathogenic", and the components of leadership and responsibility were named as "protective" (Keltikangas-Järvinen & Räikkönen, 1989). It is worth noting that in observable child behavior, pathogenic Type A behavior (including impatience and aggression) is almost equivocal to a difficult temperament (hyperactivity and negative emotionality). The core characteristics in both pathogenic Type A behavior and difficult temperament seem to be the child's aggression and poor self-control. On the other hand, the protective Type A components and the protective temperament characteristics have in common a high level of sociability and positive emotionality of the child.

In addition to studies linking temperament and Type A behavior with metabolic risk factors for CVD, there are studies that relate childhood temperament with health compromising behaviors in adulthood. The Dunedin Multidisciplinary Health and Development Study has shown that children who were undercontrolled (impulsive, irritable, emotional) at the age of 3 were significantly more likely to be involved in multiple health-risk behaviors such as alcohol dependence and unsafe sex at the age of 21 (Caspi et al., 1997). The Jyväskylä Longitudinal Study of Personality and Social Development has shown that aggression in childhood was related to problem drinking and long-term unemployment in adulthood (Kokko & Pulkkinen, 2000; Pulkkinen & Pitkänen, 1994). Barman and others (2004) have shown in the FinnTwin12 data that the temperament trait "inattentiveness" at the age of 11-12 predicted smoking experimentation and current smoking at the age of 14. Yang and others (1998; 2000) have shown that low scores on a temperament-related character called "responsibility" in adolescence predicted drop-out from organized sports and hobbies including physical activity. Furthermore, longitudinal studies have shown that childhood aggressions and impulsivity predict onset of alcohol abuse, smoking, and risky lifestyles (Cooper, Wood, & Orcutt, 2003; Dobkin, Tremblay, Mâsse, & Vitaro, 1995; Masse & Tremblay, 1997; Wills, DuHamel, & Vaccaro, 1995).

To sum up these findings, it seems that *poor impulse control and negative emotionality* are the key temperament factors predicting adherence to risky lifestyles. These behaviors are central components of the difficult temperament constellation, indicating poor manageability of the child. As shown by the abovementioned findings, childhood temperament has been connected with cardiovascular risk factors in childhood and adolescence, but much less is known about the predictive role of childhood temperament for cardiovascular risk factor in adulthood. Furthermore, population-based studies extending from childhood to adulthood are lacking. Obviously there is a need for longitudinal studies on the influence of childhood temperament on adulthood CVD risk.

1.4.4. Adult temperament-related personality characteristics and CVD

Of behavioral characteristics in adulthood, stress, depression, hopelessness, hostile attitudes, and poor interpersonal relations are known to be risk factors for CVD (Kivimäki et al., 2002; Krantz & McCeney, 2003; Kubzansky & Kawachi, 2000; Rozanski et al., 1999; Schneiderman et al., 2001; Schneiderman et al., 1989; Vahtera et al., 2004). As the experience of negative emotions is a common factor for all the aforementioned characteristics, it has been suggested that negative emotionality may be a central personality-related factor contributing to CVD (Gallo & Matthews, 2003; Kubzansky & Kawachi, 2000). *Hostility* is a much studied construct in cardiovascular research that is closely associated with temperament and negative emotions (Cates, Houston, Vavak, Grawford, & Uttley, 1993).

Hostility, which may be defined as the coronary-prone element in Type A behavior (Matthews & Haynes, 1986), has been defined as comprising and individual's relatively stable cognitive, affective, and behavioral tendencies to experience and manifest negative emotions (T W Smith, 1992). Hostility includes the experience of anger and irritability, negative cognitions about others, and overt behaviors such as physical or verbal aggression (Miller, Smith, Turner, Guijarro, & Hallet, 1996; T W Smith, 1992). Often the term hostility is used to refer to cynical attitudes, reflecting an expectancy that other people are motivated by selfishness and that people are likely to hurt each other (Miller et al., 1996; T W Smith & Ruiz, 2002). Traditionally, hostility has been assessed by either a paper-and-pencil questionnaire or a structured interview. Of the paper-and-pencil methods, the Cook-Medley Ho scale from the Minnesota Multiphasic Personality Inventory (Cook & Medley, 1954) is the most used one. The Ho scale assesses several dimensions of hostility, reflecting most strongly the cognitive component of hostility including cynical mistrust as its core construct (Barefoot & Lipkus, 1994).

Having its roots in childhood temperament and being based on a "constitutional disposition" (Krantz & Durel), hostility may be viewed as a temperament-related personality character (Cates et al., 1993). The association of hostility with temperament becomes relevant given that the temperament theory of Buss and Plomin (1975) views hostility as the cognitive component of negative emotionality. Hostility may also be seen as a component of the Big Five Neuroticism factor (Caspi, 1998). Hostility has been shown to be rather stable across time among children and adolescents (Woodall & Matthews, 1993), and the Ho scale has been shown to have significant stability in adults (Barefoot, Dahlström, & Williams, 1983; Siegler et al.). Genetic factors play a role in the development of hostility (Kaprio et al., 1995; Rose, 1988), and it has been suggested that the cynicism subscale may be the one with highest genetic heritability (Carmelli, Rosenman, & Swan, 1988; Weidner et al., 2000). Moreover, adulthood hostility is predicted by childhood difficult temperament (Keltikangas-Järvinen, 2000; Räikkönen, Katainen, Keskivaara, & Keltikangas-Järvinen, 2000). To sum up, this evidence suggests that hostility is a personality characteristics with strong temperament-related origins.

Cynical hostility has been shown to be a cardiovascular risk factor in most (Hemingway & Marmot, 1999), but not all (Hearn, Murray, & Luepker, 1989; Helmer, Ragland, & Syme, 1991; Leon, Finn, Murray, & Bailey, 1988), prospective studies. Cynical hostility predicts myocardial infarction (Barefoot, Larsen, von der Lieth, & Schroll, 1995; Everson et al., 1997; Koskenvuo et al., 1988), coronary heart disease incidence (Barefoot et al., 1983; Barefoot et al., 1995; Niaura et al., 2002), total and coronary mortality (Everson et al., 1997), and the extent of coronary atherosclerosis (Iribarren et al., 2000; Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994). In their review on several psychosocial coronary risk factors, Hemingway and Marmot (1999) posit that hostility is a robust predictor of cardiovascular disease. In addition to being associated with clinical CVD events, cynical hostility is associated with several risk factors for CVD. Research has shown that cynical people smoke more, drink alcohol more heavily, have unhealthier diets, and are more sedentary than their non-hostile counterparts (Houston & Vavak, 1991; Niaura et al., 2000; Räikkönen & Keltikangas-Järvinen, 1991; Romanov et al., 1994; L W Scherwitz et al., 1992; Siegler, Peterson, Barefoot, & Williams, 1992). Everson and others (1997) showed that health behaviors were to a significant degree responsible for the association of cynical hostility with mortality and myocardial infarction.

As reviewed exhaustively, there are several pathways whereby hostility may be associated with cardiac disease (Miller et al., 1996; T W Smith, 1992, 1994). Firstly, the association between hostility and CVD may be mediated by psycho-physiological pathways. Hostile persons tend to respond to novel situations with larger psycho-physiological reactivity, including sympathetic nervous system activity and release of stress-related hormones, including epinephrine, norepinephrine, cortisol, glucagons, and growth hormones (Manuck, Kasparowicz, Monroe, Larkin, & Kaplan, 1989; Manuck

et al., 1995; Schneiderman, 1987; Schneiderman & Skyler, 1996). It has been suggested that these stress hormones mobilize lipid strores from adipose tissues. If not removed by vigorous physical activity, the free fatty acids are not cleared as rapidly, and they may become converted to triglycerides and subsequently to LDL cholesterol which, in turn, is pathogenic for CHD (Howard, Schneiderman, Falkner, Haffner, & Laws, 1993; Schneiderman & Skyler, 1996). Secondly, hostile persons are likely to experience a taxing interpersonal environment with low social support and high levels of interpersonal conflicts (Kivimäki et al., 2003; Kivimäki, Vahtera, Koskenvuo, Uutela, & Pentti, 1998). Therefore, hostile individuals are likely to create, by their own actions, an unsupportive environment, which, in turn, is likely to make the hostile individual even more vulnerable to stressors (Kivimäki et al., 1998; T W Smith, 1992; Vahtera, Kivimäki, Uutela, & Pentti, 2000). Thirdly, health-related behaviors may mediate the association between hostility and CVD as shown by Everson and others (1997). Finally, there may be some basic biological factor, for instance related to the central nervous system, which causes both hostility and increased CHD risk (Miller et al., 1996; T W Smith, 1994). This so-called "constitutional hypothesis" is acknowledged by the literature but it has been tested relatively little.

1.5. RECIPROCAL ASSOCIATIONS BETWEEN SOCIOECONOMIC STATUS AND TEMPERAMENT

As reviewed above, there have traditionally been two separate lines of research on the factors that contribute to increased CVD risk, the one focusing on the association of socioeconomic factors on CVD, the other addressing psychosocial risk factors for CVD. However, it is conceivable that some of these socioeconomic and psychosocial factors may be related to each other. Not until recently have attempts been made to integrate these two lines of research (Gallo & Matthews, 2003). Following the framework proposed by Gallo and Matthews (2003), we elaborate three different possible pathways whereby psychosocial factors and socioeconomic circumstances may act together or reciprocally to increase cardiovascular risk. Firstly, socioeconomic status may have an impact on the development of temperament-related personality factors, which, in turn, may confer risk for CVD. Secondly, early emerging temperament-related factors may contribute to one's occupational success and socioeconomic trajectories, thereby leading to poor cardiovascular health. Thirdly, the effects of the socioeconomic environment on an individual's health may differ as a function of some third factor, which may protect from, or make the individual more susceptible to, cardiovascular disease.

1.5.1. Socioeconomic status as a predictor of negative emotions

Adverse socioeconomic conditions in early life have been suggested to kindle disproportionate levels of negative emotions, negative attitudes, and even psychiatric symptoms. Gallo and Matthews (2003) suggest that because low SES individuals are exposed to a high number of chronic and acute psychosocial stressors, they consequently develop negative emotions and cynical attitudes towards the world and other people. These negative emotions may be an important pathway through which low SES affects later cardiovascular disease.

Indeed, several studies have shown that low socioeconomic status in childhood and in adulthood is associated with negative psychosocial orientations such as depression and hostility. In the Kuopio Ischemic Heart Disease Risk Factor study it has been shown that men with both parents who had less than a primary school education or who both had unskilled manual jobs had higher levels of cynical hostility, hopelessness, and depressive symptoms in adulthood than men whose parents had higher educational or occupational status (Harper et al., 2002). In the same study group, low parents'

socioeconomic position was associated with poor cognitive functioning in adulthood (G A Kaplan et al., 2001). In the Almeda County Study it has been shown that sustained economic hardship was associated with high levels of depression and cynical hostility, and reduced cognitive functioning (Lynch, Kaplan, & Shema, 1997). Furthermore, Bosma and others (1999) have shown in a Dutch population that low social class in childhood was associated with unfavorable personality profiles in adulthood, including high levels of neuroticism and lack of future orientation. Furthermore, several studies have shown an association between low SES and high levels of hostility (Barefoot et al., 1991; Elovainio, Kivimäki, Kortteinen, & Tuomikoski, 2001; Haukkala, 2002; L Scherwitz et al., 1991). These negative life orientations have been shown to partly explain associations between low SES and increased risk for CVD (Bosma et al., 1999; Cohen, Kaplan, & Salonen, 1999; Wamala et al., 1999).

Because previous research shows that low SES is associated with cynical attitudes, on one hand, and with cardiovascular risk behaviors, on the other, it seems reasonable to hypothesize that exposures to adverse socioeconomic conditions earlier in life may be a potential "third factor" that explains the covariance of negative psychosocial factors and risk for disease later in life. The present study examined whether the association between cynical hostility and cardiovascular risk behaviors would be attributable to socioeconomic experiences at different stages of the lifecourse.

1.5.2. Temperament characteristics as contributors to downward socioeconomic drift

Contrary to the view that low SES causes adverse psychological states, the opposite may also be true, that is, psychological states may contribute to one's socioeconomic position. The *social drift hypothesis* suggests that psychological disorders may impair one's ability to attain higher socioeconomic position or they may cause one to drift down in the socioeconomic hierarchy. As shown by the social drift -path in the Gallo and Matthews (2003) model, cognitive and emotional factors may impair one's ability to attain a higher social class. While it seems understandable that severe psychiatric disorders such as schizophrenia and severe depression impair an individual's socioeconomic success (B P Dohrenwerd, 1990; Kessler, Foster, Saunders, & Stang, 1995), it has been shown that even variations within the range of normal childhood behaviors may thrust individuals into unfortunate life trajectories. Because childhood is a critical time for the development of CHD, childhood temperament would be worth studying in this context.

Prospective studies have shown that childhood temperament-related characteristics may be risk factors for downward socioeconomic drift. Caspi, Elder and Bem (1987) have shown that aggressive and impatient children with negative emotions and frequent explosive tantrums were likely to drop out of school, become unemployed and have high levels of substance use as adults. The Jyväskylä longitudinal study has shown that aggressiveness in childhood predicted long-term unemployment, career instability, low career orientation, and divorce in adulthood (Kinnunen & Pulkkinen, 2003; Kokko & Pulkkinen, 2000; Pulkkinen, Ohranen, & Tolvanen, 1999; Rönkä, Kinnunen, & Pulkkinen, 2000). Tremblay and others (Masse & Tremblay, 1997; Tremblay, Masse, & Leblanc, 1992; Tremblay, Phil, Vitaro, & Dobkin, 1994) have shown that hyperactive children were at risk for poor school achievements, antisocial behaviors, and substance abuse later in life. A recent study by Keltikangas-Järvinen and others (2003) showed that impulsivity was a risk factor for dropping out of higher education. Altogether, these findings suggest that certain temperamental characteristics, especially those related to negative emotions and hyperactivity, may set forth a lifelong cycle of mal-adaptation which leads to an accumulation of several adulthood risks.

As these same temperamental traits have been acknowledged as coronary risk factors (as summarized in chapter 1.4.), it is reasonable to hypothesize that individuals with a certain personality type are more at risk for the accumulation of multiple health risks. Thus, the co-occurrence of low SES and CVD risk may be the common end point resulting from early problematic personality, including features such as aggression and poor self-control. We examined whether the association between educational level and cardiovascular risk behaviors in adulthood would have a common denominator in childhood temperament.

1.5.3. Interactive effects between socioeconomic status and psychosocial factors

Instead of independently influencing negative outcomes, it is possible that socioeconomic and psychosocial factors work together (in interaction) in producing later CVD risk. It has been suggested that given a similar stressor, people with low SES are more vulnerable to it than individuals with higher SES (Adler et al., 1996; McLeod & Kessler, 1990; Stronks, Van de Mheen, Looman, & Mackenbach, 1998). SES may *moderate* the impact of psychological factors on CVD so that individuals with low SES suffer more from additional stressors than their counterparts with higher SES. This may be due to the fact that individuals with low SES are less well equipped to cope with stressors because of a lack of personal control, inadequate social support, a high number of interpersonal conflicts, and limited cognitive resources (Adler et al., 1996; Gallo & Matthews, 2003; McLeod & Kessler, 1990; Steptoe & Marmot, 2003; Stronks et al., 1998; Taylor & Repetti, 1997). In Gallo and Matthew's (2003) terminology, individuals with low SES have a smaller bank of resources to deal with stressful events compared to those with higher SES, and are therefore more affected by stress.

In a Finnish study, psychosocial stress affected the progression of carotid atherosclerosis more strongly in individuals with low SES than in those with high SES (Lynch, Everson, Kaplan, Salonen, & Salonen, 1998). In another study, negative emotions affected ambulatory blood pressure more strongly in individuals with low occupational prestige than in individuals with high occupational prestige (Matthews et al., 2000). A series of studies conducted by Karen Matthews and Edith Chen has shown that adults and children with lower SES interpret life events as threatening and they show more intense emotional and cardiovascular reactions to these potential threats than individuals with higher SES (Chen & Matthews, 2001; Gump, Matthews, & Räikkönen, 1999; Matthews, Gump, Block, & Allen, 1997; Matthews et al., 2000). This evidence suggests that low SES is a predisposing factor that may increase vulnerability to stress and thereby increase disease-proneness.

It has been shown that some children may be protected against an adverse environment because of their genetic makeup. Recently, it has been shown that both genetic and environmental factors increase children's resilience toward socioeconomic deprivation. Maternal warmth, stimulating activities, and the child's outgoing temperament promoted positive adjustment in children exposed to low SES (Kim-Cohen et al., 2004). A certain genetic constitution has been shown to protect the child against the adverse consequences of child maltreatment (Caspi et al., 2002; Caspi et al., 2003). As reviewed by McClearn (2004), the influence of environmental factors may depend on genotype, or, alternatively, the differential effect of genotype may depend upon the environment.

Even though it is known that stress affects children differently, depending on other protective features in the environment, no previous study has examined whether this is also true for predicting cardiovascular risk factors - such as cholesterol, relative weight, and insulin levels - in children. Examining this question could help in understanding the early environmental roots for future cardiac disease. Thus we examined whether children from low SES would be somatically more vulnerable to family stress, as measured by the mother's hostile attitudes toward the child.

1.6. AIMS OF THE STUDY

1.6.1. Hypothetical framework of the study

Arising from the model by Gallo and Matthews (2003), we present a lifecourse framework for studying socioeconomic and psychosocial influences on cardiovascular disease (Figure 1). The model suggests that adverse socioeconomic circumstances, a negative family atmosphere (manifested as poor parenting), and temperament-related factors in childhood may give rise to CVD risk factors later in life, either independently or by working jointly. There may also be covariance between the childhood factors, that is, SES in childhood, child's temperament, and parental responses to child's behavior. Low socioeoconomic status may make the individual more vulnerable to additional stressors, thereby working in interaction with psychological factors. The model suggests that the origins of adult CVD lie in early-life exposures, and that the combined effects of several adversities may be more detrimental in terms of cardiovascular risk than exposure to only one adversity.

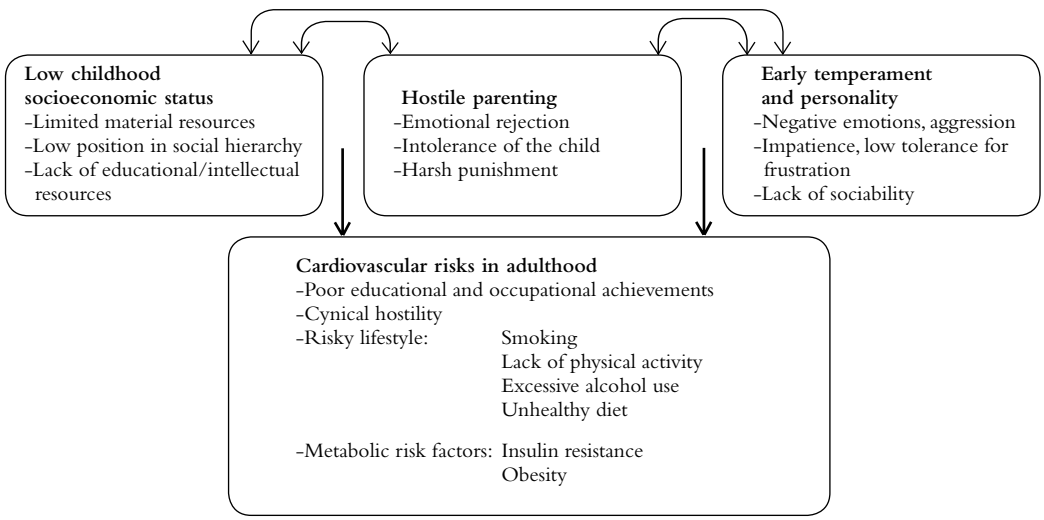

Figure 1

A lifecourse framework for socioeconomic and psychosocial influences on cardiovascular disease.

1.6.2. Research questions

The study is divided into the following three main research questions. Figure 2 shows how each study question was operationalized into separate studies.

1. First, we examined whether low SES in childhood is an underlying factor responsible for the associations between cynical hostility and cardiovascular risk behaviors (smoking, alcohol use, unhealthy diet, sedentary lifestyle) in adulthood. We hypothesized that individuals who have had a poor socioeconomic start in life have more cynical attitudes and unhealthier lifestyles in adulthood, and that early SES would explain later associations between cynical hostility and cardiac risk behaviors.

2. Secondly, we tested the assumption that temperament-related personality characteristics underlie subsequent negative socioeconomic outcomes and cardiovascular risk factors. We examined

a. Whether components of Type A behavior (including impatience, aggression-competitiveness, engagement-involvement, and hard-driving) in adolescence and early adulthood explain the associations between low educational level and cardiovascular risk behaviors (smoking, diet, alcohol consumption, physical inactivity) in adulthood.
b. Whether temperament characteristics in childhood are able to predict body mass and central adiposity 18 years later in adulthood.

We hypothesized that characteristics indicating a high level of negative emotionality and impulsivity would predict lower level of education and a high level of health-risk behaviors in adulthood. We also hypothesized that children with a high level of negative emotionality would have higher body mass as adults than children with lower levels of negative emotionality.

3. Thirdly, we asked whether children's levels of metabolic cardiovascular risk factors were differently affected by poor quality of parenting, depending on the socioeconomic situation of the child. We hypothesized that SES may act as a moderator so that children from low SES backgrounds are more affected by poor parenting than children from higher SES backgrounds.

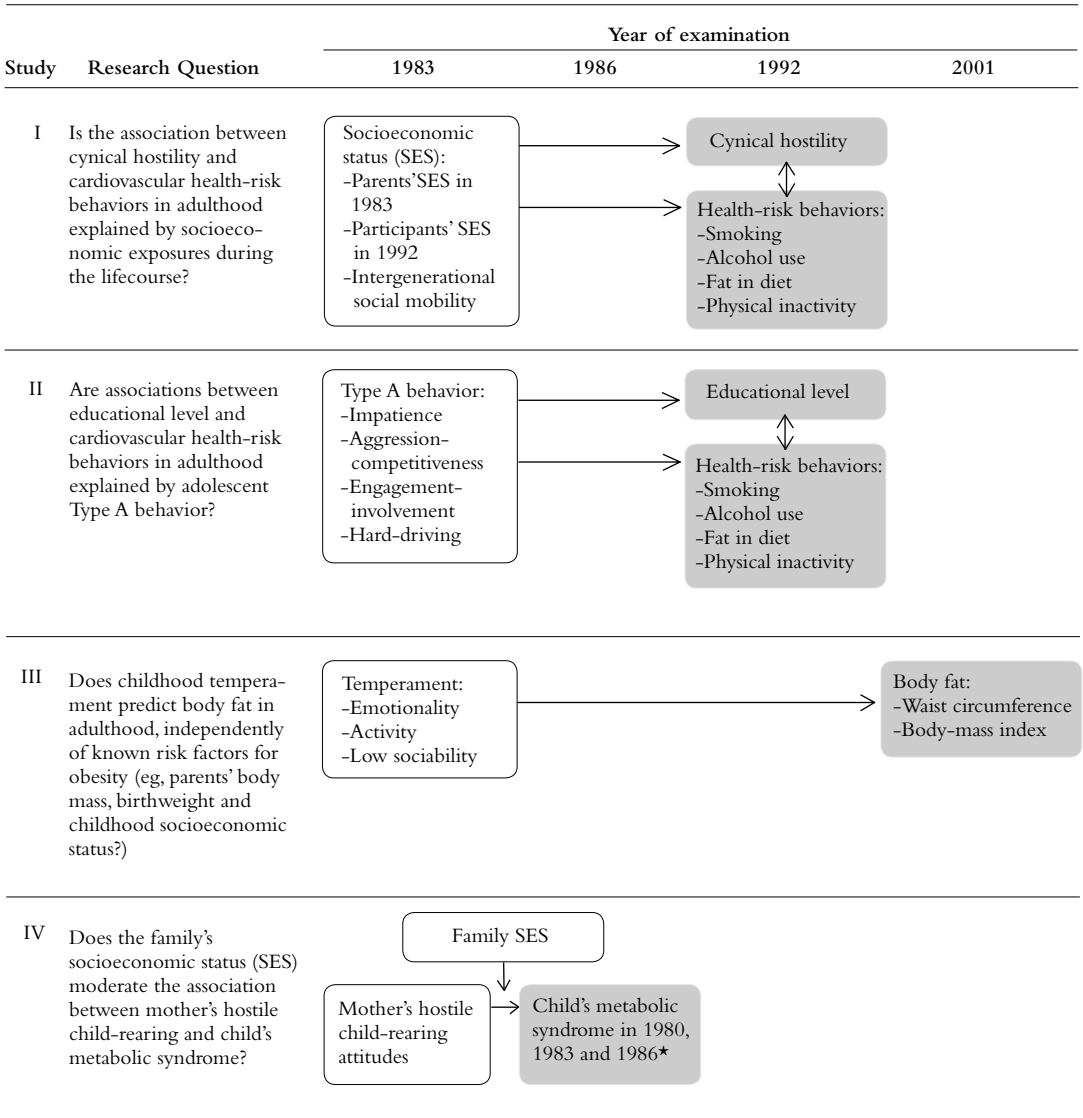

Figure 2.
Research questions and designs of studies I-IV. Predictor variables are in white boxes and outcome variables are in gray boxes.
Note.
Table does not show variables that were used as covariates in the studies.

* Consists of systolic blood pressure, body-mass index, serum triglycerides, serum insulin, and high-density lipoprotein cholesterol.

2. METHODS

2.1. OUTLINE OF THE CARDIOVASCULAR RISK IN YOUNG FINNS STUDY

The multi-center study, at first called *Atherosclerosis Precursors in Children*, and later renamed *The Cardiovascular Risk in Young Finns* (CRYF), was launched in Finland in 1979. The CRYF was designed as a collaborative effort between all university departments of pediatrics and several other institutions in Finland to study the risk factors of cardiovascular diseases and their determinants in children and adolescents of various ages in different parts of the country (Åkerblom et al., 1991). The study was motivated by the World Health Organization Recommendation of 1978 and by earlier studies indicating that atherosclerotic vascular changes start quite early in life (Åkerblom et al., 1991). The main objectives of the CRYF study have been to: 1) study risk factor levels and their possible regional and socioeconomic differences; 2) study the determinants of CHD risk factors and the mechanisms by which risk factor levels in childhood change into adult levels; 3) explore the tracking and clustering of CHD risk factors; 4) study the behavioral and psychological risk factors for CHD; 5) study the effect of life-style and life-style changes on CHD risk factors; 6) study new risk factors for CHD and genetic variation in CHD risk factors (Åkerblom, Viikari, Raitakari, & Uhari, 1999; Viikari, 2003).

2.2. SELECTION OF THE STUDY POPULATION

In order to select participants that are broadly representative of Finnish children and adolescents in terms of living conditions and socioeconomic and demographic background, Finland was divided into five areas according to the location of the university cities with a medical school (Helsinki, Kuopio, Oulu, Tampere and Turku). In each area, urban and rural boys and girls were randomly selected on the basis of their personal social security number from the Social Insurance Institution's population register, which covers the whole population of Finland. In four areas (Helsinki, Tampere, Turku and Oulu), 60 girls and 60 boys in the age cohorts of 3, 6, 9, 12, 15, and 18 years in 1980 were selected. To ensure equal numbers of participants from the east and the west, 120 boys and 120 girls were selected in each cohort in Kuopio, the most eastern area. (Åkerblom et al., 1991; Åkerblom et al., 1985). The initially selected sample of the CRYF consisted of 4,320 children and adolescents. The first and second pilot studies were carried out in 1978 and 1979. The first cross-sectional study was performed in 1980, and it included 3,596 children and adolescents (83.2% of the invited). These cohorts have had medical and psychological follow-up examinations up in 1983, 1986, 1989, 1992, and 2001. The participation rates for the follow-up studies were 83%, 78%, 76%, 66%, and 64% of the original study sample (N=3,596) in 1980 (Åkerblom et al., 1999; Viikari, 2003).

The examinations took place at the outpatient departments of the Department of Pediatrics in the urban areas, and in the Public Health Centers in the rural areas. A few weeks before the medical examinations, in connection with the invitations for children to participate in the CRYF, the families received questionnaires by mail covering socioeconomic background of the family, psychological and psychosocial characteristics of the parents and their children, the children's general health and health behaviors, the parents' health habits and their state of health, as well as the grandparents' state of health. The participants brought these questionnaires with them to the medical examination, which included blood tests, measurement of blood pressure, and anthropometric measurements (Åkerblom et al., 1991; Viikari, 2003). Ethical committees of all participant universities accepted the study plan (Åkerblom et al., 1985).

2.3. PARTICIPANTS IN STUDIES I TO IV

The number and age of the participants in *Studies I* to *IV* is summarized in Table 1. In *Studies I* and *II*, the participants were the four oldest age cohorts of the CRYF who were examined in 1983 and in 1992. In *Study III*, the participants were the three youngest age cohorts who were followed from 1983 to 2001. *Study IV* comprised the youngest age cohort of the CRYF study that was examined in 1983 and in 1986.

Participants were required to have complete data in all the study variables in the follow-up examinations. In *Studies I, II, III,* and *IV,* complete data was available from 62%, 57%, 43%, and 41% of the respective age cohorts at the baseline of the studies in 1983. In previous studies extending up to year 1992, no systematic selection in drop-out of the participants was found (Leino et al., 1999; Räikkönen et al., 2000). Our analyses revealed that compared to the original population in 1980 (the nationally representative sample), and to those who had dropped out between 1980 and 2001, the participants in the year 2001 examination were more likely to be women (57% in the present study vs. 50% in the original population vs. 50 % of drop-outs), more likely to have parents belonging to the highest occupational (25% vs. 18% vs. 16%), highest educational (36% vs. 22% vs. 22%) and highest income class (27% vs. 21% vs. 19%), were less likely to be categorized as physically passive (9% vs. 17% vs. 20%), and had lower BMI in childhood (means 15.8 kg/m2 vs 17.8 kg/m2 vs. 18.2 kg/m^2). Thus, it seems that dropout is systematic; those who had stayed were somewhat healthier and wealthier than those who had dropped out.

Table 1
Number and age of participants at the baseline and at the follow-up examinations in studies I-IV.

	Number of participants			Age (years)			
	Males	Females	Total	In 1983	In 1986	In 1992	In 2001
Study I	531	688	1219	12-21		→ 21-30	
Study II	477	648	1125	12-21		→ 21-30	
Study III	296	385	681	6-12			→ 24-30
Study IV	90	120	210	6	→ 9		

2.4. MEASURES

2.4.1. Socioeconomic status

The SES of the family of origin was measured by three indices: parents' educational level, parents' occupational status, and family income. The participants' SES was measured by educational level and occupational status.

Educational level. *In Studies I, II* and *III,* parental educational level was classified according to self-reports as high (academic; studying at or graduated from university), intermediate (secondary education but not academic), and low (comprehensive school as the highest level of education), by using the parent with a higher educational level. Educational level of the participants was categorized following a similar procedure. In *Study IV,* the number of years of education of both parents was used.

Occupational status in *Studies I* and *III* was measured according to the classification of Central Statistical Office of Finland, 1979, and categorized as upper non-manual, lower non-manual, and manual. An additional category for entrepreneurs was used in *Study I*, whereas entrepreneurs in *Study III* were classified based on their educational level in the non-manual or manual group.

Income of the parents was measured as family gross annual income. In *Studies I* and *III*, family income was categorized as low (< 50,000 FIM), intermediate (50,000 FIM to 80,000 FIM) or high (> 80,000 FIM), based on the lowest, middle, and highest tertiles derived from the entire CRYF population (the corresponding categories in USD are low < 9,650 USD, intermediate 9,650 – 15,420 USD, and high > 15,420 USD). In *Study IV*, income was measured by a standardized score from an 8-point scale, the lowest category representing "gross annual income under 20,000 FIM" and the highest category being "gross annual income over 100,000 FIM" (corresponding to < 4,000 USD and > 20,000 USD). These criteria are in accordance with the income register of Statistics Finland (Statistics Finland, 1983) which shows that median household income in 1983 was USD 12,920. No information on the participants' income as adults was available at any of the examinations.

2.4.2. Early-life stressful family environment

The mother's negative and hostile attitudes toward her child were used as an indicator of the child's stressful family environment. **Maternal child-rearing attitudes** were self-rated by the mothers in 1983 when the child was 6 year of age. The scale was derived from the "Operation Family" study (Makkonen et al., 1981). This scale consists of 9 items measured on a 5-point scale. The items assessed three dimensions of hostile child-rearing: 1) the child's *low emotional significance* to the mother (e.g., "The child is significant to me"; 1 = very significant, 5 = not significant), 2) the *strict disciplinary* style of the mother (e.g., "Disciplinary actions are regularly needed"; 1 = totally disagree, 5 = totally agree), and 3) the mother's *low tolerance* towards the child (e.g., "In difficult situations, the child is a burden"; 1 = totally disagree, 5 = totally agree). These three dimensions closely correspond to the elements of a constellation that Schaefer (1959) has named *hostile child-rearing attitude*, which includes the mother's emotional rejection of the child and the mother's perception of the child as burdensome and in need of strict disciplinary actions. The Cronbach α's in the CRYF data were .77 for low emotional significance, .67 for strict discipline, and .71 for low tolerance. This measure of mother's hostile child-rearing attitudes has been shown to predict personality-related features of the child such as negative emotionality (Katainen, Räikkönen, & Keltikangas-Järvinen, 1997), hostile personality (Räikkönen et al., 2000), and depressive tendencies (Katainen, Räikkönen, Keskivaara, & Keltikangas-Järvinen, 1999).

2.4.3. Temperament and temperament-related characteristics

The psychological variables included the participants' temperament-related characteristics as a child (Type A behavior and temperament) and as an adult (cynical hostility).

Cynical hostility was measured in 1992 when the participants were 21 to 30 years of age. The measure was derived from the Minnesota Multiphasic Personality Inventory questionnaire (MMPI; the Individual Card Form) through factor analyses of the items on the paranoia and depression scales (Comrey, 1957, 1958). Sample items: "I think nearly anyone would tell a lie to keep out of trouble", "Most people will use somewhat unfair means to gain profit or an advantage rather than to lose it", "Most people inwardly dislike putting themselves out to help other people". The original true/false response scale was reformatted into a five-point scale ranging from totally disagree (1) to totally agree (5), because this coding scheme was thought to be more effective in bringing out variance in hostility in this young, non-clinical study sample. Cronbach α for the scale was .73 for men and .76 for women, and confirmatory factor analyses have shown that the items of the scale load significantly on the same factor (Räikkönen et al., 2000). This scale has previously been shown to correlate significantly with scales measuring hostility-related constructs such as paranoia and anger (Buss & Durkee, 1957; Keltikangas-Järvinen & Ravaja, 2002). The CRYF study has previously shown that high scores on the cynical hostility scale predict low social support, hostile maternal child-rearing attitudes, difficult temperament, and physiological coronary risk factors (Keltikangas-Järvinen & Ravaja, 2002; Räikkönen et al., 2000; Räikkönen, Keltikangas-Järvinen, & Hautanen, 1994).

Type A behavior was both self-rated by the participants and evaluated by their mothers in 1983 when the participants were aged 12 to 21. The Type A behavior for the Finnish Multicenter Study (AFMS) questionnaire was used, which is described in detail by Keltikangas-Järvinen and Räikkönen (1990b). The AFMS questionnaire consists of 17 items on a five-point scale from 1 (totally disagree) to 5 (totally agree), of which 14 items are derived from the Matthews Youth Test for Health (Matthews & Angulo, 1980). To make the AFMS more suitable for adolescents and young adults, four items of

the original MYTH were replaced by four items derived from the Swedish version of the Jenkins's Activity Survey for Students (Lundberg, 1980). We used orthogonal linear composite scales based on factor analysis to measure the following dimensions of Type A behavior: *Impatience* (sample item: "When I have to wait for others I become impatient" (from the self-report scale), "When my child has to wait for others he/she becomes impatient" (from the mother-rating scale), *Aggression-competitiveness* (sample item: "I tend to get easily into fights" (from the self-report scale), "My child tends to get easily into fights" (from the mother-rating scale), *Engagement-involvement* (sample item: "I am frequently asked to be a leader", (from the self-report scale), "My child is frequently asked to be a leader" (from the mother-rating scale), and *Hard-driving* (sample items: "I consider myself more responsible than the average person", (from the self-report scale), "I consider my child to be more responsible than the average person" (from the mother-rating scale). Correlations between self-ratings and mother-ratings were .34, .22, .30, and .22 ($ps < .01$) for impatience, aggression-competitiveness, engagement-involvement, and hard-driving. These correlations are in line with previous reports on mother-child correlations in evaluating the child's characteristics (Achenbach, McConaughy, & Howell, 1987). The AFMS questionnaire has been shown to correlate significantly with a well-known Type A scale called the Wolf-Hunter A-B Rating Scale (Keltikangas-Järvinen & Räikkönen, 1990b). The AFMS shows significant continuity over different developmental periods (Keltikangas-Järvinen, 1989), and the 3-year test-retest stability was .32 ($p < .05$) for young adult men and .42 ($p < .01$) for young adult women.

Child's temperament. The mothers rated the temperament of their 6 to 12-year-old children in 1983. The temperament scale was derived from the Health Examination Survey (E Wells, 1980). *Emotionality,* as operationalized by negative emotionality and aggression, was evaluated based on responses to six items ranked on a 5-point Likert-type scale (e.g. "Other children's parents often complain about the child's behavior"; "The child often hits, pushes or provokes other children"; Cronbach alpha = .78). *Activity* of the child referred to motor activity, and was assessed on a 4-point continuum: (1) "Always controlled," (2) "Overactive or restless only occasionally, for instance when tired", (3) "Continuously more active than the average child or youth", (4) "Always extremely active and energetic, even restless". Sociability was reverse scored in order to reflect low sociability of the child. *Low sociability* was assessed with a three-point continuum: (1) "Always very co-operative and responsive to others," (2) "Sometimes problems with peers, but mostly co-operative", (3) "Continuous problems with peers". Intercorrelations (Pearson's *r*) between the temperament traits ranged between .23 and .28 for boys, and between .10 and .15 for girls. The Activity scale has been shown to predict the impatience-aggression component of Type A behavior over a 6-year period in the CRYF study (Räikkönen & Keltikangas-Järvinen, 1992). Child's difficult temperament, as assessed by a high level of emotionality and activity and a low level of sociability, has been shown to predict hostility over 12 years (Räikkönen et al., 2000) and self-rated difficult temperament over 17 years (Pesonen, Räikkönen, Keskivaara, & Keltikangas-Järvinen, 2003).

2.4.4. Health-risk behaviors

Health-risk behaviors, including smoking, alcohol use, physical inactivity, and type of fat in diet (vegetable fat versus animal fat), were self-reported by the adult participants in 1992 in *Studies I* and *II,* and in 2001 in *Study III.* In *Study III,* physical inactivity and fat in diet were also measured in childhood. Fat in diet was measured by parental reports, while physical inactivity was measured by mother-reports for the 6-year-olds, and by self-reports for children 9 years of age or older. It has been shown that from age 9 onwards, self-assessments of physical inactivity are reliable (Telama et al., 1997). The following health-risk behaviors and cut-off points have previously been associ-

ated with serum lipids, insulin concentrations, and blood pressure in previous studies of the CRYF sample (Raitakari, Porkka, Räsänen, & Viikari, 1994).

Smoking. In *Study I,* smoking in adulthood was measured by the number of cigarettes smoked per day. In *Studies II* and *III,* those smoking daily in adulthood were classified as "smokers" and others were classified as "non-smokers".

Alcohol use. In *Study I,* the participants were asked to indicate the frequency of consumption of alcoholic beverages or wine per week by the question: How often do you drink alcoholic beverages (beer, cider, long drinks, wine, or spirits)? The choices were: 1=daily, 2=at least once a week, 3=at least once a month, 4=less than once a month, 5=not at all. In *Study I,* alcohol consumption was measured by the average number of occasions per week when alcoholic beverages were consumed. For this purpose, the number of occasions of alcohol used per year was calculated on the basis on the question of frequency of alcohol use (the question was recoded as: daily=365, at least once a week=52-364, at least once a month=12-51, less than once a month=1-11, never=0). The medians of these classes were divided by the number of weeks per year, resulting in a continuous variable indicating average weekly frequency of alcohol use. In *Study II,* a dichotomous variable was used where those who reported using alcoholic beverages at least once a week were considered as regular alcohol users. This measure of alcohol has been used in previous studies on the Cardiovascular Risk in Young Finns study (Leino et al., 1999; Raitakari, Porkka, Räsänen et al., 1994). In *Study III,* participants were classified by the frequency of heavy alcohol use (more than 6 units per session) into the following categories: less than monthly, at least once a month, or at least once a week. This measure was used because recent evidence has shown that binging style of drinking is more risky for cardiovascular health than evenly distributed high total alcohol consumption (Kauhanen, Kaplan, Goldberg, & Salonen, 1997; Kauhanen, Kaplan, Goldberg, Salonen, & Salonen, 1999; Rehm, Sempos, & Trevisan, 2003).

Physical inactivity. Physical inactivity was assessed by a physical activity sum index consisting of frequency, intensity, and duration of exercise (Telama et al., 1985), consisting of the following questions: How often do you engage in leisure-time physical activity at least half an hour per time? How much are you breath-taken and sweating when you engage in physical activity and sport? How many times a week do you usually engage in the training sessions of a sport club? Do you participate in regional competition? What do you usually do in your leisure time? A sum index was formed with the help of these questions, following the method by Telama and others (Telama et al., 1997). The reliability and validity of this measure have been shown previously (Raitakari, Porkka, Taimela et al., 1994; Telama et al., 1997). In *Study I,* the physical inactivity index was used as a continuous variable. In *Study II,* participants in the lowest quartile of the physical activity index were defined as physically inactive (Leino et al., 1999). In *Study III,* physical inactivity in childhood and in adulthood was classified as active, average, and passive, using a method reported by Telama and others (Telama et al., 1997).

Type of fat in diet. In *Studies I, II,* and *III,* the participants reported whether they used vegetable oil, margarine, or butter as the primary type of fat in food preparing (only one type of fat was to be chosen) (Leino et al., 1999). Additionally, *Study III* included parent-reports of the family's fat use as an indicator of the participants' diet in childhood.

2.4.5. Metabolic cardiovascular risk factors

Body fat *(Study III)* and metabolic syndrome *(Study IV)* were used as the metabolic indicators of cardiovascular risk. Indices of body fat were body-mass index and waist circumference, which were measured in adulthood. Metabolic syndrome was measured in childhood by body-mass index, serum insulin, HDL cholesterol, triglycerides, and systolic blood pressure, as recommended by the diagnostic criteria proposed by Reaven (1988), DeFronzo and Ferrannini (1991), and the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (2001).

Body-mass index was calculated as weight (kg)/[height (m^2)]. Weight was measured with a Seca weight scale, and height was measured with a Seca anthropometer.

Waist circumference was measured in duplicate at the level of the twelfth rib (level with the navel in thin subjects) to an accuracy of one millimeter.

Serum insulin. After an overnight fast, venous blood samples were taken from the right antecubital vein of recumbent subjects. Serum insulin was measured by a modification of the immunoassay method of Herbert et al.(Herbert, Lau, Gottlieb, & Bleicher, 1965) in the Research Laboratory of the Department of Pediatrics, University of Oulu, Finland. The intra-assay CV was 1.6% and the interassay CV 8.4%, with the sensitivity of the assay at 0.5 mU/L.

HDL cholesterol and triglycerides. All lipid determinations were done in duplicate, with standard enzymatic methods used for serum cholesterol (Boehringer CHOD-PAP) (J Siegel, Schlumberger, Klose, Ziegerhorn, & Wahlfeld, 1981) and TG (Boehringer) (Wahlefeld, 1974) in the laboratory of the Rehabilitation Research Centre of the Social Insurance Institute, Turku, Finland. This laboratory continuously crosschecks the lipid determinations with the World Health Organization laboratory in Prague, Czech Republic. Serum HDL-C concentrations were measured from the serum supernatant after precipitation of VLDL and LDL lipoproteins with dextran sulphate 500,000 (Kostner, 1976). Where possible, all analyses were performed simultaneously. The average within-run and between-run CVs were 1.7% and 3.8% for HDL-C, and 2.6% and 4.4% for serum TGs. A more detailed description of the assessment protocol has been reported by (Viikari et al., 1991).

Systolic blood pressure was measured with a standard mercury gravity sphygmomanometer on the right arm, after a rest of at least 3 minutes, to the nearest even figure (Uhari et al., 1991).

2.5. STATISTICAL METHODS

Study I. Following the recommendation of Baron and Kenny (1986), a mediating effect is considered to be found if the association of the independent variable on the dependent variable is significantly attenuated after controlling for the potential mediator. In line with Baron and Kenny (1986), to support the hypothesis that SES contributes to the association between cynical hostility and health-risk behaviors, the following criteria should be met: (1) SES should be associated with hostility and health-risk behaviors (2) hostility should be associated with health-risk behaviors, and (3) this association should attenuate after controlling for SES.

The association of SES with hostility and health-risk behaviors was examined by analysis of variance by testing age-adjusted differences in the mean levels of hostility and cardiovascular risk behaviors between SES categories. The association between adulthood hostility and adulthood health-risk behaviors was examined by linear regression analysis. To examine the contribution of SES to the association between hostility and health-risk behaviors, the regression analysis was hierarchically adjusted for age, parents' SES, participants' SES, and intergenerational social mobility. To validate the findings on other SES indicators besides educational level, we additionally replicated the analyses using occupational level and income as SES indicators.

Study II. To support the hypothesis that Type A behavior contributes to the association between low educational level and a high level of health-risk behaviors the following criteria should be met (Baron & Kenny, 1986): (1) Type A behavior should be associated with subsequent educational level and with subsequent health-risk behaviors (2) educational level should be associated with health-risk behaviors, and (3) this association should attenuate after controlling for Type A behavior.

The association of Type A behavior with subsequent educational level was examined by analysis of variance. The association between adult educational level and adult health-risk behaviors was examined by logistic regression analysis. Odds ratios (OR) and their 95% confidence intervals (CI) were adjusted for age and parental education. The contribution of Type A behavior was examined by entering it into these models as a covariate. Following Bosma and others (1999), the contribution of Type A behavior was estimated by the percentage reduction in the OR of the participant's educational level using the following formula: $[OR_{(\text{adjusted for age and parental education})} - OR_{(\text{adjusted for age, parental education and type A})}]/[OR_{(\text{adjusted for age and parental education})} - 1]$.

Study III. Correlations were used to examine the bivariate relationships between temperament, childhood variables, and adult health behaviors. To examine the multivariate relationship of the temperament dimensions to the indices of body fat, we used a series of linear regression analyses with BMI and WC as continuous dependent variables. We controlled for the overall Type I error rate by adjusting the critical *p*-value with the number of temperament dimensions being used ($p = 0.05 / 3$ temperament dimensions), yielding a critical alpha level of 0.017. Finally, we examined adult health behaviors as potential mediators in the relationship between temperament and body fat (result reported in the original publication). Following the recommendation of Baron and Kenny (1986), a mediating effect was considered to be found if the association of the independent variable on the dependent variable was significantly attenuated after controlling for the potential mediator.

Study IV. Of the parameters constituting metabolic syndrome (body-mass index, serum insulin, HDL cholesterol, triglycerides, and systolic blood pressure), a metabolic syndrome factor was formed by principal component analysis. The factor accounted for 32% and 39% of the total variance of these parameters in 1983 and 1986.

The associations between parental SES, hostile maternal child-rearing attitudes, and participants' metabolic syndrome factor were computed by Pearson's correlations separately by gender, using the Bonferroni inequality in order to control for Type I error rate. The interactive effects between parental SES and hostile maternal child-rearing attitudes in predicting participants' metabolic syndrome factor were performed by moderated regression analysis (Aiken & West, 1992; Baron & Kenny, 1986). A *moderator* is a factor that specifies under what conditions another variable will operate to produce an outcome. Thus, a moderator is a variable that affects the direction and/or strength of the relation between an independent and a dependent variable (Baron & Kenny, 1986; Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001). A basic moderator effect is represented as an interaction between the independent variable and the potential moderator variable. We entered the variables in three steps: (a) the predictor variable (hostile maternal child-rearing attitudes), (b) the moderator variable (parental SES), and (c) the Predictor × Moderator interaction term.

3. RESULTS

3.1. CONTRIBUTION OF LIFECOURSE SOCIOECONOMIC STATUS TO THE ASSOCIATION BETWEEN HOSTILITY AND HEALTH-RISK BEHAVIORS IN ADULTHOOD

Low parental SES was associated with high levels of cynical hostility and a preference for butter over margarine and vegetable oil in adult men and women aged 21 to 30 (ps $\leq .001$ for men and ps $\leq .003$ for women). Irrespective of sex, the lower the participants' own SES (measured by educational level) in adulthood was, the more cynical they were, the more cigarettes they smoked, and the more often they were categorized as butter users (ps $\leq .000$ for men and ps $\leq .011$ for women). In women, low adulthood SES was also associated with a tendency to be physically inactive ($p = .010$). In both genders, the upwardly mobile group and the stable low group (that is, groups who had low SES in childhood) belonged more often to the butter-user group than those who had grown up in high SES families (ps $= .000$ for men and women). Women with stable low SES had higher hostility levels than other women ($p = .001$), and men who had drifted downward in socioeconomic status consumed alcohol more often than other men did ($p = .022$).

Table 2 shows that cynical hostility was positively associated with the number of cigarettes smoked per day and with frequency of alcohol use in men and in women. Adding parental SES and intergenerational social mobility to the model did not attenuate these associations. Adjustment for participants' educational level decreased the association between cynical hostility and smoking in women (change in β was from .09 to .06, change in p from .019 to .103). Replications of these analyses using parents' and participants' occupational status and parents' income as SES indicators showed no attenuations in the hostility-health behavior relationship.

Table2

Standardized regression coefficients of cynical hostility on cardiovascular risk behaviors, hierarcically adjusted for age, parents' and participants' socioeconomic status and intergenerational social mobility.

	Cardiovascular risk behavior											
	Cigarettes per day			Physical inactivity index			Type of fat in diet[a]			Frequency of alcohol use[b]		
Cynical hostility	β	*t*	*p-value*	β	*t*	*p-value*	β	*t*	*p-value*	β	*t*	*p-value*
Men												
Covariates:												
None	0.16	3.64	0.000	0.06	1.44	0.151	0.01	0.22	0.829	0.10	2.26	0.024
Age	0.16	3.67	0.000	0.08	1.71	0.088	0.02	0.33	0.740	0.11	2.46	0.014
+ parents' SES	0.16	3.69	0.000	0.07	1.62	0.107	-0.01	-0.10	0.918	0.12	2.68	0.008
+ participants' SES	0.14	3.23	0.001	0.07	1.54	0.123	-0.02	-0.38	0.702	0.11	2.58	0.010
+ intergenerational social mobility	0.13	2.99	0.003	0.06	1.36	0.175	-0.02	-0.36	0.719	0.12	2.69	0.007
Women												
Covariates:												
None	0.09	2.35	0.019	-0.02	-0.49	0.624	0.05	1.34	0.181	0.03	2.40	0.017
Age	0.09	2.32	0.021	-0.01	-0.35	0.727	0.07	1.68	0.094	0.04	2.66	0.008
+ parents' SES	0.09	2.19	0.029	-0.02	-0.56	0.579	0.04	1.10	0.270	0.04	2.70	0.007
+ participants' SES	0.06	1.63	0.103	-0.04	-0.94	0.350	0.04	0.92	0.359	0.04	2.79	0.005
+ intergenerational social mobility	0.06	1.63	0.103	-0.04	-0.91	0.362	0.04	0.94	0.347	0.04	2.78	0.006

[a] 1= vegetable oil, 2=margarines, 3=butter

[b] Number of occasions per week when alcoholic beverages were consumed.

3.2. CONTRIBUTION OF ADOLESCENT TYPE A BEHAVIOR TO THE ASSOCIATION BETWEEN EDUCATION AND HEALTH-RISK BEHAVIORS IN ADULTHOOD

A high score on impatience (mother-rated) and a low score on hard-driving (self- and mother-rated) in participants' aged 12 to 21 predicted low educational level in adulthood when the participants were 21 to 30 years of age (*p*s ≤ .006 for men and *p*s ≤ .019 for women). In addition, aggression-competitiveness (mother-rated) was negatively associated with adult educational level in men ($p = .024$), and engagement-involvement (self-rated) was positively associated with adult educational level in women ($p = .002$).

Associations of Type A behavior with adult health behaviors showed that male and female smokers had higher levels of impatience (mother-rated) and lower levels on hard-driving (mother- and self-rated for men, mother-rated for women) in adolescence than did non-smokers (*p*s ≤ .015 for men and *p*s ≤ .032 for women). In addition, female smokers scored lower on engagement-involvement (self-rated) than non-smokers ($p = .040$). Physically inactive women scored higher on impatience (mother-rated) and lower on engagement-involvement (self-rated) than active women (*p*s = .035 and .001). Only the Type A dimensions that were significant at this step were used in the final analysis.

Smoking and physical inactivity were considered in the final model because they had significant associations with participants' education, while alcohol use or butter use in diet were not patterned by educational level. Table 3 shows that participants belonging to the lowest educational group were approximately 8 times more likely to be smokers than participants belonging to the highest educational group. When self- and mother-rated Type A behavior were added to the model, the decreases in ORs were 20.5% in women and 28.5% in men. As regards physical inactivity, the least educated women were approximately 5 times more likely than women with an academic education to be physically inactive. After adjusting the model for self- and mother-rated TABP, the ORs decreased by 17.7%.

Table 3

Odds ratios (and 95% confidence intervals) for smoking status and physical inactivity by educational level in adulthood, hierarchically adjusted for Type A behavior in adolescence and young adulthood.

Health-risk behavior	Education I	Education II	Education III
Women			
Daily smoking			
Model 1[a]	1	3.80 (1.76-8.20)	7.44 (3.12-17.71)
Model 2[b]	1	3.72 (1.72-8.04)	7.00 (2.92-16.76)
Model 3[c]	1	3.55 (1.63-7.71)	6.50 (2.70-15.66)
Fully adjusted[d]	1	3.47 (1.60-7.56)	6.12 (2.53-14.81)
Physical inactivity			
Model 1[a]	1	2.35 (1.22-4.52)	4.95 (2.32-10.59)
Model 2[b]	1	2.26 (1.17-4.37)	4.45 (2.06-9.58)
Model 3[c]	1	2.29 (1.19-4.42)	4.71 (2.20-10.11)
Fully adjusted[d]	1	2.22 (1.15-4.28)	4.25 (1.97-9.18)
Men			
Daily smoking			
Model 1[a]	1	8.28 (3.33-20.56)	11.80 (3.99-34.91)
Model 2[b]	1	7.59 (3.05-18.91)	10.56 (3.58-31.70)
Model 3[c]	1	7.19 (2.88-17.74)	9.18 (3.07-27.45)
Fully adjusted[d]	1	6.86 (2.74-17.14)	8.72 (2.91-26.17)
Physical inactivity			
Model 1[a]	1	1.37 (0.74-2.55)	1.39 (0.57-3.36)
Model 2[b]	1	1.36 (0.73-2.53)	1.44 (0.59-3.52)
Model 3[c]	1	1.32 (0.71-2.47)	1.29 (0.53-3.17)
Fully adjusted[d]	1	1.31 (0.70-2.45)	1.34 (0.54-3.32)

Note.

Educational level: I =Academic, II =Secondary education, III =Comprehensive school.

[a] Adjusted for age and parental education.

[b] Adjusted for age, parental education and self-rated Type A behavior.

[c] Adjusted for age, parental education and mother-rated Type A behavior.

[d] Adjusted for age, parental education, self-rated Type A behavior and mother-rated Type A behavior.

3.3. CHILDHOOD TEMPERAMENT AS A PREDICTOR OF BODY MASS AND ABDOMINAL FAT IN ADULTHOOD

The correlations showed that a high level of emotionality and a low level of sociability in children aged 6 to 12 were associated with high BMI and WC in adulthood, at the ages 24 to 30 (rs = .12 and .11, ps < .01 for emotionality, rs = .09 and .09, ps < .05 for low sociability). Other predictors for high adult BMI and WC were high age, being male, high parents' BMI, high birthweight, high participants' BMI in childhood, low occupational and educational level of the parents, and adult sedentary lifestyle.

Model 2 in Table 4 shows that adding emotionality as a dependent variable to the model already adjusted for childhood and adulthood covariates resulted in an 1% increase in R^2 (significance of change in R^2 was F(1,606)=9.30, p=0.002, β for emotionality =0.10, p=0.002). In the model already adjusted for childhood and adulthood covariates, activity did not have any additive explanatory effect on BMI (increase in R^2 was 0.0 in Model 3), while adding low sociability to the model raised the R^2 by 0.5% (significance of change in R^2 was F(1,606)=5.23, p=0.023 in Model 4). The multivariate regression analyses showed similar, yet slightly weaker, associations between the temperament dimensions and WC, than those between temperament dimensions and BMI. Adding emotionality, activity, and sociability separately into the models already adjusted for childhood covariates resulted in 1%, 0%, and 0.5% increases in R^2, respectively (βs= 0.06, 0.03, and 0.07, ps=0.048, 0.288, and 0.020 for emotionality, activity, and sociability in predicting WC). However, the associations between temperament and WC became non-significant after adjustment for BMI (βs= -0.02, and 0.01, ps=0.158 and 0.499 for emotionality and low sociability in predicting WC).

Tests for potential mediating effects of adult health behaviors showed that after adjustment for adult health behaviors, the association between emotionality and BMI remained significant (β changed from 0.10 to 0.09, p changed from 0.002 to 0.006), while the association between low sociability and BMI became non-significant (β decreased from 0.07 to 0.05, p increased from 0.023 to 0.106). This reduction of significance was caused by adjusting the model with physical inactivity, which was a significant predictor of BMI in the model adjusted for childhood variables and adult health behaviors (β= 0.10, p=0.002).

Finally, we tested for possible Temperament × Gender interactions in predicting body fat, but we found none either for BMI (ps = 0.172, 0.716, and 0.660 for Emotionality × Gender, Activity × Gender, and Low sociability × Gender, respectively) or WC (ps = 0.239, 0.532, and 0.706, respectively).

Table4

Multivariate regression analyses of childhood variables and temperament dimensions in predicting body-mass index in adulthood.

Variable	Model 1			Model 2			Model 3			Model 4			Model 5		
	β	*p*	R^2	β	*p*	$\triangle R^2$	β	*p*	$\triangle R^2$	β	*p*	$\triangle R^2$	β	*p*	$\triangle R^2$
Childhood variables															
Age	-0.15	<0.001		-0.12	0.001		-0.14	<0.001		-0.14	<0.001		-0.12	0.001	
Gender[a]	0.13	<0.001		0.14	<0.001		0.16	<0.001		0.15	<0.001		0.14	<0.001	
Father's BMI	0.10	0.001		0.11	0.001		0.12	<0.001		0.12	0.001		0.11	0.001	
Mother's BMI	0.13	<0.001		0.12	<0.001		0.12	<0.001		0.12	<0.001		0.12	<0.001	
Birthweight	-0.06	0.090		-0.05	0.165		-0.06	0.091		-0.05	0.104		-0.04	0.402	
BMI in childhood	0.59	<0.001		0.58	<0.001		0.58	<0.001		0.59	<0.001		0.58	<0.001	
Parents' occupation[b]	-0.06	0.220		-0.05	0.335		-0.06	0.224		-0.05	0.305		-0.04	0.402	
Parents' education[c]	-0.02	0.607		-0.03	0.520		-0.03	0.567		-0.02	0.609		-0.03	0.557	
Parents' income[c]	0.02	0.562		0.02	0.572		0.01	0.716		0.02	0.635		0.02	0.526	
Type of fat in diet[d]	-0.01	0.793		-0.01	0.868		0.00	0.936		0.00	0.904		0.00	0.996	
Physical inactivity[e]	-0.06	0.047		-0.05	0.092		-0.05	0.106		-0.06	0.073		-0.06	0.085	
Temperament in childhood															
Emotionality				0.10	0.002								0.09	0.009	
Activity							0.03	0.357					0.01	0.787	
Low sociability										0.07	0.023		0.06	0.086	
			42.0%			1.0%			0.0%			0.5%			1.2%

Note.

ΔR^2 indicates the magnitude of change in R^2 compared to Model 1.

[a]1=women, 2=men.

[b]1=manual, 2=lower nonmanual, 3=upper nonmanual.

[c]1=low, 2=intermediate, 3=high.

[d]1=vegetable oil, 2=margarine, 3=butter.

[e]1=active, 2=average, 3=passive.

3.4. MODERATING ROLE OF PARENTAL SOCIOECONOMIC STATUS IN THE ASSOCIATION BETWEEN CHILD-REARING ATTITUDES AND CHILD'S METABOLIC SYNDROME

The correlation analysis showed that strict maternal discipline was associated with higher values of metabolic syndrome factor in boys in 1983 (r = .29, p = .006). SES was not significantly associated with the metabolic syndrome factor in boys or girls.

The interaction term Parental SES × Hostile maternal child-rearing × Child's gender was significant, and therefore following analyses were run separately by gender. In boys, there was no significant interaction between parental SES and hostile maternal child-rearing attitudes in predicting the metabolic syndrome factor at any year. In girls, the Parental SES × Hostile Maternal Child-rearing interaction significantly predicted the metabolic syndrome factor both cross-sectionally (when the children were 3 years old) and at the 3-year-follow-up (when the children were 6 years old; βs = - .031 and - .026, change in R^2 = .107 and .076, ps = .000 and .003, respectively). Because the interactions were similar at both years of measurement, only the interaction in 1983 is displayed in Figure 3. As shown in Figure 3, maternal child-rearing attitudes were differentially related to the girls' metabolic syndrome, depending on the family's socioeoconomic standing. Hostile maternal child-rearing (measured when the child was 3 years of age) was positively associated with the metabolic syndrome in girls from low SES, while hostile maternal child-rearing was negatively associated with the metabolic syndrome in girls with high parental SES.

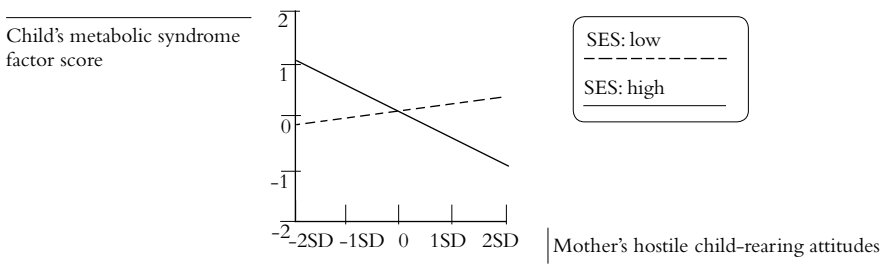

Figure 3.

The relationship between hostile maternal child-rearing attitudes and metabolic sydrome in girls with low and high parental socioeconomic status (SES). The range of the scale on the x- and y-axes is mean ± 2 SD.

4. DISCUSSION

As illustrated in the lifecourse framework presented in Figure 1, we hypothesized that the foundation for adult cardiovascular risk is laid by socioeconomic and psychosocial experiences in childhood and adolescence. By using the theoretical framework presented by Gallo and Matthews (2003), we found support for the following hypotheses: a) adverse socioeconomic circumstances in early life may give rise to negative emotions and CVD risk factors, b) temperament-related factors in early life may cause individuals to drift down in the socioeconomic hierarchy and to adopt unhealthy lifestyles, c) psychological and socioeconomic factors may interact in such a way that psychological factors may have different impacts on CVD depending on the socioeconomic environment in which they are manifested. A summary of the key findings and implications is presented in Table 5. The participants were derived from a well-characterized population-based cohort of Finnish children and adolescents followed into adulthood. The study included a large variety of different aspects of socioeconomic status, personality, and indicators for CVD risk. The measurements were based on parental reports, the participants' self-reports, and medical examinations at the hospital, thereby reducing risk of potential common-variance problems.

4.1. LIFECOURSE SOCIOECONOMIC EXPOSURES AND CARDIOVASCULAR RISK

We examined whether there was an association between cynical hostility and risky health behaviors in adulthood, and whether this association would be explained by adverse socioeconomic exposures in childhood or adulthood. We hypothesized that socioeconomic exposures may act as a "third factor" underlying the subsequent associations between hostility and cardiovascular risk behaviors. In accordance with previous findings (Everson et al., 1997), we found that participants with high adult cynical hostility smoked more and used alcohol more frequently than participants with lower levels of cynical hostility. Our hypothesis was not supported in the sense that the positive association between cynical hostility and health-risk behaviors in adulthood was not attenuated by childhood SES, adulthood SES, or social mobility. Thus it seems that the association between cynical hostility and risky lifestyles is independent of socioeconomic exposures. Even though no confounding effect by SES was found, it is, however, noteworthy that SES was strongly associated with both cynical

hostility and health-risk behaviors in adulthood.

The finding that low SES in childhood and adulthood was associated with higher levels of cynical hostility in adulthood is in accordance with studies showing that living under adverse socioeconomic conditions is likely to foster negative psychosocial orientations such as cynical hostility, hopelessness, depression, and low self-esteem in adulthood (Bosma et al., 1999; Gilman, Kawachi, Fitzmaurice, & Buka, 2002; Harper et al., 2002; Lynch, Kaplan, & Salonen, 1997; Lynch, Kaplan, & Shema, 1997). It has been suggested that low SES fosters cynical attitudes because individuals with low SES encounter more frequent and more severe stressors than individuals in more favourable socioeconomic circumstances. Low SES has been associated with such stressors as interpersonal conflicts, divorce, violence, financial strain, and negative psychosocial work characteristics (Adler et al., 1996; B S Dohrenwerd & Dohrenwerd, 1981; Gallo & Matthews, 2003; Theorell, 1989; Vahtera, Virtanen, Kivimäki, & Pentti, 1999). Gallo and Matthews (2003) propose that negative emotions and cynical attitudes are a response of the individual to a constantly taxing environment.

As expected, we found direct associations between low SES and adverse health-risk behaviors. Participants who had grown up in families with low SES had unhealthier diet as adults than those who had grown up in families with higher SES. This finding is in line with previous ones showing that dietary habits are largely inherited from the early home environment (Brunner et al., 1999; Keski-Rahkonen, Viken, Kaprio, Rissanen, & Rose, 2004; Leino et al., 1999; Leino et al., 2000; Lynch, Kaplan, & Salonen, 1997). Participants' low educational level in adulthood, in turn, was associated with a higher prevalence of smoking. Low childhood SES has previously been associated with adverse adult health-behaviors in several studies. A Dutch study has shown that excessive alcohol intake and lack of exercise explained approximately 10% of the association between low childhood SES and poor perceived health in adulthood (van de Mheen et al., 1998). In the Scottish 1958 cohort, low SES at birth was strongly associated with smoking and a fruitless diet in adulthood (Jefferis et al., 2004; Power & Matthews, 1997). The Kuopio Ischemic Heart disease study has shown that childhood SES predicted unhealthy diets, a sedentary lifestyle, and ischemic heart disease in middle-aged men, and that this association was not accounted for by adult SES (G A Kaplan & Salonen, 1990; Lynch, Kaplan, & Salonen, 1997). Adverse socioeconomic exposures in early life have also been associated with adult levels of obesity (Brunner et al., 1999; Lawlor et al., 2002; Leino et al., 1999; Leino et al., 2000; Poulton et al., 2002; Power & Matthews, 1997), cholesterol (Blane et al., 1996; Leino et al., 1999; Leino et al., 2000), blood pressure (Blane et al., 1996; Poulton et al., 2002), and metabolic syndrome (Lawlor et al., 2002).

Inter-generational social mobility, that is, the individual's current social position compared to that of his/her family of origin, did not have a major impact on health-risk behaviors. This is in accordance with findings showing that low SES in childhood has an effect on adulthood risk, independently of socioeconomic conditions in adulthood (Barker & Osmond, 1986; Davey Smith et al., 1997; Davey Smith et al., 1998; G A Kaplan & Salonen, 1990; Kuh et al., 2002; Poulton et al., 2002; Wannamathee et al., 1996). These findings reflect the lifecourse view, which suggests that social mobility has only a minor role in creating inequalities in health or health-related behaviors, and that the *cumulative effects* of socioeconomic circumstances, starting already in childhood, are the key factors in understanding health inequalities (Davey Smith et al., 1998; Karvonen, Rimpelä, & Rimpelä, 1999; Power & Matthews, 1997).

In this context it worth noting that there has been debate over the direction of the cause-and-effect link between SES and health. It is possible that low SES causes poor health, but poor health may also cause drift to low SES (Lunberg, 1991; West, 1991). A common view is the *social causation model,* where low SES is suggested to cause poor health. Our finding that exposure to low SES predicted adverse psychological and health-related outcomes support the social causation model. How-

ever, *the health selection model* – also called social selection – signifies a process where factors in early phases of life influence the social class that a person is going to achieve in adulthood. The *direct selection* hypothesis argues that socioeconomic health inequalities result from the fact that healthy people are likely to move up and sick people are likely to move down in the social hierarchy (Townsend & Davidson, 1982; West, 1991). The *indirect selection* hypothesis suggests that some early background factor determines both the chances of higher social class and health status in adulthood (West, 1991; Wilkinson, 1986). Such background factors include education, mental health, childhood deprivation, and health-related behaviors. For instance, a Finnish study has shown that adolescents who lived in health-compromising ways (smoking, alcohol use, eating a lot of sweets and not exercising) were less educated and fell in the less well-off social strata in adulthood (Koivusilta, Rimpelä, & Vikat, 2002). Our findings support the health selection model by showing that early temperament may "select" individuals into adverse socioeconomic and health-related trajectories.

It has been suggested that negative emotions may be a mediator that explains the relationship of low SES with later adverse health outcomes (Gallo & Matthews, 2003). In the present study, hostility did not mediate the association between low SES and adverse adult health behaviors (result shown in the original publication of *Study I*). However, Bosma and others (1999) have shown that external locus of control, neuroticism, and absence of active problem-focused coping explained about half of the association between low childhood socioeconomic status and poor health in adulthood. Cohen, Kaplan and Salonen (1999) have shown that anger, hostility, depression and hopelessness contributed to the association between SES and perceived health and cardiovascular mortality in samples from the United States and Finland. In Swedish women, the combination of unhealthy lifestyles and psychosocial factors such as hopelessness, social isolation, and job stress accounted for approximately 50% of the association between low educational level and CHD (Wamala et al., 1999). It may be concluded that low SES is a hazard for psychological well-being which may ultimately be manifested as deteriorating cardiovascular health.

Our findings show that individuals with low SES tend to form negative interpretations of the world, have poor academic and economic success, smoke more, and have unhealthier eating habits. All in all, these findings confirm previous suggestions that exposure to low SES, starting already early in life, predisposes to several adversities, all of which may increase susceptibility to CVD.

4.2. EARLY TEMPERAMENT AS ANTECEDENT OF CARDIOVASCULAR RISK

We hypothesized that certain temperament characteristics in childhood and adolescence may predispose individuals to adverse life-trajectories which, in turn, may make the individual more vulnerable to CVD in adulthood. Indeed, we found that the Type A dimensions "impatience" and "lack of responsible hard-driving" in adolescence explained approximately one fifth of the association between low educational level and smoking (in men and women), and low educational level and a sedentary lifestyle (in women). This finding is in line with those of previous studies in the Cardiovascular Risk in Young Finns study where it has been shown that impatience is a "pathogenic" component of Type A behavior, while responsibility is associated with benign levels of metabolic cardiovascular risk factors in children and adolescents (Keltikangas-Järvinen, 1992; Keltikangas-Järvinen & Räikkönen, 1989, 1990a, 1990b). The contribution of our findings was that they showed that the pathogenic Type A characteristics in childhood and adolescence have relevance for cardiovascular risk levels *in adulthood*.

In predicting adulthood body fat, a high level of negative emotionality (including verbal and physical aggression) and a low level of sociability (difficulty in co-operation with peers) in school-aged children predicted increases in body mass from childhood to adulthood. A high level of negative emotionality and a low level of sociability are aspects of the difficult temperament construct. Our study was the first to prospectively show that negative emotionality in childhood may have consequences for *adult* body mass. In previous studies, aspects of the difficult temperament construct have been associated with *childhood* body fat. Negative mood and hostile affect have been shown to be risk factors for weight gain and high skinfold thickness in children (Ravaja & Keltikangas-Järvinen, 1995). We also found an association between low sociability in childhood and adult BMI, which attenuated after adult health behaviors (especially physical inactivity) were taken into account. This suggests that health-risk behaviors are one mechanism through which early temperament can affect later metabolic cardiovascular risk.

Our findings on temperament are in line with literature showing that externalizing behaviors, including a high level of negative emotionality and poor self-control, are associated with unfortunate outcomes in adulthood. Caspi, Elder and Bem (1987) have found that aggressive and impatient children with negative emotions and frequent explosive tantrums were likely to drop out of school,

become unemployed and have high levels of substance use as adults. The Jyväskylä longitudinal study suggests that aggression in childhood predicts long-term unemployment and low career orientation in adulthood (Kokko & Pulkkinen, 2000; Pulkkinen et al., 1999). Tremblay and others (Masse & Tremblay, 1997; Tremblay et al., 1992; Tremblay et al., 1994) reported that hyperactive children were at risk of poor school achievement, antisocial behaviors, and substance use later in life. In the Cardiovascular Risk in Young Finns data it has previously been shown that a high level of impatience and a low level of responsibility were associated with underachievement in school (Keltikangas-Järvinen, 1992; Keltikangas-Järvinen & Räikkönen, 1990a). A recent study by Keltikangas-Järvinen and others has also shown that impulsivity was a risk factor for dropping out of higher education (Keltikangas-Järvinen et al., 2003). Several other studies suggest that children with high levels of aggressiveness and impulsivity are likely to engage in risky lifestyles including smoking and a sedentary lifestyle as adults (Caspi et al., 1997; Cooper et al., 2003; Masse & Tremblay, 1997; Pulkkinen & Pitkänen, 1994). Put together, our findings on temperament and Type A behavior indicate that early temperament/Type A behaviors are related especially to smoking, a sedentary lifestyle, and accumulation of body mass. Therefore, discouraging smoking and encouraging active lifestyles in youth with temperamental aggressions and impatience could lead to improvements in later cardiovascular health.

It should be noted that despite being statistically significant, the contributions of temperament to CVD risk factors were rather small. For instance, all the temperament dimensions together explained 1.2% of BMI in addition to the childhood variables. Even though this effect was small, it is noteworthy that it was found in addition to the established predictors for obesity such as parental body mass and participants' body mass in childhood. Because there are no previous studies connecting childhood temperament or personality characteristics to adult body mass, we cannot directly compare the robustness of our findings with those of previous studies. Pine, Cohen, Brook and Coplan (1997) showed that conduct disorder symptoms in adolescence explained 2% of the variance in BMI in adulthood. In the present study, children with high negative emotionality were on average 1.62 kg/m^2 larger in BMI as adults than were children who belonged to the lowest emotionality group. Accordingly, Pine, Goldstein, Wolk and Weissmann (2001) found that children with major depression had an average 1.9 kg/m^2 higher BMI as adults than children without depression. It seems that the magnitude of our findings is comparable to those found previously.

The association between temperament-related behaviors and CVD risk factors may be mediated by factors not measured in the present study. Temperament characteristics indicating frequent anger, irritability, and a lack of social skills, are part of a constellation called a difficult temperament (Thomas et al., 1968) which refers to behavior that is challenging to a child's caretakers. Such difficult temperament may create psychosocial stress which, in turn, has been associated with CVD (Hemingway & Marmot, 1999; Kivimäki et al., 2002; Mc Ewen & Seeman, 1999; Räikkönen et al., 1996; Rosengren et al., 2004; Rozanski et al., 1999; Vahtera et al., 2004) and obesity (Keltikangas-Järvinen, Räikkönen, Hautanen, & Adlercreutz, 1996; Kivimäki et al., 2002; Korkeila, Kaprio, Rissanen, Koskenvuo, & Sörensen, 1998). By evoking negative reactions in their caregivers and other people, difficult children are at risk of experiencing conflicts and resentment from the environment which may, in turn, increase stress of child (Chess & Thomas, 1984; Goldsmith et al., 1987). Secondly, negative emotionality may increase stress levels, because temperament affects how an individual interprets different situations; whether they are seen as positive challenges or stressful situations that should be avoided (Heponiemi, Keltikangas-Järvinen, Puttonen, & Ravaja, 2003; J Smith & Prior, 1995; Strelau, 1998).

The suggested pathway between stress and obesity is through changes in sympathetic nervous system activation and the hypothalamus pituitary-adrenocortical axis (Björntorp, 1991; Howard et

al., 1993; Manuck et al., 1995; Schneiderman & Skyler, 1996). Stress may affect physiological reactions directly through hemodynamic reactions and the release of stress hormones, or stress may affect obesity through unhealthy lifestyles. For instance, stress may be associated with increased intake of dietary sugars which, in turn, contribute to increased plasma insulin, sympathetic nervous system activity, and subsequently to hypertension, accumulation of body fat, and insulin resistance (Howard et al., 1993; Schneiderman & Skyler, 1996)

It is also possible that a constitutional factor underlies associations between temperament characteristics and obesity. For instance, allelic variants of the dopamine receptors have been shown to underlie temperamental characteristics (Keltikangas-Järvinen et al., 2003; Reif & Lesch, 2003) as well as obesity (Poston et al., 1998;Yasuno et al., 2001). Moreover, family, twin and adoption studies provide evidence for familial and genetic influences on individual differences in disease risk and in human behavior (Kaprio, Pulkkinen, & Rose, 2002). The association between certain temperament-related behaviors and CVD risk factors in children may result from parental transmission of genes to their offspring. The role of specific genes in explaining associations between personality and cardiac risk is a research topic of current interest.

It seems that temperament and personality characteristics that are detectable already in childhood and adolescence may set a "cycle of mal-adjustment" into motion that leads to a clustering of psychosocial and somatic adversities over a lifetime and which may ultimately lead to poor cardiovascular health. Children having a difficult temperament, including anger and poor self-control, may be at risk for downward mobility, and subsequent cardiac risk. These temperament characteristics may reflect individual variations in normal child behavior, but they may also be early markers of externalising and adjustment problems (reviewed in Caspi, 1998; Rothbart & Bates, 1998). Our findings suggest that temperamentally difficult children may be at heightened cardiovascular risk in adulthood through various pathways, such as social maladjustment, poor socioeconomic achievements, and health-compromising lifestyles.

4.3. INTERACTIVE EFFECTS BETWEEN SOCIOECONOMIC STATUS AND PSYCHOLOGICAL FACTORS

In addition to the independent effects of socioeconomic and psychological factors in predicting adult cardiovascular risk, we hypothesized that there may be synergistic effects between psychological and socioeconomic factors. SES may modify the impact of psychological factors on CVD so that individuals in low SES may suffer more from additional stressors than those who have higher SES. Indeed, girls who were from families with low SES, *and* whose mother's had negative attitudes toward them (an index of environmental stress) had higher levels of metabolic syndrome than girls who were subjected to negative parenting but who came from families with higher SES. Thus it seems that low SES is a vulnerability factor, so that a negative family atmosphere is disadvantageous especially to girls living under adverse socioeconomic conditions.

Our findings may be interpreted according to the reserve capacity model presented by Gallo and Matthews (2003) which suggests that individuals with low SES have a smaller bank of resources - material, social, and cognitive - to deal with stressful events compared to individuals with higher SES, and they are therefore more affected by stress. Previous studies have shown that individuals with low SES are more vulnerable to stressful situations than those with higher SES (Gump et al., 1999; Lynch et al., 1998; Matthews et al., 2000; Stronks et al., 1998). A recent study has shown that maternal warmth promoted positive adjustment in children exposed to low SES (Kim-Cohen et al., 2004). We, in turn, showed that SES may interact with parenting behaviors so that belonging to high SES may protect the child from the adverse health effects of less-than-optimal parenting, while belonging to low SES may increase the child's vulnerability to compromised parenting.

The lack of interactive effects between parental SES and mothers' negative child-rearing attitudes in predicting boys' metabolic syndrome is difficult to explain. There was, however, a direct positive association between the mother's strict disciplinary style (seeing the boy as in need of disciplinary action and using harsh punishments) and boys' metabolic syndrome. Previous research suggests that boys may be more vulnerable to the direct effect of the family's emotional atmosphere than girls (Keltikangas-Järvinen, 2000; Räikkönen et al., 2000; Weidner et al., 1992), which may also be reflected in our findings.

It has been suggested that children raised in adverse family circumstances are vulnerable to physical disease because chronic stress may negatively affect the development of the neuroendocrine system. Moreover, children from such families may not learn sufficient self-regulatory skills

to develop satisfying interpersonal relations. They also are predisposed to behavioral problems and substance use (Repetti et al., 2002). Disadvantageous socioeconomic environment may have its origins already during gestation, when poor nutrition and the mother's stress may lead to permanent neuroendocrine and metabolic changes in the fetus (Barker, 1995; Davey Smith et al., 2002). Chronic stress has been suggested to affect the metabolism through stress-induced over-activity of the sympathetic nervous system and the hypothalamus pituitary-adrenocortical axis followed by excess release of cortisol and catecholamines in the circulation (Björntorp, 1991; Björntorp & Rosmond, 1999; Howard et al., 1993; McEwen & Stellar, 1993; Räikkönen, Keltikangas-Järvinen, Hautanen, & Adlercreutz, 1997; Schneiderman & Skyler, 1996). Overactivity of these stress axes has been suggested to lead to metabolic syndrome through a redistribution of body fat characterized by obesity, hypertension and insulin resistance (Björntorp, 1991; Björntorp & Rosmond, 1999; Räikkönen et al., 1996; Räikkönen et al., 1997; Schneiderman & Skyler, 1996).

To sum up, our findings support the hypothesis that children who live in families with few social and material resources are more vulnerable to stress, which is reflected by their higher levels of somatic risk factors. As exposure to a cascade of adversities may be more important for CVD risk than a single adversity, children living in environments with multiple risks should be primary targets of measures designed to prevent later cardiovascular disease.

4.4. METHODOLOGICAL CONSIDERATIONS

The Cardiovascular Risk in Young Finns study is a population-based cohort study representative of Finnish children, adolescents, and adults (Åkerblom et al., 1991). Due to its long follow-up period, we were able to follow the same individuals from childhood into adulthood. Drop-out was systematic so that in the latest follow-up in 2001 the participants were somewhat healthier (lower body fat and healthier lifestyles) and socioeconomically better-off (higher SES) than the original sample in 1980. As this homogeneity of our sample may have led to reduction in variance, our findings may be underestimates rather than overestimates of the associations between psychosocial factors, socioeconomic status, and cardiovascular risks. Our sample was nationally representative of Finns, and thus ethnically and culturally homogeneous in nature. This may limit generalizability of our findings to other populations.

In measuring childhood SES, we had the advantage of using a first-hand source of information, that is, the participants' parents' reports of their own SES at the time when the participants were children. In most other studies, such as the Whitehall Study (Brunner et al., 1999; Marmot, Shipley, Brunner, & Hemingway, 2001), the Kuopio Ischaemic Heart Disease Study (Lynch et al., 1994, 1994b; Lynch, Kaplan, & Salonen, 1997), and the West of Scotland Collaborative Study (Blane et al., 1996), participants have retrospectively reported their parents' SES as an indicator of their SES in childhood. We consider our method more reliable, since retrospective reports may suffer from unreliability due to second-hand reports and memory problems. We defined parental SES based on the parent with the higher educational or occupation position, whether that be the mother or the father. In majority of studies conducted outside Finland (Blane et al., 1996; Brunner et al., 1999; Davey Smith et al., 1997; Davey Smith et al., 1998; Lawlor et al., 2002; Marmot et al., 2001; Power & Matthews, 1997; Power, Matthews, & Manor, 1996; Wannamathee et al., 1996), only the father's social class has been used as the indicator of the family's SES. That method would give a misleading picture in Finland, where women's participation in the labor force is one of the highest in the world (Statistics Finland, 2003).

We used educational level as the main indicator of participants' SES. This was done because some participants, especially those who were 21 or 24 years of age, were in the process of entering the labor market and did not have an established occupational position. Education may be considered a viable indicator of SES, because it is the primary mechanism through which occupational status is achieved in Finland (Lynch, Kaplan, & Salonen, 1997), it is rather reliable even when self-reported (Adler & Ostrove, 1999; G A Kaplan & Keil, 1993; Liberatos et al., 1988), and it relates well with

different health indicators in different populations (G A Kaplan & Keil, 1993). A replication of the analyses by using income and occupational status as SES indicators produced results similar to those obtained using educational level as the SES indicator.

There were large birth cohort differences in the level of education between parents and their children. Of the parents, more than 70% did not have any education after comprehensive school, while only 10% of their children were without further education. This phenomenon is explained by the rapid increase in the average number of years of education in Finland after the 1960's. Consequently, the number of participants who were classified as "downwardly mobile" was 25, and therefore the results on the effect of social mobility may have limited power. The results may also give an overly positive picture of social mobility of the study cohort; that a majority of the participants were upwardly mobiles reflects rather the parents' low average level of education than the participants' high level of education.

Our measures of temperament had obvious limitations. Because at the time of measurement, methods for evaluating child's temperament were not as developed as nowadays, we used a non-standardized temperament questionnaire. The questionnaire was, however, constructed on the basis of the well-established temperament theory of Buss and Plomin (Buss & Plomin, 1975, 1984). As the three temperament dimensions in their theory, i.e., emotionality, activity and sociability, are included in various forms in almost every model of temperament, they may be considered to have considerable generality (Goldsmith et al., 1987; Prior, 1992). Our temperament questionnaire has been shown to have validity in predicting subsequent temperament-related characteristics such as Type A behavior, (Räikkönen & Keltikangas-Järvinen, 1992), hostility (Räikkönen et al., 2000; Räikkönen & Keltikangas-Järvinen, 1992), and the cluster of difficult temperament (Pesonen et al., 2003) measured by the EAS questionnaire of temperament designed by Buss and Plomin.

The child's personality as rated by its mother was a stronger predictor of educational level and health-risk behaviors than self-rated personality. It is possible that mother-ratings of personality are colored by the mother's perception of her child's behavior rather than the child's actual behavior. Mothers characterized by a hostile parenting attitude tend to view even normal variations in their child's behavior as reflecting difficulty and a need for harsh disciplinary actions. Such a hostile maternal attitude toward the child may be a marker of dysfunctional family relationships and a high level of family stress (Keltikangas-Järvinen, 2000; Repetti et al., 2002). The mothers may also be biased in assessing their children's temperament due to their own psychopathology. It has been shown that parental depressive symptoms are associated with perceiving the infant's temperament as more negatively and less positively tuned (Pesonen, Räikkönen, Strandberg, Keltikangas-Järvinen, & Järvenpää, 2004). Thus, mother-ratings of the child's personality as difficult may in fact reflect abnormalities in family relationships which, in turn, may contribute to child's poor physical health (Lissau & Sorensen, 1994; Parker et al., 1999; Ravaja & Keltikangas-Järvinen, 1995; Repetti et al., 2002).

The use of several different temperament and Type A characteristics may seem confusing, and it raises the question of their relation to broader personality constructs. The hierarchical structure of personality and the relations between different personality theories is a much studied, yet not resolved, topic in personality psychology (Caspi, 1998; Clark & Watson, 1999; Poulton & Caspi, 2003). As Poulton and Caspi (2003) have recently pointed out, there is a need to synthesize disparate models of personality within the same framework. To date, much research in behavioral medicine has been conducted with lower-order constructs, such as hostility, without integrating the findings to broader personality constructs. Thus, "the result is confusion about which personality traits - if any - matter for health (Poulton & Caspi, 2003, p.976)".

It must be admitted that the present study cannot solve the complex question of the structure of

personality. It has been suggested that personality is formed as the result of interplay between child's unique temperamental characteristics and environmental influences (Buss, 1989; Caspi, 1998). Temperament may be seen as an antecedent of later personality, and therefore it may be helpful to compare temperament characteristics with broader personality traits. A majority of personality researchers have accepted the theory that the most important adult personality characteristics may be summarized in the broad "Big Five" superfactors (Extraversion, Neuroticism, Conscientiousness, Agreeableness, and Openness to experience). The temperament dimension of negative emotionality has been linked with a high level of Neuroticism and a low level of Agreeableness, and it is known to be associated with later externalizing and antisocial problems (Buss & Plomin, 1984; Caspi, 1998; Rothbart et al., 2000). The temperament trait of impatience/hyperactivity has been associated with low Conscientiousness and later problems in empathy and pro-social skills (Caspi, 1998; Rothbart et al., 2000). Low sociability, in turn, is best described as low Extraversion and low Agreeableness, and it may predict later depression and internalizing problems (Buss & Plomin, 1984; Caspi, 1998; Rothbart et al., 2000). All in all, temperament may serve as a disposing factor for later personality and even psychopathology. Although we considered temperament characteristics as variations in normal personality, extremes in temperament characteristics may reflect underlying psychopathology. It is possible that our findings were driven by the pathological manifestations of temperament. Clearly, there is a need for collaboration between epidemiologists, clinical psychologists, and personality researchers in creating a common framework for the understanding of the role of personality in health outcomes.

4.5. CONCLUSIONS AND PRACTICAL IMPLICATIONS

We found that both exposure to low SES and a temperament that may be labeled as "difficult" predict later cardiovascular risk levels, as shown in the framework in Figure 1. Furthermore, the health-effects of psychological factors seem to depend on the socioeconomic environment in which they are manifested. Our findings are in line with the assumption derived from Gallo's and Matthews' (2003) model suggesting that exposure to adverse socioeconomic conditions and negative psychosocial factors work reciprocally in producing CVD risk. Our findings cannot provide an answer to the question whether the environment or the individual is more important regarding cardiovascular risk. It may not even be important to solve that question, because both the environment and the child's characteristics may contribute to a stochastic process where several risk factors are interrelated to each other (Williams, 2003). Typically, multiple risk factors cluster within the same family, and they may be transmitted through generations. All this underlines that early childhood may be a critical time to take measures in order to promote later cardiovascular health.

The main implications of our study are summarized in Table 5. Firstly, it should be recognized that some children are born into better social and material circumstances than others. Therefore, societal measures that aim at reducing socioeconomic differences between families could be one way to prevent later cardiovascular disease. Educating and supporting parents, especially those who have limited educational and/or occupational resources, could be another means to better health of their children. This may be a difficult task, however, because low SES parents with cynical and hostile attitudes may be resistant to attitude change even if offered health-related information.

Although it is useful to know that early temperament has predictive validity in terms of cardiovascular risk, one might well ask whether this knowledge helps us in improving public health. Firstly, it may help in identifying those children who should receive special care at home, at school, and in the community. The practical significance of our findings lies in the fact that if one cannot change one's inherited temperament, one can, however, modify the environment in which the genes are expressed. It is known that the phenotypic expression of genes is dependent on the environment in which they are manifested (McClearn, 2004). It has been shown, for instance, that the role of genes in novelty seeking behavior (Keltikangas-Järvinen, Räikkönen, Ekelund, & Peltonen, 2004) and adolescent alcohol consumption (Rose, Dick, Viken, & Kaprio, 2001) is greater in certain environments, whereas the role of genes is dampened in more protective environments. In terms of cardiovascular prevention, the influence of temperaments on CVD risk factors may be

less detrimental if the environments are construed to respond to the specific temperament of the child. One environmental feature that may be worth considering is the role of parenting as a buffer against poor outcomes. Child-centered parenting has been shown to protect the aggressive child from unemployment in adulthood (Kokko & Pulkkinen, 2000), and it would be worth studying if high-quality parenting protects the temperamentally difficult child from adverse health outcomes, as well.

First and foremost, the society, including schools, kindergartens, and the health care system, should take into account that children are temperamentally different and therefore they make different demands on the environment. A certain environment may be a risk factor for one child but not the other, depending on whether there are protective factors that increase the child's resilience against environmental adversities. As the psychosocial origins of cardiovascular disease are complex, prevention should focus on treating multiple variables simultaneously. Thus, helping temperamentally difficult children to acquire better social and coping skills, educating parents about child-rearing, and giving health education to the child and his/her parents, should all be considered as methods to improve cardiovascular health and psychological well-being of children. Future research should examine temperament-environment interactions in order to find out which aspects of the environment would most efficiently protect the temperamentally vulnerable child against adverse cardiovascular outcomes.

Table 5
Key findings and implications of the study.

Key findings:

- Adverse socioeconomic exposures during the lifecourse contribute to adult cardiovascular risk factors, including cynical attitudes, smoking, and unhealthy diet.
- Childhood temperament characteristics indicating negative emotionality and poor self-control predict adverse socioeconomic trajectories and high levels of cardiovascular risk factors in adulthood.
- Mother's negative attitude toward the child is associated with higher levels of metabolic syndrome in girls from lower SES than in girls from higher SES families.

Implications:

- Reducing socioeconomic differences in early life may prevent cardiovascular risk factors from accumulation in later life.
- Temperamental difficulty in childhood may be an early indicator of subsequent cardiovascular risk. Interventions aimed at reducing behavioral problems in childhood and adolescence could contribute to public health.
- Children living in adverse socioeconomic circumstances may be especially vulnerable to additional stressors, such as negative family atmosphere. Special efforts should be made to focus on preventative measures for children who are simultaneously exposed to socioeconomic and psychological adversities.

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