

Facets of negative affectivity, health behaviour, and risk factors of cardiovascular diseases

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ABSTRACT

Previous research has shown that negative affectivity (NA) and lifestyle associate with CVD/CHD risk factors. In the present thesis the relationships between some key indicators of NA (depressive symptoms, vital exhaustion, hopelessness, and anger expression) and lifestyle factors, body mass index, serum lipids and blood pressure were investigated.

All the four studies that constitute this investigation were based on data from the Helsinki Metabolic Syndrome Prevention Trial, which was an uncontrolled preventive trial aimed at improving prevention of metabolic syndrome, type 2 diabetes and cardiovascular diseases by developing a practical method for primary health care. The main idea was to screen and identify men with cardiovascular risk clustering and give them individual counselling based on their risk profile. Evaluation of risk was based on a Risk Index that included five factors: body mass index, total serum cholesterol, blood pressure (systolic and diastolic), smoking habits, and physical inactivity.

The participants in the screening were 40-55 year-old-men residing in the north-east Helsinki. Data collection was carried out between May 2001 and June 2004, and all the participants were asked to answer questionnaires dealing with lifestyle factors and psychosocial risk factors. Study nurses from the Helsinki Heart District interviewed all the participants and recorded basic biomedical measurements (e.g. blood pressure, waist circumference, weight). The size of the sample in studies I-IV was 893, 705, 444, and 710, respectively.

The findings of this study indicate that depressive symptoms had a consistent statistically significant correlation with adverse lifestyle factors and triglycerides. For cholesterol fractions, path analyses indicated the parallel existence of two main pathways: from depression through the adverse health behaviour to an unfavourable cholesterol fraction balance, and from depression through a direct, probably physiological, link to favourable cholesterol levels.

Vital exhaustion and depression were found to be closely correlated constructs with comparable relationships to known vascular disease risk factors. However, vital exhaustion was more strongly related to triglycerides, and only vital exhaustion showed a direct link to body mass index. No significant direct associations with blood pressure could be found for depressive symptoms or vital exhaustion. Although vital exhaustion is less often assessed as depression, the findings of this research support its importance as a health-related psychological risk factor.

Moreover, different dimensions of anger expression related differently to adverse lifestyle factors and also to systolic and diastolic blood pressure. The association of anger-in with elevated blood pressure seems to be mediated by adverse life styles, for the anger-control findings indicated statistically significant direct pathways to elevated blood pressure but no significant links with lifestyles. Open expression of anger seemed to have a negative association with high blood pressure.

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- II. Igna, C.V., Julkunen, J., & Vanhanen, H. (2009). Anger expression styles and blood pressure: evidence for different pathways. *Journal of Hypertension*, 27(10), 1972–1979.
- III. Igna, C.V., Julkunen, J., & Vanhanen, H. (2011). Vital exhaustion, depressive symptoms and serum triglyceride levels in high-risk middle-aged men. *Psychiatry Research*, 187(3), 363–369.
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ABBREVIATIONS

AHA	anger-hostility-aggression
AIC	Akaike information criterion
BDI	the Beck Depression Inventory
BMI	body mass index
BP	blood pressure
CAD	coronary artery disease
CFI	comparative fit index
CHD	coronary heart disease
CVD	cardiovascular diseases
DBP	diastolic blood pressure
EM	expectation-maximization
GFI	goodness of fit index
HDL	high density lipoprotein
HMSP	Helsinki Metabolic Syndrome Prevention Trial
HUS	Helsinki University Central Hospital
KIHD	Kuopio Ischemic Heart Disease
LDL	low-density lipoprotein
MI	myocardial infarction
NA	negative affectivity
NFI	the normed fit index
NKS	North Karelia Study
PGFI	the parsimony goodness of fit index
RMSEA	the root mean square error of approximation
SBP	systolic blood pressure
SEM	structural equation modelling

TG	serum triglycerides
TLI	Tucker-Lewis index
VE	vital exhaustion
χ^2	the chi-square test

1 INTRODUCTION

1.1 Cardiovascular diseases

1.1.1 Prevalence of cardiovascular diseases

Cardiovascular diseases (CVD) are one of the main causes of death. Around 18 million deaths worldwide are related to cardiovascular diseases (Nieuwlaat, Schwalm, Khatib, & Yusuf, 2013), and in Europe they account for over 4.3 million deaths, nearly half of all deaths on the continent (Esper & Subramaniam, 2012).

In Finland the first serious discussions and analyses of the causes of CVD emerged in the 1950s and 1960s. At that time, Finland had the highest rates of heart disease mortality in the world. Because of this, the so called East-West Project began in 1959, with the aim of investigating the causes of CVD and testing the cholesterol hypothesis (Puska, Vartiainen, Laatikainen, Jousilahti, & Paavola, 2009). From 1970, the start of the North Karelia Project, until 2005 the prevalence of CVD deaths in Finland fell by 80% and this trend has continued (Puska et al., 2009). CVD share of all deaths decreased from 41.1% in 2007 to 39% in 2011 (*Official statistics of Finland (OSF): Causes of death [e-publication]*). CVD morbidity and mortality, however, are still major challenges for public health in Finland, and new methods for effective preventive work are especially needed in primary health care.

There are differences in CVD between men and women, both in mortality and incidence. The 2003 statistics for Finland reveal that ischemic heart disease was the leading cause of death for men (18.5% of all deaths) with cerebrovascular disease in sixth position with 358 cases (4.9% of all deaths). However, the same statistics show that ischemic heart disease was the third largest cause of death for women (7% of all deaths) with cerebrovascular disease in the same position as for men but accounting for a higher share of deaths (6% of all deaths) (*Official statistics of Finland (OSF): Causes of death [e-publication]*).

Furthermore, several studies found that women experience their first myocardial infarction (MI) 9 years later than men and their first stroke 4.3 years later (Institute of Medicine, 2010).

1.1.2 Risk factors of cardiovascular diseases

Between 1960 and 1970, at the start of the North Karelia Study (NKS), the authors found evidence identifying the main CVD risk factors: serum cholesterol, blood pressure and smoking (Puska et al., 2009). Further research has shown that there is enough evidence to also other variables on the list of CVD risk factors, including: diabetes, diet (unhealthy aliments, salt consumption), alcohol, inactivity, overweight, air pollution, genetics, non-optimal birth weight, rapid weight gain after infancy, adverse childhood experiences, poor social circumstances in childhood, and psychosocial factors such as depression, anxiety, anger, hostility, chronic and acute stress (Theorell et al., 2006), lack of social support, high job stress (Kivimäki et al., 2012) and major life events (Institute of Medicine, 2010). Most of these variables have been tested in different studies. In the INTERHEART study, nine risk factors (smoking, history of hypertension or diabetes, waist-hip ratio, dietary patterns, physical activity, consumption of alcohol, blood apolipoproteins and psychosocial factors) accounted for 90% of the population attributable risk in men and 95% in women (Sheps, Frasure-Smith, Freedland, & Carney, 2004). Further, in the INTERSTROKE study, ten risk factors (history of hypertension, current smoking, waist-to-hip ratio, diet risk score, regular physical activity, diabetes mellitus, alcohol intake, psychosocial stress and depression, cardiac causes and ratio of apolipoproteins B to A1) were all significantly related to ischemic strokes. The authors concluded that these ten risk factors accounted for 90% of the risk of strokes and that interventions for reducing blood pressure and smoking, and promoting physical activity and a healthy diet, could reduce the prevalence of strokes (O'Donnell et al., 2010).

CVD risk factors can be listed in several different categories. Some of them are related to genetics. Moreover, in utero or early infancy non-optimal developments can increase the risk of CVD very early in life. There are also external variables, such as social factors (availability of health services), economical factors and environmental factors. In addition there are a number of lifestyle related risk factors including unfavourable consumption habits (food, alcohol, smoking) and physical inactivity). Finally, there are several psychological and personality related factors, including negative affectivity, which are considered risk factors for cardiovascular diseases. Over a person's lifetime, these genetic, external or lifestyle risk factors lead to adverse health conditions (obesity, elevated blood pressure and unfavourable lipid balance, blood glucose, etc.) that contribute further to the development of CVD.

1.2 Preventable CVD predictors

1.2.1 Role of prevention in CVD

The burden of CVD on health care is considerable, and it is expected to increase further during the coming decades (Weintraub et al., 2011). This not only leads to higher financial costs but also to increasing societal problems and, at an individual level, to shorter life expectancy or diminished quality of life.

Evidence gathered over many years has supported the argument that CVD can even be prevented through prevention of other conditions, such as type 2 diabetes, which as the Finnish Diabetes Prevention Study showed can be prevented by changes in lifestyle (Tuomilehto et al., 2001). Furthermore, another study suggested that several variables can be used as a screening tool for detecting undiagnosed type 2 diabetes to improve the chances at intervening before the onset of the disease (Saaristo, 2011). These findings indicate that prevention efforts

can be aimed at avoiding an initial cardiovascular event or protecting people against a recurrent cardiovascular event. Moreover, two practical intervention approaches have emerged, one based on using preventive medication and the other one based on implementation of lifestyle changes.

Conforming Geoffrey Rose's theory (Rose, 1981), an interesting and useful phenomenon was identified by the NKS. It was repeatedly confirmed that not only individuals with high risk levels but also those with moderate risk levels are at risk of CVD events. Furthermore, findings from the NKS showed that individuals with clinically high risk factor levels account for only a small percentage of those who suffer CVD events, these high risk cases being outnumbered by those with more than one risk factor at a moderate level. This group of people with several moderate risk factors constitutes the majority of those who suffer CVD events in a community (North Karelia Study, page 31) (Puska et al., 2009).

Based on this finding, it has been argued that preventive measures should be addressed to the whole population at a community level. However an alternative strategy, called the high risk strategy, claims that the most cost effective prevention is achieved by focusing on high risk individuals, who can be screened out with routine health checks (Manuel et al., 2013). In fact, the high risk strategy has added value to primary prevention and it is more individual based. High risk individuals are found by screening, and the positive impact at an individual level is greater than is the case when using primary prevention. Furthermore, the impact of the high risk strategy has increased with the use of more effective medication and more precise knowledge about healthy lifestyles (e.g. nutrition and exercise, etc) (Cecchini et al., 2010).

1.2.2 Lifestyle: positive and negative relations to psychological and biomedical variables

Relationships between lifestyle variables and psychological variables are often bidirectional. The presence of a mental disease, or its symptoms, may have a negative impact on a person's lifestyle. This can include higher levels of substance abuse (tobacco, alcohol, and drugs), unhealthy diet, inactivity, sleep disturbances or a low level of social interaction.

On the other hand, lifestyle variables may have contradictory effects on general or mental health. Generally, the effects of smoking and substance abuse (except perhaps for the moderate use of alcohol) are considered harmful. However, exercising and outdoor activities are considered beneficial at a light-moderate level. Diet is an important lifestyle factor in relation to health. Dietary recommendations include the low consumption of salt, sugar, fat, eggs or meat (red/processed) and a healthy diet that includes vegetables (preferably fresh), fruit, fish (oil), whole grain cereals and seeds. It is recommended that coffee and tea are consumed in moderation.

1.2.3 Overweight

Overweight and obesity are defined by a non-optimal height/ weight ratio that is characterized by excessive fat accumulation.

Overweight and obesity are matters of concern because they have been repeatedly shown to associate with CVD, type 2 diabetes, respiratory problems, musculoskeletal problems and some cancers. The distinction between overweight and obesity is relevant, but both increase the probability of health problems (Cooney, Cooney, Dudina, & Graham, 2011). Overweight and obesity are the result of several factors: genetics, family history, metabolism, health status, dietary habits, other lifestyle factors, and the environment. Beyond the fact that

some factors can not be changed, still, in most cases the condition is preventable and reversible.

1.3 Negative affectivity

1.3.1 Negative affectivity- conceptualization

Negative affectivity (NA) has been described as a trait tendency or temperamental sensitivity to experience intense negative stimuli or feelings. It has been conceptualized as a higher order construct centring around three affective dispositions: depression, anxiety, and anger-hostility (Suls & Bunde, 2005). However, in an extensive study analysing the effects of negative affectivity on health, Watson & Pennebaker (1989) concluded that negative affectivity is a rather more general trait of psychosomatic distress (Watson & Pennebaker, 1989).

The key features of NA are actually quite broad constructs with different elements and indicators. For example, depression is a complex notion that has strong correlations with other related concepts, such as vital exhaustion and hopelessness (Iliceto et al., 2011).

Similarly, hostility and anger are closely related, but still different, concepts. Hostility is defined as a personality trait, mainly characterized by the negative evaluation of other people which has cognitive, affective or behavioural facets. Hostility is usually described as a stable cognitive attitude (Eckhardt, Barbour, & Stuart, 1997). On the other hand, anger together with disgust and contempt are perceived as affective components of hostility and are not personality traits but affective states. Anger is depicted as a negative emotion caused by frustration or threats and leading to the negative appraisal of self or society (Schieman, 1999). A higher level of hostility facilitates the expression of anger (Eckhardt et al., 1997).

1.3.2 Depression/ Depressive symptoms

Depression is a mental disorder, and there are different types of depression. The term also refers to a mood characterized by several negative emotions. Only when negative emotions have more intense or prolonged manifestations and impair a person's normal way of life, they are considered symptoms of mental illness. Symptoms of depression include not only negative emotions but also other symptoms, such as weight loss or change in appetite, disturbed sleep patterns, difficulties in thinking or concentration, and inhibited natural urges, for example. For a psychiatric diagnosis of depression, several symptoms have to be experienced almost continuously and for a longer period of time (ICD-10, pg. 94-95) (World, 1992).

On the other hand, depressive symptoms that do not meet the specific requirements for a psychiatric diagnosis are present more often in the general population. However, these symptoms are manifested less frequently, at a lower intensity and do not severely impair social life, the ability to work or other executive functions; nevertheless, these symptoms are not without importance. There are more instruments focused on evaluating depressive symptoms, and over time these symptoms have been found to associate with different diseases or non-optimal physiological indicators.

1.3.3 Vital exhaustion

After severe CVD events most patients feel tired and experience negative feelings. However, studies that have tried to identify the precursors of myocardial infarction have shown evidence that those feelings were not the result of the CVD event but were present before it. Appels considered that this prodromal state reflected the exhaustion of adaptation resources and named it Vital Exhaustion (Appels, 1990). Its most common characteristics are extreme fatigue associated with

loss of energy, irritability and demoralization. Because Vital Exhaustion was perceived as an independent risk factor, researchers investigated whether it could contribute to the identification of people at higher risk of CVD events and thus add to the predictive power of already known risk factors. Consequently, Appels modified the Maastricht Questionnaire to evaluate vital exhaustion symptoms, allowing it to better predict future CVD events. The tiredness factor appears to be an enhanced predictor of recurrent coronary events, and lack of hope is marginally associated with new cardiac events.

Vital exhaustion is a concept closely related to depression, but depressed mood, low self-esteem and feelings of guilt are exclusive to depression. Vital exhaustion has also been found to precede depression, and some have argued that it represents a broader concept (Kopp, Falger, Appels, & Szedmak, 1998).

The effects of Vital Exhaustion have been explained through several pathophysiological mechanisms. Furthermore, the combination of VE and smoking can amplify the risk of CVD events.

1.3.4 Hopelessness

Hopelessness is a negative feeling characterized by negative expectations of any future development and associated with a negative perception of a person's own resources or abilities.

Generally, hopelessness is perceived as a symptom of depression, and it is included in the definition of depression (Joiner et al., 2001). However, the association of hopelessness with known depression scales is not very strong, and the presence of hopelessness is not necessarily related to depression (Abela, 2001; Greene, 1989), suggesting that hopelessness could, to some extent, be a different concept from depression (Everson et al., 1996).

Feelings of hopelessness have several consequences, such as loss of motivation, inactivity and amplification of other depressive symptoms. Consequently, there have been different attempts to measure hopelessness. One such attempt was initiated by Beck (Beck et al.,

1961), who proposed a scale for measuring the three factors considered most relevant for hopelessness: feelings about future, loss of motivation and expectations. The purpose was to measure pessimism about the future and thereby ultimately to predict the risk of suicide. More recently, other shorter scales have been designed with only one or two questions; they too have confirmed hopelessness as a relevant predictor of CVD events (Everson et al., 1996) and coronary artery disease (CAD) risk (Barefoot et al., 2000).

1.3.5 Anger expression

Due to its mainly negative consequences, anger and its expression have received much attention, both in society at large and later in different health disciplines. The sources of anger are various, and the debate about the preponderance of genetic or environmental factors remains open. On the other hand, the effort to deal with depressive symptoms can lead to anger. The most common functioning pattern of anger is to focus on the elements that activated it and anticipate other circumstances that are associated with the initial trigger or could cause further anger. In psychology, anger expression can be perceived as an intermediate concept between anger and aggression. If anger is an emotional state or a trait, and (self-)aggression is the behaviour resulting from it, anger expression is the concept that helps conceptualise the point where an anger management option is finally chosen.

In recent decades, scales of anger expression have accounted for different dimensions of expression. This research used Spielberger's 24 items Anger Expression Scale (Spielberger et al., 1985). Spielberger defined three types of anger expression: inwardly expressed anger (i.e. anger-in), outwardly expressed anger (anger-out) and control of anger expression (anger-control). This categorization has since been further developed, with anger control was divided into two components: anger-control for anger-in and anger-control for anger-out. Anger expression

styles can be described as coping mechanisms that try to restore the affective balance (Kerr & Schneider, 2008). In most studies, these three factors have been found to be associated with health outcomes (Gouin, Kiecolt-Glaser, Malarkey, & Glaser, 2008; Haukkala, Konttinen, Laatikainen, Kawachi, & Uutela, 2010)

1.4 Associations between psychological variables and main CVD predictors

1.4.1 Associations between negative affectivity and lifestyle

Components of negative affectivity have been shown to associate with unhealthy diet and other lifestyle factors (Kelloniemi, Ek, & Laitinen, 2005). For example, there is evidence showing the negative impact of depression on life-style factors (Goldston & Baillie, 2008). Furthermore, both depressed and hopeless people alike are more likely to engage in smoking, a sedentary lifestyle and over-consumption of alcohol (Valtonen et al., 2009).

Vital exhaustion is another depression-related notion thought to have an influence on lifestyle, but the findings have been less conclusive. There are studies that suggest a positive relationship between vital exhaustion and smoking (Kopp et al., 1998; Schwartz, Carlucci, Chambless, & Rosamond, 2004), alcohol consumption (Conduit, Appels, & Lewis, 1998) and inactivity (Brezinka, Dusseldorp, & Maes, 1998), but in some other studies no relationship between vital exhaustion and these variables has been found (Bages, Appels, & Falger, 1999; Koertge, Ahnve, Schenck-Gustafsson, Orth-Gomer, & Wamala, 2003).

Furthermore, anger expression is a concept associated with negative affectivity that has an influence on lifestyle factors. Previous research has shown that smoking, alcohol consumption, exercise habits, and diet (Anton & Miller, 2005; Eng, Fitzmaurice, Kubzansky, Rimm, &

Kawachi, 2003; Musante & Treiber, 2000) are related to anger expression.

1.4.2 Associations of negative affectivity with BMI

The presence of depression (with or without other diagnoses) is related to a higher body mass index when compared to healthy people (Trief, Ouimette, Wade, Shanahan, & Weinstock, 2006). In spite of the fact that numerous studies have shown that body mass index is related to depression (Skilton, Moulin, Terra, & Bonnet, 2007; Toker, Shirom, & Melamed, 2007; Vaccarino et al., 2008), depressive symptoms (Aijänseppä et al., 2002; Skilton et al., 2007; Vaccarino et al., 2008) and vital exhaustion (Prescott et al., 2003), only a small number of studies have tested the mediational role of body mass index (Horsten, Wamala, Vingerhoets, & Orth-Gomer, 1997; Koertge et al., 2003).

1.4.3 Associations of negative affectivity with blood pressure and blood lipids

Depression related variables - BP

Research suggests that depression is an important risk factor in the onset and development of CVD (Musselman, Evans, & Nemeroff, 1998; Suls & Bunde, 2005; Wulsin & Singal, 2003), and especially in CAD (Barth, Schumacher, & Herrmann-Lingen, 2004; Carney, Freedland, Miller, & Jaffe, 2002; Lett et al., 2004). Furthermore, even depressive symptoms in the sub-clinical range have been shown to confer an increased risk of recurrent CAD events (Kubzansky, Davidson, & Rozanski, 2005; McCaffery, Niaura, Todaro, Swan, & Carmelli, 2003a; Suls & Bunde, 2005).

Although vital exhaustion and hopelessness correlate strongly with depression, they do not always have identical relationships with health outcomes. For example, some studies have indicated that depression and vital exhaustion associate differently with coronary disease risk

factors (Lahlou-Laforet et al., 2006); However, in another study, (Wojciechowski, Strik, Falger, Lousberg, & Honig, 2000) no significant differences were found.

In contrast, Everson et al. (Everson, Kaplan, Goldberg, & Salonen, 2000) found that hopelessness, but not depression, predicted the incidence of hypertension in middle-aged men and concluded that, over a period of four years, initially normotensive men with high baseline levels of hopelessness had a three-fold risk of becoming hypertensive. In line with the results published by Everson, another study (Valtonen et al., 2008) using the same hopelessness items and classes indicated that high systolic blood pressure was associated with high levels of hopelessness and there was a significant mean difference in systolic blood pressure between the three hopelessness groups. However, a significant association between hopelessness and hypertension has not been found in all studies (Stern, Dhanda, & Hazuda, 2009).

Depression variables – blood lipids

It has been found that depression and BMI are associated with serum lipids (Barth et al., 2004; Kelley, Kelley, & Vu Tran, 2005; Lett et al., 2004), and the incidence of CAD.

However, the association between cholesterol and depressive symptoms is characterized by contradictory research findings and a lack of consensus (Suarez, 1999; Yang, Jick, & Jick, 2003). For example, in a special issue of *Psychosomatic Medicine* on depression and heart disease (Depression and heart disease: Epidemiology pathophysiology & treatment, 2005), the potentially important question of relationship between depression and cholesterol was not discussed. Furthermore, there are studies strongly suggesting that total cholesterol measurement should be broken down into LDL and HDL measurements or other such cholesterol fractions (Aijänseppä et al., 2002; Gary, Crum, Cooper-Patrick, Ford, & Brancati, 2000; Wulsin et al., 2005).

Regarding triglycerides, another important blood lipid, the results of one study suggested that the simultaneous incidence of different mental problems (post-traumatic stress disorder and depression) was related to higher triglyceride levels as compared to those who had only one diagnosis or none (Trief et al., 2006). At the same time, growing research evidence suggests that depression (Brunner et al., 2006; Huang & Chen, 2004; Trief et al., 2006) or depressive symptoms and vital exhaustion (Kop, Hamulyak, Pernot, & Appels, 1998) correlate with triglyceride levels. From this perspective, triglycerides – together with other serum lipids – seem to be a probable mediator in the relationships between depression/exhaustion and CAD. In general, elevated triglyceride concentrations are considered a valid marker of unhealthy lifestyles.

Anger expression - BP

Previous research suggests that not only trait anger but also acute anger expression are related to blood pressure levels (Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003). Furthermore, anger expression factors have been found to be associated with health outcomes (Gouin et al., 2008; Haukkala et al., 2010). Several studies have suggested that repeated experience and expression of anger are related to vascular pathology (Everson et al., 1999; Kawachi, Sparrow, Spiro, Vokonas, & Weiss, 1996) and that elevated blood pressure is one plausible mediating mechanism (Eng et al., 2003; Starner & Peters, 2004). The classic hypothesis claims that anger-in is related to higher blood pressure. However there are some more specific findings relating to BP. Anger-in has been found to relate to lower diastolic blood pressure (DBP) (Spicer & Chamberlain, 1996; Steele & McGarvey, 1997) and higher systolic blood pressure (SBP) (Bongard & al'Absi, 2005; Helmers, Baker, O'Kelly, & Tobe, 2000). Furthermore, in one study anger-out was related to lower diastolic blood pressure (DBP) (Schum et al., 2003; Steele & McGarvey, 1997) and higher systolic blood pressure (SBP) (Bongard & al'Absi, 2005). Moreover, anger-

control has been shown to relate to higher DBP (Steele & McGarvey, 1997).

However, despite the fact that all the above studies have shown a significant relationship between some component of anger expression and blood pressure, there are also studies that have failed to find any significant relationships (Markovitz, Matthews, Kannel, Cobb, & D'Agostino, 1993; Mueller, Grunbaum, & Labarthe, 2001).

Associations between lifestyle and BMI and blood pressure and blood lipids

One study (Reiff, Schwartz, & Northridge, 2001) found that lifestyle factors, but not depressive symptoms, were associated with blood pressure (BP). Excessive alcohol consumption (Jo et al., 2001; Niskanen et al., 2004), active smoking (Halimi et al., 2002; Narkiewicz, Kjeldsen, & Hedner, 2005; Niskanen et al., 2004), a low level of exercise (Atlantis, Barnes, & Singh, 2006; Brock, King, Wofford, & Harrell, 2005) and unhealthy diet (Carollo, Presti, & Caimi, 2007; Hermansen, 2000) are commonly related to higher levels of blood pressure.

The relationship between smoking and BP seems to deserve a more detailed analysis. Although it is well known that smoking produces a chronic increase in BP for active smokers (Halimi et al., 2002; Narkiewicz et al., 2005; Niskanen et al., 2004), previous literature displays several contradictory findings on the association between smoking and BP. Although it has sometimes been found that smoking increases BP (Halimi et al., 2002; Narkiewicz et al., 2005; Niskanen et al., 2004), some studies have discovered no relationship between smoking and BP (Green & Harari, 1995; Okubo, Suwazono, Kobayashi, & Nogawa, 2004). A few studies have been suggested that smoking is related to a decrease in BP (Okubo et al., 2004; Webber et al., 1982) or that giving up smoking is related to higher BP (D. H. Lee, Ha, Kim, & Jacobs, 2001).

Coffee consumption is higher among those with a history of myocardial infarction (Appels, Falger, & Schouten, 1993) and relates to BP (Nurminen, Niittynen, Korpela, & Vapaatalo, 1999).

Lifestyle factors also explain variations in BMI (Chiolero, Faeh, Paccaud, & Cornuz, 2008; Croezen, Visscher, Ter Bogt, Veling, & Haveman-Nies, 2007; John, Hanke, Rumpf, & Thyrian, 2005), and BMI relates to higher blood pressure (Brock et al., 2005; Jo et al., 2001; Niskanen et al., 2004).

Furthermore, lifestyle factors, especially exercise (Halverstadt, Phares, Wilund, Goldberg, & Hagberg, 2007; Stefanick et al., 1998), have a direct relationship with serum lipids (Schubert et al., 2006; Yamamoto et al., 2003). The association between cholesterol and BMI (Egusa & Yamane, 2004; Kelley et al., 2005; Schubert et al., 2006), and cholesterol-lifestyle factors has been extensively studied and clarified (Martinez-Gonzalez et al., 1998; Schubert et al., 2006; Yamamoto et al., 2003).

Triglyceride levels are influenced by body mass index (BMI) (Dunbar & Rader, 2005; Knox, Jacobs, Chesney, Raczynski, & McCreath, 1996; McCaffery, Niaura, Todaro, Swan, & Carmelli, 2003b), smoking (Tirosh et al., 2007), inactivity, alcohol consumption (Dunbar & Rader, 2005) and diet (Aligeti, Gandhi, Braden, Rezk, & Elam, 2007; Bhargava, 2006; Gorinstein et al., 2006). However, one study found that alcohol consumption did not have a linear relationship with triglycerides (Wakabayashi & Kobaba-Wakabayashi, 2002).

Associations between confounding factors

Several variables, such as education, marital status and, especially, age, have shown to have associations with negative affectivity, lifestyle, BMI or health outcomes.

Age is associated with negative affectivity (Jorm, 2000) and with anger expression (Eng et al., 2003; Schieman, 1999; Steele & McGarvey, 1997). Age has also been shown to affect several lifestyle factors, including smoking behaviour (Osler et al., 1998), diet

(Drewnowski, Henderson, Driscoll, & Rolls, 1997) and influence BMI (Heo, Pietrobelli, Fontaine, Sirey, & Faith, 2006; Hsieh, Yoshinaga, Muto, & Sakurai, 2000). Age also has multiple effects on health outcomes, through its association with BP (Anderson, 1999; Jo et al., 2001; Niskanen et al., 2004) and triglycerides (Keltikangas-Järvinen, Ravaja, & Viikari, 1999; Wakabayashi & Kobaba-Wakabayashi, 2002), although for triglycerides one study found that the relationship was not linear (Wakabayashi & Kobaba-Wakabayashi, 2002).

According to previous literature, socio-economic status, level of education, and marital status are usually seen as confounding factors which need to be controlled for. On the other hand, sociologically oriented health research underlines marital status (S. Lee et al., 2005; Nystedt, 2006; Schoenborn, 2004) and education (Wardle & Steptoe, 2003; Woo et al., 1999; Yarnell et al., 2005) as important explanatory factors which determine health behaviour. A high level of education and the presence of a permanent relationship are claimed to act as protective factors against depressive feelings or detrimental lifestyle behaviours.

Possible physiological mechanisms

Theories based on previous studies suggest that the impact of depression on CAD could be mediated in two ways. In the direct link model, it is supposed that depression influences physiological mechanisms such as platelet activity, inflammation, diabetes, obesity or hypertension (Barth et al., 2004; Carney et al., 2002; Lett et al., 2004). The indirect model assumes that depression acts through lifestyle factors (Barth et al., 2004; Lett et al., 2004). Although our knowledge of how depression is related to CAD has rapidly growing we are still far from fully understanding the interplay of all the mediating mechanisms (Depression and heart disease: Epidemiology pathophysiology & treatment.2005).

The possible mechanisms through which hopelessness can cause hypertension include endothelial dysfunction (Do, Dowd, Ranjit,

House, & Kaplan, 2010) or atherosclerosis (Everson, Kaplan, Goldberg, Salonen, & Salonen, 1997; Pollitt et al., 2005). However this issue also awaits further studies.

Candidate mechanisms by which VE predicts CAD events are impaired fibrinolysis and inflammatory changes, with the latter also affecting haemostasis. A possible biological mechanism that relates vital exhaustion to diet, body mass index and triglycerides is explained in a study by Koertge (Koertge et al., 2003).

A widely supported theory explains that raised levels of stress/negative feelings activate the sympathetic-adrenal-medullary system and the hypothalamic-pituitary-adrenal-cortical axis system, leading to increased serum levels of catecholamines and cortisol, which has an influence on blood pressure (BP) and heart rate (Kubzansky & Kawachi, 2000).

The direct inverse relationship of smoking with BP could be explained through some physiological mechanisms not yet fully understood. One study found evidence that genotypic variations influence the relationship between smoking and the probability of developing hypertension (Abe et al., 2002). Another report suggests that smoking behaviour is related to higher BP only if smoking induces degenerative changes or remodelling of small arteries (Takashima et al., 2002).

In terms of a direct physiological link between depression and cholesterol, possible mediating mechanisms include the impact of depressive symptoms on the autonomic nervous system, imbalance and activation of the hypothalamic-pituitary-adrenocortical axis, dysregulation of immunologic mechanisms, coagulation abnormalities and vascular endothelial dysfunction (Barth et al., 2004). However, the specific mechanisms through which a direct relationship between depression and high HDL and low LDL could be explained cannot be found in previous literature.

Taken together, the review of previous research presented above shows that although many previous studies have contributed to our

knowledge of the interplay between psychological, lifestyle, and health variables, these relationships are only partially understood. There are several contradictory findings results in this area, and usually only a small number of relevant variables have been covered in any single study. It would be especially important to clarify the extent to which lifestyle factors could explain the association between facets of negative affectivity and health outcomes. To answer these questions and to avoid at least some of the pitfalls of previous studies, it seems necessary to apply structural equation modelling (SEM) based on a more comprehensive database including several psychological, lifestyle, and CVD risk factor variables.

2 AIMS OF THE PRESENT STUDY

The general aim of the present study was to investigate the direct and lifestyle mediated relationship between negative affectivity, i.e. depressive symptoms, hopelessness, vital exhaustion and anger expression, and well-known CVD risk factors. To achieve this, four studies were completed. The basic general design of the studies is illustrated in Figure. 1.

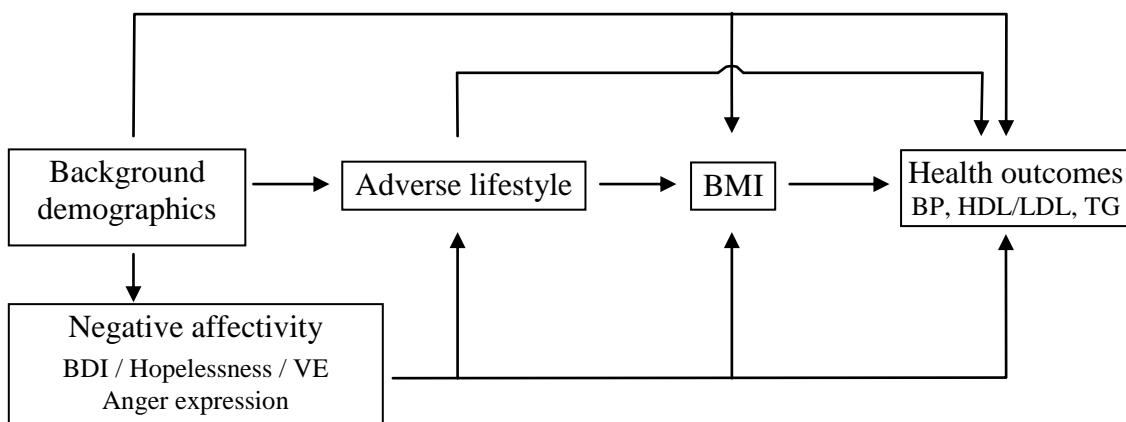


Figure 1. Theoretical model of the relationship between facets of negative affectivity and health outcomes.

Study I: The aim of this study was to investigate a basic model and an alternative model for clarifying the relationship between depressive symptoms and serum cholesterol levels.

Both models investigated partial mediation and direct effects. In the basic model, the impact of depression on cholesterol fractions was supposed to act through the mediating factors of lifestyle and BMI. In addition, an independent, direct link from depression to cholesterol fractions was expected to emerge. Age, education, and marital status were included as possible confounding factors. This model, however, is not the only plausible one, for serum cholesterol could also have an effect on depressive symptoms.

Sociologically oriented health research underlines marital status and education as important explanatory factors which determine health behaviour; a high level of education and the presence of a permanent relationship act as protective factors against depressive feelings or detrimental lifestyle behaviours. Furthermore, variations in lifestyle explain variations in depression and BMI, which through their influence on serum lipids also affect the incidence of CAD. Lifestyle factors thus have a direct relationship with serum lipids. A model based on this argument is investigated as an alternative model.

Study II: The aim of this study was to investigate a hypothetical model, separately applied for each of the three anger expression styles (anger-in, anger-out and anger-control), that attempts to provide a better understanding of the contribution of each type of anger expression to the level of BP. Moreover, this model was intended to allow evaluation of whether different anger expression styles have a direct relationship to BP, or if it is mediated through lifestyle factors and BMI.

It was hypothesized that anger expression styles are differently linked to BP, and both mediated and direct relationships were expected.

Study III: The aim of this study was to investigate the possible direct or indirect relationships between depressive symptoms and vital exhaustion and serum triglyceride levels.

It was expected that body mass index would prove an important mediating factor between lifestyle and triglyceride levels. It was also expected that depressive symptoms and vital exhaustion would have, to some extent, similar relationships with lifestyle factors and serum triglyceride levels.

Study IV: The aim of the fourth study was to investigate the possible direct or indirect relationships between depressive symptoms, hopelessness and vital exhaustion and blood pressure.

It was expected that depressive symptoms, vital exhaustion and hopelessness would have, to some extent, similar relationships with lifestyle factors and BP.

Table 1. List of variables included in each study

	Psychological predictor	Lifestyle variables	Covariates	Outcome variables
Study I (N = 893, population sample)	Depressive symptoms	Alcohol Smoking Inactivity Healthy diet Sweets	Age Education Marital status	HDL LDL
Study II (N = 705, population sample)	Anger expression	Alcohol Smoking Inactivity Healthy diet	Age Education	DBP SBP
Study III (N = 444, high risk sample)	Depressive symptoms Vital exhaustion	Alcohol Smoking Inactivity Healthy diet	Age Education	TG
Study IV (N = 710, population sample)	Depressive symptoms Vital exhaustion Hopelessness	Alcohol Smoking Inactivity Healthy diet Coffee	Age Education Marital status	DBP SBP

HDL - high density lipoprotein, LDL – low density lipoprotein, DBP – diastolic blood pressure, SBP – systolic blood pressure, TG - triglycerides

3 METHODS

3.1 Outline of the study

The present study is a sub-study of the HMSP (Helsinki Metabolic Syndrome Prevention Trial). This was an uncontrolled preventive trial aimed at improving prevention of metabolic syndrome, type 2 diabetes and cardiovascular diseases by developing a practical method for general health care. Its goals were to screen and identify men with a cluster of cardiovascular risk factors and offer them individual counselling.

An invitation letter and questionnaire was posted to 2990 middle-aged male residents of Helsinki. It was a population sample where all men aged 40-55 living in the north-eastern district of the city of Helsinki were invited to participate. A total of 1288 (43.1 %) participated in a screening visit and were asked to answer questionnaires dealing with lifestyle factors and the psychosocial risk factors of CVD. Study nurses from the Helsinki Heart District interviewed all the participants and recorded basic biomedical measurements.

Data collection was carried out between May 2001 and June 2004. The Ethical Committee of the Helsinki University Central Hospital (HUS) approved the study on the 24th April 2001.

3.2 Participants

3.2.1 Population sample

A total of 1288 men belonging to four age cohorts: 40, 45, 50 and 55-year-olds, were screened for the preventive trial. In studies I, II and IV all those below 45 years of age (22.9 %) were excluded from the present analysis because, for logistical reasons, complete psychosocial

data was only available for high-risk cases in that cohort. Thus, the remaining number of cases included in studies I, II and IV was 1005.

Study I

Subjects with uncertain smoking status (n =39) or those receiving cholesterol treatment (n =74) were excluded. The final study sample was 893 men; the mean \pm standard deviation age was 49.4 ± 3.8 years. In this study sample, 22 men had diabetes and 12 had angina pectoris and/or coronary heart disease symptoms.

Study II

Cases with uncertain smoking status (n=39) were excluded, as were those on BP medication (n=163), those with missing anger expression scores (n=105), and one person with missing BP values. The final study sample was 705; the mean age was 49.33 years (S.D., 3.74).

Study IV

Furthermore, were excluded those with missing depression, hopelessness or vital exhaustion scores, those with unclear smoking status and those taking blood pressure medication. The final study sample was 710 men; the mean age was 49.41 years (S.D., 3.78).

Using the 4-step classification of the Beck Depression Inventory (cut-off values: 0-9 = no depression, 10-18 = mild depression, 19-29 = moderate depression, 30-60 = severe depression), 76.1% of the subjects had no depression, 16.9% had mild depression, 5.8% had moderate depression, and only 1.3% had symptoms of severe depression. In the study sample thirteen men had diabetes and ten had angina pectoris and/or symptoms of coronary heart disease.

3.2.2 High risk sample

Evaluation of risk was based on a Risk Index that included five factors: body mass index, total serum cholesterol, blood pressure (systolic and

diastolic), smoking habits, and physical activity. The risk evaluation method originated from the North Karelia project and was further developed by Ketola and Klockars, whose cut-off for high risk of 4.5 points or above (Ketola & Klockars, 1999) was used in the current study. Supplementary laboratory tests were performed for high-risk participants.

Study III

All cases with a risk index below 4.5 were excluded from the present analysis because triglyceride (TG) values were not available. From the remaining sample (N=537), those with missing TG values, missing depression or vital exhaustion scores, or unclear smoking status were then excluded. The final study sample was 444 men (82.68% from high-risk cases); the mean age was 47.83 years (S.D., 5.33).

The means and standard deviations of the five factors in the risk index in this sample were body mass index, M=28.468; S.D.=4.74, total serum cholesterol, M=5.64 mmol/L; S.D.=1.19, blood pressure — systolic, M=140.08; S.D.=16.11, blood pressure — diastolic, M=91.04; S.D.=10.44, smoking habits, M=2.61; S.D.=2.88, and physical activity, M=3.07; S.D.=1.28.

Using the 4-step classification of the BDI (cut-off values: 0–9=no depression, 10–18=mild depression, 19–29=moderate depression, and 30–60=severe depression), 68.7% of the subjects had no depression, 23.2% had mild depression, 6.3% had moderate depression, and only 1.8% had symptoms of severe depression. In the study sample 12 men had diabetes and nine had angina pectoris and/or symptoms of CHD.

3.3 Psychological measures

3.3.1 Depression/depressive symptoms (Studies I, III, IV)

Depressive symptoms were assessed using the 21-item Beck Depression Inventory (BDI). It is a widely accepted measure of depression, and it has been used in the assessment of psychological risks in relation to coronary artery disease (Richter, Werner, Heerlein, Kraus, & Sauer, 1998).

In the fourth study Cronbach's alpha coefficient for internal consistency was 0.89.

3.3.2 Vital exhaustion (Studies III, IV)

Vital exhaustion was assessed using the 21-item Maastricht Questionnaire. The scale has been previously used as a predictor of myocardial infarction (Appels, 1990). Internal consistency (Cronbach's alpha) has previously been shown to be good or excellent, and it was 0.92 in the third study and 0.87 in the fourth article, indicating an excellent reliability for the scale.

3.3.3 Hopelessness (Study IV)

Hopelessness was measured with a two-item Hopelessness Scale (Everson et al., 1997). The items were "I feel it is impossible to reach the goals I would like to strive for" and "The future seems hopeless to me and I cannot believe things will change for the better". These items were first used in Finland in the large Kuopio Ischemic Heart Disease (KIHD) study, and they have been shown to be associated with the risk of coronary heart disease (Everson et al., 1997). Participants responded using a five-point scale (0 - absolutely disagree, 1 - somewhat disagree, 2 - cannot say, 3 - somewhat agree, or 4 - absolutely agree). A hopelessness score with a range of 0 to 8 was created by summing the

items. The reliability for the sample of this study was $\alpha = 0.60$ (Cronbach's alpha). For a two-item scale this was considered acceptable.

3.3.4 Anger expression (Study II)

Anger expression was assessed using the Finnish adaptation of the 24-item Spielberger Anger Expression Scale, which is a widely used assessment scale with well established psychometric properties (Spielberger et al., 1985). The scale was developed to measure characteristic styles of coping with anger arousal and contains three 8-item subscales that separately measure self-reported levels of anger-out (anger outwardly expressed), anger-in (anger suppressed) and anger-control (controlled anger). Each item is rated on a four-point Likert-scale and is assigned a score of 1 to 4.

The unweighted sum of the eight items in each subscale yields the study variables. Representative items for each subscale are presented in the Appendix.

The Finnish adaptation of the scale, developed by Greenglass and Julkunen (1991), has been used in several earlier studies, both of healthy subjects and also of CHD patients, where the reliability of the translation has been tested (Julkunen & Ahlström, 2006; Julkunen, Salonen, Kaplan, Chesney, & Salonen, 1994; Greenglass & Julkunen, 1991).

In the present sample the internal consistency (Cronbach's alpha coefficients) of the subscales was anger-in = .76, anger-control = .89, anger-out = .77.

3.4 Biomedical measures

Study nurses from the Helsinki Heart District interviewed all participants and recorded basic biomedical measurements (e.g. blood pressure, blood lipids, waist circumference, weight).

3.4.1 Obesity

Body mass index (BMI) is a simple index of weight-for-height that is commonly used to classify overweight and obesity in adults. It is defined as a person's weight in kilograms divided by the square of height in meters (kg/m^2).

3.4.2 Blood pressure

Blood pressure was measured according to best practice at the time of data collection. A single measurement was taken by the trained study nurses according to standard protocol after a minimum of 30 minutes of rest. During the procedure the men were in a sitting position, and the measurement was taken using a calibrated mercury sphygmomanometer with the cuff around the right arm. The patients were asked not to smoke, drink coffee, tea or beverages containing caffeine or perform any heavy tasks for at least 60 minutes prior to measurement.

3.4.3 Blood lipids

A fasting blood sample was taken, and serum was separated for analysis in the central laboratory of the Helsinki University Hospital, which meets the highest quality standards. Serum cholesterol was measured by the enzymatic method. HDL-cholesterol was measured directly without any precipitation by the homogenic enzymatic method. Serum triglycerides were measured by the enzymatic method from li-

heparin plasma. LDL-cholesterol was calculated Friedewald's formula when concentrations of total cholesterol, HDL-cholesterol and serum triglycerides were known (Friedewald, Levy, & Fredrickson, 1972). Blood samples were taken in the morning, with at least 12 hours of fasting before.

3.5 Lifestyle measures

- *Smoking* was evaluated with one question about the number of cigarettes consumed per day (0 = not at all, 1 = from time to time, 2 = 1-4 cigarettes per day, 3 = 5-9 cigarettes per day, 4 = 10-14 cigarettes per day, 5 = 15-19 cigarettes per day, 6 = 20-24 cigarettes per day, 7 = 25-29 cigarettes per day, 8 = 30 or more cigarettes per day).

- *Inactivity* was assessed with one question about the frequency of exercise per week (1 = 3 or more times per week, 2 = 1-2 times per week, 3 = 1 time per week, 4 = from time to time, 5 = not at all).

The inactivity measure was taken from the North-Karelia project protocol (Matilainen et al., 1994; Pekkanen, Tuomilehto, Uutela, Vartiainen, & Nissinen, 1995; Puska et al., 2009).

- *Alcohol* consumption was evaluated with two questions referring to the frequency of alcohol consumption (1 = not at all, 2 = once or less per month, 3 = 2-4 per month, 4 = 2-3 per week, 5 = over 3 times per week) and quantity of alcohol consumed per drinking episode (1 = not at all, 2 = 1-2 drinks, 3 = 3-4 drinks, 4 = 5-6 drinks, 5 = 7-9 drinks, 6 = over 9 drinks). The alcohol variable used in these analyses is the product of code numbers from the questions on the frequency and quantity of alcohol consumption.

The alcohol measure was taken from the North-Karelia project protocol (Matilainen et al., 1994; Pekkanen et al., 1995; Puska et al., 2009).

- *Coffee* consumption was evaluated with one question asking how many cups of coffee were consumed each day.

- *Diet* was evaluated with questions asking how often a specific aliment was consumed per week (0= not at all, 1=1-2 days, 2= 3-5 days, 3= 6-7 days). The healthy diet variable was calculated as the sum score of these answers for specific aliments consumed (muesli/cereals, rice/pasta, fish, fresh vegetables, fruit).

The questions on diet were based on Nordic and Finnish dietary recommendations (Finnish nutrition recommendations, 2005; Fagt et al., 2012).

The Healthy Food variable included the answers to 5 questions about the consumption of muesli/cereals, rice/pasta, fish, fresh vegetables and fruit. Depending on the consumption frequency, a score between 0 and 3 was obtained for each type of food (0= not at all, 1=1-2 days, 2= 3-5 days, 3= 6-7 days). The variable score was calculated as the sum of the scores obtained by each type of aliment assessed. This variable can have a range of 0 to 15, with the null value indicating zero consumption of any of the food types assessed and a value of 15 indicating consumption almost everyday (6-7 days per week) of all the food types assessed.

- The sugary food variable (*sweets*) was calculated as the sum score of these answers for specific aliments consumed: sweets, ice cream, and fruit juice.

3.6 Statistical methods

3.6.1 Descriptive methods and correlations

Descriptive and correlation analyses were performed using SPSS 15.0 software. The associations between psychosocial and health behaviour factors and blood pressure were analysed using Spearman correlation coefficients.

Study I

For two covariates, education and marital status frequencies were calculated and presented in a separate table. For all other variables, including age (also as a covariate), mean and SD values were calculated.

The associations between psychosocial and health behaviour factors and cholesterol fractions were analysed using Pearson's correlation coefficients. Due to missing data, N varied between 605 and 893.

Study II

For education, variable frequencies were calculated and presented in the Results section. For all other variables, including age, mean and SD values were calculated.

The associations between anger expression types, covariates and health behaviour factors and blood pressure were analysed using Pearson's correlation coefficients. Due to missing data, N varied between 685 and 705.

Study III

For education, variable frequencies were calculated and presented in the Measures section. For all other variables, including age, mean and SD values were calculated.

The associations between BDI, VE, covariates and health behaviour factors and triglycerides were analysed using Pearson's correlation coefficients. Due to missing data, N varies between 424 and 444. Partial correlations controlled for age and education were also performed.

Study IV

For education and marital status, variables frequencies were calculated and presented in the Results section.

The associations between the BDI, VE, Hopelessness, covariates and health behaviour factors and blood pressure were analysed using Pearson's correlation coefficients. Due to missing data, N varied between 675 and 710. Partial correlations, controlled for age, education and marital status, were also calculated (N = 648).

3.6.2 Structural equation modelling (SEM)

Path analysis is closely related to multiple regressions and tests the fit of the correlation matrix of observed variables against a proposed theoretical model. The method analyses correlations, and the regression weights resulting from the models are judged against the observed correlation matrix of the variables, which yields different kinds of goodness-of-fit statistics.

The hypothetical models of the predictors were evaluated using the path analysis in Lisrel 8.50 software (Jöreskog & Sörbom, 1993) or MPLUS version 5.2 software (Muthén & Muthén, (1998-2007)) (study IV).

Before evaluation of the path models with Lisrel 8.50 or MPLUS version 5.2 software, all missing data were processed using the EM (expectation-maximization) algorithm (Enders, 2006) available in SPSS 15.0 software.

All the factors in the path analyses were used as continuous variables.

The chi square test (χ^2), the root mean square error of approximation (RMSEA), the comparative fit index (CFI), and the goodness-of-fit index (GFI) were used to judge the goodness-of-fit of the models. The RMSEA value < 0.06 , CFI > 0.95 , GFI > 0.90 , TLI > 0.90 and a non-significant ($p > 0.05$) χ^2 -test indicate an acceptable model (Kline, 2005).

The normed fit index (NFI) and the Akaike information criterion (AIC) are comparative fit measures. For the NFI, a value between .90 and .95 is acceptable, and above .95 is good. The AIC is used for

comparing non-nested models and a smaller value indicates a better model.

The parsimony goodness of fit index (PGFI) is a parsimonious fit measure. Its value ranges from 0 to 1, with higher values indicating a better fit.

This study was based on a cross-sectional database, and therefore it is inappropriate to infer causality relations between any variables of the model's.

Study I

The hypothetical models of the predictors of cholesterol fractions were evaluated using the path analysis in Lisrel 8.50 software.

Age was included in all path analyses as a possible confounding factor. In the first model, education and marital status were also controlled for. In the second, alternative, model, these two variables were included as explanatory factors influencing health behaviour, which, through BMI and depression as mediators, affects cholesterol fractions.

Path analyses were performed on the two models, the basic model and the alternative model, because this study was based on a cross-sectional database and could not infer causality between variables in the models.

Study II

The hypothetical models of the predictors of anger expression were evaluated using path analysis in Lisrel 8.50 software.

Before evaluation of the path models, all missing data were processed using the expectation-maximization algorithm available in SPSS 15.0 software. Missing values were replaced in four variables: three for education, seven for inactivity, fifteen for alcohol consumption and five for diet.

Age and education were used in path analyses as possible confounding factors.

To investigate the hypotheses of this study, separate models for each anger expression type (anger-in, anger-out and anger-con) as the main predictor of lifestyles and blood pressure were evaluated.

Study III

The associations between depressive symptoms and vital exhaustion and triglyceride levels were analysed using correlation analyses and path analyses.

The hypothetical models of the predictors (depressive symptoms, vital exhaustion, lifestyle factors and covariates) of triglycerides were evaluated using path analysis.

Before evaluation of the path models, all missing data (3 cases for education, 6 cases for exercise, 7 cases for alcohol consumption, and 9 cases for diet) were processed using the EM (expectation-maximization) algorithm.

Education and age were included in the path analyses as possible confounding factors.

Because the distributions of depression and vital exhaustion were positively skewed, these variables were transformed for path analysis using the root square procedure. For skewness and kurtosis the transformed variables had values below 1.0.

To investigate the hypotheses of this study, separate models for depression and vital exhaustion as main predictors of lifestyle and triglycerides were evaluated. Finally, a model including both depressive symptoms and vital exhaustion was tested.

Study IV

The hypothetical models of the predictors of BP were evaluated using the path analysis in MPLUS version 5.2 software. Before evaluation of the path models, all missing data (16 cases for healthy food, 7 cases for inactivity, 7 cases for alcohol, 22 cases for coffee) were processed using the EM (expectation-maximization) algorithm available in SPSS 14.0 software. Education, age and marital status were

included in the path analyses as possible confounding factors. Because distributions of depression, hopelessness and vital exhaustion were positively skewed, these variables were transformed for path analysis using the root square procedure. For skewness and kurtosis the transformed variables had values below 1.0.

To investigate the hypothesis of this study, six separate models were evaluated: depression predicting SBP and then DBP, vital exhaustion predicting SBP and then DBP, hopelessness predicting SBP and then DBP.

The indirect mediation effects of the main predictors on systolic and diastolic blood pressure were also evaluated.

4 RESULTS

Although depressive symptoms were evaluated with the BDI in our sample, the scores were mainly at a subclinical level. In the first study, using the 4 step classification of the BDI, 75% had no depression, 17.8% had mild depression, 5.9% had moderate depression, and only 1.3% had severe depression. Virtually identical results were presented in the remaining two studies that used the BDI as an indicator of depression.

Correlations were used in all our studies. In the first, using zero-order correlations among study variables (except consumption of sweet food), depressive symptoms consistently correlated statistically significantly with adverse lifestyle factors, but at the same time, they correlated positively with HDL ($p < .05$). In the anger expression study (Study II), alcohol consumption correlated positively with anger-in and anger-out. DBP and SBP had an inverse correlation with healthy food and a direct, positive correlation with alcohol consumption. In the third study, in the correlation matrix of all variables controlled for age and education, as well as in the zero-order correlations, triglycerides correlated with vital exhaustion and depression, but depression showed a significant correlation only when controlled for age and education. The strongest correlation was observed between depression and vital exhaustion scores ($r = 0.83$). In the last study, blood pressure values had no significant crude correlations with depression, hopelessness or vital exhaustion. A strong correlation between depression and vital exhaustion scores was confirmed ($r = .84$).

Path analyses were used to evaluate the relationships between facets of negative affectivity and CVD risk factors: the BDI and HDL/LDL, Anger expression and SBP/DBP, the BDI and VE in relation to TG, and the associations between the BDI, VE, and hopelessness and SBP/DBP. In all path models education and age were used as

covariates; furthermore, marital status was added as a covariate in the first and last study.

Age had strong direct associations with psychological variables such as the BDI, VE, Anger-In and health outcomes (HDL, LDL, SBP).

Education had a significant direct impact on the healthy food variable and an especially strong inverse relationship with smoking behaviour. Education also had an inverse relationship with the BDI, VE and Hopelessness. Moreover, education, through its associations with Healthy food and Alcohol, had beneficial mediated associations with cholesterol fractions and blood pressure.

Marital status seemed to act as sturdy factor against smoking and also had a strong inverse relationship with the BDI, VE and Hopelessness.

4.1 Direct associations of negative affectivity

4.1.1 Direct associations between negative affectivity and lifestyle

Except for the consumption of sweet food and coffee, depressive symptoms consistently correlated statistically significantly with adverse lifestyle factors ($p < .05$). Among the important direct associations between negative affectivity and lifestyle factors, the BDI was the most significant, being related negatively to healthy diet and positively to behaviour prone to smoking, inactivity and alcohol consumption. Vital exhaustion, as a related concept to depression, displayed almost identical associations as the BDI, except in regard to smoking behaviour. Hopelessness, another concept related to depressive symptoms, only had an inverse relationship with the healthy food variable.

Anger expression types displayed significant direct associations with alcohol consumption (Anger-In and Anger-Out) and were inversely associated with healthy diet (Anger-In).

The results are summarized in Table 2. See also Figures 2-9 (page 56-59), where the main findings of the original studies are illustrated.

Table 2. Direct associations between negative affectivity and lifestyle

	Predictor	direction of association	Lifestyle
Study I	BDI	-	Healthy food
		+	Inactivity
			Smoking
			Alcohol
Study II	Anger-In	-	Healthy food
	Anger-Out	+	Alcohol
		+	Alcohol
Study III	BDI	-	Healthy food
		+	Alcohol
			Inactivity
			Smoking
	VE	-	Healthy food
		+	Inactivity
Study IV	BDI	-	Healthy food
		+	Inactivity
			Smoking
			Alcohol
	VE	-	Healthy food
		+	Inactivity
			Alcohol
	Hopelessness	-	Healthy food

BDI – Beck depression inventory, VE – vital exhaustion

4.1.2 Direct associations between negative affectivity and BMI

Only two closely related concepts, the BDI and VE, were directly associated with BMI. In the first study, the BDI showed a significant positive relationship to BMI. However, in the third study, based on the high risk sample, vital exhaustion showed a similar association with BMI, while for depression no significant direct impact on BMI was indicated (see Table 2). In contrast, in the last study none of these variables displayed a significant direct relationship to BMI.

Table 3. Direct associations between negative affectivity and BMI

	Predictor	direction of association	Body mass index
Study I	BDI	+	BMI
Study II	Not tested		
Study III	VE	+	BMI
Study IV	None		

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index

4.1.3 Direct associations between negative affectivity and blood pressure and blood lipids

In the first study, depression showed statistically significant direct relationships with HDL and LDL, but the direct links indicated a beneficial impact on cholesterol fractions. Anger-out displayed significant inverse relationships with BP and, conversely, anger-control had a significant positive relationship with BP. The BDI and VE also showed direct associations with TG, but in the last model of this study, when both the BDI and VE were included, the relationship between depression and TG was reduced to zero. Meanwhile, the association between vital exhaustion and TG became non-significant (T-value 1.68, P=0.31, d.f.=4) but had the same standardized solution (0.13) as in the first model. VE as compared to BDI also had a stronger correlation with TG.

For hopelessness the path models indicated a relatively weak, albeit statistically significant, direct inverse association with systolic BP (Figure 9); for diastolic BP the value of the standardized coefficient (-0.05) remained statistically non-significant. For depression and vital exhaustion no significant direct impact on systolic or diastolic blood pressure was indicated.

Table 4. Direct associations between negative affectivity and blood pressure and blood lipids

	Predictor	direction of association	CVD risk factors
Study I	BDI	+	HDL
		-	LDL
Study II	Anger-Out	-	DBP
			SBP
	Anger-Con	+	DBP
			SBP
Study III	VE	+	TG
	BDI	+	TG
Study IV	Hopelessness	-	SBP

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index, HDL - high density lipoprotein, LDL – low density lipoprotein, DBP – diastolic blood pressure, SBP – systolic blood pressure, TG - triglycerides

4.2 Mediated associations of negative affectivity

The components of negative affectivity presented not only direct relationships with body mass index or CVD risk factors but also mediated associations. First, these mediations were two kinds: against BMI and against CVD risk factors. Moreover, in regard to CVD risk factors, there were mediations by lifestyle factors only, mediations by BMI only or mediations including both lifestyle factors and BMI.

4.2.1 Mediated associations between negative affectivity and BMI

Anger expression types displayed no kind of significant mediated relationship with BMI. On the other hand, particularly the BDI, but also VE, presented several significant relationships. Among these, the axis BDI/VE – Inactivity – BMI was most frequent and revealed the link through which depressive symptoms could negatively influence BMI.

Table 5. Mediated associations between negative affectivity and BMI

	Predictor	direction of association	Lifestyle factors	direction of association	Body mass index
Study I	BDI	+	Inactivity	+	BMI
			Smoking	-	BMI
Study II	None				
Study III	BDI	+	Smoking	-	BMI
			Alcohol	-	BMI
	VE	+	Alcohol	-	BMI
Study IV	BDI	+	Smoking	-	BMI
			Inactivity	+	BMI
	VE	+	Inactivity	+	BMI

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index

4.2.2 Mediated associations between negative affectivity and blood pressure and blood lipids

The results of the original studies gave further weight to the view that lifestyle factors are important variables in the relationship between negative affectivity and blood pressure or blood lipids.

Inactivity is not only related to higher BMI values, but in this study the results indicate that it is also an important factor in the relationship between depression and cholesterol fractions.

Depressive symptoms mediated by alcohol consumption resulted in a beneficial effect on HDL, but there was a negative effect on blood

pressure. Vital exhaustion, Anger-In and Anger-Out had similar relationships with blood pressure measurements.

Smoking revealed both expected and unexpected mediating effects. In the first study, the BDI was directly associated with smoking, while smoking had an inverse relationship with HDL. In the fourth study, smoking was inversely linked to DBP.

In these results some of the most interesting associations were those related to the Healthy Diet variable. The BDI, VE, Hopelessness and Anger-In had an inverse relationship with healthy food, and this variable had also an inverse relationship with blood pressure. Furthermore, in the first study, the results suggest that healthy diet is associated with beneficial cholesterol fractions.

In the third study, which investigated the associations between psychological constructs and TG, lifestyle variables mediated no kind of relationship.

Table 6. Life style mediated associations between negative affectivity and blood pressure and blood lipids

	Predictor	direction of association	Lifestyle	direction of association	CVD risk factors	
Study I	BDI	-	Healthy food	-	HDL	
					LDL	
		+	Inactivity	-	HDL	
					+	LDL
			Smoking	-	HDL	
Alcohol	+	HDL				
Study II	Anger-In	+	Alcohol	+	DBP	
					SBP	
	-	Healthy food	-	-	DBP	
					SBP	
Anger-Out	+	Alcohol	+	DBP		
				SBP		
Study III	None					
Study IV	BDI	-	Healthy food	-	SBP	
					DBP	
		+	Smoking	-	DBP	
			Alcohol	+	SBP	
	VE	+	Alcohol	+	SBP	
					DBP	
		-	Healthy food	-	-	SBP
						DBP
Hopelessness	-	Healthy food	-	-	SBP	
					DBP	

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index, HDL - high density lipoprotein, LDL – low density lipoprotein, DBP – diastolic blood pressure, SBP – systolic blood pressure, TG - triglycerides

In some cases, solely BMI was able to mediate the associations between negative affectivity and blood lipids. In the first study, it mediated the relationship between the BDI and cholesterol fractions, and in the third study it mediated the relationship between VE and TG levels.

Table 7. Body mass index mediated associations between negative affectivity and blood pressure and blood lipids

	Predictor	direction of association	Body mass index as mediator	direction of association	CVD risk factors
Study I	BDI	+	BMI	-	HDL
				+	LDL
Study II	Not tested				
Study III	VE	+	BMI	+	TG
Study IV	None				

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index, HDL - high density lipoprotein, LDL – low density lipoprotein, TG - triglycerides

In the first study, the results based on the basic path model supported the hypothesis that there might be two independent pathways in the depression-HDL/LDL relationship. In the indirect path, depressive symptoms were associated with adverse health behaviours (inactivity, smoking) and BMI, which in turn had a significant negative impact on HDL and a positive impact on LDL. In the direct path, depressive symptoms had a positive association with HDL and an inverse association with LDL. In the third study, vital exhaustion and the BDI had identical associations with the Alcohol consumption variable, BMI and TG. In addition, the BDI was similarly related to the Smoking variable. The findings of this research indicated that only the BDI and VE were predictors with significant links to blood pressure and blood lipids mediated by both lifestyle factors and the BMI.

The detrimental mediation role of Inactivity and BMI is also confirmed in the association between BDI or VE and blood pressure.

Table 8. The lifestyle and body mass index mediated associations between negative affectivity and blood pressure and blood lipids

	Predictor	direction of association	Lifestyle factors	direction of association	Body mass index	direction of association	CVD risk factors
Study I	BDI	+	Inactivity	+	BMI	-	HDL
						+	LDL
			Smoking	-	BMI	-	HDL
						+	LDL
Study II	None						
Study III	VE	+	Alcohol	-	BMI	+	TG
	BDI	+	Smoking	-	BMI	+	TG
			Alcohol	-	BMI	+	TG
Study IV	BDI	+	Inactivity	+	BMI	+	SBP
							DBP
	BDI	+	Smoking	-	BMI	+	SBP
							DBP
	VE	+	Inactivity	+	BMI	+	SBP
							DBP

BDI – Beck depression inventory, VE – vital exhaustion, BMI – body mass index, HDL - high density lipoprotein, LDL – low density lipoprotein, DBP – diastolic blood pressure, SBP – systolic blood pressure, TG – triglycerides

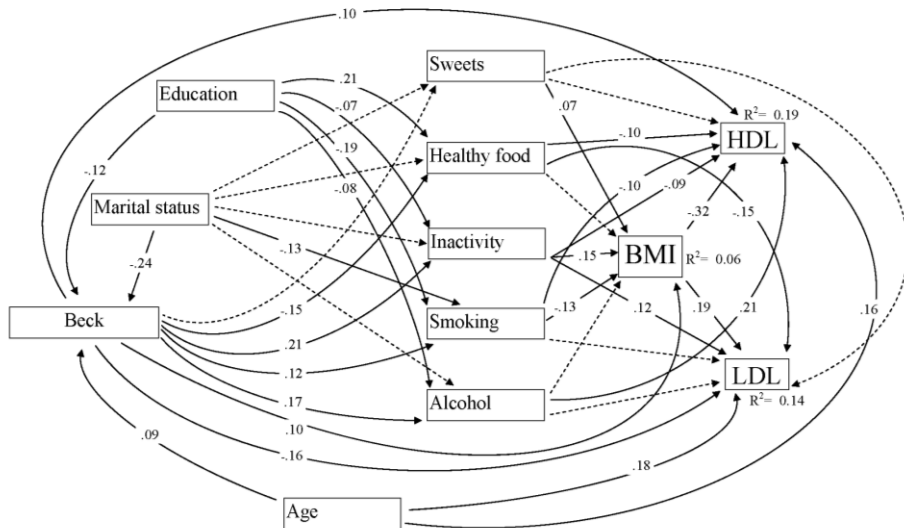


Figure 2. Study I: Basic theoretical path model with standardized regression coefficients. All tested paths are included. Nonsignificant paths are presented with dots.

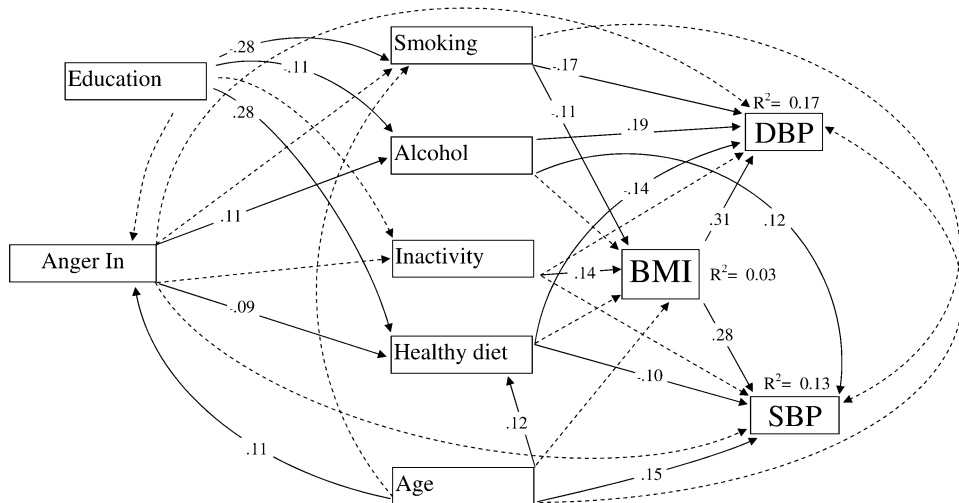


Figure 3. Study II: Standardized regression coefficients of the Anger-in path model. All tested paths are included. Nonsignificant paths are presented with dotted lines.

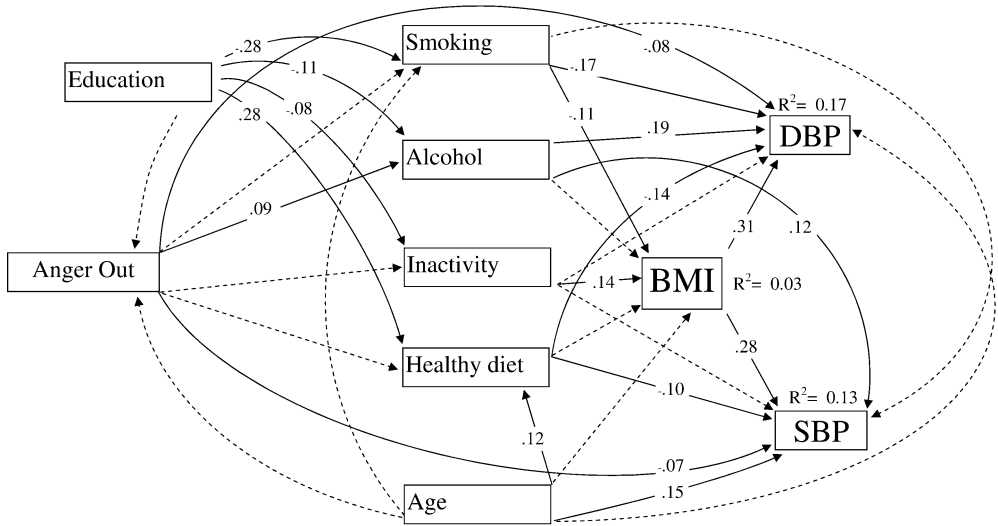


Figure 4. Study II: Standardized regression coefficients of the Anger-out path model. All tested paths are included. Nonsignificant paths are presented with dotted lines.

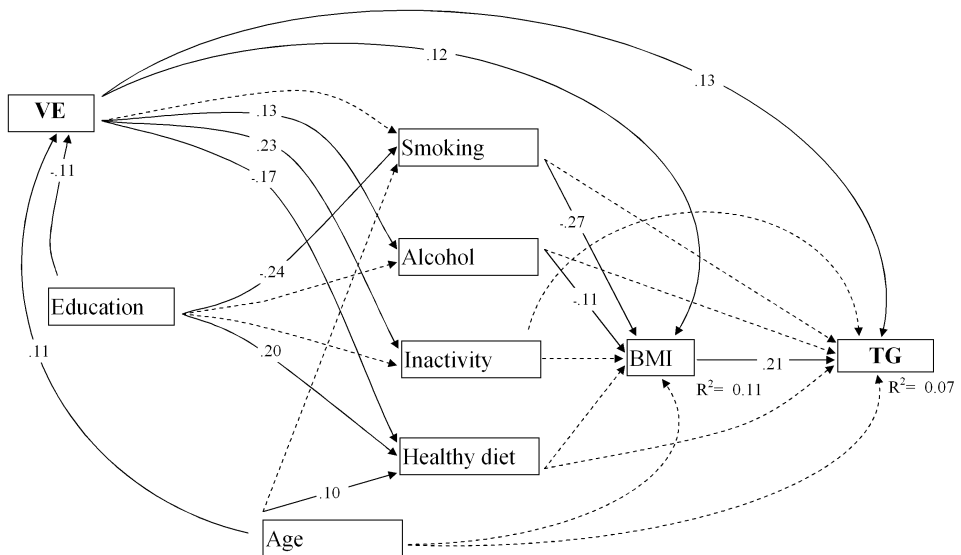


Figure 5. Study III: Significant (solid lines) and non-significant (dotted lines) relationships of the path model with vital exhaustion; only significant coefficients are presented.

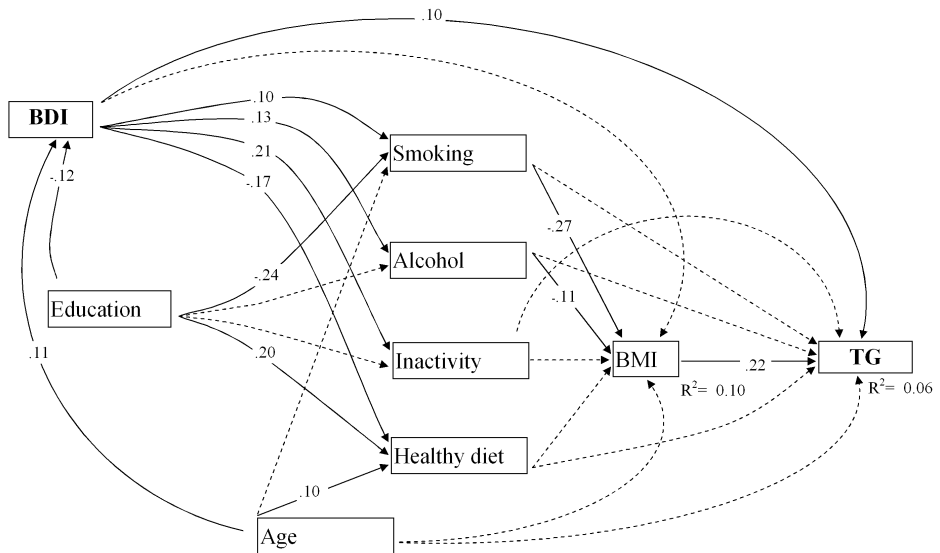


Figure 6. Study III: Significant (solid lines) and non-significant (dotted lines) relationships of the path model with depression; only significant coefficients are presented.

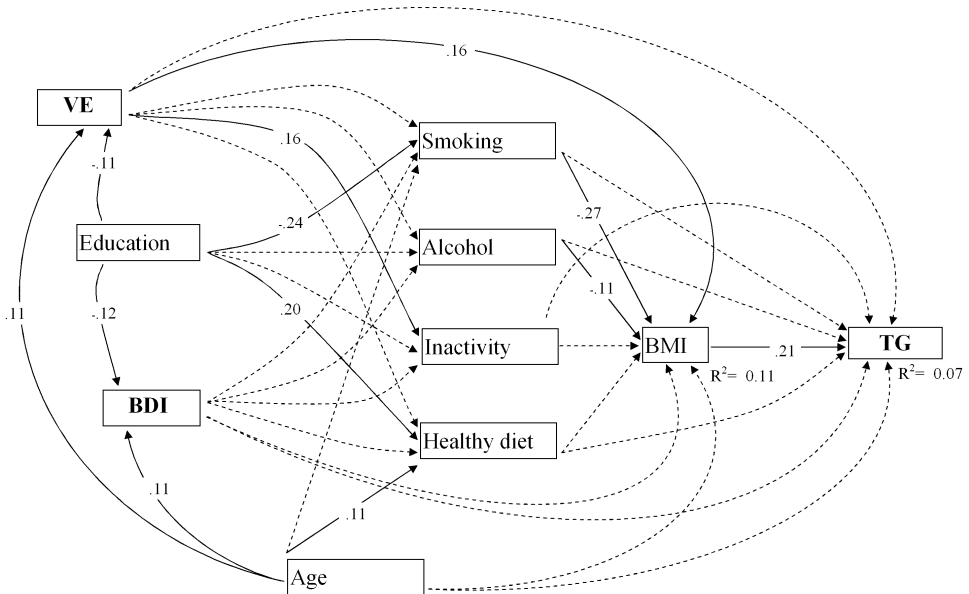


Figure 7. Study III: Significant (solid lines) and non-significant (dotted lines) relationships of the path model with vital exhaustion and depression; only significant coefficients are presented.

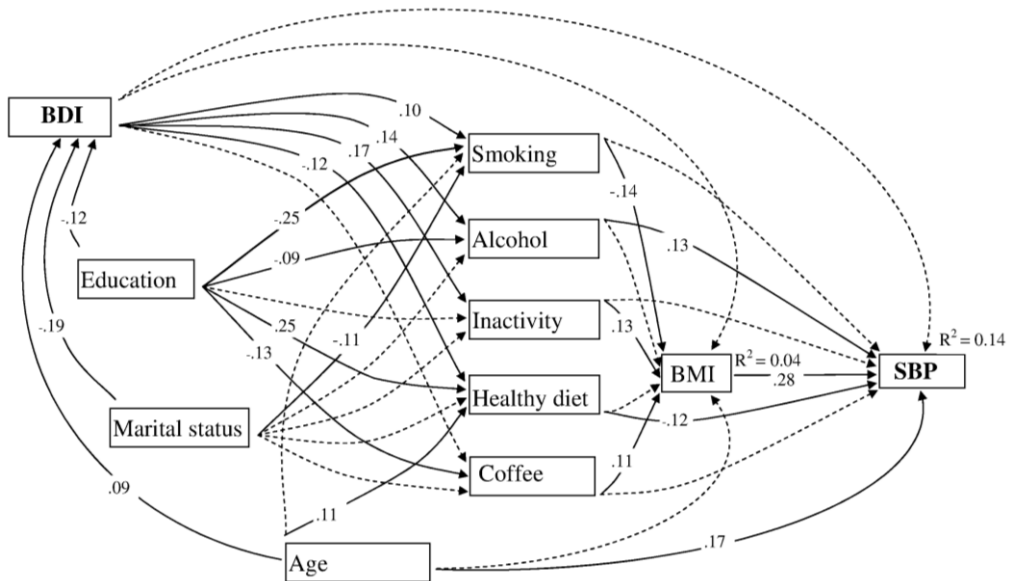


Figure 8. Study IV: Significant (solid lines) and non-significant (dotted lines) relationships of the path model BDI – systolic BP; only significant coefficients are presented.

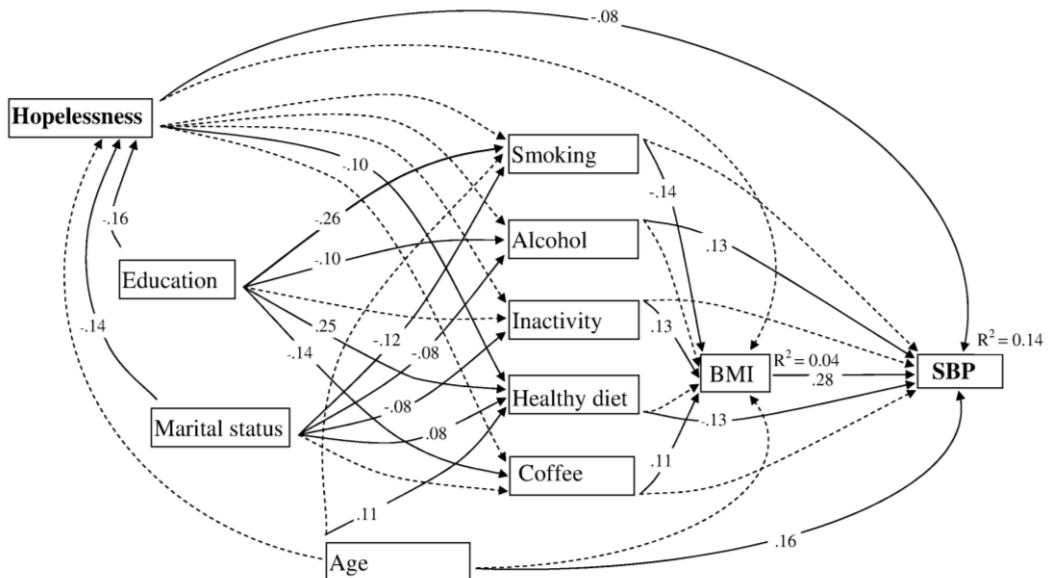


Figure 9. Study IV: Significant (solid lines) and non-significant (dotted lines) relationships of the path model Hopelessness – systolic BP; only significant coefficients are presented.

5 DISCUSSION

Despite numerous studies on facets of negative affectivity and CVD risk factors, there is still no consensus on the relationship between symptoms of depression, vital exhaustion, hopelessness or anger expression and some of the best-established risk factors of CVD, such as serum lipids or BP. In addition, failing to include other relevant variables - confounding or mediating - in study protocols has hampered the achievement of reliable results.

The present study aimed to increase knowledge of these issues by analysing and comparing different path models which included indicators of health behaviour and overweight as mediators.

5.1 Main findings related to blood lipids and blood pressure

5.1.1 Associations between negative affectivity and blood lipids

In our study, the mean value of BDI scores in the population sample was rather low, but even depressive symptoms of a subclinical range have been shown to confer an increased risk of recurrent CAD events (Kubzansky et al., 2005; McCaffery, Niaura, Todaro, Swan, & Carmelli, 2003a; Suls & Bunde, 2005).

When investigating the relationship between HDL/LDL and depressive symptoms the results of this research indicated that there might be two main pathways, a mediated pathway and a direct pathway. In the mediated pathway, depressive symptoms are clearly associated with adverse health behaviours, such as inactivity, smoking, heavy drinking, and unhealthy diet as well as with BMI. In this model, BMI is the key mediating factor, with BMI above the normal range adversely affecting HDL and raising LDL levels. One exception in the

adverse depression-lifestyle cholesterol chain is the already previously documented link between depression, through alcohol consumption, and improved HDL levels. In the direct pathway, depressive symptoms seem to have a direct, beneficial impact on both HDL and LDL cholesterol fractions.

Two previous studies have reported a positive association between depression or vital exhaustion (a close parallel variable to depression) and HDL (Gary et al., 2000; Golden et al., 2004). In both studies, the results were seen as an unexpected finding and no further discussion was provided. Similarly, to our knowledge, there are only two previous studies reporting a significant negative association between depression and LDL. In a sample of men aged 70 to 89 years, Aijänseppä et al. (Aijänseppä et al., 2002) found an independent association between low levels of LDL-cholesterol and depression and similar results Ariyo et al. (Ariyo et al., 2000) reported for a sample of women aged over 65. Both of these samples were older than the present study sample. More importantly, in a recent longitudinal study including people over the age of 65, the relationship between cholesterol fractions and depression was widely investigated. It was found that men with low LDL-cholesterol had a significantly higher risk of clinical depression, and this was particularly the case for men displaying a vulnerability caused by a serotonin transporter gene. Finally, the study suggests that LDL-cholesterol is a sensible indicator for men, with high levels related to the risk of CVD and low levels related to the risk of depression (Ancelin et al., 2010). The present results also support and extend the validity of those findings in younger cohorts of middle-aged men.

When exploring the comparative path models of depressive symptoms and vital exhaustion to TG, the results demonstrated similar relationships for both variables with lifestyle variables, with the exception of smoking. Both were related to increased alcohol consumption, lack of exercise and less healthy diet. Although BMI is usually influenced by these lifestyle factors, only a few studies have tested the mediational role of BMI in this context (Horsten et al., 1997;

Koertge et al., 2003). This is surprising, because numerous studies have shown that the BMI is related to depression (Skilton et al., 2007; Toker et al., 2007; Trief et al., 2006; Vaccarino et al., 2008), depressive symptoms (Aijänseppä et al., 2002; McCaffery, Niaura, Todaro, Swan, & Carmelli, 2003b; Skilton et al., 2007; Vaccarino et al., 2008) as well as to vital exhaustion (Prescott et al., 2003), and also the fact that, higher BMI is related to elevated TG levels (Dunbar & Rader, 2005; Knox et al., 1996; McCaffery, Niaura, Todaro, Swan, & Carmelli, 2003b).

The correlations between depression and vital exhaustion and TG indicate that in our sample the relationship between vital exhaustion and TG is stronger than the relationship between depression and TG. Moreover, in the path analyses only vital exhaustion had a direct relationship with BMI. These results support the argument that these two constructs share a significant common area but do not overlap completely, as some previous studies have already suggested (Kudielka, von Kanel, Gander, & Fischer, 2004; Lahlou-Laforet et al., 2006). Depression has some specific symptoms (e.g. guilt and loss of self-esteem) that differentiate it from vital exhaustion. Therefore, as vital exhaustion lacks the cognitive aspects specific to depression, one could speculate that the vital exhaustion scale is more focused on aspects of physiology and mood (fatigue, sleep disturbances, and irritability), and in this way it could be more closely related to BMI and TG levels.

In the path analysis that included both vital exhaustion and depression, only vital exhaustion retained a significant relationship with inactivity, while depression had no significant relationship with lifestyle variables. Furthermore, the depression–TG relationship was reduced to zero, while the relationship between vital exhaustion and TG remained close to significant suggesting a stronger relationship between vital exhaustion and TG. In sum, it seems that including both vital exhaustion and depression in the same model does not improve the

model and may actually disguise the specific associations between these constructs and health behaviour.

A possible biological mechanism relating vital exhaustion to diet, BMI and TG is proposed in a study by Koertge et al. (Koertge et al., 2003). They suggest that long-term stress and unhealthy diet can, through several links, generate hyper-insulinemia, and this may lead to a higher level of TG. In another study, the results suggested that the simultaneous incidence of different mental problems (post-traumatic stress disorder together with depression) was related to higher TG levels compared to those who had only one diagnosis or none (Trief et al., 2006). It was also found that depressive people (with or without another diagnosis) also had a higher BMI than healthy people.

From these results it is difficult to discern how much of the impact of mental problems on TG levels acted through direct physiological mechanisms and how much was mediated through lifestyle factors that modified the BMI. However, our results from separate models for depression and vital exhaustion as predictors of lifestyle and TG indicate that both links are plausible.

When comparing the path models of depressive symptoms and vital exhaustion to TG, both a direct and a mediated relationship between vital exhaustion and depression and TG was found. In short, vital exhaustion seemed to be a stronger predictor of TG. Taken together these results offer support for the importance of vital exhaustion as a health-related psychological risk factor and clarify some of the possible mediating mechanisms in the previously established VE–CAD relationship.

The significant relationship between vital exhaustion, BMI and TG supports the argument for the importance of the BMI to triglycerides. The fact that the BMI and vital exhaustion can be managed through specific counselling and health programmes gives hope for an alternative approach towards reducing levels of TG and the incidence of cardiovascular disease.

5.1.2 Associations between negative affectivity and blood pressure

In this study both beneficial and harmful associations were identified. Some of the findings supported previous research, while some clarified earlier findings or raised new question in the field of health psychology.

By studying the relationship between different forms of anger expression and lifestyle factors, BMI and BP, the study tested whether anger expression variables have mediated or direct relationships with BP. The results suggest that each type of anger expression has a different pattern of relationships with lifestyle factors and BP. Anger-control had no significant relationship with lifestyle factors; however, it had a significant positive direct relationship with BP. In contrast, anger-out was statistically significantly related to alcohol consumption, which was associated with elevated DBP. At the same time, however, anger-out also had a significant direct inverse relationship with BP. Anger-in was statistically significantly related to alcohol consumption and unhealthy diet. There were no significant direct relationships with BP. However, there seems to be a clear pathway from anger-in to BP through alcohol consumption and diet.

The lack of a significant relationship between anger-in and BP seems to contradict some previous studies, but there are several methodological factors that hamper comparison. For example, many previous studies (Gentry, Chesney, Gary, Hall, & Harburg, 1982; Harburg et al., 1973; Harburg, Blakelock, & Roeper, 1979) did not use the Spielberger scale to evaluate anger-in. There are also considerable differences in the samples used in terms of age (Schneider, Egan, Johnson, Drobny, & Julius, 1986; Somova, Connolly, & Diara, 1995), gender (Helmers et al., 2000) and ethnicity (Harburg et al., 1973; Somova et al., 1995). Among studies that have used the Spielberger scale, only one (Everson, Goldberg, Kaplan, Julkunen, & Salonen, 1998) has reported a relationship between anger-in and BP, while other

studies (Mueller et al., 2001; Steele & McGarvey, 1997) have failed to reveal any significant relationship.

When considering the findings from the path analyses used in the present research, the contradictory results reported in earlier literature for anger expression, DBP and SBP could be partly explained as an effect of complex interactions and the influence of mediating factors and covariates. Most importantly, however, the present results indicate that the various ways of expressing feelings of anger may have distinctly different implications for BP levels.

Anger-in and anger-out had a statistically significant positive relationship with alcohol consumption, which was related to higher levels of BP. This suggests that both anger-out and anger-in might confer an elevated risk of hypertension through alcohol consumption, despite their different direct relationships with BP. Another interesting finding is the negative relationship between anger-in and healthy diet, and through the diet variable the unfavourable effect of anger-in on BP. Previous studies have shown that negative emotions associate with unhealthy diet and other lifestyle factors (Kelloniemi et al., 2005), and on the basis of the current study's findings, one could speculate that anger-in and adverse lifestyle might have a common, personality-related background. Moreover, depression could be considered a factor leading to suppressed anger and an unhealthy lifestyle.

Exploring these relationships, however, goes beyond the scope of the present study, and further research is needed to clarify these issues.

Symptoms of depression, vital exhaustion and hopelessness are three depression-related constructs, but it is unclear if these concepts have identical relationships with health behaviour and blood pressure. The results showed that two, depressive symptoms and vital exhaustion, had very similar significant associations with unfavourable lifestyle variables. Both were related to increased alcohol consumption, lack of exercise and a less healthy diet. The only exception seemed to be smoking, which associated with depression, but not with exhaustion. At the same time, we found no direct association between depressive

symptoms or vital exhaustion and BP. Testing of the indirect mediation effects showed that alcohol consumption, diet, and inactivity could be possible mediators in the relationship between depression or exhaustion and blood pressure. These results suggest that the previously reported positive association between depression and exhaustion and BP could be explained through the mediating factor of adverse lifestyle. One previous study (Reiff et al., 2001) also found that lifestyle factors, but not depressive symptoms, were associated with BP. Furthermore, their findings seemed to exclude the possibility that lifestyle factors could act as mediators in the depression-hypertension relationship. Together with the present findings this suggests that relationships between these variables may be more complex than was previously thought. Clearly, the possible mediating role of health behaviour in the depression-BP relationship requires more detailed investigation, with a particular need for studies with a longitudinal design (Spencer, Zanna, & Fong, 2005).

In conclusion, for our sample of middle-aged men, the results indicate that vital exhaustion and depressive symptoms associate with several unfavourable lifestyles, but have no direct relationship with BP values. Together with the observed indirect effects, these results suggest that previously reported relationships between depression and vital exhaustion and BP could have been mediated by unfavourable lifestyles.

In contrast to the results for depressive symptoms and vital exhaustion, hopelessness displayed a rather weak association with health behaviour. Instead, a significant direct, but inverse, relationship between hopelessness and SBP was found. However, the negative relationship between hopelessness and DBP was non-significant. The inverse relationship between hopelessness and SBP, corroborated by the absence of any significant relationships between the BDI or VE and BP values, supports the argument that depression-related constructs might work differently in the context of health. This also supports the argument that hopelessness is to some extent a different concept from depression (Everson et al., 1996).

In their prospective study, Everson et al. (Everson et al., 2000) used the same two hopelessness questions used in the present study, and concluded that, over a period of four years, initially normotensive men with high levels of baseline hopelessness had a three-fold risk of becoming hypertensive. Despite their use of the same hopelessness measure and some common covariates in a middle-aged male sample, it is difficult to compare the contradictory results of these two studies. For example, the present sample also included hypertensive men, and hopelessness was at a lower level. In Everson's study the mean value of hopelessness was 2.4, and those with a high level of hopelessness represented 8.44% of their study sample, while in our study the mean value of hopelessness was 1.96, and those with a high level of hopelessness represented only 4.1% of the sample. In line with this argumentation, the results from another study (Valtonen et al., 2008) using the same hopelessness items and classes as Everson et al. found that high systolic BP was associated with high levels of hopelessness and there was a significant mean difference in systolic BP between the three hopelessness groups. Again, those with a high level of hopelessness represented a large percentage (34.5%) of their study sample.

Furthermore, even Everson et al. found that those reporting a moderate level of hopelessness were not at a significantly increased risk of hypertension. Possibly for the same reason, another study (Stern et al., 2009) could not find any association between hopelessness and hypertension. One possible explanation for these results could be that there is a threshold effect in the association between hopelessness and BP, where a positive association can only be found among the most hopeless subjects. This question remains to be answered in future studies.

The possible mechanisms through which hopelessness can cause hypertension include endothelial dysfunction (Do et al., 2010) or atherosclerosis (Everson et al., 1997; Pollitt et al., 2005). This issue also awaits further studies.

In sum, hopelessness is not so closely related to adverse health behaviours as depression or vital exhaustion, and in one study it had an inverse association with SBP. The results of this study do not support the importance of hopelessness as a psychological risk factor for elevated BP.

5.1.3 Effect of lifestyle and BMI on blood pressure and blood lipids

The results of the present research supported previous findings showing that lifestyle factors and BMI are important variables affecting BP or the level of blood lipids.

Inactivity, healthy diet and BMI had statistically significant links with cholesterol fractions, healthy diet being the only beneficial variable. Alcohol had a positive relationship and smoking an inverse association with HDL. In our basic model, inactivity is part of a strong axis that leads from depression to BMI and then to cholesterol fractions. In this model also, inactivity is the only lifestyle variable that has significant direct links to BMI and both cholesterol fractions. Together these findings confirm the results of other studies that indicate the importance of exercise as a relevant variable in the relationship between depression (Pilu et al., 2007; Smith et al., 2007), BMI (Atlantis et al., 2006; Cooper, Page, Fox, & Misson, 2000) and serum lipids (Halverstadt et al., 2007; Stefanick et al., 1998).

In the path analyses of BDI/VE and TG, none of the lifestyle variables, and only BMI, had a significant relation to TG. Furthermore, BMI associated negatively with alcohol consumption and smoking behaviour. However these inverse relationships between lifestyle factors and BMI are not a new finding. As shown in previous research, smoking can reduce appetite and in this way may lead to a reduced BMI (Chiolero et al., 2008), and alcohol can also have the same effect (Suter, 2005).

Furthermore, one finding that we did not expect was the inverse relationship between smoking and DBP, because it is well known that smoking produces a chronic increase in BP for active smokers (Halimi et al., 2002; Jo et al., 2001; Narkiewicz et al., 2005; Niskanen et al., 2004). Nevertheless, previous literature includes several contradictory findings for the relationship between smoking and BP. Many studies (Halimi et al., 2002; Jo et al., 2001; Narkiewicz et al., 2005; Niskanen et al., 2004) have found that smoking increases BP, but some other studies have found no relationship between smoking and BP (Green & Harari, 1995; Okubo et al., 2004). Some previous studies even suggest that smoking is related to a decrease in BP (Okubo et al., 2004; Webber et al., 1982) or that giving up smoking is related to higher BP (D. H. Lee et al., 2001). One explanation for these contradictory findings could be related to the complex interactions between smoking behaviour and lifestyle and health. BP is influenced by BMI (Brock et al., 2005; Jo et al., 2001; Mueller et al., 2001; Niskanen et al., 2004; Spicer & Chamberlain, 1996), and, through higher energy expenditure and lower appetite, smoking can lead to a lower BMI (Chiolero et al., 2008). In our sample, smoking had a statistically significant inverse relationship with BMI and also with DBP. The direct inverse relationship between smoking and BP could also be explained through some physiological mechanisms yet to be fully understood. One study (Abe et al., 2002) found evidence that genotypic variations influence the relationship between smoking status and the probability of developing hypertension. Another study (Takashima et al., 2002) has suggested that smoking behaviour is related to higher BP only if smoking induces degenerative changes or the remodelling of small arteries.

Although the psychological and lifestyle variables used in our studies had significant relationships with BMI, the level of explained variance of BMI was only between 3-11%. On the other hand, the strong associations between BMI and BP and blood lipids can be a

possible argument for considering BMI an important independent variable in relation to CVD risk factors.

5.2 Methodological considerations

The main limitations of this study are related to the cross-sectional nature of the survey and reliance on self-reports of depressive symptoms and lifestyle. Furthermore, the present study only included middle-aged men. Future studies should include women and a larger variation of age and risk factor levels as well as prospective designs.

Despite the numerous statistically significant paths in our models, the amount of explained variance for BP, cholesterol fractions, TG and BMI remained rather low. 14-17% of BP variance was explained, indicating that some important determinants of BP could not be assessed in this study. One could also argue that only using a single BP measurement weakens the reliability of our dependent variable. However, because most of our analyses are based on correlations and considering the findings presented by Boveta (Boveta et al., 2003) a double or triple measurement of BP would probably have only marginally changed the results of this study. It seems probable that our results are actually underestimates of the “true” associations. However, for cholesterol fractions, the explained variance was close to the percentages found for BP (HDL 19%, LDL 14%). Nevertheless, the values for TG (6-7%) and BMI (3-11%) remained low in our models. Perhaps adding new variables, such as unhealthy diet, would mean a larger proportion of the variance in BMI and blood lipids could be explained. Items considered as high cholesterol food were also assessed, but due to contradictory answers to the questions, a reliable variable could not be calculated. Moreover, in this study assessment of nutrition was based on the frequency of consuming certain food products. Due to the study design it was not possible to control for the

overall amount of food consumed, and therefore our measure should only be considered a tentative estimate of healthy nutrition.

Considering that education presented significant associations with psychological variables and lifestyle, it would be interesting to include more socioeconomic status variables in future studies.

Another specific limitation refers to our data collection. The lifestyle variables, which naturally occur as continuous variables, were made discontinuous in the data collection process.

Among the fit indices used in our studies, the PGFI had a relatively small value, although there is no exact cut-off point for this fit index. As with the high GFI value, the small value for PGFI is probably due to the complexity of the path models (Sivo, Fan, Witta, & Willse, 2006). It seems likely that this complexity emerged from the numerous relationships that were indicated by the hypothesized model, which affected the parsimony of the models because not all the theoretical relationships were confirmed by path analyses. All other indices, however, indicated a good fit for the tested models with the data.

In this study symptoms of depression were assessed with the BDI, because this scale has also been the most widely used method in studies of CVD risk factors offering validated reference values, and it has been shown in numerous studies to be a reliable and valid instrument for assessing symptoms of depression. Furthermore, the prevalence of depression observed in this study is comparable to larger population studies conducted with standard interview methods (Pirkola et al., 2005). However, the use of this scale contains a potential limitation, since it was originally developed for psychiatric patients. In future studies the use of other scales developed for medical patients should be considered.

However, it is likely that the combined effect of these possible sources of error variance in our study variables have was only to reduce the conclusiveness of the study and the strength of observed associations. Finally, because this study was based on a cross-sectional database and utilised statistical techniques from which no directionality

can be suggested, one can only speculate on the causal role of any variable (Reynolds, 1999).

5.3 General conclusions and clinical implications

Beyond these methodological considerations, there are several strengths in our research. First, it was based on a fairly large sample of individuals, and this allowed for better detection and testing of associations. Second, it compared the importance of different facets of depression (depressive symptoms, vital exhaustion and hopelessness) and anger expression as related to BP, cholesterol fractions and TG. Third, to our knowledge, these studies were the first to investigate the role of different health behaviours as mediators in the relationship between facets of negative affectivity and CVD risk factors while controlling for confounding variables such as age, education, and marital status.

Perhaps the most important theoretical finding in our studies was the usefulness of the basic hypothetical model. In all studies the model enabled the exploration of both direct and mediated links from key psychological variables of negative affectivity to health outcomes. This revealed the complex interaction between psychological, sociodemographic and lifestyle variables and health status. The results also offered possible explanations for relatively isolated or contradictory findings from this area of research.

For serum lipids, the negative effect of depression on the HDL-LDL balance was partly mediated through adverse health behaviours. At the same time, the results indicated a direct, probably physiological, link between depressive symptoms and cholesterol, but this time the effect on the HDL-LDL balance was beneficial. Depressive symptoms and vital exhaustion also had both a direct and a mediated association with TG concentrations.

Concerning BP measurements, the results indicate that vital exhaustion and depressive symptoms associate with several unfavourable lifestyles but have no direct relationship with BP values. Together with the observed indirect effects, these results suggest that the previously reported relationships between depression and vital exhaustion and BP could have been mediated by unfavourable lifestyles. On the other hand, anger expression styles are differently linked to BP. The association of anger-in with elevated BP seems to be mediated by adverse life styles. For anger-control we found statistically significant direct pathways to elevated BP but no significant links with lifestyles. Open expression of anger seemed to associate with low BP.

The assessment of several depression-related concepts enabled the evaluation of their specific associations with biomedical variables. Depressive symptoms, measured with BDI, revealed a different pattern of correlations with biomedical variables from that of vital exhaustion or hopelessness. The results suggest that although vital exhaustion is not so commonly assessed as depression, the direct link to BMI and the stronger relationship with TG strengthen the evidence for the role of vital exhaustion as a potentially important risk factor for metabolic syndrome and CAD. Hopelessness, on the other hand, was not so closely related to adverse health behaviours as depression or vital exhaustion, and it had an inverse association with SBP.

In this research the effect of main predictors was influenced by the interplay of background, lifestyle and BMI variables. Furthermore, some of these variables presented independent associations that allowed for a better understanding of the mechanisms involved in health maintenance. Confirming previous results, the correlations and results from path analyses between the BDI, VE and Hopelessness scores, marital status, education, and lifestyle variables show that a high level of education and the presence of a permanent relationship act as protective factors against depressive feelings or detrimental lifestyle behaviours. For example, the existence of family relationships relates to non-smoking behaviour. Education had the same influence, and it was

also linked to all the other lifestyle variables considered, except for the consumption of sugary food.

Considering that the BMI and vital exhaustion can be managed through specific counselling and health programmes there is hope for an alternative approach to lowering TG levels and reducing the incidence of cardiovascular diseases.

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Appendix

Example of items for subscales of Spielberger scale:

Anger-in

I keep things in

I pout or sulk

Anger-out

I express my anger

I make sarcastic remarks of others

Anger-control

I control my temper

I am patient with others

Each subscale has 8 items:

Anger-in subscale measures the frequency of hold in or suppressed anger by the respondent.

Anger-out subscale measures the frequency of expressed anger against other people or objects by the respondent.

Anger-control subscale measures the respondent attempts to control own expression of anger.