Effort–Reward Imbalance, Overcommitment, Perceived Control and Health Behaviours in Central and Eastern Europe

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Declaration

I, Sung–Wei Chen, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Abstract

Aims Health behaviours – alcohol drinking, smoking, poor diet and physical inactivity – are influenced by various psychosocial factors. Despite evidence linking work stress and personality constructs independently to health behaviours, only limited literature is available on the relationship between work stress, personality and health behaviours. The aims of the thesis are: (1) to examine the potential role of overcommitment (OC) personality in the relationship between work stress defined by the Effort–Reward Imbalance (ERI) model and health behaviours; (2) to investigate the potential role of perceived control (PC) in the relationship between ERI, OC and health behaviours.

Methods This project used data from the HAPIEE (Health, Alcohol and Psychosocial factors In Eastern Europe) study, which randomly selected people aged 45 to 69 years from population registers in Russia, Poland and the Czech Republic. A two–wave cohort study for drinking and smoking outcomes (n= 7,513) and a cross–sectional study for dietary outcomes (n= 11,012) were analysed by logistic regression and structural equation modelling.

Results In terms of the potential role of OC in the relationship between ERI and health behaviours, OC and ERI may have bi–directional relationship; the effect of OC on ERI was stronger than the other direction in the middle–aged and older populations. Thus, antecedent role of OC in the relation between ERI and health behaviours was statistically significant, but mediator role of OC was not. With regards to the potential role of PC in the relationship between OC, ERI and health behaviours, both ERI and PC partially mediated the effects of OC on health behaviours; ERI and PC may have bi–directional relationship.

Conclusion This thesis will contribute to deeper understanding of intersecting pathways by which work stress (ERI) and personality constructs (OC and PC) jointly influence health behaviours, thereby providing insight into research, practice and policy.

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List of abbreviations

CEE	Central and Eastern Europe
CHD	Coronary heart disease
CI	Confidence interval
DC model	Demand–Control model
ERI model	Effort–Reward Imbalance model
ER ratio	Effort–reward ratio
HAPIEE study	Health, Alcohol and Psychosocial factors In Eastern Europe study
HDI	Healthy Diet Indicator
LOC	Locus of control
OC	Overcommitment
OR	Odds ratio
PC	Perceived control
PBC	Perceived behavioural control
SCM	Social Cognitive Model
SEM	Structural Equation Modelling
TPB	Theory of Planned Behaviour
WHO	World Health Organisation

Chapter 1. Introduction

Health behaviours, such as alcohol drinking, smoking, poor diet and physical inactivity, have been found to increase the risks of chronic diseases – major causes of morbidity and mortality across the world.[1] Health behaviours are influenced by a wide range of psychosocial factors – measurements that link psychological phenomena to social environment and physiology, such as chronic stress, personality constructs, psychological distress, and protective aspects of social environment.[2,3] Despite empirical support linking each psychosocial factor independently to health behaviours, few studies have attempted to examine potential relationships between different psychosocial factors in relation to health behaviours. In particular, the relationships between psychosocial factors – combined influences of work stress and personality on human behaviours have attracted researchers' interest and debate. A profound understanding of these relationships is crucial to promote accumulation of knowledge and to inform effective interventions on health behaviours.

Stressors are demands made by internal or external environment that upset balance in an individual, thereby affecting physical and psychological well-being and requiring one's action to restore balance.[4] Work stress, as defined by the Demand–Control (DC) model and the Effort–Reward Imbalance (ERI) model, has been repeatedly reported to predict worse profiles of health behaviours and health outcomes in empirical studies.[5,6] The DC model suggests that job task profiles defined by low control and high demand (job strain) may elicit sustained stress reactions. The ERI model proposes that violation of social reciprocity in terms of high effort and low reward at work may elicit negative emotions and sustained stress responses.[7]

Personality represents a dynamic organisation, inside the person, of psychophysical systems that create a person's characteristic patterns of behaviours, thoughts and feelings.[8] Various personality constructs have been repeatedly reported to predict health behaviours in several empirical studies.[9,10] Overcommitment (OC)

personality was proposed in the ERI model; OC displays attitude, behaviour and emotion characterized by excessive striving at work and strong motivation for approval. OC was originated from the concept of Type A behaviour, which was derived from perceived control (PC). Rosenman proposed that Type A persons have higher need for control over environment and tend to perceive lower PC; their response is enhanced coping to assert and maintain control over environment.[11] Although the effects of OC on health behaviours have rarely been examined, many studies have supported the effects of OC–related personality (Type A behaviour, Neuroticism, and Hostility) on health behaviours.[12,13,14]

Despite empirical evidence linking work stress and OC–related personality independently to health behaviours, little literature is available on the relationship between work stress, OC personality and health behaviours. The potential role of OC in ERI–outcome relationship was originally suggested as main effect or modifying effect.[15] However, this original assumption on OC remains inconclusive in existing literature and appears relatively simple compared to accumulated research on diverse roles of personality in stress processes (modifying, antecedent, mediator, or direct effects).[16] To evaluate the potential role of OC in ERI–outcome relationship more rigorously, these four possible roles should be examined simultaneously.

It is plausible to suggest a potential role of perceived control (PC) in the relationship between OC, ERI and health behaviours; PC might mediate the effects of OC on health behaviours. Rosenman proposed that Type A persons have higher need for control (OC) over environment and tend to perceive lower PC.[11] Greenberger and Strasser suggested that the higher need for control a person has, the lower PC one perceives.[17] In fact, it is suggested that social–cognitive constructs (e.g. PC) provide a more active and specific process account of individual differences that complements the broader and more static personality traits (e.g. OC).[18]

Based on gaps identified in existing research, the aims of the thesis are: (1) To examine the potential role of OC personality in the relationship between work stress

defined by the ERI model and health behaviours, including modifying, antecedent, mediator, and direct effect of OC. (2) To investigate the potential role of PC in the relationship between ERI, OC and health behaviours. A two–wave cohort study for drinking and smoking outcomes (n= 7513) and a cross–sectional study for dietary outcomes (n= 11012) are conducted in the middle–aged and older populations in Central and Eastern Europe. It is hoped that this thesis would contribute to growing understanding of the combined influences of work stress (ERI) and personality constructs (OC and PC) on health behaviours.

The thesis is structured as follows. The second chapter provides a general literature review on psychosocial factors and health behaviours, particularly focusing on the associations between work stress and health behaviours, the associations between OC–related personality and health behaviours, and the associations between PC and health behaviours. Furthermore, the chapter discusses the potential role of OC in the relationship between ERI and health behaviours, and the potential role of PC in the relationship between OC, ERI and health behaviours.

The third chapter outlines the aims, objectives and hypotheses of the thesis. The fourth chapter gives detailed explanation for the methods of the thesis, including description of the HAPIEE (Health, Alcohol and Psychosocial factors In Eastern Europe) study, study samples, description of variables used in this project, statistical power, and statistical analysis. Specific details in the methodology for Structural Equation Modelling (SEM) are also provided.

Chapters five to seven describe the analytical methods and the results for drinking outcomes, smoking outcomes and dietary outcomes, respectively. In general, each result chapter is divided into three parts. First, descriptive statistics for study populations and outcomes are shown. Second, the potential role of OC in ERI–outcome relationship is analyzed, including direct, antecedent, mediator and modifying effects of OC. Third, the potential role of PC in the relationship between OC, ERI and outcomes is examined. Finally, main findings relevant to the hypotheses are

summarized.

Chapter eight focuses on general discussion of the thesis findings. The main results for three health–behaviour outcomes are summarized. The methodological issues are also addressed. Next, the results are discussed according to the following topics: (1) work stress and health behaviours; (2) OC personality and health behaviours; (3) the potential role of OC in the relationship between ERI and health behaviours; (4) PC and health behaviours; (5) the potential role of PC in the relationship between OC, ERI and health behaviours.

Chapter nine provides an overall discussion regarding the implications for research, practice and policy based on the findings obtained from the thesis. Finally, Chapter ten summarizes the general conclusions of the whole thesis.

Chapter 2. Literature Review

2.1 Health Behaviours

2.1.1 Health behaviours – definition and their impact on health

It has been increasingly recognized that individuals can contribute to their health by adopting or avoiding particular behaviours. Since the 1970s, health behaviours have become a paramount issue in epidemiology through a series of empirical studies. For instance, the impacts of health behaviours on mortality and morbidity were investigated in Alameda County in the United State (1979).[19] Health behaviours were added to traditional physiological and environmental factors to predict health outcomes in the British Whitehall II study (1991).[20] In contemporary research, health behaviours – including alcohol drinking, smoking, poor diet and physical inactivity – are considered the main causes of morbidity and mortality (chronic disease such as heart disease, stroke, or cancer) across the world.[1]

While health behaviours are undoubtedly important, their definitions are diverse. Conner and Norman defined health behaviours as: "*any activity undertaken for the purpose of preventing or detecting disease or for improving health and well-being*".[21] By this definition, there are a wide range of health behaviours including medical service usage, compliance with treatment, and self-directed health behaviours (e.g. smoking). The definitions have evolved with mounting evidence for the impacts of psychosocial factors on health behaviours. Emphasizing psychological factors, Gochman defined health behaviours as: "*personal attributes like beliefs, expectations, values, and other cognitive elements; personality characteristics, including emotional and affective states and traits; and overt behaviour patterns, actions, and habits that relate to health maintenance, health restoration, and health improvement*".[22] Emphasizing social factors, Cockerham proposed the collective patterns of health behaviours – health lifestyles, which are based on individuals' life choices from available options according

to their life chances (social structures, collective patterns of living related to societies, institutions, or social classes that constrain or enable individuals to act).[23]

Despite diversity in the definitions, a common way of classifying health behaviours in epidemiology is to distinguish between risky behaviours and protective behaviours.[24] Risky behaviours have harmful effects on health or predispose individuals to diseases; protective behaviours enhance health or protect individuals from diseases. It depends on existing evidence to define whether a specific behaviour is a risky or protective behaviour; empirical studies have shown that risky behaviours such as alcohol drinking, smoking, poor diet and physical inactivity can increase the risks of chronic diseases across the world, including Central and Eastern Europe (CEE).[25,26] Because of their impacts on morbidity and mortality, the term health behaviours adopted in this thesis represent four risky health behaviours: alcohol drinking, smoking, poor diet and physical inactivity. The four health behaviours are discussed in Chapter 2, but physical inactivity will be excluded from my analyses due to the limitations of available data.

The four risky health behaviours and their impacts on health are summarized in the next part of this section. Although not all researchers agree on the J–shape relationship between higher alcohol consumption and poorer health outcomes, moderate alcohol consumption is generally associated with decreased mortality due to cardio-protective effects, but high alcohol consumption is associated with increased mortality and morbidity.[27,28] For acute effects, drinking progressively impairs cognition, attention, judgement and coordination, resulting in increased risks of accident, injury, violence, and suicide. For chronic effects, high alcohol consumption adversely affects nearly every organ of the body, resulting in coronary heart disease, cardiomyopathy, liver cirrhosis, gastritis, pancreatitis, infection, neurological disorders, psychiatric disorders, and cancers of upper digestive tract, liver and breast.[29,30]

The toxic components in tobacco smoking include hydrogen cyanide, carbon monoxide, and nitrogen oxide, which cause damage of cells and tissues in a variety

of organs. Smoking is found to increase the risks of coronary heart disease, myocardial infarction, stroke, chronic obstructive pulmonary disease, and cancers of lung, mouth, pharynx, larynx and oesophagus.[31,32] For example, after long duration of smoking (20 to 50 years), mortality rate of middle–aged current smokers was found to increase to 3 times higher than that of non–smokers.[33] Lung cancer death rates are 10–12 times higher in current smokers than in non–smokers across the world.[34]

Dietary intakes are reported to predict a variety of chronic diseases; adherence to a healthy diet is generally associated with reduced mortality and morbidity.[35,36] For example, high intakes of fruit and vegetable (major sources of vitamins and minerals) may reduce the risks of coronary heart disease, stroke, hypertension, diabetes, obesity and cancer.[37] In contrast, high intakes of saturated fat and cholesterol are associated with high levels of blood lipids (low–density lipoprotein fraction of cholesterol and triglycerides), which increase the risks of coronary heart disease and atherosclerosis. High intakes of free sugars are also associated with increased risk of coronary heart disease.[38]

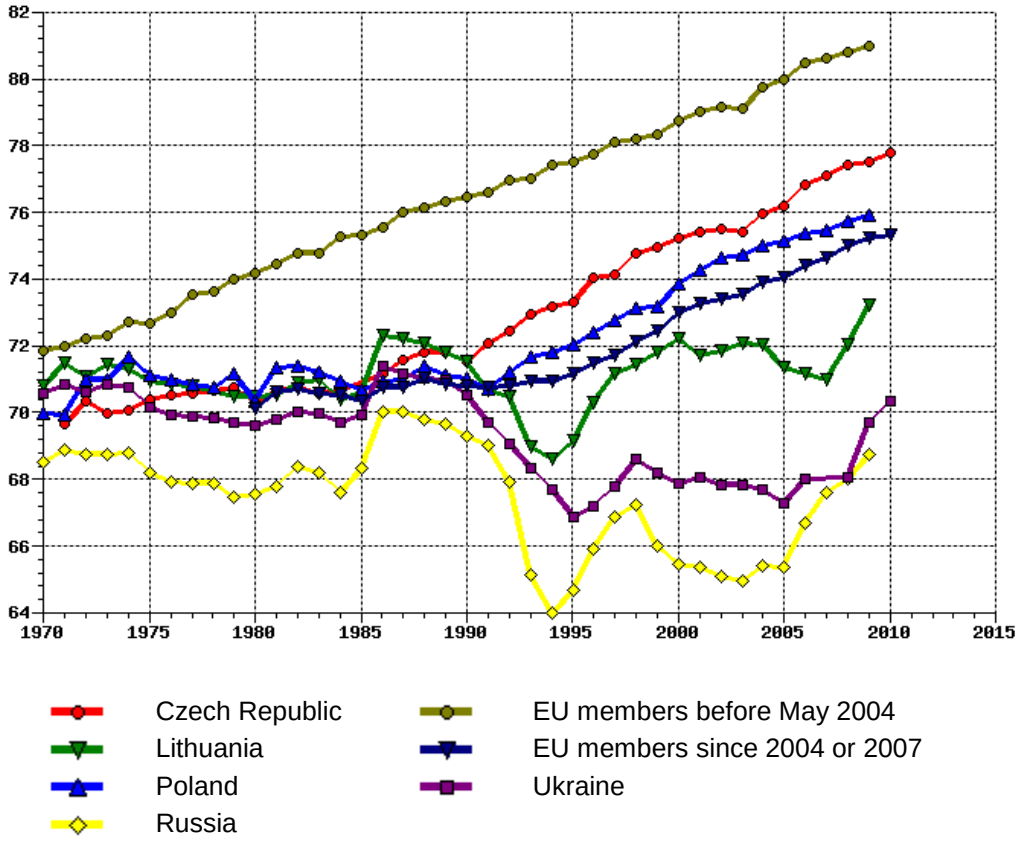
Engaging in regular physical activity can increase metabolism of fats and carbohydrates, increase artery diameter and coronary blood flow, and lower blood pressure, thereby resulting in reduced cardiovascular morbidity and mortality.[39] For adults and elders, at least 150 minutes of moderate–intensity aerobic physical activity weekly or at least 75 minutes of vigorous–intensity aerobic physical activity weekly is recommended by WHO.[40] In contrast, physical inactivity poses physiological influence on cardiovascular system, leading to increased risks for coronary heart disease, myocardial infarction, hypertension, stroke, obesity, hyperlipidaemia, diabetes mellitus, and sudden death.[41,42]

2.1.2 Health behaviours and health in Central and Eastern Europe

(1) Mortality gap between Eastern Europe and Western Europe

Since the 1970s, life expectancy continued to rise in Western Europe while it began to fall in CEE. After 1989, there was more divergence in mortality within CEE countries; life expectancy continued to decrease and fluctuate in Russia and Former Soviet Union, but it increased gradually in the Czech Republic, Poland, Hungary, and Slovenia (Figure 2.1). In the mid–1990s, there was a life expectancy gap of 6 years for men and 5 years for women between Eastern and Western Europe. This East–West mortality gap was mainly attributable to cardiovascular diseases (54%) and external causes of death (23%) in middle–aged populations, particularly in men.[43]

Figure 2.1 Trends in life expectancy at birth (years) for selected countries (1970–2010). Sources: WHO Regional Office for Europe (2011).[44]



Bobak and Marmot (1996) proposed that the East–West mortality gap might be mainly explained by health behaviours (e.g. drinking, smoking or diet) and psychosocial factors (e.g. work stress, perceived control, or social support). Material factors (e.g. environmental pollution or poor medical care) might contribute to only

20% of this gap. Psychosocial factors are important in determining the inequalities in health among and within countries.[45] Between CEE countries, the mortality changes after 1989 were associated with both changes in income and income inequality. In poor countries, income was associated with mortality primarily via material factors (e.g. malnutrition or unclean water); in rich countries, mortality was more strongly related to income inequality mainly via psychosocial factors.[46] Within CEE countries, the social gradient in morbidity and mortality has been identified as the gradient observed in the Whitehall II study for British civil servants.[47] A gradient in mortality among those not poor argues for the importance of psychosocial factors linked to social position.[48]

McKee and Shkolnikov (2001) indicated that the leading causes of high mortality in CEE were injury, violence and cardiovascular disease, particularly in men before age 65. High alcohol consumption, binge drinking, smoking and poor nutrition were considered important underlying factors. As men with least educational levels and least social support were affected the most, they suggested that psychosocial factors might play pivotal roles in explaining health behaviours and mortality gap.[49]

Mackenbach et al (2008) reported that mortality and poor self-rated health were substantially higher in groups of lower socioeconomic status among 22 European countries, but the magnitude of inequalities between higher and lower socioeconomic status was much larger in CEE countries. The authors reported that these country variations were attributable to causes of death related to health behaviours (smoking or drinking) and quality of medical care; psychosocial factors were not measured in this study.[50] In summary, health behaviours appear to be major pathways that partially explain the East–West mortality gap.

(2) Health behaviours and health in Central and Eastern Europe

In general, CEE has higher prevalence of risky health behaviours than Western Europe. From 1960s to 1980s, there had been a dramatic rise in alcohol consumption and cigarettes smoking in CEE.[51] Between 1995 and 2005, Eurocadet Project across

30 European countries found that the highest daily consumption of alcohol per capita was in men in the Czech Republic (56.9 g/l), followed by Luxembourg and Hungary (46.1 g/l). The highest prevalence of current smokers was in men in Latvia (61.6%), Lithuania (58.8%) and Estonia (55.1%). The lowest levels of physical activity were among women in Hungary, Poland and the Czech Republic, and among men in Hungary, Estonia and Bulgaria. In terms of low fruit and vegetable consumption (< 150 g/d), Slovakia, the Czech republic and Latvia were among the fourth to sixth in Europe.[52] Notably, this project excluded Former Soviet Union, which might have even worse profiles of health behaviours than other CEE countries (e.g. 63.0% of Russian men were current smokers in 2004).[53]

The evidence on the links between four health behaviours and health in CEE is summarized. In terms of alcohol drinking, there has been strong evidence suggesting that drinking explained the mortality fluctuations in Russians over the past 20 years. Gorbachev's anti–alcohol campaign (1984 to 1987) was associated with increased life expectancy by 3.2 years for men and 1.3 years for women.[54] However, increased alcohol consumption was associated with rising mortality after the 1990s during social transition.[55] Several studies found that heavy drinking and binge drinking increased cause–specific mortality in cardiovascular disease, accident, violence and liver cirrhosis in Russia.[56,57] Bobak et al found that Russian men had higher prevalence of binge drinking and alcohol–related problems than Czech and Polish men, despite lower annual intake of alcohol.[58] In the Czech republic, Poland and Hungary, the temporal relationships between social policies (e.g. anti–alcohol campaign), alcohol consumption and mortality were also observed in several longitudinal studies.[59,60]

With regards to smoking, it has been reported that smoking accounts for 30% of all deaths at 35–69 years old and 14% at older ages in CEE; mortality from smoking–attributable diseases in Russia is among the highest of the world.[61] From 1960 to 1989, trends in cigarette sales were associated with mortality rates from lung cancer in CEE – the highest rates in Europe.[62] In publications from 2006, mortality rates from lung

cancer among male current smokers in CEE (20% to 28%) were significantly higher than those in Western Europe (16%).[63] In Russia, the dramatic rise of smoking prevalence during the 1990s and 2000s was explained by expansion of trans–national tobacco companies.[64] Perlman and Bobak reported that current smokers had increased risks of mortality in men (OR= 1.80) and women (OR= 2.63) in the Russia Longitudinal Monitoring Survey.[65] In the Czech Republic, smoking prevalence in men declined from 50% in 1985 to 44% in 1992, and this decline was accompanied by reduced cardiovascular mortality at the same period.[66] Notably, smoking prevalence among women in CEE countries has been increasing since the 1980s.[67]

In terms of diet, high consumption of saturated fat and low intake of fruit/vegetable are common in CEE, probably due to poverty, social norms or winter shortages of food.[68] Between 2002 and 2005, Boylan et al found higher intakes of saturated fat and sugar but lower intakes of fruit/vegetable in Russia, the Czech Republic and Poland compared to WHO dietary guidelines for the prevention of chronic diseases.[69,70] In Russia, most people maintained adequate levels of nutrition, but their dietary patterns were characterized by high levels of animal fat, low levels of high–quality protein, and low intakes of fruit and vegetable.[71] In Poland and the Czech Republic, dietary patterns were similar to Russia before 1989; however, ecological studies found that changes in diet quality (e.g. decreased consumption of carbohydrate and saturated fat and increased intake of fruit/vegetable) were associated with decline in cardiovascular mortality in the late 1990s.[72,73]

With regards to physical activity, Steptoe and Wardle showed that 70% of young adults in Eastern Germany, Poland and Hungary had lack of regular exercise (sports or physically active games less than 4 times over past 2 weeks) compared to 64% of Western counterparts.[74] Palosuo reported that 43% of men and 59% of women in Russia had leisure–time exercise less than once a month, compared to 12% of Finnish counterparts.[75] In Russia, only 21% of men and 12% of women engaged in regular leisure–time exercise (sports) in the 1990s.[76] A national survey in Poland showed that

only 14% of the population reported regular leisure–time exercise in the 1990s.[77] In the Czech Republic, a national survey reported that only 27% of men and 18% of women engaged in regular leisure–time exercise in the 1990s.[78]

(3) Social determinants of health behaviours in Central and Eastern Europe

Cockerham explained the patterns of health behaviours in CEE by the social contexts under communist regimes and social transformations after 1989.[79] Using the works of sociologists Max Weber and Pierre Bourdieu as theoretical grounds, he defined collective patterns of health behaviours as health lifestyles based on life choices from options available according to life chances in social structures.[80,81] Weber proposed that life choices (from individual) and life chances (from social structures) interact in a dialectical way; individual's life choices are constrained or enabled by life chances based on socioeconomic factors (class and status). Bourdieu proposed that knowledge of social structures produce enduring orientations towards routine actions (habitus – schemes of perception, thought and action). Habitus provides a process assimilating social structures into individual subjectivity; people choose lifestyles without free will, as habitus predisposes them to limited choices.

To demonstrate Cockerham's analyses in CEE, health lifestyles of Russian working–class men are taken for example. Heavy episodic drinking (e.g. high doses of vodka in a short time) was a strong tradition of Russian peasant culture, which spread into cities as industrialization transformed peasants into industrial workers. Traditionally, heavy drinking took place only on holidays, but it gradually became common throughout the year. In social drinking, one is expected to drink as much as the others regardless of one's own will. Social norms and interpersonal dynamics may force one's choice to drink. Similarly, there were social norms for smoking and eating more fat and less fruit and vegetable among these men.[82]

In socialism, there is the priority of state goals (e.g. military or heavy industry) over personal needs (e.g. health care). This paternalism of the state induced a false

sense of security; people believed that the state would take care of their health. Although the state invested in secondary prevention (e.g. contact with physicians for early detection of diseases), primary prevention (e.g. people take responsibility for their health lifestyles) was not impimented or encouraged. In summary, unhealthy lifestyles of Russian working–class men were determined by habitus derived from the wider society – social norms, experience and reality of class circumstances (de-emphasizing the individual and over-emphasizing the state).[83]

According to sociological theories, Cockerham elaborated social determinants of health behaviours in the CEE contexts. As mentioned earlier, the Eastern–West mortality gap was mainly attributable to psychosocial factors and health behaviours based on epidemiological evidence. Indeed, Cockerham's works implied that various psychosocial factors embedded in social contexts might influence individual's health behaviours. The associations between psychosocial factors and health behaviours will be discussed in detail in the following sections.

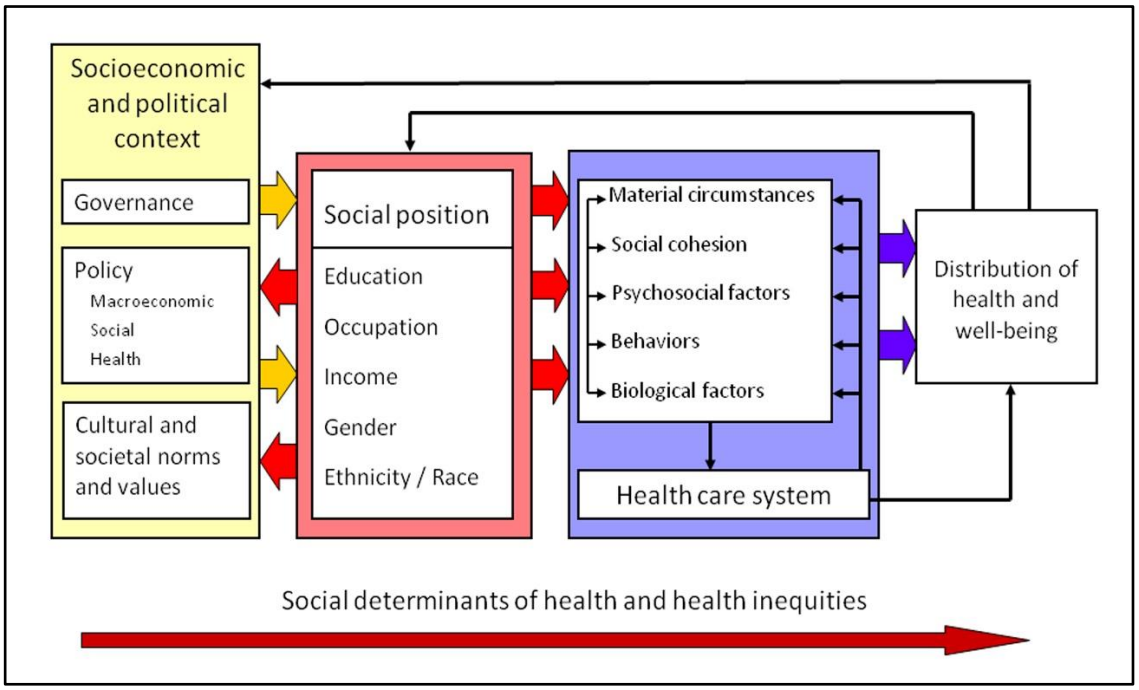
2.2 Psychosocial Factors

2.2.1 Psychosocial factors – definition and their impact on health

Psychosocial factors were defined by Hemingway and Marmot (1999) as measurements that potentially link psychological phenomena to social environment and physiological changes.[84] Martikainen et al (2002) defined psychosocial factors as pertaining to the influence of social factors on an individual's mind or behaviour, and to the interrelation of behavioural and social factors. This definition might have important implications for social epidemiologists and health researchers, because it implies that psychosocial factors can be viewed as: (1) mediators in the effects of social structural factors on individual health outcomes, or (2) modified by the social structures and contexts in which they exist.[85]

There have been numerous empirical studies presenting causal pathways from social structures to individual health.[86] The macro–level social, political and economic context contributes to unequal distribution of resource, power and prestige within a society, which then affects the meso–level socioeconomic position (social position) defined mainly by occupation, education and income. A social gradient in health across a society was reported in the British Whitehall II study and later replicated across the world; those in higher social position tend to have better health status.[87,88] Moreover, the influences of social position on individual health are mediated by psychosocial, behavioural (health behaviour), material, and biological factors.[89] In the theoretical framework for social determinants of health (Figure 2.2), psychosocial factors are regarded as mediators in the effects of social position on individual health.

Figure 2.2 Theoretical framework for social determinants of health. Source: WHO Commission on Social Determinants of Health (2008).[90]



At micro–level individuals, psychosocial factors can influence health directly via psychobiological processes or indirectly via choices of health behaviours. Psychobiological processes are the pathways via which psychosocial factors stimulate central nervous system activation of autonomic, endocrine, immune and inflammatory

responses.[91] First, social processes influence individual's psychological processes, involving the brain structures in limbic system and prefrontal cortex to influence lower neural pathways (hypothalamic–pituitary–adrenal axis). Second, these neural pathways regulate autonomic nervous systems by neurotransmitters (e.g. catecholamine) and hormones (e.g. cortisol), thereby influencing peripheral physiological activities. Finally, physiological consequences are tissue damage in inflammation, inhibited immune function, metabolic changes, and oxidative stress reactions which contribute to a variety of chronic diseases.[92]

Based on existing literature, psychosocial factors related to health are categorized by Steptoe et al.[93] First, chronic stress exposures such as work stress, neighborhood stress, caregiver strain, economic hardship, and life events have been shown to increase risk of cardiovascular disease, depression and poor health.[94,95,96] In terms of work stress, the Demand–Control model and the Effort–Reward Imbalance model have gained strong support to predict a variety of health outcomes.[97,98] Second, several personality constructs have been found to reduce (e.g. perceived control or self-efficacy) or increase (e.g. Type A behaviour, hostility or neuroticism) the risk of morbidity and mortality.[99,100] Third, psychological distress and depression have been reported to be associated with cardiovascular morbidity and mortality.[101] In contrast, positive affect was shown to improve health.[102] Fourth, protective aspects of social environment such as social support, social network and social capital have been found to reduce morbidity and mortality.[103,104]

Although numerous studies have linked each psychosocial factor independently to health outcomes, relatively few studies have attempted to examine potential inter-relationships between different psychosocial factors (e.g. work stress and personality) in relation to health behaviours. It is widely recognized that psychosocial factors rarely occur in isolation, so it is important to expand the breadth of studies to address this multiplicity of psychosocial factors – this thesis will focus on this issue.[105]

2.2.2 Psychosocial factors and health in Central and Eastern Europe

As mentioned earlier, Bobak and Marmot (1996) proposed that the East–West mortality gap might be mainly explained by health behaviours (e.g. drinking, smoking or diet) and psychosocial factors (e.g. work stress or perceived control).[106] McKee and Shkolnikov (2001) also indicated that health behaviours and psychosocial factors played pivotal roles in explaining the East–West mortality gap.[107] The links between health behaviours and health in CEE have been introduced in Section 2.1.2. In this section, the relations between psychosocial factors and health in CEE are discussed.

Previous research has showed that psychosocial factors such as work stress, depression, low perceived control, and poor social support are serious risk factors for morbidity and mortality in CEE countries.[108,109] In the following paragraphs, the two psychosocial factors related to this thesis – work stress and perceived control, and their influence on health in the CEE contexts are introduced. Note that the impact of OC on health has rarely been examined in CEE.

(1) Work stress and health in Central and Eastern Europe

The social contexts of working conditions in CEE are described. From 1947 to 1989, the communists adopted centrally planned economies across CEE countries. The socialist enterprise was state–owned and oriented to an input–output plan rather than any market. The enterprise played a role in implementing the state's social welfare policies; efficiency and productivity were not major concerns for managers. This legacy of socialist era may influence employee's passive attitude and low efficiency even in post–communistic period.[110]

There have been rapid and profound changes in labour markets in CEE since 1989; new economic mechanisms created quite different contexts from communistic regimes. After 1989, labour market differences between CEE and Western Europe were substantial in terms of gross domestic product per capita (GDP – a country's

standard of living) and unemployment rate. From 1990 to 1995, CEE countries which joined European Union later (e.g. Czech Republic or Poland) went through transitional recession with GDP decreased by 4.7%; after 1995, economic began to recover with annual GDP growth at 3.7%. However, Former Soviet Union still lagged behind. Employees often had low wage levels, unstable pay, dual earning careers and holding a third job, partcularly in those with low social position.[111]

In terms of unemployment, cumulated decline in employment from 1990 to 2003 was 17.0% in CEE countries, in contrast to cumulated increase in employment by 7.3% in European Union. Despite growth of productivity in CEE after 1995, restructuring processes led to layoffs of redundant workers, resulting in considerable job losses in agricultural and industrial sectors. High unemployment rates also aggravated job insecurity in active employees.[112]

Globalisation led to deregulation of labour markets and increased competition; many organisations undertook restructuring and downsizing.[113] Global division of labour has transformed CEE countries into manufacturing locations as subcontractors for Western European and US firms. They were transformed from unskilled and labor–intensive to skilled and capital–intensive production (e.g. automotive or information technology).[114] To be globally competitive in production efficiency, organisations might adopt strategies to meet aggressive production goals, probably resulting in high levels of work stress.[115] For example, information technology employees in CEE were found to have stressful work conditions, low wage and temporary employment.[116]

As the biggest contribution to the East–West mortality gap was from working–aged men, psychosocial factors at work might contribute to this gap.[117] Several studies have shown that work stress measured by the DC and the ERI models predicted cardiovascular morbidity and mortality,[118,119] poor self-rated health,[120] menstrual pain,[121] depression,[122] and high alcohol consumption in CEE.[123] The adverse effects of high ER ratio on health in CEE were at least as strong as those found in Western countries.[124] Laszlo et al compared the prevalence of job insecurity

(one item in the ERI model) in 23,245 adults among 16 European countries; the highest levels of job insecurity were in Poland (41.7%), Czech Republic (41.0%) and Hungary (40.4%), compared to the lowest in Spain (14.2%) and France (17.6%). Job insecurity was associated with poor self-rated health across European countries.[125] Therefore, work stress appears to be a serious public health issue in CEE.

(2) Perceived control and health in Central and Eastern Europe

In socialism, there is the priority of state goals (e.g. military or heavy industry) over personal needs (e.g. health care). This paternalism of the state induced a false sense of security; people believed that the state would take care of their health. Although the state invested in secondary prevention (e.g. visiting physician for early detection of diseases), primary prevention (e.g. people take responsibility for their health lifestyles) was not encouraged or implemented.[126] Perceived control (PC) is defined as: the extent to which one can intentionally produce desired outcomes and prevent undesired ones.[127] The paternalism of socialism may reduce people's PC for their health behaviours and health.

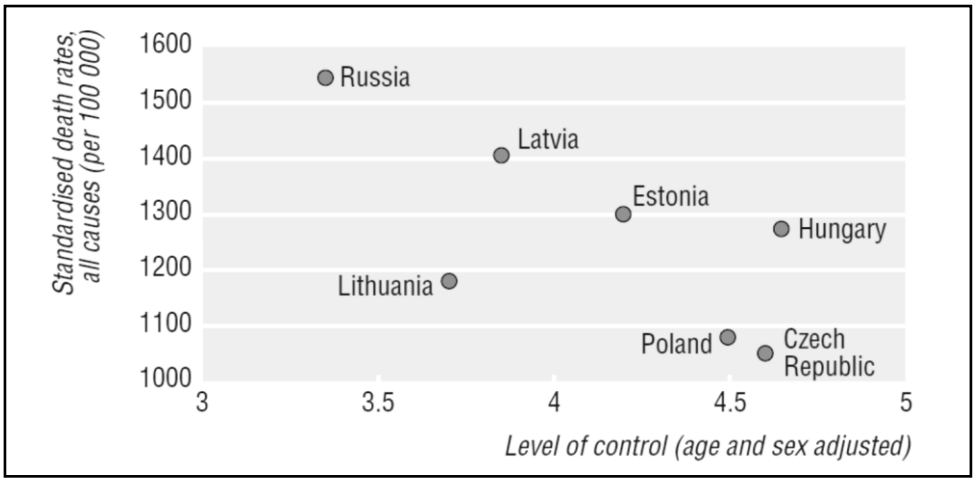
Since 1989, socio-economic transformation in CEE might challenge one's beliefs about how the world works, which were primarily based on economic and political understanding in communist periods. The past is not an adequate guide to the present, and some people might have low sense of control (PC) over life. The stressors from transformation not only challenged individuals to cope actively and to find opportunities, but also threatened some people to suffer from uncertainty and low PC over life.[128]

Due to transformational contexts in CEE, reserachers were particularly interested in one psychosocial factor – PC and its relation to health. Several empirical studies have examined the relationships between PC and health outcomes in CEE. Bobak et al conducted a cross–sectional survey (n= 5,330) in 7 CEE countries (Russia, Lithuania, Latvia, Estonia, Poland, Czech Republic and Hungary). They found that low PC was significantly associated with poor self–rated health; PC can partially mediate

the effects of material deprivation on poor health.[129] Lundberg et al observed that the levels of PC in Russia were significantly lower (n= 9,237) than those in Sweden (n= 1,007); lower PC was associated with poorer self-rated health in both countries.[130]

Carlson used data from the 1992 World Value Survey, showing that lower PC was associated with poorer self-rated health within and between 23 national samples of men and women; PC can partially explain the East–West divide in self-rated health.[131] At the population level, Pikhart found lower levels of PC were associated with higher rates of all–cause mortality in 7 CEE countries; Pikhart indeed provided the ecological evidence that group levels of PC predicted population rates of mortality (Figure 2.3).

Figure 2.3 Mean levels of control for 7 population samples plotted against all–cause mortality for the countries from which these population samples were drawn. Source: Pikhart (2002).[132]

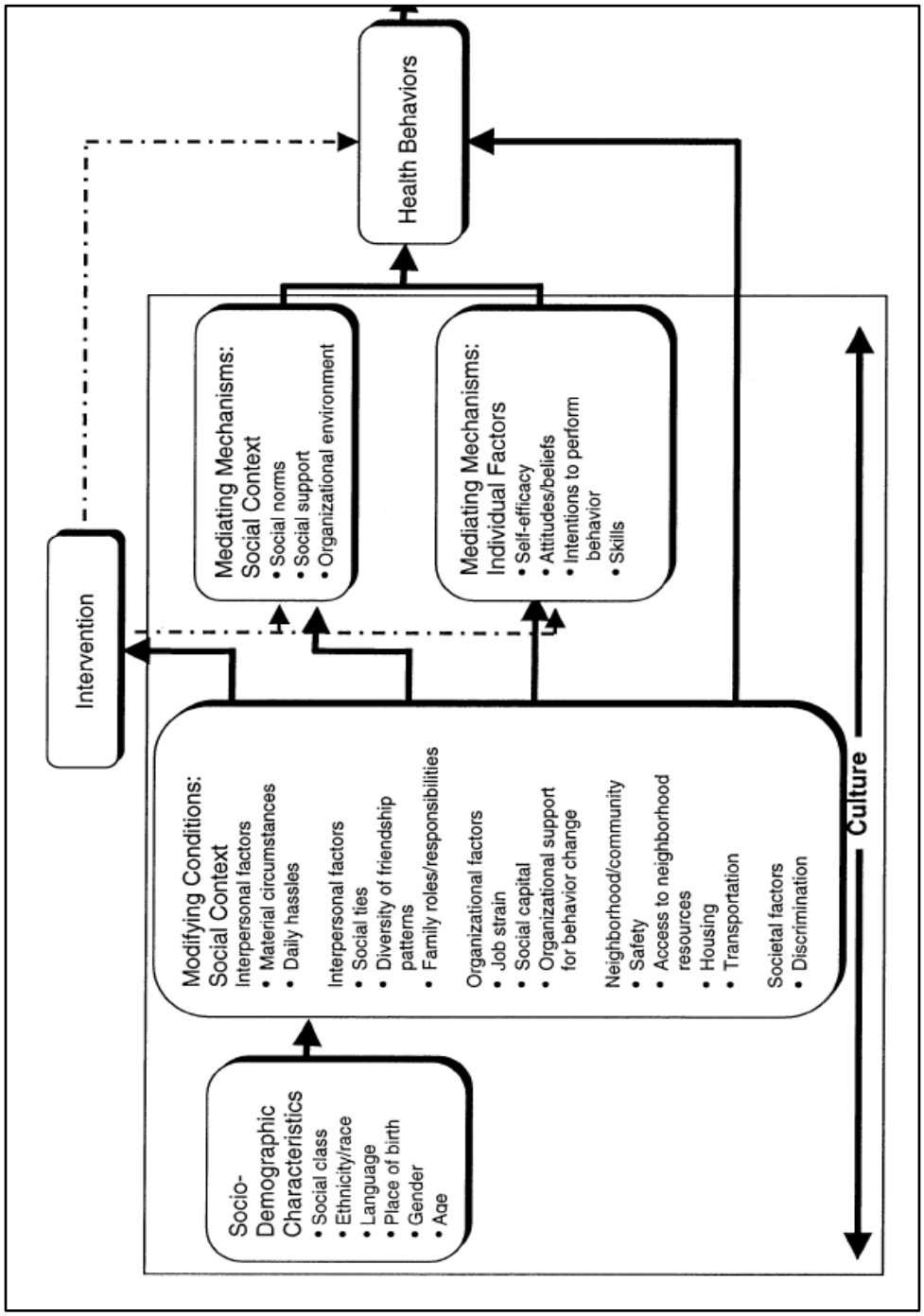


2.2.3 Psychosocial factors and health behaviours

Across multiple health behaviours, the risk patterns by social position remain relatively constant: people with higher levels of income, occupation or education tend to engage in fewer risky health behaviours than those with lower social position.[133] Social epidemiologists attempted to identify the psychosocial factors linking social position to health behaviours. Sorensen and colleagues in Harvard School of Public

Health proposed a conceptual framework for the social context of health behaviours, which combined the strength of several disciplinary perspectives in the framework of social ecological model (Figure 2.3). First, they adopted the rich tradition of psychological research, building on behavioural theories (e.g. Social Cognitive Models) to identify critical psychosocial factors predicting health behaviours – self-efficacy, attitude or intention.[134] Second, they incorporated the input from social epidemiology, in which numerous studies have supported the causal pathways from social position to health behaviours. Finally, they identified mediating mechanisms that are important to behaviour change and are potentially modifiable by interventions based on theory and research.

Figure 2.4 Conceptual framework for social contexts of health behaviours. Source: Sorensen, Emmons and Hunt, et al (2003).[135]



Social ecological model has become a main model for health behaviours since the 1980s, when the limited effectiveness of individual interventions to change health behaviours led to a paradigm shift to consider broader social contexts in which people live and work. Social ecological model provides a comprehensive framework for understanding the multiple and interacting determinants of health behaviours and can

be used to develop interventions targeting at several levels. A general acceptance of this model is reflected by international authorities guiding public health.[136,137] The multi-level determinants of health behaviours are intrapersonal, interpersonal, organisational, community, and public policy levels. Public policy level includes local and state policies and laws that regulate or support healthy actions for disease prevention. Community level involves social networks and norms, which exist among individuals, groups and organisations. Interpersonal level refers to interpersonal processes – family and friends that provide social identity and social support.[138] Organisational and intrapersonal levels are introduced in more details below.

Organisational level means the level of workplace; an organisation is a group of people intentionally organized to accomplish an overall set of goals (e.g. products or services). Given how important work is in everyday life, workplace is both a resource for health promotion and a source of stress exposure influencing health. Since the 1970s, organisational policies and practices are often the target of health promotion. For examples, worksites may offer smoke–free office buildings, smoking cessation classes, facilities for physical activity, healthy food served at cafeterias, or health examinations for employees.[139] Most large corporations now provide health promotion programs for employees. In contrast, accumulating evidence has shown that work stress can influence health directly via psychobiological processes or indirectly via health behaviours (for more details, see Section 2.3).[140]

Intrapersonal level refers to the level of psychological factors in an individual. Although there have been a wide range of psychological models predicting health behaviours, no single theoretical model has dominated research and practice in health behaviours. Glanz et al conducted reviews of publications from 1986 to 2005; they found that the most commonly used theories of health behaviours at intrapersonal level are Social Cognitive Models (SCMs). SCMs specify cognitive and affective factors as proximal determinants of health behaviours; the common construct across all SCMs is self-efficacy (a component of PC), belief that one can successfully perform

the behaviour.[141] In addition, specific personality traits (Type A behaviour, Neuroticism or Hostility) have received empirical support to predict health behaviours. Various personality factors – personality traits and social–cognitive constructs have been found to predict health behaviours (for more details, see Section 2.4).

The distinction between personality traits and social–cognitive constructs (e.g. PC or self-efficacy) is introduced here. By integrating diverse perspectives in psychology, "personality" is defined by Carver and Scheier as: *a dynamic organisation, inside the person, of psychophysical systems that create the person's characteristic patterns of behaviours, thoughts and feelings*. First, characteristic patterns suggest continuity and consistency uniquely identified in an individual. Personality traits – proposed by *dispositional, biological, and psychoanalytic perspectives* are biologically based temperaments less susceptible to the influence of environments. Second, dynamic organisation implies ongoing readjustment and adaptation in an individual; *social learning, cognitive, socio-cognitive, and humanistic perspectives* view personality as an accumulated set of thoughts and behaviours learned from environments.[142] Social–cognitive constructs (expectancy or appraisal) are more susceptible to the influence of environments (e.g. social position or work stress).[143,144] Social–cognitive constructs provide a more active and specific process account of individual differences that complements the broader and more static description in personality traits; they provide better prediction for behaviours and more modifiable targets for interventions.[145]

In social ecological model, there has been consistent support for the multi–level influences (independent effects of various psychosocial factors) on health behaviours, but the interactions between different levels – such as organisational level and intrapersonal level deserve further research.[146] In this thesis, the dimension between organisational and intrapersonal levels will be addressed. To further elaborate this issue, literature review will particularly focus on work stress, personality constructs

(personality traits and social–cognitive constructs), and their influences on health behaviours in the following sections.

2.3 Work Stress and Health Behaviours

For most population in early, middle and old adulthood, work plays a significant role as it is generally a prerequisite for a regular income, an opportunity for learning and achievement, and a variety of life opportunities. It is mainly through work that core social identity outside family and social status are acquired. Thus, the qualities of work conditions in terms of prospect and security are crucial for health and well-being.[147] In contrast, physical and psychological hazards at work may lead to adverse health consequences. With technological progression, the nature of work has undergone fundamental changes from industrial production to service sector; current jobs are often sedentary works involving information processing and coordination, rather than physically strenuous works. Thus, work stress becomes a central concern in modern societies; most employees are exposed to psychological demands rather than physical hazards.[148] Among theoretical models of work stress, the Demand-Control (DC) model and the Effort-Reward Imbalance (ERI) model have gained considerable attention and support.[149]

2.3.1 The Demand–Control model

Karasek and Theorell traced the history of modern work patterns to industrial revolution in the 19th century. The dramatic changes of work structures generated the political and economic power of enterprises, which exert strict control over employees' work processes. Karl Marx (1867) indicated the alienating and dehumanizing nature of work patterns. Frederick Taylor (1911) wrote "Principles of Scientific Management"; workers' tasks were simplified into element skills required, which were recombined into

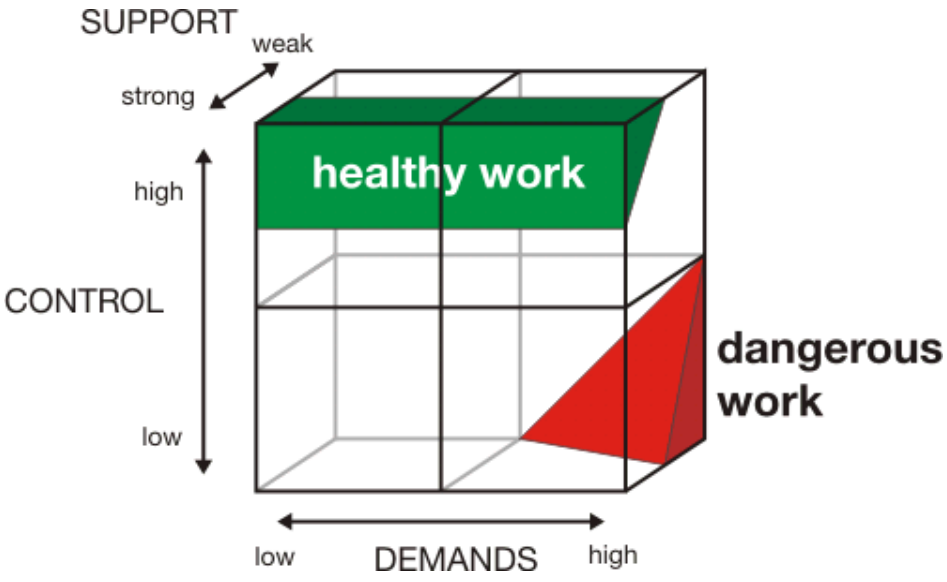
complex tasks by a machine–paced assembly line. A set of managers and engineers control workers' behaviours in a way that coordinate their specialized tasks. This precise division of labour can increase work speed and produce maximum profit; however, workers may lose control and get high demands, as Karasek confirmed by empirical research in machine–paced operatives and low–status service operatives.[150] Since the 1960s, social movements against work conditions occurred in United States and Europe. Nevertheless, the new division of labour was formed on a global scale, with similar work patterns replicated across the world.

Karasek and Theorell proposed the DC model based on accumulating literature on psychological demands and control. In the research on demands, Selye (1936) proposed a U–shaped association between demands and performance; some level of demands is necessary for effective performance and job satisfaction, but higher demands are disastrous.[151] Hinkle (1968) conducted the first prospective study on the link between high demands and the risk of myocardial infarction.[152] Since then, there have been a growing number of studies on psychological demands and health.[153] In the research on control, Karasek attributed his concepts to the similar origins as perceived control: White's effectance motivation, Rotter's locus of control, and Bandura's self-efficacy (for more details, see Section 2.4.3). He emphasized social learning processes: active learning occurs in situations requiring high demands and high control in order to choose how best to cope with a new stressor.

The DC model is initially composed of two dimensions. One dimension is control (decision latitude) indicating employees' control over their tasks and how these tasks are executed. Control consists of skill discretion (a variety of tasks, low repetitiveness, occasions for creativity, and opportunities to learn new things) and decision authority (ability to make decisions about their own job, and ability to influence team and company). Another dimension concerns psychological demands representing psychological stressors in work environment (time pressures, pace of work, interruption rate, conflicting demands, amount of work, degree of concentration

required). Interactions of high and low levels of control and demands generate four psychosocial work characteristics. Job strain (high demands and low control) is the worst situation; when individuals have low control to cope with overwhelmingly high demands, the stress would produce adverse health outcomes. Passive job (low demands and low control) is the second worst situation; employees become passive at work. Active job (high demands and high control) is a favorable situation; when individuals have high control to cope with high demands, they can learn actively and develop competence to deal with challenges. Low strain (low demands and high control) is a favorable situation regarding health outcomes.

Figure 2.5 Low control, high demand and low work support cause adverse health outcomes. Source: Adapted from Johnson and Hall (1988).[154]



Johnson and Hall added a third dimension – workplace support from supervisors and colleagues into the original framework, because accumulating research showed that social support can influence health outcomes or buffer stress–health relationships. Social support generally includes instrumental and emotional support. Instrumental support is extra resources or direct assistance in work tasks given by supervisors or colleagues. Emotional support is social and emotional integration and trust between colleagues and supervisors. The combination of low control, high demands, and low

workplace support (called iso–strain) is a stressful psychosocial work condition producing adverse health outcomes (Figure 2.4).

2.3.2 The Effort–Reward Imbalance model

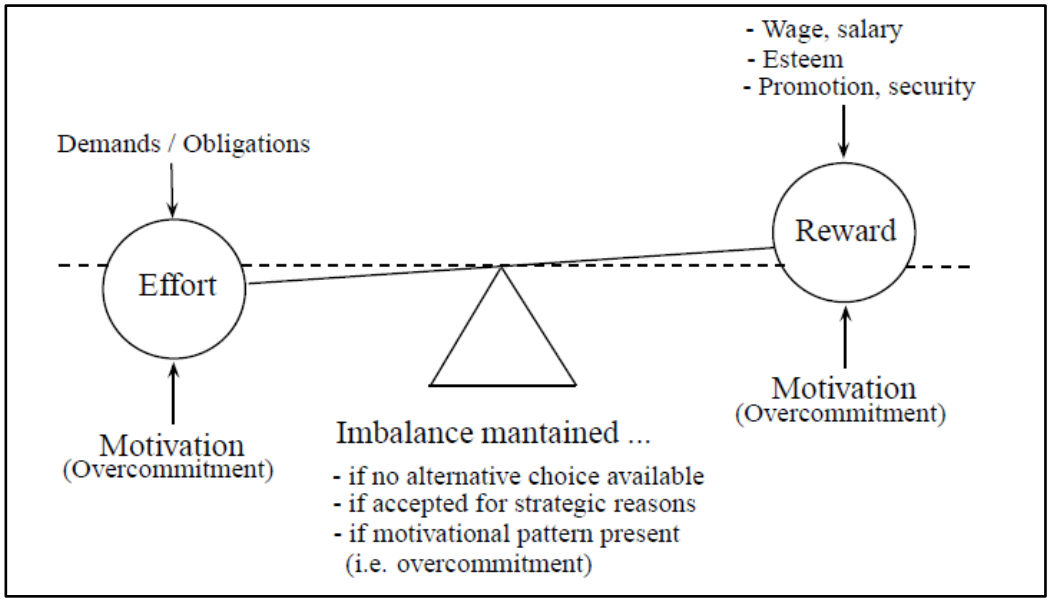
The ERI model was proposed by Johannes Siegrist (1990) based on medical sociology and the concept of social reciprocity, a basic principle of social exchange process. Social reciprocity lies at the core of employment contract, which assumes that tasks and obligations to be performed by employees in exchange for adequate rewards from employers. Contractual reciprocity operates via norms of return expectancy. Violation of reciprocity in terms of high effort and low reward can elicit strong negative emotions, which subsequently trigger sustained stress responses (sustained autonomic and neuroendocrine activation) involved in the pathogenesis of coronary heart disease and adverse health outcomes. In contrast, adequate reward can promote positive emotions, well-being and good health.[155] Notably, the ERI model particularly emphasizes the social roles of work; the workplace provides opportunities to acquire self-regulatory needs in terms of self-efficacy (e.g. successful job performance), self-esteem (e.g. recognition form supervisors or colleagues), and self-integration (e.g. belonging to a social group).[156]

The ERI model is composed of three dimensions. The first dimension is extrinsic effort that represents time pressure, interruptions, responsibility, overtime work, physical demands, and increasing demands. The second dimension is reward that represents salary, esteem (respect from superiors, colleagues and work, adequate support, and unfair treatment) and social status control (promotion prospects, adequate position, adequate work prospects, undesirable change, and job security). Effort–Reward ratio is the ratio of the score for extrinsic effort (E) to the score for reward (R). ER ratio > 1 is considered to indicate effort–reward imbalance (Figure 2.5).

The ERI model incorporated a personality dimension into a situational model. Overcommitment (OC) is a personality construct reflecting the personal need for control in dealing with work demands. A high OC person displays a pattern of attitudes, behaviours, and emotions characterized by an excessive striving at work and a strong motivation for esteem and approval at work. High OC people strive towards high achievement, have difficulty withdrawing from work, and maintain excessive effort under inadequate reward, thereby resulting in prolonged non–reciprocal exchange.[157]

Figure 2.6 High effort and low reward at work cause adverse health outcomes.

Source: Adapted from Siegrist (2000).



The following three hypotheses are derived from the ERI model: (1) Extrinsic ERI hypothesis: the mismatch between high effort and low reward can lead to adverse health outcomes. (2) Intrinsic effort hypothesis: a high level of OC per se can increase the risk of adverse health outcomes; that is, there is a direct effect of OC on health. (3) Interaction hypothesis: those who are characterized by both conditions (1) and (2) have even higher risks of adverse health outcomes; that is, there is interaction between OC and ERI.[158]

Some researchers argue that the ERI model has more general explanatory value than the DC model in current working contexts. The DC model was initially addressed in blue–collar industrial workers and it reflected social concerns emphasizing workers' control in the 1970s.[159] In this era of globalization, tight managerial control in industry is shifted to flexibility, self-regulation and decentralization.[160] Empirical evidence supports that the ERI model emphasizing "reward" in career prospects and esteem is more sensitive in explaining work stress in modern occupations than the DC model.[161,162] In addition, although the two models overlap to some extent in "extrinsic effort" and "demands", they identify different aspects of work stress in "reward" and "control". Evidence has supported that combination of the ERI and the DC models, or at least dimensions of them (e.g. ERI and control), can produce stronger predictive power on health outcomes than adopting either model alone.[163]

2.3.3 Empirical studies on work stress and health behaviours

Many studies have showed that the DC and the ERI models can predict mental disorders (e.g. depression, anxiety), cardiovascular diseases (e.g. coronary heart diseases, hypertension, stroke), metabolic diseases (e.g. diabetes, hyperlipidaemia), musculoskeletal diseases, poor self-rated health and sickness absence.[164,165,166] For example, Kivimäki et al conducted a meta-analysis on 13 European cohort studies; the hazard ratio for coronary heart disease was 1.23 (95% CI= 1.10–1.37) for job strain versus no strain.[167] Stansfeld and Candy conducted a meta-analysis on 11 cohort studies; the effects of job strain (OR= 1.82, 95% CI= 1.06–3.10) and ERI (OR= 1.84, 95% CI= 1.45–2.35) were substantial on depression and anxiety.[168] Van Vegchel et al reviewed 45 studies and found that most studies supported predictive validity of ER ratio for various health outcomes.[169]

The impacts of work stress (psychosocial factor) on health are mainly explained by direct psychobiological processes or by indirect pathways via health behaviours.

Siegrist reviewed 46 studies and found moderate support for consistent associations between the DC/ERI models and health behaviours; relatively strong relationships were found in alcohol drinking and overweight (a proxy measure for diet and physical activity) in men.[170] Heikkilä et al conducted a meta-analysis based on 11 European cross–sectional studies (n= 118,701) and 4 cohort studies (n= 43,971); individuals with job strain were more likely than those with no strain to have co–occurrence of several health behaviours– heavy drinking, current smokers, physical inactivity and overweight (OR= 1.25, 95% CI= 1.12–1.39).[171] Kouvonen et al reported the dose–response relationship between the extent of work stress (measured by the DC/ERI models) and the number of health behaviors (alcohol, smoking, physical inactivity, and overweight) in the Finnish Public Sector Study (n= 36,127).[172,173]

The potential mechanisms linking work stress to health behaviours are suggested based on existing evidence. In terms of biological pathways, work stress might lead to biological responses (e.g. dysfunction of mesolimbic dopamine system in the brain), which cause substance addictions (alcohol drinking or smoking).[174] Work stress can influence physiological responses (e.g. increased activities of hypothalamus–pituitary–adrenal axis and elevated levels of cortisol and insulin), resulting in food choice towards high-fat and high-carbohydrate content.[175] In terms of psychological pathways, work stress has been found to predict psychological distress like anxiety and depression; individuals might engage in risky health behaviour (emotion-focused coping) to temporarily relieve or avoid their psychological distress and to distract their attention from stressful situation.[176]

In the following paragraphs, empirical evidence on the relationships between the DC/ERI models and the four health behaviours will be discussed.

(1) Work stress and alcohol drinking

In terms of the DC model, Heikkilä et al conducted a meta-analysis on 12 cross–sectional (n= 142,140) and 4 longitudinal studies (n= 48,646) on the DC model and

alcohol drinking. Compared to moderate drinkers (1–210 g/week of ethanol in men; 1–140 g/week in women), heavy drinkers (>= 280 g/week in men; >= 210 g/week in women) had significantly higher odds of job strain (OR= 1.12, 95% CI= 1.00–1.26).[177] Siegrist also found 4 out of 6 longitudinal studies supporting the link between the DC model and alcohol consumption.[178] In general, the moderate associations between the DC model and alcohol drinking have received support. Most of studies reported that job strain (high demands and low control) was associated with high alcohol consumption or alcohol abuse.[179,180] However, several studies had slightly different findings. For example, Gimeno et al reported that passive job (low demands and low control) was related to heavy drinking in a US cross–sectional study (n= 3,099).[181] Niedhammer et al found that low control was associated with high alcohol consumption in men, but low work support was related to high alcohol consumption in women in a French cross–sectional survey (n= 20,625).[182] It is possible that the associations between the dimensions of work stress and drinking are relatively specific for each study population, thereby leading to differences between various studies.

In terms of the ERI model, several studies have showed promising results to support the links between high ER ratio and drinking outcomes. Head et al reported that high ER ratio was associated with alcohol dependence in men (OR= 1.93) after adjustment for age and employment grade in British Whitehall II cohort study (n= 7,372); this association in women was not as remarkable as that in men. In contrast to men, women with higher employment grade tended to drink more.[183] Puls et al found that ERI was associated with high alcohol consumption in a German cross–sectional study.[184] Bobak et al conducted a cross–sectional study in men (n= 694) in 3 CEE countries; they found that high ER ratio was associated with binge drinking (OR= 1.36), problem drinking (OR= 1.37), negative consequences of alcohol (OR= 1.22), high annual intake of alcohol (OR= 1.29), and high annual number of drinking sessions (OR= 1.34).[185] To establish stronger evidence on the link between the ERI model and drinking, more longitudinal studies are still needed.

(2) Work stress and smoking

In terms of the DC model, Albertson et al reviewed 22 prospective studies on the DC model and smoking outcomes. There was strong evidence for the effect of high demands on smoking intensity (the amount smoked) in current smokers. High control increased the probability of smoking cessation, and high demands increased the probability of smoking relapse.[186] In addition, work social support was positively associated with smoking cessation and negatively associated with smoking intensity and relapse. Heikkilä et al conducted a meta-analysis on 15 cross–sectional data (n= 166,130) and 6 longitudinal data (n= 52,024) from European studies. Current smokers had higher odds of job strain than never–smokers (OR= 1.11, 95% CI= 1.03–1.18); there was no difference in job strain between ex–smokers and never–smokers. For smoking intensity, current smokers with job strain smoked three cigarettes more per week than those without job strain. However, there was no clear evidence for longitudinal associations between job strain and taking up or quitting smoking (changes in smoking status).[187]

In terms of the ERI model, Kouvonen et al reported that high ER ratio was associated with being current smokers (OR= 1.28) in a Finnish cross–sectional study (n= 46,190); among current smokers, high ER ratio was associated with high smoking intensity (OR= 1.19).[188] Peter et al observed a positive association between ER ratio and smoking intensity in a German cross–sectional study of middle-aged men.[189] In addition, an Australian cross–sectional study (n= 1,101) showed that higher ER ratio was associated with higher smoking intensity in women, but not in men.[190] However, Ota et al found that ER ratio at baseline did not predict smoking cessation at 2–year follow-up in 1,423 middle-aged men in Japan.[191] Despite promising results to support the associations between ERI and smoking outcomes in several cross–sectional studies, more longitudinal studies are needed to provide better evidence.

(3) Work stress and diet

The associations between chronic stress and dietary outcomes have been extensively studied in epidemiological and laboratory studies.[192,193] In terms of the DC model, Hellerstedt and Jeffery found that high demands were associated with high fat intakes in men, but not women, in a US cross–sectional study (n= 3,843).[194] In a British cohort Study (n= 3,397), passive job was associated with unhealthy food habits (e.g. not eating vegetables/fruit at least twice a day, or not choosing wholegrain bread and low–fat milk).[195] In a Finnish cohort study (n= 6,243), low job strain was associated with a healthier diet (fresh vegetables and fruit daily, whole grain bread daily, fish at least twice a week, using vegetable–based margarine, and usually using oil in cooking) among women, but not men.[196] A Finnish cross–sectional study (n= 6,369) reported that job strain was associated with frequent use of packed meals (less in line with nutritional recommendations) among men.[197] In Japan, job strain was positively associated with more fat and cholesterol intakes among men in a cohort study (n= 25,104);[198] job strain was associated with less vegetables (n= 6,759) and more calorie intakes (n= 1,183) in two cross-sectional studies.[199,200] However, no significant association between job strain and diet was reported in a cohort study in Netherlands (n= 3,309).[201]

Despite empirical support on the link between the DC model and diet, no research is available on the ERI model and diet. The ERI model has been found to predict other health behaviours (drinking or smoking), and it is reasonable to suggest a potential link between ERI and diet. In addition, the measurements of dietary outcomes varied considerably between previous studies (e.g. foods, nutrients, or food habits); those studies with less precise measures (e.g. fewer food items) should be interpreted cautiously. The method of diet quality takes into account the intakes of various foods and nutrients, thereby providing more accurate pictures of diet than single food/nutrient intake. Diet quality is often defined by the adherence to dietary guidelines associated with health outcomes, such as the WHO guidelines for the prevention of

chronic diseases.[202] Thus, empirical studies regarding work stress and diet quality would provide more solid evidence for this topic.

(4) Work stress and physical activity

With regard to the DC model, Fransson et al conducted a meta-analysis based on 14 European cohort studies (n= 170,162); in prospective analyses, the odds for physical inactivity during leisure time were higher for those with job strain (OR= 1.21, 95% CI= 1.11–1.31) and passive job (OR= 1.20, 95% CI= 1.11–1.30) compared to those with no strain.[203] Kirk and Rhode conducted a systematic review and found that job strain was related to physical inactivity during leisure time in 6 out of 8 cross–sectional and prospective studies.[204] For example, Lallukka et al reported that job strain was prospectively associated with physical inactivity (OR= 1.88) among white–collar men in British Whitehall II Study (n= 3,397) and women (OR= 1.25) in Helsinki Health Study (n= 6,070).[205] Gimeno et al found that passive job was related to physical inactivity at 5–year follow-up (OR= 1.16) in men in Whitehall II Study (n= 4,291).[206]

In terms of the ERI model, Kouvonen et al reported that high ER ratio was associated with physical inactivity among women (OR= 1.08) and men (OR= 1.17) in a Finnish cross–sectional study (n= 35,918).[207] In contrast, Kuper et al reported that higher ER ratio was prospectively associated with more physical activity measured by time spent in moderate to vigorous activity in Whitehall II study.[208] It is implied that high ERI might decrease physical activity in some people (e.g. those prone to depression) but increase physical activity in others (e.g. those with active coping adopt physical activity to reduce stress).[209] Further evidence is needed regarding the relationship between the ERI model and physical activity.

In summary, there has been adequate support for the moderate associations between the DC model and four health behaviours. Additionally, existing studies have shown promising results to support the relationship between the ERI model and health behaviours, but no literature is available on ERI–diet relationship. There are some

limitations to the generality of existing literature on work stress and health behaviours. First, there have been relatively few longitudinal and intervention studies in this topic, and further evidence with methodology better than cross–sectional design is needed. Second, the associations between the work stress and specific health behaviours varied across sexes and populations; this inconsistency may be explained by sex and other psychosocial factors (mediators or modifiers) like personality constructs.[210] To predict health behaviours more accurately, it is helpful to incorporate personality constructs into the investigation of relationships between work stress and health behaviours.

2.4 Overcommitment Personality and Health Behaviours

In the ERI model, Siegrist proposed the personality construct of overcommitment (OC) which describes individual attitudes, behaviours and emotions reflecting excessive work–related striving and high need for approval and esteem; they have difficulty withdrawing from work and maintain excessive effort under inadequate reward.[211] Siegrist initially described OC as “need for control” – a distinct individual pattern of coping with work demands, which evolved from Type A behaviour.[212] Rosenman traced the origins of Type A behaviour to perceived control (PC) in psychology; Type A persons have higher need for control over environment and tend to perceive lower PC, and their response is enhanced coping to assert and maintain control over environment.[213] Thus, the origins of OC might be traced to Type A behaviour (see Section 2.4.1) and PC (see Section 2.4.3).

Siegrist and colleagues initially assessed “need for control” by a 29–item scale with 6 dimensions (need for approval, competitiveness, disproportionate irritability, inability to withdraw from work, hard work, and perfectionism).[214] However, several studies cannot replicate the factorial structure of need for control.[215,216] A shorter version of OC score was then developed by exploratory and confirmatory factor

analyses, consisting of inability to withdraw from work (5 items) and disproportionate irritability (1 item). In terms of internal consistency, coefficient alpha for the 6-item OC measure were ranged from 0.79 to 0.82 in the European samples.[217] In terms of discriminate validity, OC score correlated very weakly with other Big Five personality traits except Neuroticism.[218]

Personality psychology is in debate over broad versus specific personality traits. Although more and more specific personality traits have been identified, new personality constructs are suggested to be compared with existing personality traits for a possible common core.[219] Due to theoretical links, OC may overlap with other personality traits related to Type A behaviour – Hostility (negative attitude and mistrust towards others, predisposing a person to anger and aggression) and Neuroticism (stable and pervasive individual differences in the tendency to experience negative emotions). Researchers proposes that OC represents an aspect of Neuroticism manifested in the work context. In empirical studies, OC correlated significantly with Type A behaviour (r= 0.39) and Neuroticism (r= 0.30–0.38).[220,221,222] Neuroticism correlated significantly with Hostility (r= 0.66) and Type A behaviour (r= 0.34).[223,224]

2.4.1 Overcommitment – origins from Type A behaviour

The origins of OC personality are traced to Type A behaviour and PC. In this section, Type A behaviour and related personality traits (Hostility and Neuroticism) are introduced, and their relationships with health outcomes are summarized.

(1) Type A behaviour

Historically, research on the impact of personality on health had a rich and long tradition in medicine and psychology; Type A behaviour has been one of the most influential constructs in the studies regarding psychosocial factors and health. Friedman and Rosenman (1959) firstly described Type A behaviour as an emotion–

action complex characterized by hostility and aggression, sense of time urgency, competitiveness, and ambitiousness.[225] Rosenman et al (1975) conducted the Western Collaborative Group Study in 3,524 employed men aged 39–59 years old, a prospective study with a follow–up period of 8.5 years. The study found that Type A behaviour was strongly associated with the incidence of coronary heart disease (CHD) (ORs= 1.87 in younger group and 1.98 in older group) after adjustment for classical risk factors (diabetes, blood pressure, smoking or blood lipids).[226] Subsequently, these results have been replicated in the Framingham Heart Study and other prospective studies.[227] In the late 1970s, the review panel of US National Heart, Lung and Blood Institute endorsed Type A behaviour as an independent risk factor for CHD.

However, since the late 1980s, several studies failed to show any association between Type A behaviour and CHD. For examples, a longer follow–up of Western Collaborative Group Study observed no association between Type A behaviour and CHD mortality.[228] In the Framingham Heart Study, Type A behaviour was associated with incidence of angina pectoris, but not myocardial infarction or fatal cardiac events.[229] Therefore, researchers suggested that previous evidence on Type A behaviour and CHD should be interpreted more cautiously. Several explanations for inconsistent associations between Type A behaviour and CHD were raised; one main explanation is that the global Type A construct is too broad, and only specific components may be pathogenic to CHD. Hostility has been identified as the “toxic” element in Type A behaviour, as this element can predict CHD most strongly.[230]

(2) Hostility

Hostility is a multi–faceted construct incorporating cognitive (cynicism and negative beliefs about human nature), affective (anger, annoyance, and resentment), and behavioural components (aggression, antagonism and uncooperativeness). These attitudes and cognitions predispose a person to intensive emotion (anger) coupled with physiological arousal, leading to verbal or physical aggression.[231] There

are several widely–used measures of Hostility (e.g. Buss–Durkee scale or Cook–Medley Scale).[232,233] A large number of prospective studies and meta-analyses have supported the association between Hostility and CHD.[234,235] A meta-analysis by Chida and Steptoe found that Hostility was associated with more CHD events in the 25 healthy population studies (hazard ratio= 1.19; 95% CI= 1.05–1.35) and with poor prognosis in the 19 CHD population studies (hazard ratio= 1.24; 95% CI= 1.08–1.42). Notably, the harmful effects of Hostility on CHD events in men were greater than those in women.[236]

There have been several potential pathways via which Hostility might affect the risk of CHD. Firstly, Hostility might simply be a marker for an "inborn structural weakness" of cardiovascular system, which causes both CHD and Hostility. Secondly, Hostility influences the body on a daily basis, forming a pattern of intense responsiveness to physical and mental stressors, which then increases the risk of atherosclerosis.[237] Thirdly, Hostility may have a negative impact on social relationship, resulting in lack of social support.[238] Fourthly, Hostility may be associated with health behaviours – smoking or alcohol consumption.[239] Finally, life course perspective views Hostility as the product of person and environment; low socioeconomic position in childhood and early adulthood can predict high levels of Hostility.[240]

The Edinburgh Artery Study (1991) of 1,592 community-dwelling people was designed to gather information on risk factors of cardiovascular disease, with the measures of Hostility and Big Five personality traits administered simultaneously; the study found that Hostility was strongly associated with Neuroticism.[241] Subsequently, Smith's review focused on the problems about measurements of Hostility, reporting that several items in Cook–Medley Hostility Inventory (e.g. cynicism or social avoidance) overlapped with Neuroticism.[242] Felsten reported that Neuroticism correlated strongly with Hostility measured by Buss–Durkee Hostility Inventory in men (r= 0.66) and women (r= 0.63).[243] Since that time, researchers have recognized that Neuroticism is a personality trait closely related to Hostility and Type A behaviour.

(3) Neuroticism

Neuroticism or negative affectivity, proposed by Eysenck and Eysenck (1964), reflects the stable and pervasive individual differences in the tendency to experience negative emotional states, including anxiety, anger, guilt and distress.[244] High Neuroticism individuals tend to be worried, easily upset, often depressed, and to focus on negative aspects of self, others and the world.[245] This dimension has been an established part of the most widely–used model of Big Five personality traits (Table 2.1). Neuroticism has been assessed by several measures: NEO Five-Factor Inventory or Eysenck Personality Questionnaire.[246,247]

Table 2.1 Big Five personality traits and their dimensions

Personality traits	Dimensions
1. Neuroticism	Anxiety, anger–hostility, depression, self-consciousness, impulsiveness, vulnerability
2. Extraversion	Warmth, gregariousness, assertiveness, activity, excitement-seeking, positive emotions
3. Openness to experience	Fantasy, aesthetics, feelings, actions, ideas, values
4. Agreeableness	Straightforward, trust, altruism, compliance, modesty, and tender mindedness
5. Conscientiousness:	Competence, order, dutifulness, achievement striving, self-discipline, deliberation

An important issue is the conceptualization and measurement of Neuroticism. This global trait includes several more specific characteristics, including depression, anxiety, irritability, anger, or low self-esteem. Scales implying the measurement of specific dimensions are often psychometrically indistinguishable from the measures of broader traits. For instance, individuals with depressive or anxiety disorders scored high on the measures of Neuroticism, and high levels of Neuroticism were associated with increased risk of depressive and anxiety.[248] Those studies on the relationships between Neuroticism and various diseases would involve the effects of undiagnosed depressive or anxiety disorders, and vice versa.[249]

High levels of Neuroticism were associated with adverse psychosocial outcomes, such as higher stress, poor mental health, poor social relationship, poor work performance, counterproductive work behaviour, and occupational injury.[250,251] Also, many prospective studies have showed that Neuroticism predicted a wide range of health problems, including cardiovascular diseases and all–cause mortality.[252,253] Finally, numerous studies have indirectly supported the effects of Neuroticism on health; various measurements of specific dimensions (depression, anxiety or low self–esteem) predicted subsequent CHD,[254] atherosclerosis,[255] diabetes,[256] and all–cause mortality in cohort studies.[257,258]

2.4.2 Empirical studies on Type A behaviour and health behaviours

To my best knowledge, the effects of OC on health behaviours have rarely been reported in empirical studies.[259] Due to theoretical links, evidence for the effects of Type A behaviour and related personality (Neuroticism and Hostility) on health behaviours can be used to partially support the links between OC and health behaviours. I summarise empirical studies on the associations of Type A behaviour with four health behaviours – alcohol drinking, smoking, diet, and physical activity.

(1) Type A behaviour and alcohol drinking

Type A behaviour has received moderate support in relation to alcohol drinking. Friedman and Rosenman found that those with Type A behaviour had higher alcohol consumption than Type B behaviour (a behavioural pattern characterized by absence of Type A behaviour; Type B persons tend to be relaxed and easy–going).[260] Koskenvuo et al reported that Type A persons drank alcohol more frequently than Type B persons in a Finnish cross–sectional study of 11,364 adults.[261] In the US cross–sectional study of 12,866 men, Folsom et al reported that those with Type A behaviour consumed 30% more alcohol and drank more frequently than Type B persons.[262] In

contrast, Glynn et al observed that Type A behaviour was not associated with high alcohol consumption in a cross–sectional study in the US (n= 1,556 men).[263]

Hostility has been found to be associated with high alcohol consumption in numerous large population–based studies. In a Finnish cohort study of 2,125 men aged 42–60 years, Everson et al reported that alcohol consumption significantly mediated the effects of Hostility on mortality and myocardial infarction.[264] Pulkki et al found that Hostility assessed at 12–21 years old predicted the frequency of alcohol use after 9 years of follow-up in a Finnish cohort study (n= 1,219).[265] Siegler et al found that Hostility assessed at late adolescence subsequently predicted high alcohol consumption in adulthood in the US (n= 4,710).[266] Scherwitz et al reported that Hostility was associated with increased alcohol consumption in a cross–sectional study of 5,115 young adults in the US.[267] Whiteman et al observed that Hostility was related to high alcohol consumption in a British cross–sectional study (n= 1,592).[268]

Neuroticism has received moderate support in relation to alcohol consumption. Kuntsche et al found that high Neuroticism was associated with high alcohol consumption in a cross–sectional study of 2,090 university students in Switzerland.[269] Almada et al reported that high Neuroticism was associated with high alcohol consumption in the US cross–sectional study of 1,871 middle-aged men.[270] However, some studies reported negative findings on Neuroticism and alcohol drinking.[271,272] A meta–analysis of 124 studies reported that Neuroticism predicted “emotion–focused coping” to minimize negative emotions via emotional expression, withdrawl or avoidance (r= 0.22–0.41), such as alcohol abuse (r= 0.28).[273] Neuroticism is associated with high rates of stress exposure and intense emotional and physiological reactivity to stress, so they tend to minimize unpleasant arousal via avoidance or drinking. As drinking is only one of maladaptive coping strategies used by Neuroticism, effects of Neuroticism on alcohol drinking might not be very strong.[274]

(2) Type A behaviour and smoking

Type A behaviour has received strong support in relation to smoking. Jenkins et al observed that Type A behaviour was found in 53% of heavy smokers (> 20 cigarettes per day), 47% of light smokers (< 20 cigarettes per day), and 41% of never smokers; Type A behaviour was associated with smoking status after 4 years of follow–up in 2,318 middle–aged men in the US.[275] Shekelle et al found that Type A behaviour was positively correlated with smoking intensity (the number of cigarettes smoked per day) in a cross–sectional study of 4,108 adults in the US, but the magnitude of correlation was not large.[276] In a Finnish population–based cohort study (n= 1,125) with 9 years of follow–up, Pulkki et al reported that Type A behaviour mediated 28.5% and 20.5% of the effects of low education on smoking in men and women, respectively.[277]

Hostility has been found to be associated with smoking in several large population–based studies. Siegler et al found that high Hostility measured in young adulthood predicted the risk of being current smokers after 22 years of follow–up in 4,710 people in the US.[278] Pulkki et al found that high Hostility measured at 12–21 years old predicted smoking intensity after 9 years of follow-up in 1,219 Finnish people.[279] Everson et al found that smoking significantly mediated the effects of Hostility on mortality in a Finnish cohort study of 2,125 middle–aged men.[280] Scherwitz et al observed that high Hostility was associated with a 1.5 times higher prevalence of current smokers in the US cross–sectional study of 5,115 adults.[281] Schrijvers et al found that high Hostility was associated with being current smokers in a cross–sectional study of 3,494 adults in the Netherlands.[282] In a British cross–sectional study (n= 1,592), high Hostility was associated with being current smokers.[283]

Neuroticism has been moderately supported to be associated with smoking. In a British cohort study (n= 5,362), high Neuroticism measured at age 16 was associated with being current smokers in adulthood.[284] Goodwin and Hamilton reported that higher Neuroticism was associated with greater risk of cigarette smoking in the US cross–sectional study of 3,032 adults.[285] In a twin study of 1,551 adults in Australia,

the association between Neuroticism and smoking was explained by genetic and environmental sources of co-variation.[286] Notably, the meta-analysis of 22 studies published between 1972 and 2001 reported that high Neuroticism was associated with an increased likelihood of being current smokers, but the effect size was modest.[287]

(3) Type A behaviour and diet

Type A behaviour has received moderate support in relation to dietary outcomes. In a cross–sectional study in Northern Ireland (n= 551), Barker et al found that Type A behaviour in men had moderate but significant associations with intakes of saturated fat and cholesterol. In women, Type A behaviour had a weak association with sugar intake.[288] In contrast, Gallacher et al found no association between Type A behaviour and fat intake, but Type A behaviour was associated with low fruit/vegetable intake in a British cross–sectional study of 532 middle-aged men.[289] In a cohort study of 10,602 men, Type A behaviour was significantly associated with high consumption of saturated fat, cholesterol, and vegetable in Northern Ireland and France.[290]

Hostility has been found to be associated with dietary outcomes in several studies. For example, Iribarren et al found that higher Hostility was associated with less intakes of polyunsaturated fat after 2 years of follow–up in 3,581 young adults in the US.[291] Scherwitz et al observed that higher Hostility was strongly associated with greater caloric intake in a cross–sectional study of 5,115 young adults in the US.[292] In the US cohort study of 629 adults, Hostility was associated with less likelihood of monitoring and controlling for dietary patterns after 1 year of follow–up.[293] There has been indirect support for the effects of Hostility on diet–related outcomes; for instance, high Hostility predicedt high body–mass index after 22 years in 4,710 young adults in the US.[294]

Neuroticism has been reported to be associated with dietary outcomes in several studies. In the Helsinki Birth Cohort Study (n= 1,681), higher Neuroticism was found to be associated with lower fish and vegetable consumption in women.[295] De Bruijn et al found that the effects of high Neuroticism on low fruit consumption in 405 adults in

the Netherlands.[296] Vollrath et al conducted a cohort study on personality and food consumption in 327 Norwegian children aged 6–12 years old; girls with lower Conscientiousness and higher Neuroticism consumed more sweet drinks, and boys with higher Conscientious and lower Neuroticism consumed more fruits and vegetables.[297] Note that literature has indirectly supported the effects of Neuroticism on diet–related outcomes such as increased body–mass index.[298,299]

(4) Type A behaviour and physical activity

Type A behaviour has generated mixed support in relation to physical activity. Pulkki et al found that Type A behaviour mediated 17.7% of the effects of low education on physical inactivity after 9 years of follow–up in women in a Finnish cohort study (n= 1,125).[300] In contrast, some components of Type A behaviuor subsequently predicted high levels of physical activity in 2,031 young adults from the Young Finns Study.[301] Abbott et al observed that those with Type A behaviour perceived themselves to be more physically active, even though objective estimates of physical activity were not associated with Type A behaviour.[302]

Hostility has received moderate support to predict physical inactivity. Schrijvers et al observed that Hostility was associated with physical inactivity in a cross–sectional study of 3,494 adults in the Netherlands.[303] Maier and James found that greater Hostility was associated with lesser physical activity in 859 college students in the US.[304] In a 9–year follow–up study of 2,125 middle-aged men in the US, physical inactivity was found to mediate the effects of Hostility on mortality.[305] In a 7–year follow–up study of 1,022 adults in the US, physical inactivity was found to significantly mediate the impacts of Hostility on recurrent CVD events.[306] However, there have been several studies with negative findings on Hostility and physical inactivity.[307,308]

Neuroticism has received strong support to be associated with physical inactivity in numerous studies. In a Norwegian population–based cross–sectional study (n= 38,743), Brunes et al found that high Neuroticism was associated with physical

inactivity.[309] In a Dutch population–based survey (n= 19,288), low Neuroticism was found to be associated with high physical activity.[310] Droomers et al found that high Neuroticism was associated with physical inactivity in a Dutch cross–sectional study of 2,598 adults and elders.[311] Tolea et al reported that high Neuroticism predicted low muscle strength in the US cohort study (n= 1,220); physical inactivity partly mediated this association.[312] Notably, a meta-analysis of 33 studies found that high Neuroticism was negatively associated with physical activity (r= –0.11).[313]

In summary, Type A behaviour and related personality (Hostility and Neuroticism) are significant, albeit not strong, predictors for four health behaviours. It is important to note that other personality predictors for health behaviours include: Conscientiousness and Extraversion. Rhodes and Smith conducted a meta-analysis of 33 studies on personality traits and physical activity; the effects of Conscientiousness (r= 0.20) and Extraversion (r= 0.23) on physical activity were slightly stronger than that of Neuroticism (r= –0.11). A meta-analysis of 194 studies reported that Conscientiousness was most consistently associated with all health behaviours; Neuroticism received moderate support to predict all health behaviours.[314]

2.4.3 Overcommitment – origins from perceived control

OC was originated from the concepts of Type A behaviour, which was derived from perceived control (PC) in psychology. Rosenman proposed that Type A persons have higher need for control over environment and tend to perceive lower PC; their response is enhanced coping to assert and maintain control over environment. The concepts of PC and its relationships with health behaviours are introduced below.

(1) Origins of perceived control

Perceived control, also named personal control or sense of control, has emerged broadly from social science and psychology over a century.[315] In social science,

concepts of control appeared in Marx and Durkheim's works. Karl Marx's concept of alienation means that workers lose control of their lives and destinies by being deprived of the right to be director of their actions. Durkheim's description of anomie indicates that society undergoes significant changes with a discrepancy between values commonly possessed and what is actually achievable in daily life, leading to people's feelings of purposelessness and powerlessness.[316]

In psychology, "control" has been one of the most pervasive and enduring ideas across diverse schools. In psychoanalysis, Freud emphasized ego's primary task in reducing conflict between external reality, superego and id. Hartmann (1939) then proposed a conflict–free sphere of ego, acting through cognitive processes to adapt to environment.[317] This idea of an autonomous ego was adopted by Robert White (1959), who proposed that people have "effectance motivation" to be effective in dealing with their environment.[318] Social psychologist Lewin (1936) argued that people strive to control the world rather than just react to it.[319] Humanistic psychologist, Deci and Ryan (1985), proposed autonomy and self-determination that described individual's fundamental motivation to act as a causal agent on environment.[320]

While early psychological theories emphasized control as a motivation to master environment, since the 1960s cognitive psychology directed researchers to focus on cognitive processes (belief, expectancy, or perception) that describe how individuals interact with environment.[321] Expectancy is the judgment about the likelihood that a given behaviour will attain the outcome; as a step away from learning perspectives to social–cognitive perspectives, expectancy emphasizes mental representation in this process. People think over available evidence (e.g. past outcomes or current situations) and judge the likelihood of future outcomes; this expectancy then influences success or failure of outcomes.[322] Rotter (1966) proposed the expectancy – locus of control (LOC), the belief about one's behaviour over the outcomes. Bandura (1977) proposed the expectancy – self-efficacy, the belief of one's ability to perform the behaviour.[323] Self-efficacy and LOC are the two components of PC.

(2) Definition and measurement of perceived control

Perceived control was defined by Skinner (1988) as: “the extent to which one can intentionally produce desired outcomes and prevent undesired ones”.[324] PC is viewed as a “self–outcome relation” that was integrated from a “self–behaviour relation” (self-efficacy) and a “behaviour–outcome relation” (LOC).[325] Thus, self-efficacy and LOC are viewed as the two components of PC.[326] Skinner’s works for PC provided an integrative framework to organize heterogeneous constructs in the concepts of control, some of which were often used in epidemiological and health research (Table 2.2). The heterogeneity among these constructs interfered with the accumulation of research findings.[327] Skinner’s integrative framework can be used to locate parallel constructs; for instance, if a construct is defined in a way as a behaviour–outcome relation, its associations with other measurements would be similar to LOC.

Table 2.2 Different constructs in the concepts of control[328,329,330,331,332]

Construct	Definitions
Locus of control (LOC) (Rotter 1966)	The belief about contingency between one’s action and actual outcome. Internal LOC refers to the conviction that outcomes are contingent upon one’s own behaviour, whereas external LOC refers to the conviction that outcomes are not contingent upon one’s action, but upon chance or powerful others.
Self-efficacy (Bandura 1977)	The belief in one’s capabilities to successfully execute the behaviour required to produce certain outcomes.
Mastery (Pearlin & Schooler 1978)	A perception that reflects one’s personal control over life outcomes. The extent to which one regards one’s life chances as being under one’s own control in contrast to being fatalistically ruled.
Learned helplessness (Overmeier & Seligman 1967)	The acquisition of expectancy is based on interaction between exposure and response to it. Learned helplessness occurs when an individual has learned that there is no relationship between his responses and outcome.
Self-control (Rosenbaum et al, 1982)	Self-control refers to the ability to monitor and inhibit one's own emotions, thoughts, and behaviours. Primary control involves taking action to get desired outcomes, and secondary control refers to changing oneself to adjust to the environment.
Sense of coherence (Antonovsky 1993)	A global orientation that indicates the extent to which one has a pervasive, enduring and dynamic feeling of confidence that one's internal and external environments are predictable.

The heterogeneous concepts of PC have generated various measurements.[333,334]

In health research, the most widely used measurement for PC is Lachman and Weaver's General Perceived Control Scale based on the concepts of mastery and PC. Pearlin and Schooler's concept of mastery is similar to Skinner's definition of PC, as mastery (Table 2.2) also indicates "self–outcome relation".[335] General Perceived Control scale has 2 dimensions: (1) personal mastery represents one's sense of effectiveness or efficacy in carrying out goals; (2) perceived constraint represents one's beliefs of the obstacles or factors beyond one's control that interfere with reaching goals.[336] This thesis will adopt this measure for PC, because it has been validated in many empirical studies including CEE populations (for more details, please see Section 2.2.2). For example, Bobak et al conducted a cross–sectional survey (n= 5,330) in 7 CEE countries; they found that lower PC was associated with poor self–rated health.[337] Lachman and colleagues found that higher PC was associated with better health status, fewer chronic diseases, fewer functional limitations, and more regular exercise in several large–sample cohort studies in the US.[338,339,340]

Due to the evidence supporting the effects of PC on health, epidemiologist Leonard Syme (1989) suggested that PC provides a parsimonious concept to understand why higher rates of disease are found among seemingly unrelated factors: poor social support, life events, migration, or low control at work. PC can transcend research boundaries to develop interdisciplinary integration in experimental, clinical and epidemiological studies.[341] In experimental studies, PC has been reported to suppress autonomic arousal, cardiovascular activation, stress hormone release, and pain perception in animals and humans.[342] In clinical and epidemiological research, as mentioned earlier, empirical evidence showed that higher PC predicted lower morbidity and mortality. It is suggested that PC can influence health directly by psychobiological processes and indirectly via health behaviours.

2.4.4 Empirical studies on perceived control and health behaviours

PC has been widely applied in predicting health behaviours in psychology. Skinner suggested that when people perceive high control, they initiate action, exert effort, try hard, and persist in the face of failures. When people perceive control as impossible, they withdraw, escape, or become fearful and depressed.[343] PC was integrated into Transactional Model of Stress by Skinner and Wellborn; individuals' appraisals of whether the stressor is controllable and whether their resources are adequate to exercise control subsequently influence coping. Appraisals of high control lead to active coping, such as information seeking, planning, efforts, and direct action. Appraisals of low control result in escape, passivity, and risky health behaviours.[344]

Bandura proposed potential mechanisms via which self-efficacy might affect health behaviours: (1) Cognition: those with high self-efficacy tend to anticipate success scenarios and to create effective means for exercising control over actions (e.g. drinking abstinence or dietary choices). (2) Motivation: those with high self-efficacy can motivate themselves and guide their actions anticipatorily through forethought; their expectancies for positive outcomes are high, and their motivations to execute actions are strong. (3) Affection: Self-efficacy influences whether a stressor is cognitively constructed in a way good for emotion. Self-efficacy regulates emotional states by supporting effective actions. (4) Selection processes: self-efficacy affects the types of environments or activities (e.g. drinking occasions) people choose to undertake or to avoid.[345]

Bandura's theory of self-efficacy has a profound impact on Social Cognitive Models (SCMs). Glanz et al conducted reviews of publications from 1986 to 2005 and found that SCMs are the most commonly used theories in predicting health behaviours at intrapersonal level.[346] SCMs specify cognitive and affective factors as proximal determinants of health behaviours based on the assumptions of self–regulation involving cognitive evaluation of beliefs, goal setting, and ongoing evaluation of goal–

directed behaviours.[347] Socio–cognitive constructs are assumed to mediate the effects of social determinants on health behaviours, and they are suggested to be more modifiable than personality traits. Notably, all SCM models have a common construct – self-efficacy, belief that one can successfully perform the behaviour.[348]

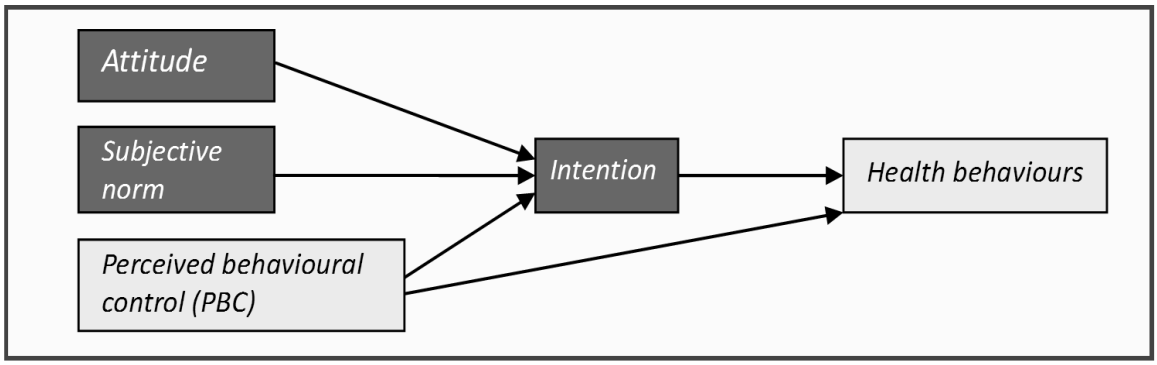
The roles of self-efficacy in SCMs are summarized. Health Belief Model proposes that health behaviours are determined by two cognitions: perceptions of illness threat and evaluation of behaviours to counteract this threat. Self-efficacy and intention were added to the model to improve predictive power.[349] Protection Motivation Theory suggests that primary determinants of performing health behaviours are threat appraisal and coping appraisal. Coping appraisals include self-efficacy and behaviour–outcome expectancy (one's expectancy that carrying out the behaviour can remove the threat). Social Cognitive Theory suggests that behaviour is determined by intention to perform the behaviour, behaviour–outcome expectancy, and self-efficacy. Trans-theoretical Model of Change proposes that different cognitive factors are important at different stages: pre-contemplation (not thinking about change), contemplation (aware of need to change), preparation, action, and maintenance; one major factor influencing stage transitions is self-efficacy.[350]

Theory of Planned Behaviour (TPB) is the most widely accepted SCM in current literature. It proposes that the proximal determinant of behaviour is intention, which is predicted by attitude (beliefs about perceived consequences of the behaviour), subjective norm (perceptions of whether salient groups or others think the person should perform the behaviour), and perceived behavioural control (PBC; perception of the extent to which performance of the behaviour is easy or difficult). In particular, PBC was derived from the concept of self-efficacy (Figure 2.7).[351] TPB has received strong support in predicting health behaviours. Armitage and Conner conducted a meta–analysis based on 185 studies; PBC significantly predicted health behaviours directly or indirectly via intention, and PBC emerged as the strongest predictor of intention.[352]

In summary, all SCMs emphasize pivotal roles of the two components of PC (self-

efficacy and behaviour–outcome expectancy) in predicting health behaviours. In the following review, PC and its components (self-efficacy, LOC, and PBC derived from self-efficacy) have been extensively supported to predict four health behaviours – alcohol drinking, smoking, diet, and physical activity.

Figure 2.7 Theoretical framework of Theory of Planned Behaviour. Source: Adapted from Ajzen (2002).



(1) Perceived control and alcohol drinking

Lower levels of PC and its components have received empirical support to predict high alcohol consumption, binge drinking and problem drinking in a wide range of Western populations from college students to elders. In terms of PC, Perlman et al observed that high PC was related to low alcohol consumption in a Russian cross–sectional study of 1,599 men.[353] Troein et al showed that low PC was associated with high alcohol consumption in a Swedish cross–sectional study of 453 men.[354]

In terms of PC's components, low self-efficacy has been found to predict problem drinking, binge drinking, and relapse of alcohol abuse in college students and adults (n= 273 to 359) in cohort studies from US, UK and Australia.[355,356,357] Grembowski et al found that low self-efficacy was related to heavy alcohol consumption in a cross–sectional study of 2,524 American elders.[358] In terms of TPB, low PBC was reported to predict high alcohol consumption and binge drinking in college students (n= 289 to 513) in cohort studies from UK and Australia.[359,360] A randomized controlled study

showed that TPB–based intervention (e.g. to promote PBC) reduced binge drinking after 1 month in 467 college students from Estonia, Finland and UK.[361]

(2) Perceived control and smoking

Lower levels of PC and its components (self-efficacy, LOC and PBC) have been found to predict more smoking status as current smokers, higher smoking intensity (number of cigarettes per day), and less smoking cessation from adolescents to adults. In terms of PC, Sigrun et al showed that higher PC at age 14 predicted less smoking intensity at age 17 in a longitudinal study in Iceland (n= 1,293 adolescents).[362] Devogli et al found that low PC was associated with more current smokers in a cross–sectional study in Italy (n= 4,002 adults).[363] Low PC was associated with high smoking intensity in a Swedish cross–sectional study of 453 middle-aged men.[364]

In terms of PC's components, Diclemente et al reported that lower self-efficacy predicted more current smokers and less smoking cessation after 5 months of follow–up in 957 American adults.[365] There are intervention studies in Norway and US (n= 244 to 642), showing that increased self-efficacy can lead to smoking cessation.[366,367] Low self-efficacy and external LOC were associated with current smokers in a cross–sectional study of 885 Korean adolescents.[368] Bennett et al found that external LOC was associated with current smokers in a cross–sectional study of 11,401 British adults.[369] With regard to TPB, lower levels of PBC were reported to predict more current smokers in several cohort studies (n= 346, 4079 and 14,434) from Canada, Netherlands and China.[370,371,372] Moan and Rise observed that high levels of PBC predicted smoking cessation after 6 months of follow–up in 698 adults in Norway.[373]

(3) Perceived control and diet

Lower levels of PC and its components (self-efficacy, LOC or PBC) have been repeatedly reported to predict less consumption of fruit/vegetable and more consumption of saturated fat and sugar in Western populations from students to elders.

In a British cross–sectional study (n= 372), Barker et al found that low PC was associated with unhealthy dietary patterns, such as less consumption of vegetables, wholegrain bread and vegetarian food, and more consumption of chips, meat, crisps, snacks, white bread, and sugar.[374]

In terms of PC's components, a review of 14 prospective and 21 cross–sectional studies found that lower self-efficacy predicted less intakes of fruit/vegetable and more intakes saturated fat and sugar.[375] Grembowski el al reported that low self-efficacy was associated with high intakes of saturated fat in the US cross–sectional study of 2,524 elders.[376] There have been large–sample cross–sectional studies (n= 7,115 to 13,045) showing that Internal LOC was related to more fruit/vegetable and less saturated fat/sugar intakes across 18 Europe countries.[377,378] Finally, low levels of PBC in TPB predicted low intake of fruit/vegetable and high intake of saturated fat in adults in cohort studies from the UK (n= 413) and the US (n= 609).[379,380]

(4) Perceived control and physical activity

Low levels of PC and its components have been found to predict physical inactivity from adolescents to adults across Western populations. In terms of PC, Lachman and colleagues found that higher PC was associated with more time in vigorous to moderate levels of physical activity in adults in two large–sample cross–sectional studies (n= 3,848 and 4,242) in the US.[381,382]

In terms of PC's components, Hagger et al conducted a meta–analysis of 72 studies and found that self-efficacy and PBC in TPB are the strongest determinants for intention and behaviour of physical activity.[383] Several longitudinal studies (n= 328 to 389) showed that higher self-efficacy predicted more time in physical activity in college students and adults from US, Australia and Finland.[384,385,386] Steptoe and Wardle found that internal LOC was associated with high levels of physical activity in a cross–sectional study of 7,115 students across 18 European countries.[387] Rhodes

and Courneya reported that PBC predicted intention and behaviour of physical activity after 2 weeks in 300 students and 272 cancer survivors in Canada.[388]

There are some limitations for the interpretation of existing literature. While most evidence has supported the effects of PC or its components on health behaviours, some studies reported negative findings.[389,390] Godin and Kok's meta-analysis found that PBC contributed an additional 12% of variance to predict health behaviours. The effects of PC on health behaviours appear significant, but other psychosocial factors (e.g. social norm, attitude, or social support) may confound the relationship between PC and health behaviours.[391] For example, Perlman et al found the effect of PC on health behaviours in Russian populations was weaker than that of Western European populations; the findings were explained by the differences in social norm and attitude embedded in Russian contexts.[392] Research regarding the effects of PC on health behaviours should take into account other psychosocial factors.

2.4.5 Relationship between overcommitment, perceived control and health behaviours

Siegrist initially developed OC as need for control. High OC persons have higher need for control over environment and tend to perceive lower PC; their response is enhanced coping to assert control over environment. In this section, the potential relation between OC, PC and health behaviours is elaborated based on two approaches in dispositional perspective on personality (need and motive; personality trait). Dispositional perspective proposes that people display consistency in actions, thoughts and feelings. Motives are fundamental desires and personality traits channel how these desires are expressed; they are different but complementary.[393]

(1) The approach of need and motive

In this approach, a need is an unsatisfactory internal condition that motivates

behaviour; a need is an internal force that determines how people seek out or respond to the environment. A motive is a cognitive–affective cluster organized around readiness for a preferred experience or goal; a motive takes the underlying need and move it a step closer to the behaviour. Henry Murray (1938) proposed that some needs are based on biological nature (e.g. needs for food or water), while others are based on psychological makeup (e.g. needs for achievement, autonomy, or affiliation).[394]

Skinner (1995) proposed a meta-theory to explain the widespread effects of PC across life domains: PC reflects the fundamental need for control in all humans.[395] This assumption is based on the accumulating literature in psychology. White (1959) proposed effectance motivation that all people have an inborn need to build an increased competence to deal with environment.[396] Piaget (1976) proposed that infants enjoy and detect contingencies in environment as soon as they have motor control over behaviours. Children and adults have their needs for competence met in playgrounds and work, respectively.[397] DeCharms (1968), Deci and Ryan (1985) described a need for autonomy – the intrinsic motivation to be the origin of one's own behaviour and to choose one's course of action.[398,399]

Greenberger and Strasser proposed a dynamic model of PC based on reactance theory, learned helplessness and two–process model of PC.[400,401] Cognitive appraisal for PC is suggested as a function of two dimensions:

PC = The amount of control possessed / The amount of control desired

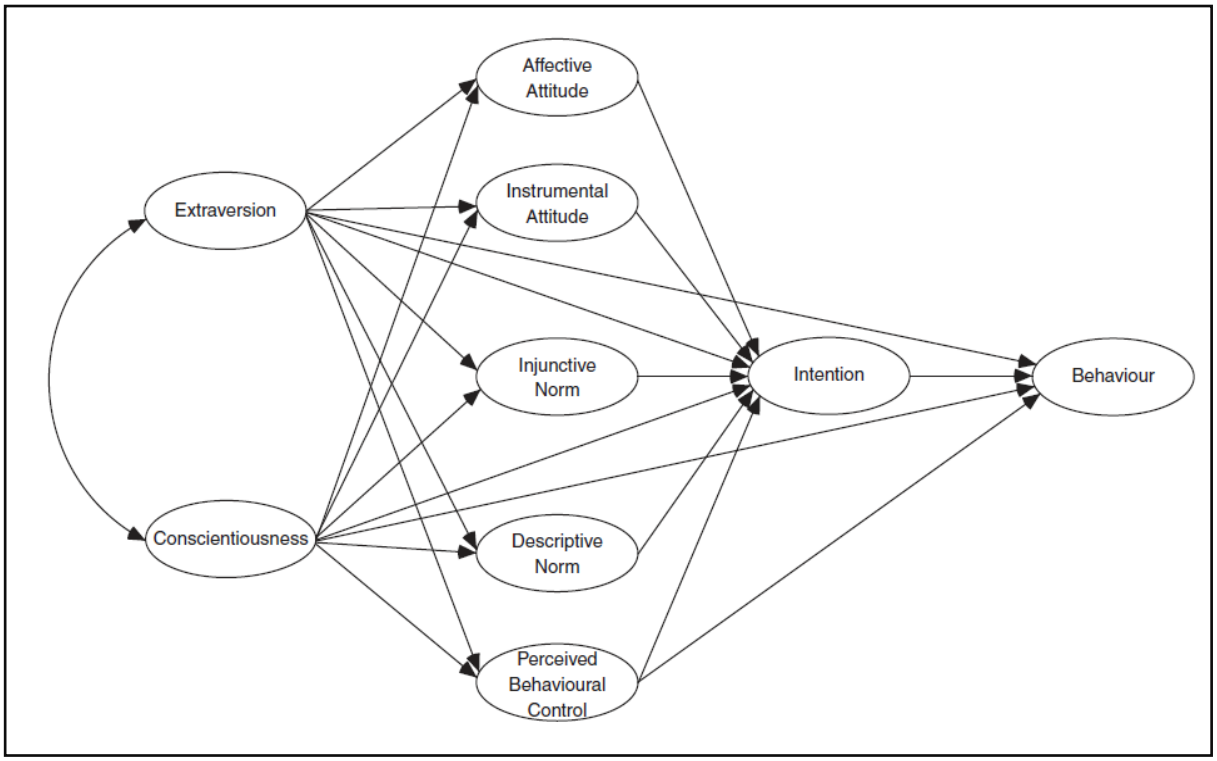
The amount of control desired is viewed as "need for control". Given the same amount of control possessed, the higher need for control a person has, the lower PC one perceives.[402] If individuals perceive lower control than they need, they are motivated to seek control by three responses: (1) Direct reaction: individuals attempt to restore control directly at environmental source. (2) Indirect reaction: they attempt to adapt to environment and change themselves by cognitive adjustment or action; for example, they may drink alcohol to satisfy feeling of control immediately. (3) Learned helplessness: after failure in direct and indirect reactions, the only way is not trying. In

short, high need for control (OC) might decrease one's PC, which might subsequently affect risky health behaviours.

(2) The approach of personality trait

By this approach, it is plausible to suggest that PC can mediate the effects of OC personality on health behaviours. In health psychology, numerous studies have adopted the analytical framework with a hierarchical structure, in which the effects of higher–order personality traits (e.g. Five–Factor Model such as Neuroticism) on health behaviours are mediated by lower–order, socio–cognitive constructs (e.g. self-efficacy or PBC in TPB).[403, 404, 405] For examples, McEachan et al used the hierarchical framework in which PBC and TPB variables mediated the effects of personality traits on intention and behaviour of physical activity (Figure 2.8).[406]

Figure 2.8 Perceived behavioural control mediated the effects of personality traits on physical activity. Source: McEachan, Sutton and Myers (2010).



The personality traits can clarify which personality factors predict health outcomes, but do less in describing how these factors are associated with cognitive, emotional, and behavioral processes that affect health outcomes. The Five–Factor Model focuses on the structure of personality that people "have", rather than personality processes that people "do". In contrast, social–cognitive constructs can provide a more active and specific process account of individual differences that complements the broader and more static personality traits. Socio-cognitive constructs are considered mediating pathways for the effects of personality traits on health outcomes; they may provide better prediction for behaviours and more modifiable targets for interventions.[407]

A similar hierarchical relationship was proposed in Transactional Model of Stress. Primary appraisal is where the individual evaluates and gives personal meaning to a stressor, and considers the significance of "what is at stake" in terms of harm, threat, loss or challenge. Secondary appraisal addresses the question "what can I do about it" by evaluating one's ability to change the situation and to manage emotional reaction (e.g. PC or self-efficacy); it is where the individual evaluates the availability of coping options and resources to deal with the stressor.[408] Higher–order personality traits are suggested to influence primary appraisal, secondary appraisal (PC) and coping, which then affect health behaviours (for more details, see Section 2.6).[409]

Section 2.4 is summarized. First, OC was originated from Type A behaviour and highly correlated with Hostility and Neuroticism, all of which have received empirical support to predict health behaviours. Second, Type A behaviour was derived from PC; PC has also received strong support to predict health behaviours. Similar to Type A behaviour, high OC persons may have higher need for control and tend to perceive lower PC, thereby engaging in risky health behaviours.[410] Despite little evidence on the effects of OC on health behaviours, the literature on the effects of Type A behaviour and PC on health behaviours is used to partially support these relationships.

2.5 Overcommitment, Effort–Reward Imbalance, and Health Behaviours

In the ERI model, Siegrist proposed the personality construct of overcommitment (OC) which describes individual attitudes, behaviours and emotions reflecting excessive work–related striving and high need for approval and esteem. Siegrist proposed 3 hypotheses for the ERI model: (1) Extrinsic ERI hypothesis: high effort and low reward lead to adverse health outcomes. (2) OC hypothesis: a high level of OC can increase risks of adverse health outcomes; there are main effects of OC on adverse health outcomes. (3) ERI x OC interaction hypothesis: those who are characterized by both condition (1) and (2) have even higher risks of adverse health outcomes; there are modifying roles of OC in ERI–outcome relationship.[411]

Current literature has been inconsistent on potential roles of OC in ERI–outcome relationship; however, research on the influence of personality on health has been accumulated in interrelated fields of behavioral medicine, health psychology and psychosomatic medicine since Friedman and Rosenman's Type A behaviour (1959). Compared to current understanding on diverse roles of personality in stress–outcome processes, original assumption of OC (main or modifying effects) appears relatively simple. To gauge potential roles of OC in ERI–outcome relationship, it would be helpful to review a wider range of literature regarding OC–related personality traits (Type A behaviour, Neuroticism and Hostility). In the following sections, potential roles of OC in ERI–outcome relationship are discussed in detail.

2.5.1 Modifying or main effect of OC in ERI–outcome relationship

Main effect implies that two or more predictor variables (e.g. ERI and OC) contribute independently to explaining variance in an outcome. Modifying effect (effect modification) means that the magnitude and direction of the effect of a predictor (e.g. ERI) on an outcome depends on the level of another predictor (e.g. OC). There are different types of modifying effects. For example, "buffering" implies that an adaptive

personality trait protects people from adverse effects of risk exposure. On the other hand, “vulnerability” implies that a maladaptive personality trait combined with a risk factor predict a disproportionately adverse outcome compared to the additive effect. Vulnerability is the suggested mechanism for the interaction between OC and ERI.

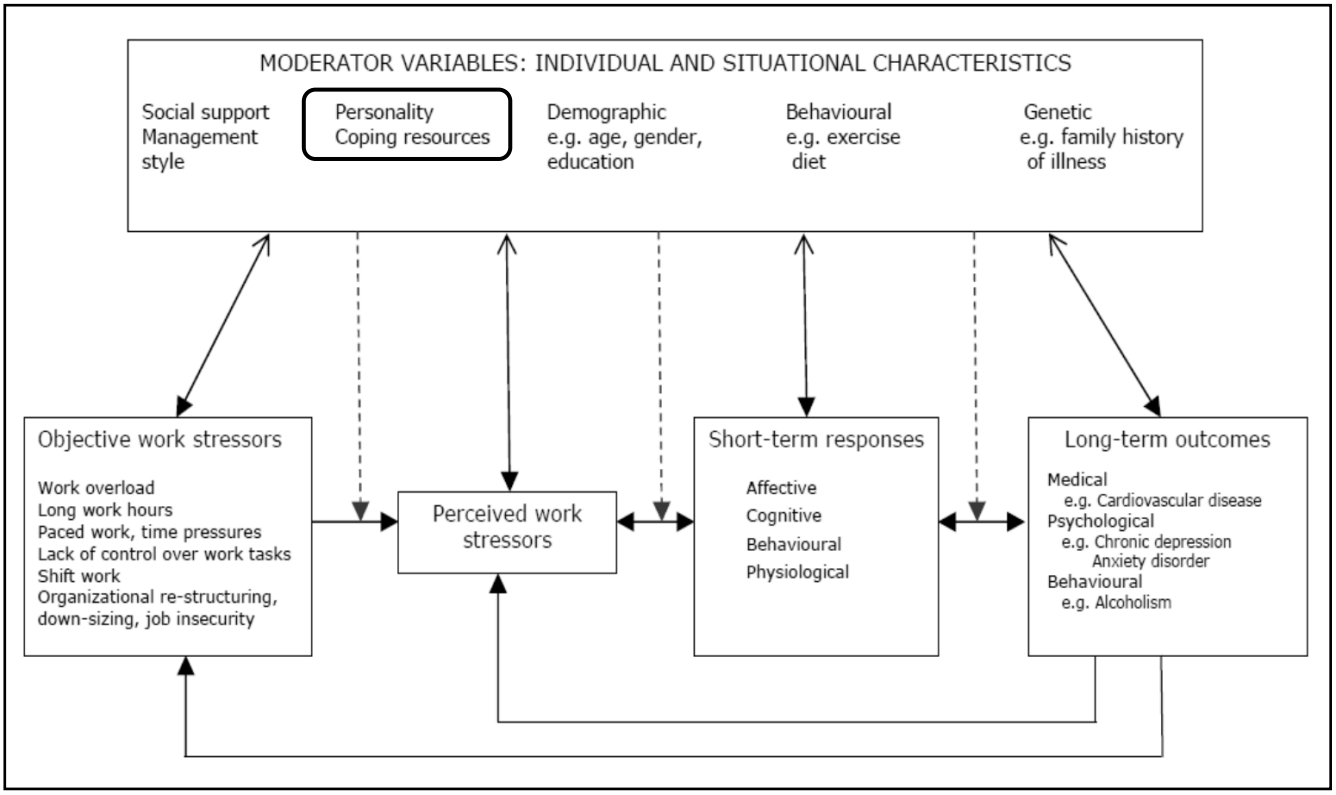
Van Vegchel et al reviewed 45 studies on the ERI model published from 1986 to 2003, including outcomes of physical health (e.g. cardiovascular diseases), psychosomatic health (e.g. depression), behaviour (e.g. sickness absence), and job–related well–being (e.g. burnout). First, the review found that extrinsic ERI hypothesis was strongly supported in 48 out of 52 studies; some studies included several outcomes and were counted twice. Second, OC hypothesis was examined in 27 of 52 studies, and “main effects” of OC on outcomes were supported in 17 out of 27 studies (63%). Third, OC x ERI interaction hypothesis was examined in only 12 of 52 studies, and “modifying roles” of OC were supported in only 3 out of 12 studies (25%).[412] As interaction hypothesis was rarely examined, they suggested that strong conclusions regarding modifying role of OC cannot be made. This review also noted that the potential roles of OC in the relationship between ERI and health–behaviour outcomes have not been tested.

Parkes reviewed 33 longitudinal studies from 2000 to 2009 on the relationships between work stress and personality constructs (e.g. OC, Neuroticism or Hostility).[413] First, “main effects” of OC were supported in 7 out of 8 studies (88%). Among men, high OC was consistently predictive of adverse health outcomes, including depression, anxiety, poor subjective health, and CHD; the reported risk ratios were generally moderate (1.5–2.0).[414,415] Among women, the effects of OC were less consistent. Main effects of Neuroticism were reported in 9 out of 11 studies (82%); main effects of Hostility were supported in 2 out of 2 studies. Second, “modifying effects” of OC were not supported in 2 out of 2 studies.[416,417] In contrast, modifying effects of Neuroticism and Hostility were supported in 4 out of 5 studies; those with higher levels of Neuroticism or Hostility were more vulnerable to work stress.[418,419,420]

The reviews by Van Vegchel et al and Parkes are summarized. First, most studies have supported “main effects” of OC and related personality traits; however, this evidence might not really confirm main effects of OC (OC and ERI contribute independently to explaining variance in an outcome), as other two possibilities had not been tested: antecedent or mediator roles of OC in ERI–outcome relationship. For mediation analyses, confirmation of the effect of OC on outcome is merely a first step for OC–ERI–outcome (OC as antecedent) or ERI–OC–outcome (OC as mediator) causal chains.[421] Thus, previous evidence for “main effects” of OC might partially support or at least not exclude other two possibilities – antecedent or mediator. Second, modifying role of OC has not been supported by the limited amount of literature. Failure to consider interaction may lead to this modifying role remaining hidden in most studies; more research to test modifying role is needed before drawing any conclusion.

As mentioned before, research on the influence of personality on health has been accumulated in psychology and medicine. Compared to current understanding on diverse roles of personality in stress processes, original assumption of the roles of OC (main or modifying effect) appears relatively simple. A more sophisticated model – the Michigan model has been particularly influential in guiding research into the combined effects of personality constructs and work stress. Objective work environments influence subjective perceptions of work stress, which affect short–term psycho–biological responses, leading to long–term health problems. The influence of personality can operate at several points in the stress process, including modifying, mediator, bidirectional or direct effects (Figure 2.9).[422]

Figure 2.9 The Michigan model describes individual and situational factors affecting the process of work stress. Source: Israel, et al (1996).[423]



The Michigan model attempted to capture potentially diverse roles of personality in work–stress processes. For example, if personality affects a person's perceptions to objective work stressors or if personality creates one's objective work stressors, personality is suggested to be an antecedent in the effects of work stress on outcomes. The model also incorporates bi–directional pathways and feedback loops. For example, if objective or perceived work stressors would affect personality which then influences health, personality is suggested to be a mediator in the effects of work stress on outcomes. Based on the framework provided by the Michigan model, four possible roles of personality can be proposed: modifying, antecedent, mediator, or direct effect. In this thesis, I attempt to test these four potential roles of OC in ERI–outcome relationship.

2.5.2 Antecedent role of OC in ERI–outcome relationship

Antecedent role of OC (OC influences ERI that then affects outcomes) has ever been supported by theoretical explanations and empirical studies. Indeed, Siegrist implied the possibility of antecedent role of OC in ERI–outcome relationship; individuals with high OC might expose themselves more often to high demands (efforts) at work, or they exaggerate their efforts beyond what is formally needed, thereby resulting in continued imbalance between high effort and low reward.[424] In contrast, individuals with low OC tend to reduce their efforts or change jobs in order to avoid effort–reward imbalance. OC might explain the duration of exposure to ERI work stress. Moreover, several researchers who investigated the ERI model suggested testing the possibility of antecedent role of OC in future research.[425]

In terms of psychological theories, personality can influence work stress via several mechanisms: (1) Stressor creation: high Neuroticism individuals may create objective work stressors for themselves by provoking interpersonal conflicts or poor work performance. High Hostility persons might have antagonistic behaviours which elicit negative behaviours from others. (2) Perception: personality influences one's perception to objective work environments. High Neuroticism individuals tend to perceive their jobs as having high levels of stressors. High Hostility persons tend to perceive threat or hostile intent from others. (3) Self selection: Type A persons may select themselves into highly competitive jobs, because they tend to set task goals too high for their abilities, leading to more failures and dissatisfaction. High Neuroticism people choose less complex jobs or they are less attractive candidates for better jobs. (4) Reaction: high Neuroticism people react exaggeratedly to work stressors in their psycho–biological processes and health behaviours; they tend to use emotion–focused coping, such as alcohol drinking.[426,427]

In terms of empirical evidence, antecedent role of OC–related personality in work stress–outcome relationship has been partially supported (in particular the effect from

personality traits to work stress). For example, Hintsa et al reported that specific dimensions of Type A behaviour (high aggression, hard-driving, and time urgency) measured at adolescence subsequently predicted both high ER ratio and high job strain at adulthood in the Cardiovascular Risk in Young Finns study (n= 752).[428] Their team in University of Helsinki also found that high Neuroticism measured at adolescence predicted both high ER ratio and high job strain after 15 years (n= 621); Neuroticism predicted low control (β= −0.129, p= 0.012), high job strain (β= 0.337, p= 0.001), and low rewards (β= −0.195, p= 0.001).[429,430]

With regards to empirical studies regarding the effect of OC personality on work stress, Allisey et al found that high OC was associated with high effort (r= 0.40) and low reward (r= –0.31) in an Australian cross–sectional study (n= 897).[431] Rennesund and Saksvik reported that high OC was associated with high job strain in a cross–sectional study in Norway (n= 924).[432] In addition, there is a growing body of evidence showing that personality traits influence the way people perceive environmental stressors (e.g. daily stressors) and subsequent responses to stressors in various experimental and epidemiological studies.[433,434]

Note that the above studies only examined the pathway from OC (or related personality traits) to perceived work stress (ERI or job strain), but the pathway from perceived work stress to outcomes has not been tested simultaneously. Nevertheless, there have been many studies supporting the effects of work stress on health outcomes (Section 2.3.3). Thus, it is of value to examine the causal path “OC–work stress–outcome” simultaneously in a longitudinal study.

2.5.3 Mediator role of OC in ERI–outcome relationship

Mediator role of OC (ERI influences OC that then affects outcomes) appears possible based on theoretical explanations and empirical studies. Personality is defined by Carver and Scheier (2000) as: “a dynamic organisation, inside the person,

of psychophysical systems that create a person's characteristic patterns of behaviours, thoughts and feelings". This definition attempts to integrate diverse perspectives in psychology. Characteristic patterns suggest continuity and consistency uniquely identified in an individual; dispositional, biological, and psychoanalytic perspectives argue that personality traits are biologically based "temperaments" not susceptible to influence of environments (e.g. work stress) and do not change over time.[435] The perspectives have been challenged by current literature showing that personality traits can change over time. Thus, personality is now defined as a dynamic organisation which implies ongoing readjustment and adaptation in an individual; social learning, cognitive, and socio–cognitive perspectives view personality as accumulated sets of thoughts and behaviours which are learned from or changed by environments.[436]

Continuity and change are often indexed by correlation between personality scores across two time points (e.g. test-retest correlation). The meta-analysis of 92 longitudinal studies reported that personality traits (measured by Big Five personality traits) change moderately before age 30 (test-retest correlation increase from 0.41 at childhood to 0.55 at age 30) and become increasingly stable but still change mildly across adulthood (test-retest correlation increase from 0.55 at age 30 to 0.70 at age 50).[437] The most remarkable changes occur in young adulthood involving more life-changing roles (e.g. a new job) and identity decisions than any other period, but modest changes continue into middle–aged (40–60 years old) and older populations (> 60 years old). Note that personality traits can change over time, but they are more stable than other psychological constructs except intelligence. In addition, literature has showed that personality traits can be changed by stressors from environments and uncontrollable situations (e.g. low social position or chronic diseases). For example, the onset of heart disease, diabetes, hypertension, and cancer can predict subsequent changes in Big Five personality traits in a meta-analysis of three US cohort studies (n= 17,493; mean age= 55.8 years).[438]

There have been several empirical studies supporting that psychosocial work

conditions predicted changes in OC–related personality (Neuroticism). A 3–year longitudinal study in the Netherland (n= 576) found that perceived work stress and life satisfaction can predict moderate changes in personality traits in adults (average age 43.9 and 41.7 years for men and women); positive work experience was related to personality maturation (e.g. decreased levels of Neuroticism).[439] An 8–year longitudinal study in the US (n= 1,130) reported that as work satisfaction increased, the levels of Neuroticism decreased.[440] Robert et al reported that de-investment in work (counterproductive behaviours such as fighting with co-workers or breaking safety rules) was associated with increased levels of Neuroticism in an 8–year longitudinal study of 907 young adults in New Zealand.[441]

In particular, empirical studies have found that OC personality was changed by work environments. DeJonge et al tested the stability of ERI constructs over time in a Dutch cohort study (n= 650); they reported that test–retest reliability for OC scale was 0.53 over 1–year interval and 0.45 over 2–year interval.[442] Tsutsumi et al found that during 1 year of organisational changes, OC scale changed significantly and moderately in 544 Japanese employees; the magnitude of changes in OC scale was less than that in situation–specific components in the ERI model (effort and reward).[443]

Some limitations should be noted in the above studies. First, the aforementioned studies only examined the pathway from work conditions to OC–related personality, but the pathway from OC to outcomes has not been tested simultaneously. However, many studies have supported the effects of OC–related personality on health behaviours (Section 2.4.2). It is of value to examine the causal path “work stress–OC–outcome” simultaneously in a cohort study. Second, the above studies measured exposure factors in work conditions, but work stress has not been measured by the ERI or DC models. Third, although OC personality may be changed by work stress, the magnitude of personality change is expected to be small if the samples come from middle–aged (40–60 years old) and older populations (> 60 years old).

2.5.4 Reciprocal relationship between OC and ERI across life–course

Antecedent (Section 2.5.2) and mediator (Section 2.5.3) roles of OC in ERI–outcome relationship may coexist. To fully elucidate how personality traits and stress processes interact over time, there is a need for researchers to adopt the life course approach, in which personality and (work) environments might have "bi–directional causal relationship" across life span.[444,445] Social environments in childhood (e.g. rearing styles or learning experiences) and adulthood (e.g. work environments) might alter an individual's personality traits. In contrast, personality traits may shape mastery of educational and work tasks (e.g. occupational attainment or job performance), cultivation of social relationship, and maintenance of physical and mental health (e.g. responses to stressors); thus, personality may influence an individual to select, encounter and create different environmental stressors.[446,447]

In empirical studies, Roberts et al found that personality traits measured at age 18 predicted objective and subjective work experiences at age 26; those with high Neuroticism at age 18 experienced difficult transitions into employment, occupied lower prestige jobs, were less satisfied with jobs, and reported financial difficulties at age 26. In contrast, work experiences were related to changes in personality traits between 18 and 26 years old; higher occupational status, more satisfying jobs, and having financial security at age 18 were associated with decreased levels in Neuroticism at 26.[448] Sutin et al showed that career success (e.g. more job satisfaction or higher incomes) predicted decreased levels of Neuroticism after 10 years; personality traits predicted changes in career success after 10 years.[449] Note that the above evidence comes from the samples in their young adulthood.

Importantly, Sutin and Costa adopted a longitudinal cross–lagged analysis in the US (n= 722) and found that Big Five personality traits have significant effects on work stress defined by the DC model; low levels of Neuroticism were associated with increases in job control and decreases in demands after 10 years. However, work

stress (job strain) was found to have only small effects on personality traits after 10 years.[450] The results may be somewhat explained by the middle–aged sample (mean age 52.3 years old at wave 2), as the meta-analysis of 92 longitudinal studies reported that personality traits become increasingly stable across adulthood (test–retest correlation 0.55 at age 30 and 0.70 at age 50).[451] This study is particularly relevant to this thesis, as my samples in the HAPIEE study are also in middle–aged adulthood.

Note that another role of OC not mentioned is a *confounder* between work stress and health behaviours. Earlier studies tended to adjust personality as a confounder for self-reported bias, suggesting that individuals with Neuroticism tend to report high levels of distress even without objective stressors.[452] However, the measure of work stress is to assess "perceived" rather than "objective" work stressor, personality traits can cause underlying construct – perception (perceived work stress). If OC personality is viewed as the antecedent or mediator in the causal path from work stress to health behaviours, OC should *not* be viewed as a confounder.

The literature review in Section 2.5 tentatively suggests four potential roles of OC in the relationship between work stress (ERI) and health behaviours: modifying role, antecedent, mediator, and direct effect of OC. Note that these potential roles of OC may not be mutually exclusive (e.g., OC is possible to have both antecedent role in ERI–drinking relation and direct effect on drinking). Finally, main effect of OC on health behaviours can be considered after the first three roles are excluded.

2.6 Overcommitment, Effort–Reward Imbalance, Perceived Control, and Health Behaviours

The relationship between ERI, OC and health behaviours has been addressed. In this chapter, the potential role of PC in the relationship between ERI, OC and health behaviours is considered by theoretical framework of Transactional Model of Stress.

2.6.1 Transactional Model of Stress

Transactional Model of Stress, proposed by Richard Lazarus and Susan Folkman (1984), is the most widely–accepted model to clarify complex relationship between the environmental stressor and the person in relation to health outcomes (health behaviours, emotional well–being, and functional status).[453] This model has been enriched by accumulating literature in personality psychology, cognitive psychology, and health psychology. In this model, stressful experiences are constructed as "person–environment transactions". When a person faces a stressor in environment, one would evaluate potential threat (primary appraisal) and one's ability to alter the situation and manage negative emotional reaction (secondary appraisal). Both appraisals can affect one's coping efforts (problem management and emotional regulation), which subsequently influence health behaviours and health outcomes.[454] The processes are described in detail in the following paragraphs.

Stressors are demands made by internal or external environment that upset balance or homeostasis in an individual, thereby affecting physical and psychological well-being and requiring one's action to restore balance or equilibrium.[455] Stressors can contribute to diseases via direct physiological effects or indirect effects via health behaviours. This model emphasizes that individual's cognitive appraisals, rather than objective stressor, would influence health behaviours and health outcomes.

Cognitive appraisals refer to cognitive processes that incorporate not only information from the stressor but also information inside the person. Primary appraisal is where the individual evaluates and gives personal meaning to the stressor, and considers the significance of "what is at stake" in terms of harm, threat, loss or challenge. Two basic primary appraisals are: (1) perceptions of *susceptibility* to the threat, which refer to beliefs about the likelihood of getting a threat (e.g. a stressful condition or an illness); (2) perceptions of *severity* of the threat, which refer to feelings about the seriousness of a threat and its possible consequences (e.g. death, disability,

or negative effects on work and social relationship). Appraisals of high severity and susceptibility of a threat not only prompt efforts to cope with the stressor, but also generate psychological distress and physiological responses.[456] As primary appraisal is one's subjective evaluation for susceptibility or severity of objective stressor, I suggest that perceived "severity" of work stress measured by the ERI model (effort–reward ratio) or the DC model (job strain) should be viewed as primary appraisal.

Secondary appraisal is an assessment of a person's availability of coping options and resources to deal with the stressor; it addresses the question "what can I do about it" by evaluating one's ability to change situation and to manage emotional reaction.[457] Key examples of secondary appraisals in psychology are: perceived ability to change the situation (PC over the threat), perceived ability to manage one's emotional reactions to the threat (PC over emotional feeling), and expectations about the effectiveness of one's coping effort (coping self-efficacy).[458] As mentioned previously, PC is defined as the extent to which one can intentionally produce desired outcomes and prevent undesired ones. Thus, PC should be viewed as secondary appraisal.

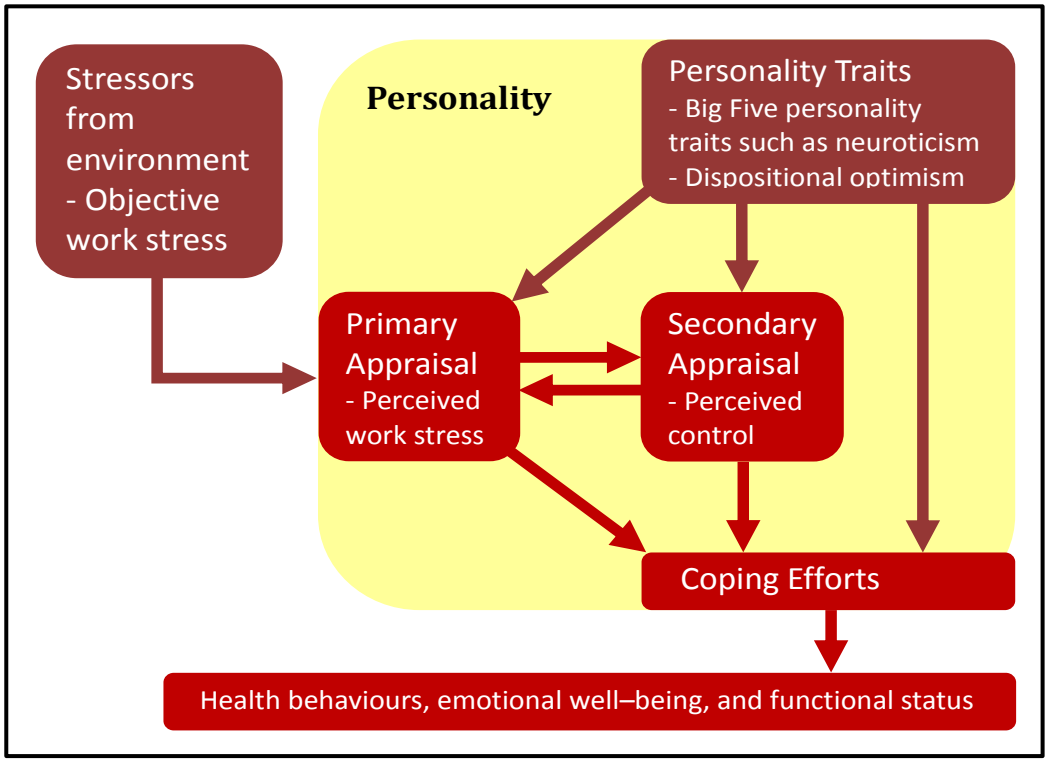
Coping effort is defined as the constantly changing cognitive and behavioural efforts a person makes to manage specific external/internal demands that are appraised as taxing/exceeding the resources of the person. The basic approach to classifying coping strategies is the dichotomy between problem–focused coping (directed at changing the stressful situation, including active coping, problem solving, and information seeking) versus emotion–focused coping (directed at changing the way one thinks or feels about a stressful situation, including emotional expression, avoidance, and denial). When a stressor is appraised as controllable and a person has high self-efficacy, one is more likely to engage in problem–focused coping. By contrast, when a stressor is perceived as highly threatening and uncontrollable, one tends to use emotion–focused coping.[459,460]

Avoidance and denial (emotion–focused coping) can shift one's attention away from the stressor and temporarily minimize psychological distress by avoiding

thoughts and feelings about the stressor (e.g. avoiding others, refusing to think about the threat, or hiding feelings); however, avoidance and denial may lead to intrusive thoughts that generate psychological distress over time and keep people from developing adaptive coping strategies. Avoidance coping is temporarily useful, but it is ineffective when confronting a stressor that poses a real threat – something that will have to be dealt with eventually.[461] Engaging in risky health behaviours (e.g. drinking or smoking) is suggested to relieve psychological distress temporarily and to distract one's attention from stressful situation; it may however cause long–term social and health problems. Thus, engaging in risky health behaviours is viewed as an emotion–focused coping, like avoidance and denial.[462]

Coping outcomes, influenced by coping efforts, indicate a person's adaptation to a stressor. Three main categories of coping outcomes are emotional well–being, functional status (health status or disease progression), and health behaviours. The theoretical framework of Transactional Model of Stress is illustrated (Figure 2.10).

Figure 2.10 Transactional Model of Stress. Source: Adapted from Glanz and Schwartz (2008).[463]



Transactional Model of Stress has been enriched by literature from personality psychology.[464] In contrast to situation–specific coping efforts, Lazarus conceptualized coping styles as stable dispositional characteristics reflecting generalized tendencies to interpret and respond to stress in particular ways.[465] Coping styles are enduring personality traits to drive primary and secondary appraisals and coping efforts; specific effects of a stressful event on adjustment may partly depend on a person's coping styles. The most widely studied coping style is dispositional optimism – the tendency to have positive rather than negative generalized expectancies for outcomes; these expectancies are relatively stable over time and across situations.[466] Evidence has supported that dispositional optimism influences the transactional process at primary appraisal, secondary appraisal and coping efforts.[467] For example, Taylor et al found that dispositional optimism was associated with lower perceived risk of disease (primary appraisal), higher PC over disease (secondary appraisal), more problem–focused coping, and less risk health behaviors.[468]

In addition to dispositional optimism, several studies reported that other personality traits (e.g. Neuroticism) can influence the transactional process at several points – primary appraisal, secondary appraisal and coping efforts (Figure 2.10). First, personality traits may alter subjective appraisal of ongoing stressor (primary appraisal); for example, high Neuroticism person tends to interpret neutral or ambiguous stimuli as a threatening event. Second, personality traits (Neuroticism) may reduce one's PC (secondary appraisal) which then affects health behaviours.[469] Third, personality traits may influence available choice of one's coping efforts; for example, high Neuroticism person tends to use more emotion–focused coping (e.g. risky health behaviours) in order to relieve higher levels of psychological distress.[470]

As Neuroticism is highly correlated with OC, it is plausible that OC may influence the transactional process at primary appraisal, secondary appraisal and coping efforts. Lazarus and Folkman have never mentioned "overcommitment" but they described "commitment", which denotes enduring motivational and cognitive process on what is

important and what has meaning for the person. In a stressful encounter, commitment determines what is at stake – to what extent the stressor harms or threatens the person (commitment influences primary appraisal). The greater the strength of a commitment, the more vulnerable a person is to psychological stress in the area of that commitment. Additionally, commitment impels a person toward a course of action that can reduce threat and sustain coping effort in the face of obstacles (commitment influences secondary appraisal).[471]

After personality traits were taken into account, the unique roles of PC (secondary appraisal) are considered. First, empirical studies have shown that PC can mediate the impacts of Five Factor personality traits on health behaviours.[472,473,474] Compared to static and broad personality traits, PC has better prediction for health behaviours by providing active and specific cognitive processes. Besides, PC is more modifiable (better target for intervention) than personality traits.[475] Second, empirical evidence has found that PC is changeable by stressors from environments (e.g. social position, work stress, or chronic illness); PC can mediate the effects of stressors on health outcomes.[476,477] Thus, Steptoe proposed that PC occupies a central position in the interplay between psychosocial demands (e.g. stressors from environments) and resources (e.g. personality traits) and poses effects on health.[478]

In summary, potential roles of PC (secondary appraisal) are considered in the relationship between ERI (primary appraisal), OC (personality traits), and health behaviours by the Transactional Model of Stress. This integration of the ERI model and Transactional Model of Stress would shed light on practical implications for both organisational and individual interventions for work stress.

2.6.2 Reciprocal relationship between perceived control and ERI

In Transactional Model of Stress, Lazarus identified primary appraisal and secondary appraisal, both of which were suggested to engage in a reciprocal and

dynamic relationship with each other.[479] Due to the dynamic nature of stress process, it is often difficult to determine whether primary appraisal causes secondary appraisal, or secondary appraisal affects primary appraisal. Both occur as part of a complex process, and both are required to shape individual's responses to a stressful encounter. Thus, Lazarus proposed that each is dependent on the other, and they should be regarded as part of the same process.[480]

In this thesis, work stress measured by ERI is viewed as primary appraisal, and PC is considered secondary appraisal. In the following paragraphs, empirical evidence on the potentially reciprocal relationship between ERI and PC, together with their relationships with outcomes, will be reviewed. This review is divided into two parts: (1) PC acts as a mediator in the effects of work stress on outcomes; (2) work stress acts as a mediator in the effects of PC on outcomes.

(1) Perceived control acts as a mediator in the effects of work stress on outcomes

The possibility of PC as a mediator in the effects of work stress on outcomes has been supported by theoretical and empirical evidence. In terms of theories, Kohn and Schooler focused on the impact of working conditions on individual's cognition and psychological health; work characteristics that allow for employee's use of independent judgment in complex matters can promote the development of PC and general intellectual functioning.[481] Pearlin et al proposed that the accumulation of experiences in which one successfully controls work environments may lead to increased perceptions of mastery (similar to PC).[482] Bandura proposed that individuals may learn and emulate skills and beliefs from workplace and bring them to other life situations; work experience may shape one's self-efficacy.[483] Siegrist suggested that work role is crucial to fulfill one's self-regulatory needs, because work offers opportunities to acquire self-efficacy (e.g. successful performance), self-esteem (e.g. recognition) and self-integration (e.g. belonging to a group). Work stress defined by ERI may impair one's self-efficacy, self-esteem and self-integration.[484]

In terms of empirical evidence, my review has found 8 empirical studies (listed in Table 2.3) supporting that PC or its components can partially mediate the effects of works tress (job strain, job insecurity, or job uncertainty) on health outcomes (health behaviours, self-rated health, or psychological distress). The exposure of work stress was measured by the DC model in 3 cohort studies[485,486,487] and by other measures of work stress (e.g. job insecurity or uncertainty) in 5 studies.[488,489,490,491,492] Note that no such literature is available on the ERI model. In addition, 6 out of 8 studies have sample size less than 500, so the results should be interpreted carefully; a cohort study with larger sample size is still needed in this topic. In general, my review shows that PC and its components may partially mediate the impacts of work stress on health outcomes.

Table 2.3 Empirical studies supporting perceived control as a mediator in the effects of works stress on outcomes

Authors (year)	Sample	Study type	Work stress measure	Perceived control measure	Outcome	Findings on the roles of perceived control
Payne et al (2002)	213 adults, UK	Cohort study, 1 week	DC model	Self-efficacy	Health behaviour	Self-efficacy partially mediates the impacts of job strain on physical activity
Payne et al (2005)	286 adults, UK	Cohort study, 2 weeks	DC model	PBC in TPB	Health behaviour	PBC partially mediates the impact of demands on physical activity
Wickrama et al (2008)	318 men, USA	Cohort study, 10 years	DC model	Perceived control	Self-rated health	PC partially mediates the effect of work stress on outcomes
Plotnikoff et al (2010)	612 adults, Canada	Cohort study, 1 Year	Perceived work stress	Self-efficacy	Health behaviour	Self-efficacy partially mediates the impact of work stress on physical activity
Vander Elst et al (2011)	211 adults, Belgium	Cross-sectional study	Job insecurity	Perceived control	Psychological distress	PC partially mediates the effect of job insecurity on outcomes
Paulsen et al (2005)	553 adults, Australia	Cohort study, 1.5 years	Job uncertainty	Perceived control	Emotional exhaustion	PC partially mediates the effect of job uncertainty on outcomes
Ito et al (2001)	204 adults, Canada	Cross-sectional study	Job uncertainty	Perceived control	Emotional exhaustion	PC partially mediates the effect of job uncertainty on outcomes
Bordia et al (2004)	222 adults, Australia	Cross-sectional study	Organisational changes	Perceived control	Psychological distress	PC partially mediates the effect of work stress on outcomes

(2) Work stress acts as a mediator in the effects of perceived control on outcomes

The possibility of work stress as a mediator in the effects of PC on outcomes has been supported by theoretical and empirical evidence. By socio–cognitive theories, it is reasonable to assume that self-efficacy can affect perceived work stress which then influences health outcomes.[493] Bandura proposed that self-efficacy affects cognitive processes, persistency of motivation, affective states and selection processes, all of which contribute to the extent of one's performances (e.g. health behaviours or coping efforts). People with high self-efficacy perceive and appraise stressors with the confidence that they can exercise control over them; they have strong beliefs in capabilities to approach difficult tasks as challenges to be overcome, rather than as threats to be avoided. Thus, they can invest high efforts in what they do and even heighten their efforts in the face of failures.[494] It is suggested that higher self-efficacy might initially result in lower level of perceived work stress; subsequently, more active coping efforts might further reduce both objective and perceived work stress.

In terms of empirical studies, Spector conducted a meta-analysis and found that higher PC was associated with lower levels of work stress – measured by role conflict in 8 studies and role ambiguity in 14 studies.[495] In addition, my review found 6 empirical studies (Table 2.4) supporting that PC or its components can affect measures of work stress (e.g. the DC model or other measures), which then influence various outcomes.[496,497,498,499,500] For example, Judge et al reported that higher self-efficacy in early adulthood predicted better profiles of perceived work conditions (e.g. autonomy or task variety), resulting in higher levels of job satisfaction in middle adulthood.[501] In general, my review implies that work stress may partially mediate the impacts of PC on outcomes. Nevertheless, there has been literature with negative findings. For example, in a 2–wave cohort study in Belgium (n= 536), PC was found to mediate the effect of job insecurity on emotional exhaustion, but job insecurity did not mediate the effect of PC on emotional exhaustion.[502]

Table 2.4 Empirical studies supporting work stress as a mediator in the effects of perceived control on outcomes

Authors (year)	Sample	Study type	Work stress measure	Perceived control measure	Outcome	Findings on the roles of perceived control
Judge et al (2000)	258 adults, USA	Cohort study, 30 years	Perceived work conditions	Self-efficacy, LOC	Job satis-faction	Perceived work conditions mediated the effect of LOC/self-efficacy on outcomes
Schwarzer et al (2008)	458 teachers, Germany	Cohort study, 1 year	Job demands, perceived work stress	Self-efficacy	Burnout	High self-efficacy decreased perceived work stress, which then reduced burnout
Spreitzer et al (2002)	350 adults, USA	Cohort study, 1 year	Threat in organization downsizing	Perceived control	Voluntary turnover	High PC reduced threat in downsizing, which then decreased turnover
Xanthopoulou et al (2009)	163 adults, Netherland	Cohort study, 1.5 year	DC model, Job control, workplace support	Self-efficacy	Work engage-ment	High self-efficacy increased job control and workplace support, which then increased work engagement
Hoge et al (2004)	205 adults, Germany	Cross–sectional study	Perceived work stress	Sense of coherence	Physical & mental health	High sense of coherence decreased perceived work stress, which improved health
Rennesund et al (2010)	924 adults, Norway	Cross–sectional study	DC model, job strain	Self-efficacy	Work per-formance	High self-efficacy decreased job strain, which promoted work performance

Several limitations need to be taken into account in the above review. First, the exposure of work stress was assessed by the DC model or other measurements, but no literature is available on the ERI model. While health and job–related outcomes were measured, there were no studies on health–behaviour outcomes. In addition, 5 out of 6 studies have sample size less than 500, and the results should be interpreted carefully; a large cohort study is still needed in this topic.

While PC may mediate the effects of work stress on outcomes, work stress may also mediate the effects of PC on outcomes; the two are not mutually exclusive. In Transactional Model of Stress, primary appraisal (e.g. perceived work stress such as ERI) and secondary appraisal (e.g. PC) are engaged in a reciprocal relationship.[503] In social cognitive theory, Bandura emphasized that the reciprocal causation of the characteristics of persons (self-efficacy) and their environments is better captured by

the transactional perspective; people are both producers and products of social environment.[504] Based on the theoretical and empirical evidence, I tentatively suggest that there is a reciprocal relationship between PC and ERI.

2.6.3 Interaction between perceived control and ERI

In heath and psychological research, interaction hypothesis (interaction between personality and stressor from environment) is a widely–used approach that cannot be ignored in this thesis. Modifying effect means that the magnitude and direction of the effect of a predictor (e.g. work stress) on an outcome depends on the level of another predictor (e.g. PC). There are different forms of interaction. “Buffering” implies that a high level of adaptive personality construct (e.g. PC) protects individuals from adverse effects of risk exposure (e.g. work stress). “Person-environment fit” indicates neither high nor low levels of a personality construct are necessarily maladaptive; adverse outcomes arise from a lack of fit between personality and environment. For instance, job control and locus of control may be congruent or incongruent, with favorable or unfavorable effects on outcomes, respectively.[505]

In terms of empirical evidence, my review found 7 cohort or cross–sectional studies supporting that PC or its components can modify the effects of work stress on outcomes (Table 2.5). There are 3 studies foucused on the DC model,[506,507,508] 3 study on other measurements of work stress,[509,510,511] and one study on the ERI model.[512] Note that there are 3 large–sample studies (n > 1000). For example, Bethge and Radoschewski reported that internal locus of control acted as a buffer between the effect of ERI on impaired work ability in a German cross–sectional study (n= 1,348).[513]

Some limitations of my review should be noted. First, the findings on the forms of modifying effects are mixed and inconsistent; both “buffering” and “person–environment fit” have received support, and both two–way (e.g. job control x PC) and three–way interactions (e.g. demand x job control x PC) have been reported. Second,

there have been other studies reporting negative findings on modifying role of PC in work stress–outcome relationship.[514,515] For instance, Marchand et al found that LOC did not modify the effect of job strain on psychological distress in a 7–year cohort study in Canada (n= 6,359).[516] Moreover, there has been literature supporting mediator rather than modifying role of PC when both possibilities were tested.[517] In a cross–sectional study in Belgium (n= 211), PC did not buffer the effects of job insecurity on outcomes, but PC can mediate the effects of job insecurity on outcomes.

Table 2.5 Empirical studies supporting interaction between perceived control and work stress

Authors	Sample	Study type	Work stress measure	PC measure	Outcome	Findings on the roles of perceived control
Olsson et al (2009)	2,246 adults, Sweden	Cohort study, 9 years	DC model	Sense of coherence	Self-rated health	SOC modified the effect of work stress on self-rated health
Rodriguez et al (2001)	542 Europeans	Cohort study, 1 year	DC model	LOC	Job satis-faction	LOC modified the effect of work stress on job satisfaction
Parkes (1991)	590 civil servants, UK	Cohort study, 1 year	DC model	LOC	Affective distress	LOC modified the effect of work stress on affective distress
Jimmieson et al (2004)	213 adults, Australia	Cohort study, 2 years	Work load, role ambiguity	Self-Efficacy	Job satis-faction	Self-efficacy modified the effect of work stress on job satisfaction
Brockner et al (2004)	1,067 adults, US	Cohort study, 1 year	Threat from organisation downsizing	PC	Work performa-nce	PC modified effect of threat from downsizing on work performance
Lu et al (2000)	581 managers in Taiwan / UK	Cross-sectional study	Perceived work stress	LOC	Job satis-faction, well-being	LOC modified the effect of work stress on job satisfaction/ well-being
Bethge et al (2010)	1,348 adults, Germany	Cross-sectional study	ERI model	LOC	Impaired work ability	Internal LOC acted buffer effect of high ERI on reduced work ability

It is premature to conclude whether PC can modify the effects of work stress on outcomes by existing literature. Based on the Transactional Model of Stress, reciprocal relationship between ERI and PC, rather than interaction between the two, is assumed as my hypothesis. However, considerable findings from previous studies imply that the possibility of interaction between ERI and PC cannot be ignored and should be empirically tested in this thesis.

2.6.4 Relationship between OC, ERI, perceived control and health behaviours

In this chapter, I attempt to hypothesize the potential relationship between ERI, OC, PC and health behaviours by integrating the theoretical frameworks of Transactional Model of Stress and the ERI model. In Transactional Model of Stress, primary appraisal is one's subjective evaluation for susceptibility or severity of objective stressor. I suggest that perceived severity of work stress measured by the ERI model (ER ratio) should be viewed as primary appraisal. The reason is that the lack of reciprocity between high effort and low reward together – rather than either effort or reward alone, defines a state of emotional distress with autonomic arousal and strain reactions in the ERI model.

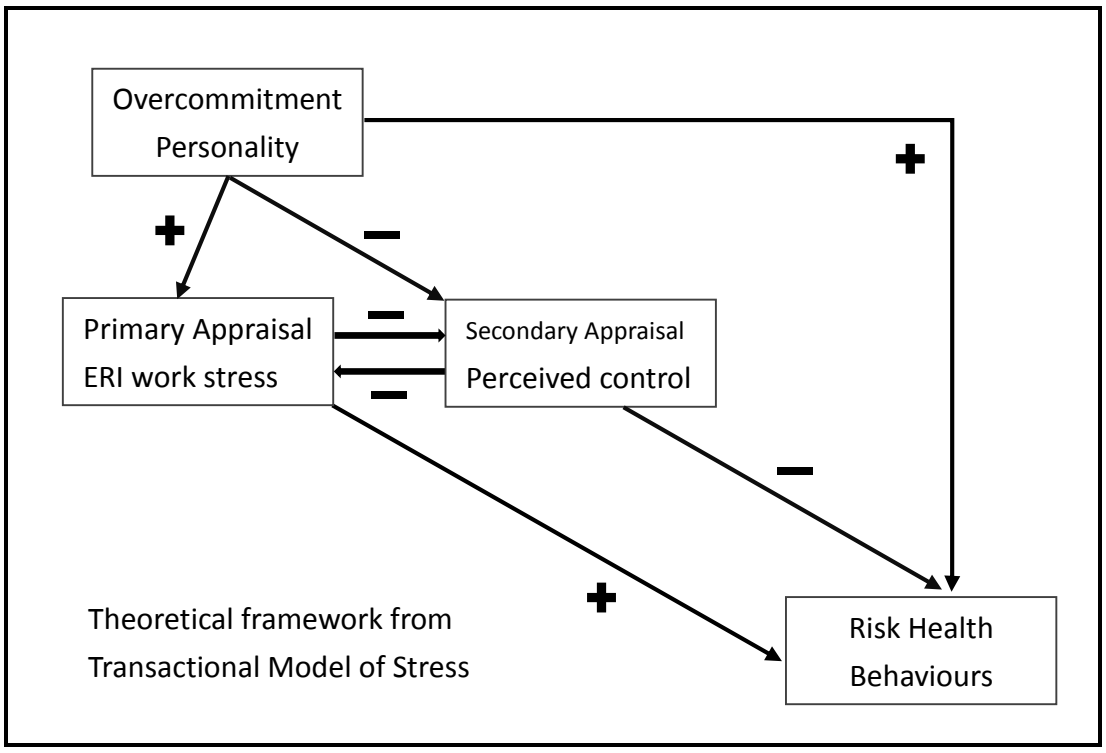
Secondary appraisal is one's assessment of availability of coping options and resources to deal with the stressor. Main examples of secondary appraisal in psychology are perceived ability to change the situation (PC over the threat), perceived ability to manage one's emotional reactions to the threat (PC over emotional feeling), and expectations about the effectiveness of one's coping effort (coping self-efficacy).[518] Thus, PC should be viewed as secondary appraisal.

Notably, my interpretation and integration for the ERI model and Transactional Model of Stress (ER ratio is primary appraisal) is slightly different from Siegrist's original ideas on the link between these two models. Siegrist (1996) recognized the influence of Lazarus's Transactional Model of Stress on the ERI model.[519] He wrote: "negative emotions are the result of a multistage appraisal process, which includes the taxing of stressor properties and of a person's coping repertoire under exposure….. This theory would predict cognitive and behavioural adjustment to a high-cost / low-gain condition as a consequence of cognitive appraisal processes". Siegrist has ever suggested that "effort / reward" were conceptually similar to "primary appraisal / secondary appraisal" in early works. On the other hand, Siegrist tried to distinguish between the ERI model and Transactional Model of Stress; he suggested that rapid

and direct pathways of affective information processing may bypass neocortical–limbic structures, so chronically everyday experience of ERI at work may not necessarily be subjected to conscious appraisal.

In Transational Model of Stress, primary appraisal and seconday appraisal subsequently affect coping efforts (health behaviours) and health outcomes. In particular, primary appraisal (ERI) and secondary appraisal (PC) engage in a reciprocal relationship. Additionally, personality traits (e.g. OC) can influence the transactional process at primary appraisal, secondary appraisal and coping efforts. The potential relationships between the constructs in this thesis are summarized in Figure 2.11, which is relatively simplified but not different from Figure 2.10.

Figure 2.11 Potential relation between OC, ERI, PC and health behaviours based on Transactional Model of Stress.



Two points should be noted in the above model. First, the directions of the effects are categorized into positive association (+) and negative association (-). In Section 2.4.4, a substantial amount of literature has supported that lower PC is associated with

worse profiles of health behaviours. In Section 2.4.5, the relationship between OC, PC and health behaviours has been addressed; higher OC is associated with lower level of PC, which is associated with worse profiles of health behaviours. PC is suggested to mediate the effects of OC on health behaviours.

Second, it is suggested that OC personality and ERI might have bi–directional causal relationship in Section 2.5.4. Based on existing evidence, the effect of OC personality on ERI would be stronger than the other causal direction in the middle–aged and older populations. In a parsimonious model, the cross–sectional "snapshot" of the bi–directional relationship between OC and ERI would be: ERI mediates the effect of OC personality on health behaviours. Transactional Model of Stress and personality psychology also supports this parsimonious model.

2.7 Summary of Literature Review

2.7.1 Gaps identified in the existing research

For several decades, researchers have been intrigued by the debate over the joint influences of work environment and personality constructs on human behaviours. A profound understanding of the complex relationship between the environment and the person is crucial to promote a steady accumulation of knowledge and to inform effective interventions on health behaviours. In Chapter 2, literature review has summarised the evidence related to different relationships between work stress, personality constructs (OC personality and PC), and health behaviours. Several gaps in existing literature have been identified and listed below.

(1) Very limited literature on the links between OC, ERI and health behaviours

Health behaviours are influenced by a wide range of psychosocial factors. The approach of many epidemiological studies has been to identify independent

contribution of each psychosocial factor. It is widely recognized that psychosocial factors rarely occur in isolation, but few studies have attempted to examine how two or more psychosocial factors interact to influence health outcomes. It is necessary to expand the breadth of studies by addressing this multiplicity of psychosocial factors.[520] Work stress (ERI and job strain) or OC–related personality (Type A behaviour, Neuroticism, Hostility, and PC) have been repeatedly reported to independently predict health behaviours (described in Section 2.3.3, 2.4.2 and 2.4.4). Despite considerable evidence linking ERI and OC–related personality independently to health behaviours, there has been very limited literature on the links between OC, ERI and health behaviours in studies where all of them are considered simultaneously.

In the review of 45 studies on the ERI model by Van Vegchel et al, there have been only two studies regarding health–behaviour outcomes (smoking and drinking); extrinsic ERI hypothesis was supported, but the potential role of OC (main or modifying effect) was not tested.[521,522] Since this review published, another two studies from Japan and Australia have reported negative findings on main effects of OC on smoking; however, modifying effect of OC has not been examined.[523,524] As very limited literature is available on the links between OC, ERI and health behaviours, it is of value to investigate this topic.

(2) Inconclusive findings regarding potential role of OC in ERI–outcome relationship

Current literature on potential role of OC in ERI–outcome relationship has been inconclusive. The main effect of OC has received strong support from empirical studies, while modifying effect of OC has gained weak support from limited literature (as described in Section 2.5.1). In the wider fields of psychology and medicine, current understanding of diverse roles of personality in stress processes includes “antecedent and mediator roles” of OC in ERI–outcome relationship, but these possibilities have not been examined for the ERI model. To evaluate potential roles of OC in ERI–

outcome relationship more rigorously, four possible roles of OC should be tested: modifying role, antecedent role, mediator role, or direct effect.

(3) No literature on the links between OC, ERI, PC and health behaviours

Perceived control (PC) and its components have gained strong support to predict health behaviours from theoretical models and empirical studies. PC is suggested to mediate the effects of OC personality on health behaviours (as described in Section 2.4.3 and 2.4.5). By Transactional Model of Stress, the potential role of PC (secondary appraisal) can be integrated into the relationship between OC, ERI (primary appraisal) and health behaviours (as described in Section 2.6.4). The integration of the ERI model and Transactional Model of Stress would enlighten practical implications for interventions for work stress. To my knowledge, no literature is available on the links between OC, ERI, PC and health behaviours in studies where all of them are considered simultaneously.

(4) Limitations of small–sample studies and cross–sectional design

Most evidence regarding the relationships between work stress, personality constructs (e.g. OC and PC), and health outcomes (e.g. health behaviours) comes from studies with sample size less than 1000. In addition, about half of these studies have cross–sectional design. These studies cannot provide strong evidence of modifying, antecedent or mediator roles of personality constructs in stress processes with a clear temporal relationship. Thus, a large–sample cohort study is needed to help establish sequential and ultimately causal nature of relationships between work stress, personality constructs and health behaviours, thereby providing more solid evidence for potential roles of personality in work stress–outcome relationship.

2.7.2 Importance of proposed project

Based on the gaps identified, this thesis aims to investigate the relationship between OC, ERI, PC and health behaviours in a large–sample and two–wave cohort study in CEE populations, particularly focusing on two aspects: the potential role of OC in the relationship between ERI and health behaviours; the potential role of PC in the relationship between OC, ERI and health behaviours. It is hoped that this thesis will contribute to growing understanding of intersecting pathways by which work stress (ERI) and personality constructs (OC and PC) jointly influence health behaviours.

The unique context of social transformation in CEE provides a natural setting for investigating the relationship between work stress, personality constructs and health behaviours. The East–West mortality gap in Europe, a major public health concern in CEE, was previously hypothesized to be mainly explained by psychosocial factors and health behaviours. It is crucial to understand the interactions between multiple levels of psychosocial factors (work stress and personality) and their combined influences on health behaviours in order to develop more effective interventions aimed at promoting health behaviours and subsequent health in CEE. Thus, the aims of this thesis might contribute to deeper understanding on the East–West mortality gap. However, I should state explicitly that this thesis using data collected from CEE populations provides an opportunity to investigate the general topic of interest – the relationship between work stress, personality constructs and health behaviours; the specific topic on contextual importance of CEE will not be addressed in detail in the following parts of the thesis.

Chapter 3. Aims, Objectives and Hypotheses

3.1 Aims

This chapter outlines the main aims, objectives, hypotheses, and conceptual framework of the thesis. In the previous chapter, the gaps in knowledge related to the relationship between work stress, personality constructs, and health behaviours were identified; the current project aims to address some of these gaps. Thus, the two aims of the thesis are:

1. To examine the relationship between effort–reward imbalance (ERI), overcommitment (OC), and health behaviours in the Central and Eastern European (CEE) populations, particularly focusing on the potential role of OC in the relationship between ERI and health behaviours.
2. To additionally investigate the potential role of perceived control (PC) in the relationship between ERI, OC and health behaviours in the CEE populations.

Three health–behaviour outcomes (alcohol drinking, smoking, and diet) will be used in this thesis. The specific objectives and relevant hypotheses related to these three outcomes are listed below.

3.2 Objectives and Hypotheses

In relation to the first aim, the focus is on the associations between ERI and health behaviours, on the associations between OC and health behaviours, and on assessing whether OC has potential role of antecedent, mediator, modifier, or direct effect in the relationship between ERI and health behaviours. The objectives and hypotheses are:

<u>Objective 1</u>

To assess crude and adjusted associations between ERI and three health behaviours

– alcohol drinking, smoking, and diet, respectively.

→ Hypothesis 1: Higher ER ratio is associated with higher levels of alcohol drinking after adjustment for covariates.

→ Hypothesis 2: Higher ER ratio is associated with higher levels of smoking after adjustment for covariates.

→ Hypothesis 3: Higher ER ratio is associated with less healthy diet after adjustment for covariates.

Objective 2

To assess crude and adjusted associations between OC and three health behaviours – alcohol drinking, smoking, and diet, respectively.

→ Hypothesis 4: Higher OC is associated with higher levels of alcohol drinking after adjustment for covariates.

→ Hypothesis 5: Higher OC is associated with higher levels of smoking after adjustment for covariates.

→ Hypothesis 6: Higher OC is associated with less healthy diet after adjustment for covariates.

Objective 3

To evaluate the potential role of OC (antecedent, mediator, modifier, or direct effect) in the relationship between ERI and health behaviours.

→ Hypothesis 7: OC and ERI have bi–directional relationship, but it is predicted that the effect of OC on ERI is stronger than the other direction in the middle-aged and older populations. OC might have antecedent role in ERI–drinking relationship.

→ Hypothesis 8: OC and ERI have bi–directional relationship, but it is predicted that the effect of OC on ERI is stronger than the other direction in the middle-aged and older populations. OC might have antecedent role in ERI–smoking relationship.

→ Hypothesis 9: OC and ERI have bi–directional relationship, but it is predicted that

the effect of OC on ERI is stronger than the other direction in the middle-aged and older populations. OC might have antecedent role in ERI–diet relationship.

The second aim focuses on the associations between PC and health behaviours, and on the potential role of PC in the relationship between ERI, OC and health behaviours. The objectives and hypotheses are listed below:

Objective 4

To assess crude and adjusted associations between PC and three health behaviours – alcohol drinking, smoking, and diet, respectively.

→ Hypothesis 10: Lower PC is associated with higher levels of alcohol drinking after adjustment for covariates.

→ Hypothesis 11: Lower PC is associated with higher levels of smoking after adjustment for covariates.

→ Hypothesis 12: Lower PC is associated with less healthy diet after adjustment for covariates.

Objective 5

To examine the potential role of PC (mediator or modifier) in the relationship between ERI, OC and health behaviours.

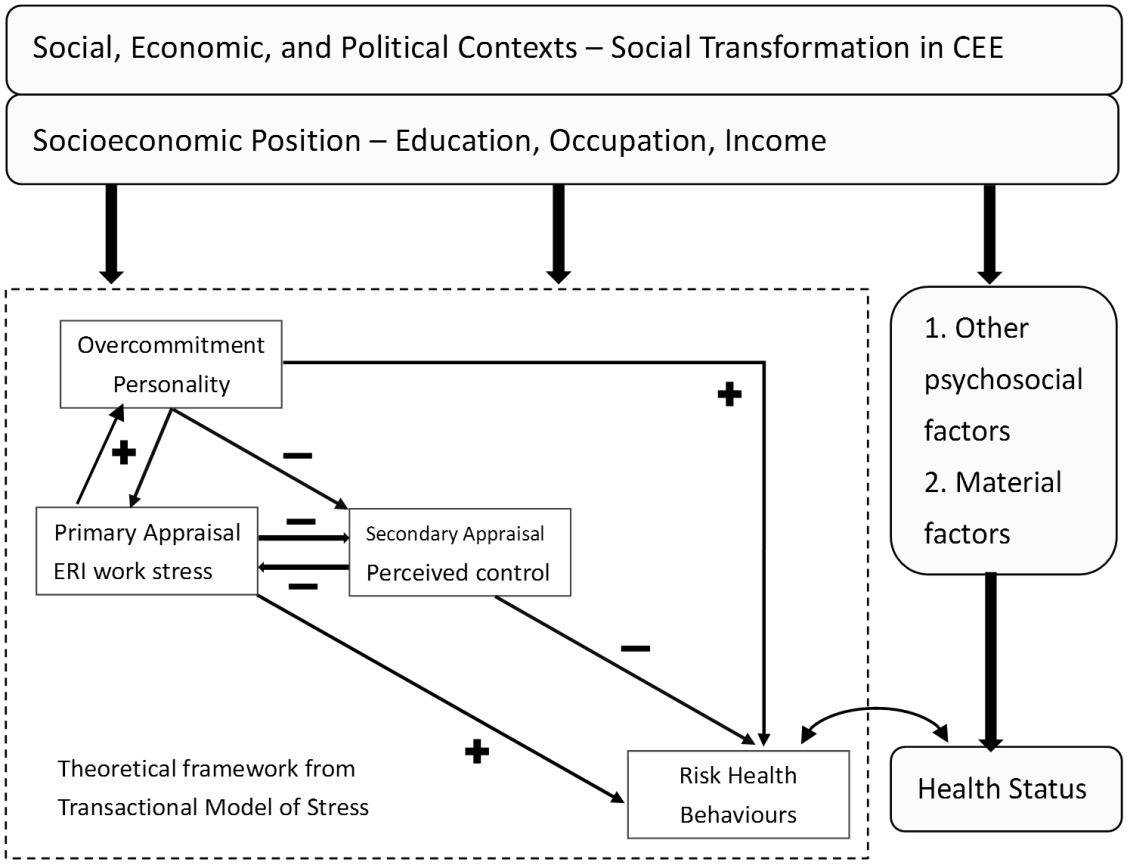
→ Hypothesis 13: PC and ERI partially mediate the effects of OC on alcohol drinking. In addition, PC and ERI might have bi–directional relationship.

→ Hypothesis 14: PC and ERI partially mediate the effects of OC on smoking. In addition, PC and ERI might have bi–directional relationship.

→ Hypothesis 15: PC and ERI partially mediate the effects of OC on diet. In addition, PC and ERI might have bi–directional relationship.

The conceptual framework (Figure 3.1) illustrates the relationships between the three psychosocial factors (OC, ERI, and PC) and health behaviours in the wider context of social determinants of health. Potential confounders that are known risk factors for health behaviours and are associated with exposure variables in the source population include: age, social position (education and occupation), material factors (deprivation), other psychosocial factors (marital status, depression, and social isolation), and health status (self–rated health); these factors will be adjusted in the regression analyses.

Figure 3.1 A conceptual framework of the thesis



The assumed relationship between OC, ERI, PC, and health behaviours is based on Transactional Model of Stress. The directions of the effects are categorized into positive association (+) and negative association (-).

Chapter 4. Methods

4.1 Study Description

This thesis is based on the data from the HAPIEE (Health, Alcohol and Psychosocial factors In Eastern Europe) study, which is a prospective cohort study designed to examine the impact of classical and non–conventional risk factors (e.g. psychosocial factors) on cardiovascular and other non–communicable diseases in CEE. The baseline data collection (wave 1) was carried out between 2002 and 2005 in six towns in Czech Republic, Novosibirsk in Russia, and Krakow in Poland. The wave 2 data collection was extended to include Lithuania (not included in the thesis) and conducted between 2006 and 2008.

In terms of study populations, the HAPIEE study originally had the 3 cohorts: (1) six towns in Czech Republic, (2) Novosibirsk in Russia, and (3) Krakow in Poland (Figure 4.1). The six Czech towns, with a total population of over 0.6 million, cover a variety of socioeconomic profiles. For example, *Hradec Kralove* is a prosperous city with chemical industry, electronics manufacturing and Information technology, with low unemployment rate (6.5%). *Havirov/Karvina* is a large city with the highest unemployment rate in the country (19.6%). *Kromeriz* and *Jihlava* are both towns with a variety of production industries.

Krakow is the second largest city – a science and technology centre in Poland with a population of one million. Krakow is more prosperous than the Polish average, the unemployment rate in 2007 was 4.8% compared to the national average (13%). The study selected four different districts ranging from blue–collar districts to middle–class districts, which should represent various socioeconomic spectrums.

Novosibirsk, the third largest city in Russia with a population of 1.4 million, is an industrial city with electric power, gas and water supply, and mechanical engineering. Novosibirsk is typical for urban populations in Russia in terms of social development. Two districts of the city with different socioeconomic profiles were selected.

Figure 4.1 The study sites in the HAPIEE study

The reported response rates were 61% in Poland and Russia and 55% in Czech Republic. As a small proportion of non–respondents had died or moved away after the sample was selected but before being invited to the study, they were ineligible for inclusion; thus, the real response rates may be higher (estimated at least 68%, 71%, and 60% for Poland, Russia, and the Czech Republic). In examining a subsample of non–respondents, they were more likely to be younger and male, with lower levels of education, with higher prevalence of smoking, and with poorer self–rated health.[525] All participants gave written informed consent, and all procedures were approved by University College London (UCL) Hospital and local ethical committees.

4.2 Study Samples

The cohorts consisted of random samples of men and women aged 45–69 years at baseline, stratified by gender and by 5–year age groups and selected from

population registers. Of the 28,947 subjects at wave 1, *ineligible subjects* such as retired persons (14,060), unemployed (1,178), housewives (307) and those with unknown employed status (131) were excluded, as only those employed at the time of the study completed a module on work characteristics (e.g. questionnaire for the ERI model) but others did not (e.g. retired persons completed a module on retirement and quality of life). In the 13,271 *eligible subjects* who were employed at the time of the study, those with missing values in exposure variables – ERI/OC (425) and PC (93) at wave 1 – were excluded. The remaining 12,753 subjects were used to generate two subsamples for the analyses of different outcomes.

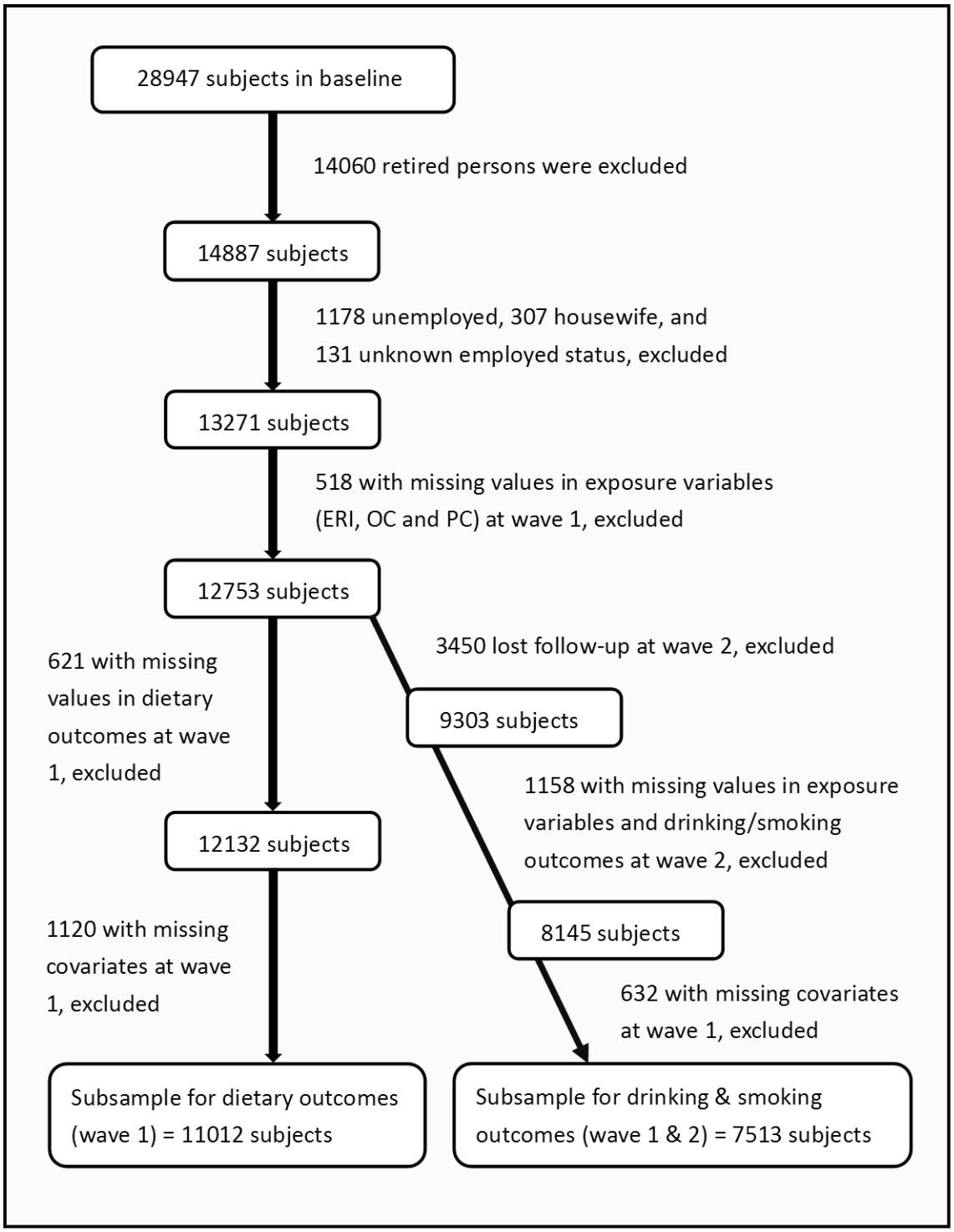
For the analyses of drinking and smoking outcomes (available at both wave 1 and 2), 3,450 subjects who were lost to follow–up at wave 2 were excluded. Then, those with missing values in exposure variables and outcomes (drinking and smoking outcomes) at wave 2 (1,158) were excluded. Finally, 632 subjects with missing covariates were excluded. This subsample with complete information for exposures and drinking/smoking outcomes at wave 1 and 2, and with complete information for covariates at wave 1, consisted of 7,513 subjects (3,782 men and 3,731 women).

For the analyses of dietary outcomes (available at wave 1 only), 621 subjects with missing values for more than 15 questions in the Food Frequency Questionnaire were excluded. Next, 1,120 subjects with missing covariates were excluded. This subsample with complete information for exposure variables, dietary outcomes, and covariates at wave 1 was composed of 11,012 subjects (5,735 men and 5,277 women).

As a form of sensitivity analysis, bivariate analyses were conducted among study samples and excluded subjects due to missing values in exposures, outcomes and covariates. In the subsample for drinking/smoking outcomes, excluded subjects (n= 13271 – 7513 = 5758) were more likely to be male (55.1% versus 50.3%), with lower educational level (29.0% versus 34.9% at university degree), with higher alcohol consumption (4139 ± 9376 versus 3652 ± 8725 g/year), with more current smokers (36.1% versus 30.9%), and with poorer self–rated health (2.8 ± 0.8 versus 2.6 ± 0.7)

than the study sample. In the subsample for dietary outcomes, excluded subjects (n= 13271 – 11012 = 2259) were more likely to be older (55.1 ± 6.1 versus 53.9 ± 5.8) and male (53.6% versus 52.1%), with lower educational level (29.6% versus 33.4% at university degree), with more current smokers (34.8% versus 32.9%), and with poorer self–rated health (2.9 ± 0.7 versus 2.7 ± 0.7) than the study sample.

Figure 4.2 Flow chart for selection of the study subsamples



4.3 Description of Variables

The baseline data collection included questionnaires, physical examination, and blood sample. The questionnaire covered socioeconomic status, demographics,

material factors, psychosocial factors, health behaviours and health. Within psychosocial factors, employed subjects completed a module on work characteristics (e.g. questionnaire for the ERI model), and retired persons completed a module on retirement and quality of life. The variables used in the analyses of the thesis are described below. All questionnaires related to the thesis are presented in the Appendix.

4.3.1 Outcome variables

The three health behaviours – drinking, smoking, and diet were adopted as outcome variables in this thesis. As the measurement of physical activity (time spent in physical activity) was relatively simple in the HAPIEE study, physical inactivity was not included in these analyses. The three outcome variables are described below.

(1) Drinking outcomes

Several measures of alcohol consumption were derived from the graduated frequency questionnaire (GFQ). The frequency of drinking occasions was assessed by 9 mutually exclusive categories, ranging from “never” to “daily”. The amounts of ethanol consumed per occasion were assessed by 6 mutually exclusive categories at wave 1 (ranging from “<1” to “10 and above” drinks) and 3 mutually exclusive categories at wave 2 (ranging from “<2” to “5 and above” drinks). The amounts were expressed in local units; 1 drink means 0.5L of beer, 0.2L of wine, and 0.05L of spirits, which approximately equal to 20g ethanol. Total annual consumption of alcohol was calculated from the frequency of drinking occasions and the amounts per occasion.[526]

Based on information from GFQ, three drinking outcomes were obtained: (1) Binge drinking: a dichotomous variable was defined by drinking at least 100g in men or 60g in women of ethanol per drinking session at least once a week, with all other respondents reporting alcohol intakes below these limits in reference category. (2) Heavy drinking: it was defined as a dichotomous variable by the cutoff point: 350

g/week or more of ethanol in men and 210 g/week or more in women, respectively.[527] (3) Problem drinking: the CAGE questionnaire (Table 4.1) was used to screen for problem drinking; it contained 4 items with 2 responses (0= no; 1= yes). With a cut-off point of 2, previously reported sensitivity ranged from 0.78 to 0.81 and specificity ranged from 0.76 to 0.96 in relation to alcohol abuse or dependence.[528] In the HAPIEE study, both GFQ–based variables and problem drinking were strongly associated with separately taken measures of alcohol consumption and serum gamma–glutamyl transferase.[529]

Table 4.1 The CAGE questionnaire

Items
1. Have you ever felt you should cut down on your drinking?
2. Have people ever annoyed you by criticising your drinking?
3. Have you ever felt bad or guilty about your drinking?
4. Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?

(2) Smoking outcomes

Smoking outcomes included smoking status and smoking intensity. Smoking status was measured in a standard way using the questions: “do you smoke cigarettes?” The four answers were: (1) yes, regularly, at least one cigarette a day on average; (2) yes, occasionally, less than one cigarette a day; (3) no, I smoked in the past but I stopped; (4) no, I have never smoked. Those who gave the first two answers were classified as *current smokers*; those who gave the third answer were classified as *past smokers*, and those who gave the last answer were *life–long non–smokers.* For these analyses, the outcome of smoking status was dichotomized as: current smokers (1) and current non–smokers (0) which included past smokers and life–long non–smokers.

Among current smokers, smoking intensity was assessed by the question: “how many cigarettes a day do you smoke now?” For these analyses, the outcome of

smoking intensity was categorized as: 1= light smoker (1–9 cigarettes a day), 2= medium smoker (10–19 cigarettes a day), and 3= heavy smoker (20 or more cigarettes a day). This classification for the levels of smoking intensity has been adopted in several previous studies.[530,531]

(3) Dietary outcomes

Dietary data were collected using the Food Frequency Questionnaire (FFQ) adapted from Willett et al,[532] which was used previously in the Whitehall II Study.[533] FFQ is the primary method for measuring average long–term diet in epidemiologic applications. Due to country–specific dishes, Czech, Polish and Russian FFQs consisted of 136, 148 and 147 food items, respectively. For each food item, a country–specific portion size was specified according to the McCance and Widdowson Food Composition Database and local food composition tables.[534] Subjects were asked how often they had consumed that amount of food during the last 3 months, with 9 responses ranged from "less than once per month" to "6 or more times per day". By multiplying frequency of food consumed per day with nutrient content of specified portion size, nutrient intakes (quantity per day) were calculated. This methodology was described in detail by Boylan et al.[535]

Diet quality is often defined by the adherence to dietary guidelines associated with health outcomes (e.g. chronic diseases or mortality).[536] The Healthy Diet Indicator (HDI) was constructed to reflect the adherence to pre–defined dietary recommendations of World Health Organisation (WHO) for the prevention of chronic diseases.[537] This approach was developed by Huijbregts et al to identify diet quality associated with chronic diseases.[538] The measurement of diet quality takes into account intakes of various foods and nutrients, thereby providing more accurate pictures of diet than single food/nutrient intake. From the WHO guideline, nine nutrient/food intakes were selected as follows: (1) nutrient density evaluated by percentages of total energy intakes (nutrient intakes divided by total energy intakes)

from saturated fats, polyunsaturated fats, total carbohydrates, free sugars, and protein; (2) nutrient intakes of non–starch polysaccharides (NSP), cholesterol, and sodium; (3) food intakes of fruit and vegetable. Macronutrients including fats, protein and carbohydrates are major sources of energy; fruit and vegetable represent main sources for micronutrients (vitamins and minerals). Next, a dichotomous variable was generated for each nutrient/food intake; if one's intake was within the WHO recommended range this variable was coded as 1 (healthy intake), otherwise it was coded as 0 (unhealthy intake). The HDI score was then calculated as the sum of nine dichotomous variables, with values ranged from 0 to 9 (Table 4.2).

Table 4.2. Nine individual HDI components as dichotomous variables

Individual HDI components as dichotomous variables	1= Within the WHO recommended range 0= Otherwise
1. Saturated fat	1= < 10% total energy intake 0= > 10% total energy intake
2. Polyunsaturated fat	1= 6–10% total energy intake 0= < 6% or > 10% total energy intake
3. Protein	1= 10–15 % total energy intake 0= < 10% or > 15% total energy intake
4. Total carbohydrate	1= 55–75% total energy intake 0= < 55% or > 75% total energy intake
5. Free sugars	1= < 10% total energy intake 0= > 10% total energy intake
6. Non–starch polysaccharides	1= > 20 g/day 0= < 20 g/day
7. Cholesterol	1= < 300 mg/day 0= > 300 mg/day
8. Sodium	1= < 2000 mg/day 0= > 2000 mg/day
9. Fruit & vegetable	1= > 400 g/day 0= < 400 g/day

4.3.2 Main exposure variables

(1) Effort–Reward Imbalance (ERI) at work

The ERI model was operationalized as a standardized self-reported measure containing 23 Likert-scaled items (Table 4.3), defining three unidimensional scales: extrinsic effort (6 items), reward (11 items), and overcommitment (6 items). In the

HAPIEE study, the ERI questionnaire was translated into all three languages and then back translated to confirm the accuracy of original translations.

"Extrinsic effort" was measured by six items on demanding aspects of work environment (3 items measuring quantitative load, 1 item measuring qualitative load, 1 item on physical load, and 1 item on increase in total load over time). For each item, the rating procedure was given on a 5-point scale: (1) no; (2) yes, not at all distressed; (3) yes, somewhat distressed; (4) yes, rather distressed; (5) yes, very distressed. "Reward" was assessed by eleven items (1 item on financial reward, 5 items measuring esteem reward, and 5 items on promotion prospects and job security). In terms of rating procedure, 4 of 11 items were rated in the same way as extrinsic effort. The other items were rated on a 5-point scale: (1) yes; (2) no, not at all distressed; (3) no, somewhat distressed; (4) no, rather distressed; (5) no, very distressed. Positively and negatively worded items were included to control for response biases; negatively worded items were reversely coded to ensure all responses in the same direction.

The extent of imbalance between extrinsic effort and reward was measured by effort–reward (ER) ratio; extrinsic effort score was in the numerator, and reward score was multiplied by a correction factor to adjust for unequal number of items in the denominator.[539] High ER ratio (> 1) indicates high levels of work stress, in which a high amount of effort spent is not met by the rewards received or expected. In my analyses, average scores were calculated if a minimum of 5 out of 6 questions on extrinsic effort (the average score of non-missing items multiplied by 6) and 9 out of 11 questions on reward contained valid answers.[540]

Overcommitment (OC) is the cognitive–motivational pattern of coping with demands characterized by an excessive work–related overcommitment and a high need for approval. OC was assessed by 6 items; for examples, I get easily overwhelmed by time pressures at work; as soon as I get up in the morning I start thinking about work problems; people close to me say I sacrifice too much for my job. Each item was rated on a 4–point scale (strongly disagree, disagree, agree, and

strongly agree). In my analyses, average scores were calculated if a minimum of 5 out of 6 questions on OC contained valid answers (the average score of non-missing items multiplied by 6).

By the commonly used analyses based on score distribution, ER ratio and OC were divided into thirds to indicate low (tertile 1), intermediate (tertile 2), and high levels (tertile 3), respectively.[541]

Table 4.3 The Effort–Reward Imbalance questionnaire

Extrinsic Effort
1. There is constant time pressure in my job due to a heavy workload
2. There are many interruptions and disturbances in my job
3. I have a lot of responsibility in my job
4. There is pressure in my job to work overtime
5. My job is physically demanding
6. Over the past few years, my job has become more and more demanding
Reward
1. Are you treated unfairly at work?
2. Are the promotion prospects in your job poor?
3. Do you expect to experience an undesirable change in your work situation?
4. Is your own job security poor?
5. Do you receive the respect you deserve from your work colleagues?
6. Do you receive the respect you deserve from your supervisors?
7. Do you experience adequate support in difficult situations?
8. Does your current job adequately reflect your knowledge, skills and training?
9. Does your salary/income adequately reflect all your past efforts and achievements?
10. Considering all your efforts and achievements, do you receive the respect and prestige you deserve at work?
11. Considering all your efforts and achievements, are your work prospects adequate?
Overcommitment
1. I get easily overwhelmed by time pressures at work
2. As soon as I get up in the morning I start thinking about work problems
3. When I get home, I can easily relax and ‘switch off’ work
4. People close to me say I sacrifice too much for my job
5. Work rarely lets me go, it is still on my mind when I go to bed
6. If I postpone something that I was supposed to do today, I’ll have trouble sleeping at night

(2) Perceived control (PC)

The PC score was based on 11 questions (Table 4.4) adapted from the Whitehall II Study and MacArthur Foundation Programme on Midlife Development.[542] This instrument was similar to the perceived constraints of General Perceived Control Scale developed by Lachman and Weaver.[543] In terms of external validity, this PC score has been found to be associated with several socioeconomic indicators and self-rated health in the context of CEE countries.[544]

In the PC score, the items 2 to 4 represented "control over health", while other items represented "control over life". The subjects were asked to what extent they agree or disagree with the statements, with the answers recorded on a 6–point scale (0 meaning low control; 5 meaning high control). All negative–worded items (items 5 and 7–11) were reverse coded to ensure that all responses were in the same direction. The final score ranged from 0 (no control) to 55 (maximum control). In my analyses, scores were calculated if a minimum of 9 out of 11 questions contained valid answers (the average score of non-missing items multiplied by 11). By the approach based on score distribution, PC score was divided into thirds to indicate low (tertile 1), intermediate (tertile 2), and high levels (tertile 3), respectively.

Table 4.4 The perceived control score

1.	At home I feel I have control over what happens in most situations
2.	Keeping healthy depends on things that I can do
3.	There are certain things I can do for myself to reduce the risk of a heart attack
4.	There are certain things I can do for myself to reduce the risk of getting cancer
5.	I feel that what happens in my life is often determined by factors beyond my control
6.	Over the next 5–10 years I expect to have many more good things than bad things happen
7.	I often have the feeling that I am being treated unfairly
8.	In the past 10 years, my life has been full of changes without my knowing what would happen next
9.	I very often have the feeling that there's little meaning in the things I do in my daily life
10.	I sometimes feel as if I've done all there is to do in life
11.	I gave up trying to make big improvements or changes in my life a long time ago

4.3.3 Covariates

(1) Demographics

The subjects were aged 45-69 years old at baseline and were grouped into 5-year age groups. Due to small proportion in 65-69 age group (retired persons were not included), it was incorporated into 60-69 age group. Gender and marital status (married/cohabiting, single, and divorced/separated/widowed) were recorded.

(2) Social position

Education, occupational grade, and material deprivation were used as indicators of social position. *Education* was categorized as: primary/less, vocational (apprenticeship), secondary (A-level equivalent), and university degree. *Occupational grade* was obtained by combing 2 questions about position (higher manager, manager/supervisor, employee, and self-employed) and description of job (sedentary occupation, standing occupation, physical work, and manual work), and it was then categorized as: manager/professional (derived from position), non-manual workers, and manual workers (derived from physical work and manual work).

Material deprivation was assessed by 3 questions about how often the subject's household had difficulties to buy enough food or clothes and to pay bills for electricity, heating and housing. The answers were ‘‘never or almost never’’, ‘‘sometimes’’, ‘‘often’’, and ‘‘always’’ (coded 0 to 3); a deprivation score was derived as the sum of three responses.[545] The score was dichotomized into low (0–3.9) and high deprivation (4–9).

(3) Other psychosocial factors

Depressive symptoms were measured by the Center for Epidemiologic Studies Depression scale (CES-D), consisting of 20 self-reported items. Each item was based on a question “how often you have felt this way during the past week”, rated from 0 (rarely/none of the time) to 3 (most/all of the time).[546] If at least 16 out of 20 items

were answered, the mean of valid questions was multiplied by 20, so the final score has values between 0 and 60. CESD >= 16 was defined as having depression.[547]

Social isolation was constructed by combining 2 questions about the frequency of contact with friends or relatives. People were classified as *socially isolated* if having regular contact with friends or relatives less than once a month.

(4) Self-rated health

It was assessed by a standard single question with answers on a 5-point scale (1= very good, 2= good, 3= average, 4= poor, and 5= very poor).[548] A dichotomized measure of self-rated health was used: very good/good/average and poor/very poor.

4.4 Statistical Power

Since this thesis was based on existing data in the HAPIEE study, sample size could not be influenced; however, statistical power can be estimated. Power is the probability of rejecting a false null hypothesis; it should be close to one. Power analysis was calculated using the formula for logistic regression in software G Power 3.1 based on the following assumptions.[549] (1) Alpha value – the probability of rejecting a true null hypothesis – was set as 0.05. (2) Sample sizes were set as 6000 and 4000, as there were two subsamples on dietary outcomes and drinking/smoking outcomes and men and women were analysed separately. (3) Baseline probability (P0) is the outcome probability at the mean of the predictor X; P0 was set from 0.05 to 0.60 due to the prevalence of health behaviours (outcomes) ranging from 5% to 60%. (4) There is loss of power in multivariate analysis when adjusting for covariates. The adjustment was given for sample size (N_m= N/1 – R^2) where R was the correlation coefficient between X (ER ratio or PC) and covariates (R is estimated as 0.20).

Table 4.5 reports the statistical power of the thesis for several odds ratios and various baseline probabilities at the significance level of 0.05. In the subsample on dietary outcomes (sample size= 6000), the results of power calculation shows that

statistical power is over 99% for odds ratio larger than 1.3 in baseline probability of 0.05. In the subsample on drinking/smoking outcomes (sample size= 4000), the results of power calculation shows that statistical power is over 95% for odds ratio larger than 1.3 in baseline probability of 0.05.

Table 4.5 Power calculation for the two study samples (N= 6000 and 4000)

Baseline Probability	Examples of outcomes	Odds Ratio (OR) 1.1	1.2	1.3	1.4
Sample size = 6000					
0.60	Polyunsaturated fat Fruit and vegetable	94 %	> 99 %	> 99 %	> 99 %
0.30	Heavy drinking in men Smoking status in men	91 %	> 99 %	> 99 %	> 99 %
0.20	Heavy drinking in women Smoking status in women	82 %	> 99 %	> 99 %	> 99 %
0.10	Binge drinking in men Problem drinking in men	58 %	98 %	> 99 %	> 99 %
0.05	Binge drinking in women Problem drinking in women Healthy Diet Indicator 5–9 Saturated fat	36 %	87 %	99 %	> 99 %
Sample size = 4000					
0.60	Polyunsaturated fat Fruit and vegetable	82 %	> 99 %	> 99 %	> 99 %
0.30	Heavy drinking in men Smoking status in men	77 %	> 99 %	> 99 %	> 99 %
0.20	Heavy drinking in women Smoking status in women	66 %	99 %	> 99 %	> 99 %
0.10	Binge drinking in men Problem drinking in men	43 %	92 %	> 99 %	> 99 %
0.05	Binge drinking in women Problem drinking in women Healthy Diet Indicator 5–9 Saturated fat	26 %	71 %	95 %	> 99 %

4.5 Statistical Analysis

The statistical analyses for drinking, smoking and dietary outcomes are described in detail in Chapter 5, 6 and 7, respectively. In general, descriptive statistics with the percentages and means for the covariates and the outcomes by country and by gender

were presented. Bivariate analyses for the associations between covariates and outcomes were conducted in men and women, respectively; chi–squared tests were used to examine the significance of differences between categories of the variable. To assess whether data of three countries would be pooled for further analyses, crude associations between exposure variables and outcomes in country–specific strata were assessed. By comparing log likelihoods for the model with the interaction term (between country and exposure variable) and the model without, likelihood–ratio (LR) test was used to test the significance of this interaction term.

The associations between exposure variables (ER ratio, OC and PC) and outcomes (drinking, smoking and diet) were evaluated, respectively. For binary categorical outcomes, the associations between exposure variables and outcomes were assessed by binary logistic regression; for ordinal categorical outcomes (smoking intensity and HDI), the associations between exposure variables and outcomes were assessed by ordinal logistic regression. These associations were assessed after adjustment for age and country (model 1) and after additional adjustment for other covariates (model 2). All above analyses were conducted by STATA 11 (Stata Corp LP, College Station, USA).

For assessing modifying role, log likelihoods for the model with the corresponding interaction term and the model without were compared, and LR test was used to test the significance of this interaction term. For assessing antecedent or mediator roles, the techniques of structural equation modelling (SEM) were applied by software Mplus 7; specific SEM models used for each study outcome are described in detail in Chapter 5, 6 and 7. The general methodology of SEM that will be applied in my analyses is explained in the next section.

4.6 Methodology for Structural Equation Modelling

Structural equation modelling (SEM) comprises two components, a measurement

model (derived from confirmatory factor analysis) and a structural model (derived from path analysis). First, a measurement model relates one or several observed variables to a latent variable, which refers to a theoretical construct that cannot be directly measured. This origin is traced to Spearman (1904) who developed the techniques of factor analysis.[550] Second, a structural model specifies relation among these latent variables and regressions of latent variables on observed variables. This origin comes from path analysis proposed by Wright (1921).[551] Factor analysis and path analysis were integrated by Joreskog (1970), who developed the first SEM software LISREL.[552] The 1980s and 1990s witnessed the development of computer programs and rapid expansion of SEM techniques. For example, Muthen (1984) developed the software Mplus and extended the applications of SEM to non-normal data (e.g. categorical variables).[553] SEM has also been applied in advanced statistical literature, such as generalized linear models or multilevel analysis.

4.6.1 Introduction to path analysis

Path analysis is a statistical technique that uses both bivariate analysis and linear regression analysis to test causal relation among the variables specified in a model. It involves 3 major steps: (1) a path diagram is drawn based on a theory or a set of hypotheses; (2) path coefficients are calculated using regression techniques; (3) total effect is decomposed into direct and indirect effects. Justification for adopting path analysis in this thesis is that, compared to regression analysis, path analysis allows simultaneous examination of several causal processes underlying observed relationships and comparison of relative importance of each path. Here, the single–mediator model is adopted to introduce path analysis and mediation analysis (Figure 4.3) with the following three equations:

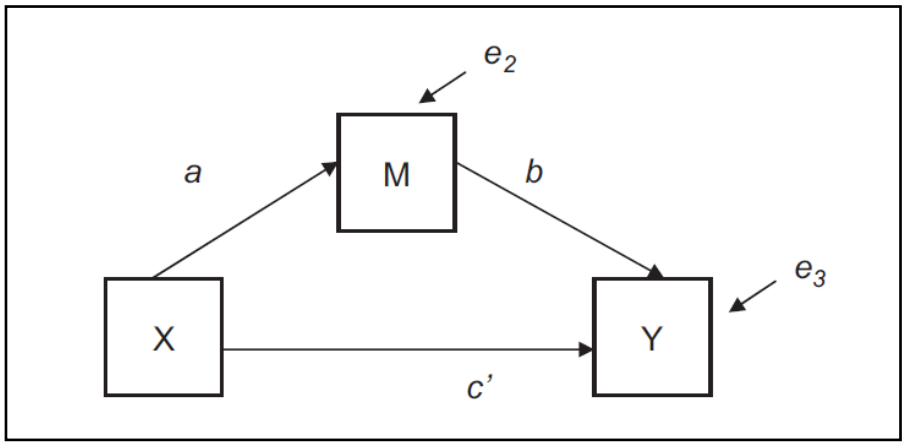
$$Y = i_1 + c\,X + e_1$$

$$Y = i_2 + c'\,X + b\,M + e_2$$

$M = i_3 + a\,X + e_3$

Where Y is the dependent variable, X is the antecedent variable, and M is the mediator variable. Path coefficients (a, b and c') obtained by regression analyses are numerical estimates of the causal relationships between variables; they are interpreted as the amount of expected change in dependent variable due to one–unit change in independent variable. The coefficient c represents the strength of prediction of Y from X; the coefficient c' represents the strength of prediction of Y from X, with the strength of M–to–Y relation removed. Next, b is the coefficient for the strength of prediction of Y from M, with the strength of X–to–Y relation removed; a is the coefficient for the strength of prediction of M from X. The intercepts in each equation (i_1, i_2 and i_3) represent the average score of each variable, respectively. The errors in each equation (e_1, e_2 and e_3) refer to the part of relationships that cannot be predicted.[554]

Figure 4.3 The single–mediator model adopted to introduce path analysis



In the model, total effect of X on Y (c) is decomposed into two parts: direct effect and indirect effect. First, a direct effect of X on Y with the strength of mediated relation removed, is represented and quantified by c'. Second, an indirect (mediated) effect of X on Y transmitted via the mediator variable, is quantified by (ab) or (c – c'). The numerical values of the mediated effect is computed by either the product of coefficients (ab) or the difference in coefficients (c – c').[555]

A mediated effect should be evaluated by the effect size and the statistical

significance. Measures of effect size provide an indication of the size and meaningfulness of an effect that does not depend on sample size. First, a unstandardized or standardized path coefficient can serve as an effect size measure for the path. Second, another common measure for effect size is the proportion of total effect that is mediated (ab / ab + c'); for instance, a researcher can state that a mediated effect explains 30% of the total effect of a predictor on an outcome.

The tests for statistical significance aid in the evaluation of whether a mediated effect is larger than expected by chance alone. One can test the null hypothesis that the indirect effect coefficient is zero in the population from which the sample data were drawn. For significance testing, test statistic is computed by dividing the product of coefficients (indirect effect) by its standard error. Sobel (1982) derived the asymptotic standard error of indirect effect using the multivariate delta method; the standard error of the mediated effect is:[556]

$$\sqrt{\alpha^2\sigma_\beta^2 + \beta^2\sigma_\alpha^2}$$

Where α is the unstandardized regression coefficient for predicting M from X, σ_α^2 is the standard error for that coefficient, β is the unstandardized regression coefficient for predicting Y from M controlling for X, and σ_β^2 is the standard error for that coefficient. The indirect effect is divided by the standard error, which is then compared to a standard normal distribution to test for significance (*H*o: $\alpha\beta = 0$).[557]

The product of two normally distributed random variables is normally distributed only in special cases, which explains the inaccuracy for assessing significance of mediation based on normal distribution (e.g. Sobel test).[558] The simulation study showed that sample sizes of 1,000 were needed for product of coefficients methods to have Type I error rates below 0.05 and adequate power to detect small effects.[559] Due to inaccuracy in assessing significance of mediation based on normal distribution, MacKinnon et al recommended to evaluate significance testing by "distribution of

product" approach or "bootstrap method" in the studies with small sample sizes or with more complicated models (e.g. multiple mediators or categorical outcomes).[560] These two approaches are discussed below.

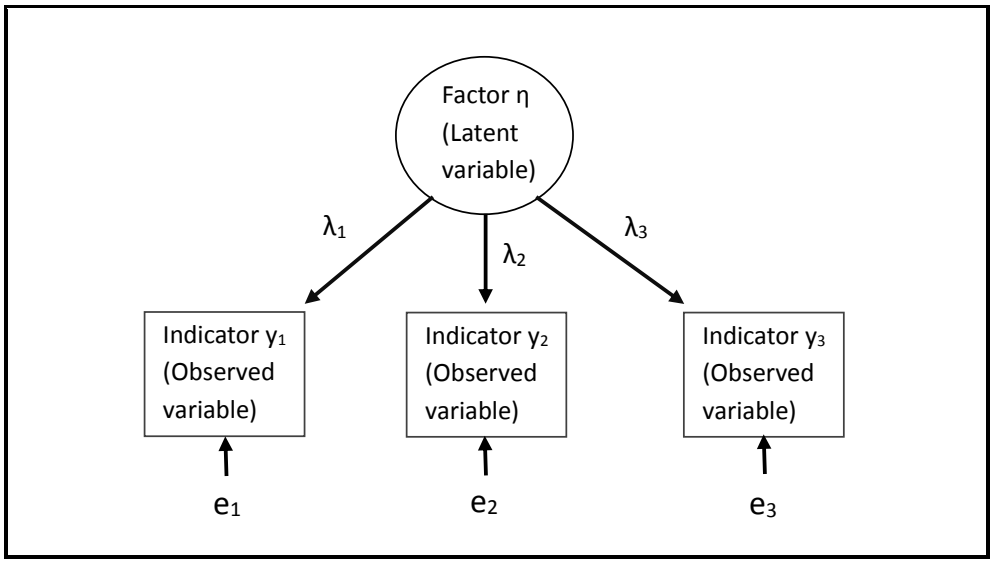
First, "distribution of product" approach bases inference on a mathematical derivation of the distribution of product of two normally–distributed variables; the distribution of product of regression coefficients is often asymmetric with high kurtosis. MacKinnon et al conducted extensive simulations to estimate the empirical sampling distribution of product. On the basis of these empirical sampling distributions, critical values of the product distribution for different significance levels were determined.[561]

Second, "bootstrap method" is a nonparametric resampling procedure widely used for testing mediation; it does not impose the assumption of normal distribution. The bootstrap method is a computationally intensive method that involves repeatedly sampling from the data set and estimating the indirect effect in each resampled data set. By repeating this process typically at least 1,000 times, an empirical approximation of sampling distribution of the product of regression coefficients is built and used to estimate confidence intervals and significance levels for the indirect effect.[562] In this thesis, I decided to test the significance of indirect effect by bootstrap method, because these SEM models are complicated models (categorical outcomes and multiple mediators) and the assumption of normal distribution may not be met.

4.6.2 Introduction to confirmatory factor analysis

As mentioned earlier, SEM comprises two components, a measurement model (derived from confirmatory factor analysis) and a structural model (derived from path analysis). Confirmatory factor analysis (CFA) analyzes a priori measurement model in which the number of factors and their correspondence with indicators are explicitly specified. A measurement model with one factor and three indicators is adopted to introduce parameter estimation in CFA (Figure 4.4).

Figure 4.4 A measurement model with one factor and three indicators



In the above measurement model, a latent continuous variable (factor η) is denoted by the three observed variables (indicators y_1, y_2 and y_3). This measurement model is also expressed by the following 3 equations

$y_1 = a_1 + \lambda_1 \eta + e_1$

$y_2 = a_2 + \lambda_2 \eta + e_2$

$y_3 = a_3 + \lambda_3 \eta + e_3$

Where a_1 to a_3 are intercepts that give the expected value of each y when the latent variable η is zero; e_1 to e_3 are unique variances. Factor loadings (λ_1, λ_2 and λ_3) are used to estimate the direct effects of a factor on each indicator, respectively, and are interpreted as regression coefficients. For example, if unstandardized factor loading is 1.5 for direct effect from the factor to the indicator, then 1–unit increase in the factor is associated with 1.5 unit of increase in the indicator. In general, one factor loading is fixed to 1.0 to scale the corresponding factor in unstandardized solution and is not tested for statistical significance due to no standard error.

Standardized factor loadings are estimated correlation between the indicators and its factor, when indicators are specified to load on a single factor. Therefore, squared standardized factor loadings are proportions of explained variance (R^2). For instance,

if a standardized factor loading is 0.7, the factor explain 49% ($0.7^2 = 0.49$) of the variance of the indicator. Ideally, a CFA model can explain the majority of the variance ($R^2 > 0.50$) of each indicator (standardized factor loading > 0.7).

Each measurement error term (e_1, e_2, and e_3) for each indicator represents unique variance, which is the indicator variance not explained by the factor. Like disturbances in path analyses, measurement errors are proxy variables for all sources of residual variation not explained by the model. Two types of unique variance are: random error (score unreliability) and all sources of systematic variance not due to the factor. The ratio of an unstandardized measurement error variance over the observed variance of the corresponding indicator equals the proportion of unexplained variance, and one minus this ratio is the proportion of explained variance (R^2, squared standardized factor loadings). Thus, unique variance or proportion of unexplained variance is estimated as ($1 – R^2$).

4.6.3 Basic steps in Structural Equation Modelling

Kline proposed that six basic steps are required in the applications of SEM.[563] Specification, the first step, means representation of a researcher's hypotheses in the form of a structural equation model. A researcher assumes the relationships among observed variables and latent variables based on literature, and draws a measurement model (confirmatory factor analysis) and a structural model (path analysis) to represent the presumed relationships. Specification includes selection of variables, directionality of causal effects, parameter status, or type of structural models. Specification requires a series of thoughtful decision; for example, these options should be considered when specifying the directionality of a causal effect: (1) to specify and test alternative models, each with different causal directionality between the two variables; (2) to include reciprocal effects to cover both possibilities; (3) to specify a model without directionality between the two variables, which are specified to be

correlated with each other.

The second step, identification, means going from the known information to the unknown parameters. A model is “identified” if it is theoretically possible to derive a unique estimate for every parameter (unknown information) based on the number of elements in observed variance – covariance matrix (known information). Identification is a property of a model, rather than the data. The difference between the known versus unknown information typically equals degrees of freedom, which should not be less than zero. There are several rules of identification for different structural and measurement models that should be paid attention before conducting SEM.

The third step is measure selection and data collection. A researcher should select a good measure for intended construct based on score reliability and validity. Before analyzing SEM, original data should be screened for problems of co–linearity, outlier, missing data, and normality. The default estimation in SEM is maximum likelihood (ML) which assumes all variables are continuous and normally distributed. If this assumption is violated (e.g. categorical outcomes), a researcher needs to consider other techniques described later.

The fourth step, estimation, involves using an SEM computer tool to conduct the following analyses: (1) Evaluation for model fit, which means to determine how well the model explains the data. If the initial model does not fit the data very well, the researcher should go to the fifth step “re-specification”. (2) Interpretation for parameter estimates, which includes magnitude, directionality and significance of each path coefficient, or decomposition of total effect. (3) Consider an equivalent model, which explains the data as well as the researcher’s preferred model with a different configuration of hypothesized relationships among the same variables.

The fifth step is re-specification. A researcher usually arrives at this step because the fit of the initial model is poor. Then, a new model should be re–specified based on theoretical consideration. Any re–specified model should go through the previous steps from identification to estimation.

The sixth step is to completely and accurately describe the SEM analysis in written reports. Researchers can refer to published guidelines for reporting results of SEM.[564]

The evaluation of model fit in the fourth step is introduced in the following paragraphs. The original test for overall model fit is chi-squared test, a test of whether the covariance matrix implied by hypothesized model is close enough to sample covariance matrix. However, chi-squared test is too sensitive to sample size. In a large sample (> 5000), chi-squared test may be significant even though only trivial differences exist between observed and predicted covariance.

RMSEA (Root Mean Square Error of Approximation) is a parsimony-adjusted index that theoretically follows a non-central chi-squared distribution, where non-centrality parameter allows for discrepancies between model-implied and sample covariance up to the level of expected values of chi-squared or degrees of freedom. RMSEA is scaled as a badness-of-fit index where declining values indicate improving fit (zero for a perfect fit). RMSEA less than 0.06 indicates “good fit”, and RMSEA 0.06–0.08 is considered “acceptable fit”.

CFI (Comparative Fit Index) is an incremental fit index which measures the relative improvement in the fit of hypothesized model compared to that of a baseline model (independent model that assumes zero covariance among observed variables). TLI (Tucker-Lewis Index) compares the mean square (the sum of squares divided by degrees of freedom) for the hypothesized model to that for a baseline model. TLI gives the distance between the baseline and target models as a proportion of distance between baseline and true models. Both CFI and TLI are goodness-of-fit indexes where increasing values indicate better fit (ranged between 0 and 1).[565]

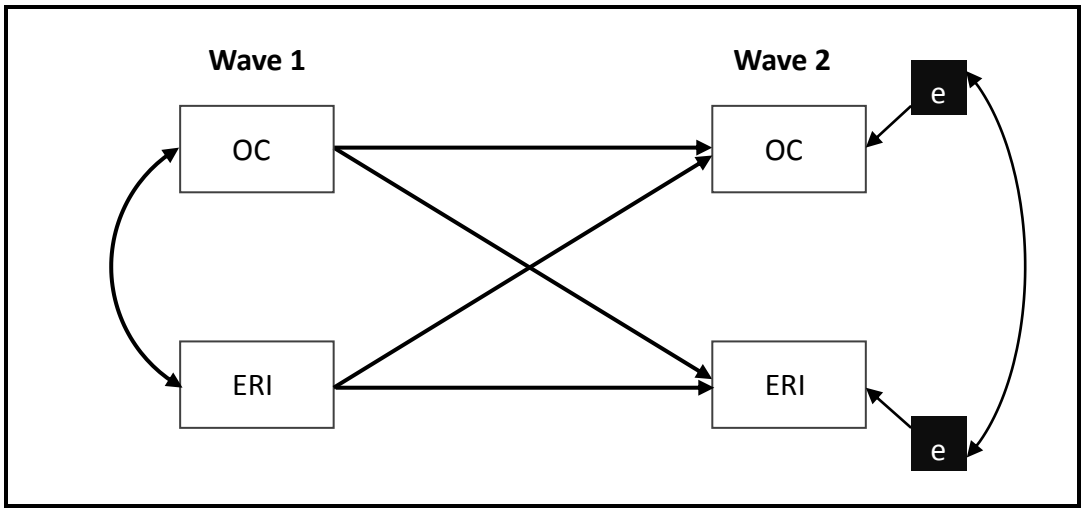
Researchers have not reached common consensus on the criteria for model fit. Hu and Bentler proposed a set of practical criteria for “good fit” of the model, including: RMSEA less than 0.06, CFI more than 0.95, and TLI more than 0.95. In this thesis, the proposed cutoff values are adopted as the guidelines for overall fit of the model.[566]

4.6.4 Structural Equation Modelling in cohort studies

SEM in longitudinal data has a rich history with the development of new analytical models and technological innovations to collect data over time. Autoregressive models were seen as the “gold standard” of methodology for analyzing longitudinal data prior to the development of latent growth curve models.[567] To date, autoregressive models are still useful for many important questions in longitudinal studies. “Autoregressive” means regressed on itself, so each variable is predicted by the same variable at an early wave; the stability of this variable (the extent to which the mean of a measure is the same across time) is evaluated by the strength of path coefficient of the same variable connecting two waves. By adjusting prior levels of the dependent variable, any unmeasured exogenous variable (confounder) that correlates with the predictor and the dependent variable can be controlled.

An autoregressive model simply assesses how a construct changes over time. The common application of an autoregressive model with a cross–lagged panel design examines how a construct changes and covariates across time “with other constructs”. The cross–lagged panel design involves the following features. First, each variable is modeled with an autoregressive structure; for example, OC at wave 2 is predicted by OC at wave 1. Second, longitudinal relation consistent with longitudinal mediation are present among the variables. For instance, the cross–lagged effects of “OC at wave 1 on ERI at wave 2” and “ERI at wave 1 on OC at wave 2” are measured. These cross–lagged relationships help to identify the directionality of potentially causal relationships. Third, although this model would not include contemporaneous causal relation among variables at the same wave, covariances among the variables at wave 1 and covariances among residual variances of the variables at wave 2 are included. The model recognizes that there are correlation among variables at the same wave but direction of relationships are unknown (Figure 4.5).[568]

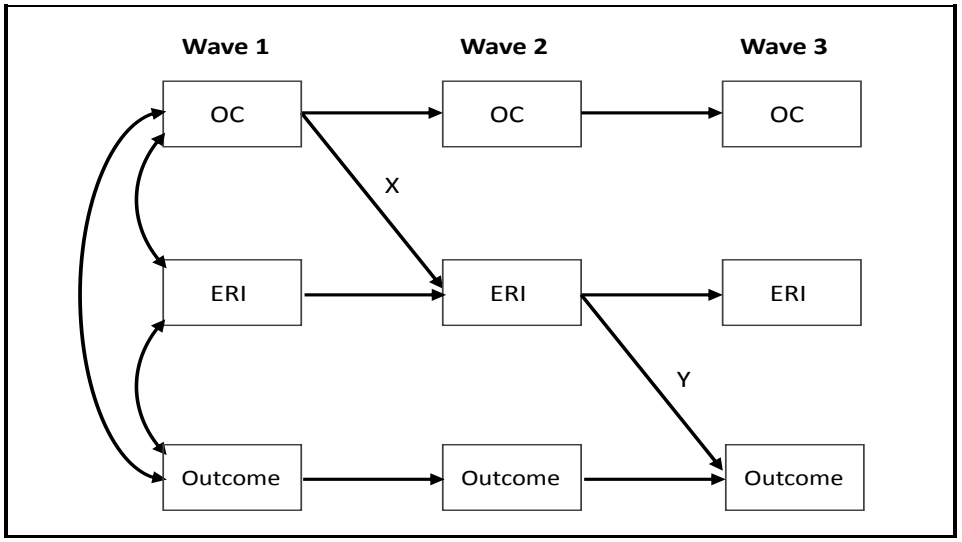
Figure 4.5 An autoregressive model with a cross–lagged panel design



e = residual variance of the variable

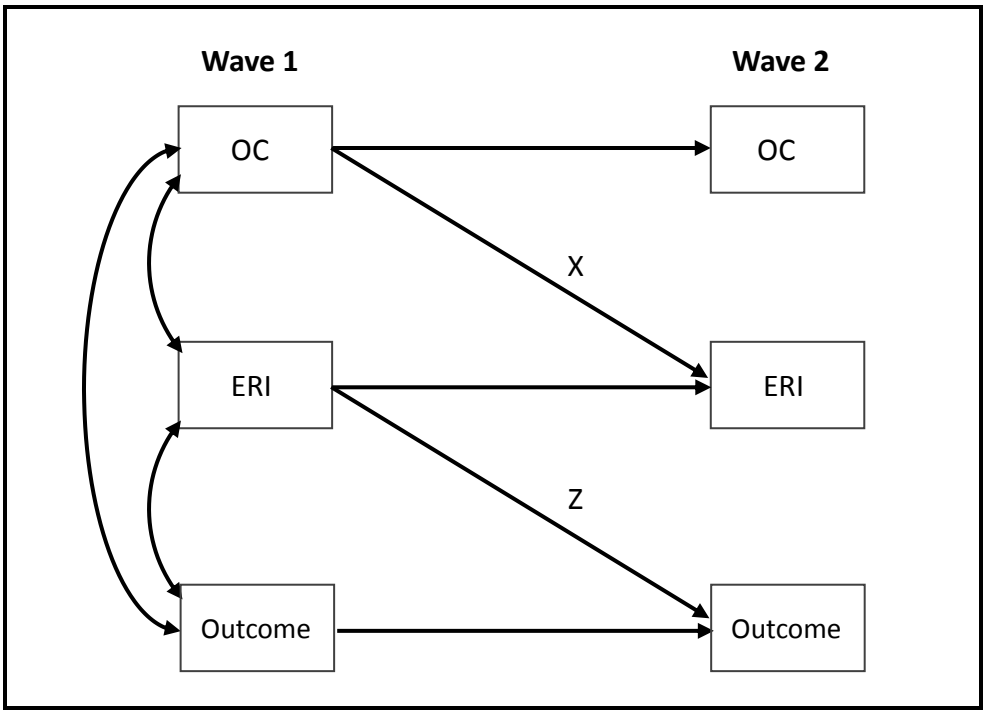
In terms of mediation analyses, ideally, a 3–wave cohort design is required to estimate a mediation model; the exposure variable should precede the mediator in time, and the mediator should precede the outcome in time. For example, if the mediation process is suggested as OC → ERI → outcome, the effect of OC at wave 1 on ERI at wave 2 (X), and the effect of ERI at wave 2 on outcome at wave 3 (Y) would be assessed. This mediator effect of ERI is estimated by multiplying the two cross–lagged effects (X * Y) (Figure 4.6). However, multiphase longitudinal studies are relatively rare in occupational health research, and a 2–wave cohort design still can test the significance of partial mediation.

Figure 4.6 A three–wave cohort design to estimate a mediation model



The distinction between partial mediation and full mediation is summarized. Baron and Kenny proposed a simple procedure to test whether a variable B acts as a mediator of the effect of a predictor A on an outcome C. The following assumptions need to be satisfied: (1) the association between A and C is statistically significant; (2) A and B are related; (3) B is significantly associated with C, after control for A; (4) the association *ac* between A and C is weaker when B is controlled, compared with the situation when B is not controlled. If *ac* becomes not significant after control for B, B fully mediates the relationship between A and C. If *ac* is weaker but still significant, B partially mediates the relationship between A and C.[569]

Figure 4.7 A two–wave cohort design to estimate a mediation model



In terms of a 2–wave cohort design, Taris and Kompier (2006) proposed that as mediation is a causal chain involving at least two causal relationships, these causal relationships can be tested separately using only two phases. For example, if the mediation process is suggested as: OC → ERI → outcome, the effect of OC at wave 1 on ERI at wave 2 (X) and the effect of ERI at wave 1 on outcome at wave 2 (Z) can be assessed (Figure 4.7). Partial mediation applies if both causal relationships are

confirmed; the product of two path coefficients (X * Z) can estimate the strength of the mediator effect.[570] Note that full mediation cannot be examined in a two–wave design; with only 2 phases of data, it is impossible to test whether the relationship between OC at wave 1 and outcome at wave 3 (Figure 4.6) is fully mediated by ERI at wave 2.

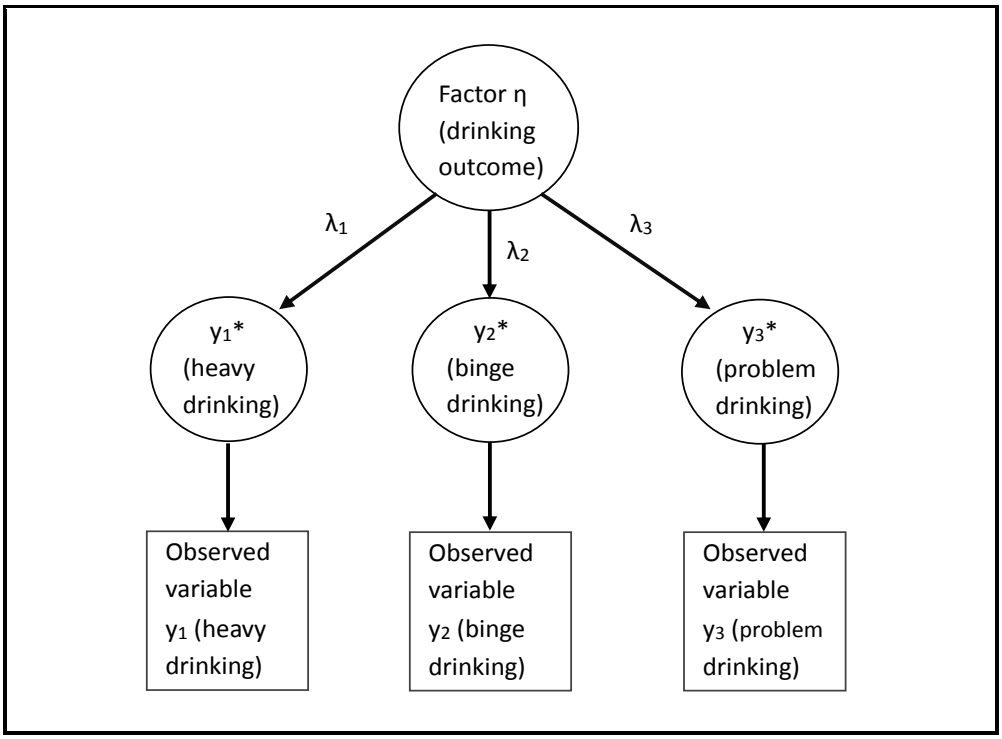
Although a 3–wave cohort study provides the best estimation for mediation, a 2–wave cohort design can offer indication for the presence of partial mediation. The evaluation for partial mediation by a psychosocial factor is reasonable, as there are often several psychosocial factors identified to influence a health outcome. Cole and Maxwell (2004) suggested that a 2–wave cohort design yielded better evidence than a cross–sectional study or a half–longitudinal design (one of the associations between predictor, mediator and outcome is cross–sectional) based on two reasons.[571] First, failing to control for prior levels of dependent variables often creates problems of unmeasured confounders. Second, a cross–sectional design is limited by difficulties in determining causal sequence. In a cross–sectional mediation study with 3 variables, for example, there are 6 possible causal sequences (3 x 2 x 1). Many researchers test only one causal sequence that fits their proposed theory, but the other sequences are neglected. Indeed, it is difficult to argue a priori that one particular causal sequence is plausible while others are not, if temporal sequence cannot be determined.[572]

4.6.5 Structural Equation Modelling with categorical outcomes

The most common method used in SEM to account for nonlinear relationship between observed categorical variables with a latent continuous variable is summarized; the hypothetical model is illustrated (Figure 4.8). A single latent variable (factor η) underlies the relationship among 3 observed categorical variables (y_1, y_2 and y_3). To solve the problem of nonlinear relationships, it is assumed that there are 3 continuous latent response variables (y_1*, y_2* and y_3*) underlying each observed categorical variable. Note that factor loadings are represented by λ_1, λ_2 and λ_3,

respectively; 1–unit increase in the latent factor (η) results in λ_1, λ_2 and λ_3 unit of increase in latent response variables y_1^*, y_2^* and y_3^*, respectively. The next question is how to obtain the latent response variable from the observed categorical variable.[573]

Figure 4.8 A measurement model for relationships between a latent continuous factor and three observed categorical variables



Muthen described the approach to estimating SEM models with any combination of continuous, dichotomous or ordinal categorical outcomes.[574,575] In the formulation, a continuous latent response variable y* expresses the amount required to respond in certain categories of an observed categorical variable. For simplicity, consider the one factor model for the continuous latent response variable y^*_i for individual i:

$$y_i^* = \nu + \lambda\, \eta_i + \epsilon_i,$$

Where ν is an intercept parameter, λ is a factor loading, η is a factor variable, and ϵ is a residual. The expectation μ* and variance σ* of y* are

$$\mu^* = \nu + \lambda\, \alpha,$$
$$\sigma^* = \lambda^2\, \psi + \theta,$$

Where α is the mean of η, ψ is the variance of η, and θ is the variance of the residual ϵ. Next, the latent response variable y* is related to the observed categorical variable y via the threshold model:

$$y_i = \begin{cases} C_i - 1, & \text{if } \tau_{i,C_i-1} < y_i^* \\ C_i - 2, & \text{if } \tau_{i,C_i-2} < y_i^* \le \tau_{i,C_i-1} \\ \vdots & \\ 1, & \text{if } \tau_{i,1} < y_i^* \le \tau_{i,2} \\ 0, & \text{if } y_i^* \le \tau_{i,1} \end{cases}$$

Where C denotes the number of categories in an observed categorical variable; τ_i denotes the location of the cut point (called threshold), with $\tau_0 = -\infty$ and $\tau_c = \infty$. This also leads to the conditional probability expression:

$$P(y \ge c|\eta) = F[-(\tau_c - \nu - \lambda\,\eta)\,\theta^{-1/2}],$$

Where *F* is typically chosen as a standard normal (probit) or logistic distribution function depending on the distributional assumption for ϵ. By the above procedures, an observed categorical variable is linked with a latent response variable by a conditional probability model (probit or logistic function).[576]

For mediation analysis in categorical variables, probit models are preferable to logistic modes for better mathematical tractability in rescaling and estimation. Probit models assume that transformation function is the cumulative density function of standard normal distribution, but logit models assume that transformation function is the logistic function. Probit and logit models are almost identical and they produce similar results; however, their β coefficients are scaled differently. In probit models, random error is assumed to be distributed normally with variance 1; in logit models, random error is assumed to be distributed logistically with variance $\pi^2 / 3$.[577] That is, probit analysis sets the standard deviation of error as 1, but logit analysis sets the standard deviation of error as 1.814 ($\pi / \sqrt{3}$). Thus, probit coefficient multiplied by 1.8 is approximately the same as logit coefficient, but they vary slightly due to small

differences between logistic distribution and standard normal distribution.[578]

For path analysis in linear regression, total effects (c) can be decomposed into direct effect (c') and indirect effects (a x b). In logit or probit models, (c – c') is not always equal to (a x b) due to fixed residual variance.[579] Importantly, Mplus software uses probit regression to estimate thresholds for categorical outcomes and provides more complicated iterative approaches based on multivariate probit distribution; as the model is standardized as part of the analysis, scaling problems can be solved. Thus, (c – c') becomes approximately equal to (a x b). However, this solution cannot make mediation analysis in logit or probit models as accurate as linear regression. Probit models produce more similar results to linear regression than logit models.[580]

Another challenge for SEM with categorical variables is estimation; maximum likelihood (ML) estimator assuming that data follow normal distribution is not accurate. One solution is limited–information estimator which uses a summary (e.g. variance) of available data. To correct for non-normal distribution of observed data, a weight matrix is used in conjunction with a least squares estimator, which chooses parameter values to minimize the distance between what is observed (data) and what is expected (model-implied covariance matrix); this combination is weighted least squares (WLS). The data matrix is an asymptotic correlation matrix of latent response variable: tetrachoric correlation matrix for dichotomous variables and polychoric correlation matrix for ordinal categorical variables. In Mplus, WLS estimator applied to ordered categorical outcomes is mean– and variance–adjusted weighted least squares (WLSMV) in probit models. WLSMV generally performs well if the sample size is larger than 200 and the distribution on ordered variables is not markedly skewed.[581]

Chapter 5. Drinking Outcomes

The aims of analyses for drinking outcomes in Chapter 5, in line with the aims listed in Chapter 3, include: (1) to examine the potential role of OC in ERI–drinking relationship, including modifying, antecedent, mediator, or direct effects; (2) to investigate the potential role of PC in the relationship between OC, ERI and drinking outcomes. The analyses use data from a 2–wave cohort study (3782 men and 3731 women aged 45–69), part of the HAPIEE study, which has been described in detail in Chapter 4 Methodology.

The results are presented in three parts. First, descriptive statistics for covariates and drinking outcomes by country and by gender are presented. Second, the associations of ERI and OC at wave 1 with drinking outcomes at wave 2, respectively, are assessed by logistic regression. Structural equation modelling (SEM) with an autoregressive and cross–lagged model is applied to examine antecedent or mediator roles of OC in ERI–drinking relationship. Modifying roles of OC in ERI–drinking relationship are also tested. Third, the associations between PC at wave 1 and drinking outcomes at wave 2 are evaluated by logistic regression. SEM with an autoregressive and cross–lagged model is used to examine the relationship between OC, ERI, PC, and drinking outcomes. Modifying effects of PC in ERI–drinking relation are also tested.

5.1 Descriptive Statistics

5.1.1 Descriptive characteristics of study populations

In this sample of 7513 subjects (3782 men and 3731 women), the means of age at wave 1 are 54.8 years (standard deviation= 6.0) in men and 53.2 years (standard deviation= 5.4) in women. The average follow–up periods between wave 1 and wave 2 are 3.5 years (standard deviation= 0.7) in men and 3.6 years (standard deviation=

0.6) in women. Descriptive statistics with percentages and means for covariates by country and by gender are presented (Table 5.1).

Table 5.1 Descriptive statistics of study sample by country and gender (N= 7513)

Variable	Czech Republic		Russia		Poland	
	Men (n= 1082)	Women (n= 1099)	Men (n= 1402)	Women (n= 1394)	Men (n= 1298)	Women (n=1238)
Age, N (%)						
45 – 49	279 (25.8)	372 (33.9)	300 (21.4)	408 (29.3)	357 (27.5)	461 (37.2)
50 – 54	306 (28.3)	422 (38.4)	353 (25.2)	453 (32.5)	380 (29.3)	423 (34.2)
55 – 59	313 (28.9)	202 (18.4)	387 (27.6)	314 (22.5)	317 (24.4)	230 (18.6)
60 – 69	184 (17.0)	103 (9.3)	362 (25.8)	219 (15.7)	244 (18.8)	124 (10.0)
Education, N (%)						
Primary or less	30 (2.8)	82 (7.5)	81 (5.8)	54 (3.9)	45 (3.5)	47 (3.8)
Vocational	391 (36.1)	277 (25.2)	324 (23.1)	454 (32.6)	256 (19.7)	141 (11.4)
Secondary	382 (35.3)	547 (49.8)	464 (33.1)	395 (28.3)	396 (30.5)	517 (41.8)
University	279 (25.8)	193 (17.6)	533 (38.0)	491 (35.2)	601 (46.3)	532 (43.0)
Occupation, N (%)						
Manager/ profession	295 (27.3)	186 (16.9)	391 (27.9)	276 (19.8)	393 (30.3)	223 (18.0)
Non-manual worker	461 (42.6)	727 (66.2)	479 (34.2)	843 (60.5)	650 (50.1)	846 (68.3)
Manual worker	326 (30.1)	186 (16.9)	531 (37.9)	275 (19.7)	254 (19.6)	170 (13.7)
Marital status, N (%)						
Married/ cohabiting	926 (85.6)	790 (71.9)	1279 (91.2)	885 (63.5)	1188 (91.5)	875 (70.7)
Single	30 (2.8)	31 (2.8)	35 (2.5)	85 (6.1)	43 (3.3)	104 (8.4)
Divorce/ widowed	126 (11.6)	278 (25.3)	88 (6.3)	424 (30.4)	67 (5.2)	259 (20.9)
Deprivation, N (%)						
Low (0 – 3.9)	946 (87.4)	909 (82.7)	1011 (72.1)	740 (53.1)	1057 (81.4)	929 (75.0)
High (4 – 9)	136 (12.6)	190 (17.3)	391 (27.9)	654 (46.9)	241 (18.6)	309 (25.0)
Depression, N (%)						
CESD < 16	974 (90.0)	885 (80.5)	1214 (86.6)	1013 (72.7)	1118 (86.1)	935 (75.5)
CESD >= 16	108 (10.0)	214 (19.5)	188 (13.4)	381 (27.3)	180 (13.9)	303 (24.5)
Social isolation, N (%)						
No (>= once a month)	699 (64.6)	789 (71.8)	632 (45.1)	655 (47.0)	597 (46.0)	614 (49.6)
Yes (< once a month)	383 (35.4)	310 (28.2)	770 (54.9)	739 (53.0)	701 (54.0)	624 (50.4)
Self-rated health, N (%)						
Very good – average	1019 (94.2)	1045 (95.1)	1290 (92.0)	1147 (82.3)	1220 (94.0)	1165 (94.1)
Poor – very poor	63 (5.8)	54 (4.9)	112 (8.0)	247 (17.7)	78 (6.0)	73 (5.9)

There are gender differences across three countries. Compared to women, men have higher proportions in age group over 55, university–educated, manager/

profession and manual workers, and married/ cohabiting; men also have lower deprivation, less depressive symptoms, and more social isolation than women.

There are country differences observed in both genders (Table 5.1). For age, Russian samples are older than Czech and Polish samples. University education in Czech samples is the least common of all countries, and Czech samples have the greatest gender inequality in education. High deprivation in Russia is the most prevalent of all countries. The proportions of depression and social isolation in Czech Republic are the lowest among all countries.

5.1.2 Descriptive characteristics of drinking outcomes

Descriptive statistics of three drinking outcomes (binge drinking, heavy drinking, and problem drinking) by country and by gender at wave 1 and 2, respectively, are shown (Table 5.2). There are gender differences across three countries. Men have remarkably higer proportions in binge drinking, heavy drinking, and problem drinking than women.

In addition, there are country differences seen in Table 5.2. At wave 1, the reported levels of annual alcohol intake in Czech men and women are about 1.5–2.0 times the levels in Russian and Polish people. The percentages of binge drinking are highest for Russian sample (14.3%) in men and highest for Czech sample (4.9%) in women. The percentages of heavy drinking are highest for Russian sample in men (38.7%) and women (16.3%). The percentages of problem drinking are highest for Russian sample in men (18.6%) and highest for Czech sample in women (4.1%). At wave 2, the highest percentages of three drinking outcomes in men and women, respectively, are in the same countries as those at wave 1.

Table 5.2 Descriptive statistics of drinking outcomes by country and gender

Variables	Czech		Russia		Poland	
	Men (n= 1082)	Women (n= 1099)	Men (n= 1402)	Women (n= 1394)	Men (n= 1298)	Women (n=1238)
Wave 1						
Annual alcohol intake, mean (g)	6146.1	1683.5	4992.8	756.7	3628.8	769.1
Annual drinking occasion, mean	142.6	64.5	82.8	24.7	94.5	34.8
Dose per occasion, mean (g)	43.1	26.1	60.3	30.6	38.4	22.1
1. Binge drinking, n (%)						
Yes	81 (7.5)	54 (4.9)	200 (14.3)	51 (3.7)	52 (4.1)	40 (3.2)
No	1001 (92.5)	1045 (95.1)	1202 (85.7)	1343 (96.3)	1245 (95.9)	1198 (96.8)
2. Heavy drinking, n (%)						
Yes	237 (21.9)	136 (12.4)	543 (38.7)	227 (16.3)	218 (16.8)	124 (10.0)
No	845 (78.1)	963 (87.6)	859 (61.3)	1167 (83.7)	1080 (83.2)	1114 (90.0)
3. Problem drinking, n (%)						
Yes	118 (10.9)	45 (4.1)	261 (18.6)	51 (3.6)	131 (10.1)	38 (3.1)
No	964 (89.1)	1054 (95.9)	1141 (81.4)	1343 (96.4)	1167 (89.9)	1200 (96.9)
Wave 2						
Annual alcohol intake, mean (g)	6024.5	1480.8	4625.3	809.1	4008.6	871.9
Annual drinking occasion, mean	130.4	58.3	79.2	25.8	94.1	37.1
Dose per occasion, mean (g)	46.2	25.4	58.4	31.4	42.6	23.5
1. Binge drinking, n (%)						
Yes	98 (9.1)	49 (4.5)	195 (13.9)	59 (4.2)	83 (6.4)	46 (3.7)
No	984 (90.9)	1050 (95.5)	1207 (86.1)	1335 (95.8)	1215 (93.6)	1192 (96.3)
2. Heavy drinking, n (%)						
Yes	306 (28.3)	178 (16.2)	513 (36.6)	287 (20.6)	301 (23.2)	183 (14.8)
No	776 (71.7)	921 (83.8)	889 (63.4)	1107 (79.4)	997 (76.8)	1055 (85.2)
3. Problem drinking, n (%)						
Yes	95 (8.8)	47 (4.3)	269 (19.2)	58 (4.2)	153 (11.8)	47 (3.8)
No	987 (91.2)	1052 (95.7)	1133 (80.8)	1336 (95.8)	1145 (88.2)	1191 (96.2)

The ways of pooling the data in subsequent analyses are described. First, men and women are analysed separately as most studies on the associations between psychosocial factors and health outcomes. Second, crude associations between exposure variables (ER ratio and OC) at wave 1 and drinking outcomes at wave 2 in country–specific strata are assessed. Next, logistic regression analyses are conducted for 3 drinking outcomes, respectively, regressed by country, ER–ratio tertile and interaction term between country and ER–ratio tertile. By comparing the log likelihoods

for the model with this interaction term and the model without, likelihood–ratio (LR) test is used to test the significance of this interaction term (Table 5.3). In a similar way, the interaction term between country and OC tertile is evaluated (Table 5.4).

There are country differences seen in Table 5.3. In men, the association between ER ratio and binge drinking in Czech Republic is the strongest of all countries. In women, the association between ER ratio and heavy drinking in Poland is the strongest of all countries; the association between ER ratio and problem drinking in Russia is stronger than other countries. Overall, crude associations between ER ratio at wave 1 and drinking outcomes at wave 2 are not very different across country–specific strata (all p–values by LR test > 0.12); no significant interaction between country and ER ratio is found.

Table 5.3 Crude associations between ER ratio and drinking outcomes in country–specific strata

Strata	ER ratio Tertile▲	Heavy drinking OR (95% CI)	Binge drinking OR (95% CI)	Problem drinking OR (95% CI)
Men				
Czech	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.31 (0.91 – 1.89)	1.78 (0.89 – 3.55)	1.25 (0.65 – 2.41)
	Tertile 3	1.63 (1.16 – 2.29)*	3.24 (1.72 – 6.12)*	1.96 (1.08 – 3.55)*
	P for trend	0.004	< 0.001	0.014
	OR by 1 tertile	1.27 (1.07 – 1.50)*	1.81 (1.35 – 2.41)*	1.43 (1.07 – 1.91)*
Russia	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.26 (0.99 – 1.60)	1.75 (1.22 – 2.51)*	1.35 (1.00 – 1.83)*
	Tertile 3	1.53 (1.15 – 2.03)*	2.81 (1.90 – 3.95)*	2.56 (1.84 – 3.56)*
	P for trend	0.002	< 0.001	< 0.001
	OR by 1 tertile	1.24 (1.08 – 1.42)*	1.68 (1.38 – 2.04)*	1.58 (1.34 – 1.87)*
Poland	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.27 (0.90 – 1.78)	1.14 (0.61 – 2.15)	1.86 (1.09 – 3.17)*
	Tertile 3	1.47 (1.08 – 2.01)*	1.87 (1.08 – 3.23)*	3.16 (2.03 – 4.27)*
	P for trend	0.016	0.017	< 0.001
	OR by 1 tertile	1.21 (1.04 – 1.41)*	1.40 (1.06 – 1.84)*	1.79 (1.43 – 2.25)*
Interaction country x ERI	LR test	P= 0.984	P= 0.673	P= 0.701
Women				
Czech	Tertile 1	1.00	1.00	1.00
	Tertile 2	0.85 (0.58 – 1.25)	1.53 (0.67 – 3.50)	1.52 (0.64 – 3.64)
	Tertile 3	1.23 (0.86 – 1.78)	2.71 (1.25 – 5.73)*	1.94 (0.85 – 4.41)
	P for trend	0.173	0.004	0.111
	OR by 1 tertile	1.14 (0.95 – 1.37)	1.64 (1.14 – 2.36)*	1.37 (0.93 – 2.02)
Russia	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.04 (0.78 – 1.40)	1.68 (0.99 – 2.83)	1.64 (0.95 – 2.79)
	Tertile 3	1.08 (0.81 – 1.42)	1.93 (1.09 – 3.40)*	2.74 (1.24 – 5.88)*
	P for trend	0.726	0.017	0.004
	OR by 1 tertile	1.04 (0.89 – 1.20)	1.39 (1.06 – 1.83)*	1.63 (1.16 – 2.26)*
Poland	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.65 (1.13 – 2.43)*	1.88 (0.81 – 5.49)	1.08 (0.46 – 2.52)
	Tertile 3	1.86 (1.30 – 2.66)*	2.67 (1.11 – 5.95)*	1.99 (0.98 – 4.07)
	P for trend	0.001	0.012	0.040
	OR by 1 tertile	1.33 (1.12 – 1.58)*	1.63 (1.06 – 2.45)*	1.46 (1.02 – 2.09)*
Interaction country x ERI	LR test	P= 0.128	P= 0.794	P= 0.358

▲ Country– and gender–specific tertiles of ER ratio. * P value < 0.05.

Country differences are found in Table 5.4. In men, the association between OC and binge drinking in Czech Republic is stronger than other two countries. In women, the association between OC and binge drinking in Poland is the strongest of all countries; the association between OC and problem drinking in Poland is stronger than other two countries. Overall, crude associations between OC at wave 1 and drinking outcomes at wave 2 are not very different across country–specific strata (all p–values

by LR test > 0.16). Due to no significant interactions between country and exposure variables (ER ratio and OC), data for three countries are pooled for further analyses.

Table 5.4 Crude associations between OC and drinking outcomes in country–specific strata

Strata	OC score Tertile▲	Heavy drinking OR (95% CI)	Binge drinking OR (95% CI)	Problem drinking OR (95% CI)
Men				
Czech	Tertile 1	1.00	1.00	1.00
	Tertile 2	0.97 (0.71 – 1.33)	1.40 (0.79 – 2.48)	1.53 (0.84 – 2.77)
	Tertile 3	1.22 (0.88 – 1.69)	2.52 (1.42 – 4.38)*	2.17 (1.21 – 3.91)*
	P for trend	0.203	< 0.001	0.008
	OR by 1 tertile	1.11 (0.94 – 1.31)	1.62 (1.24 – 2.13)*	1.47 (1.10 – 1.95)*
Russia	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.14 (0.89 – 1.47)	1.49 (1.04 – 2.13)*	1.32 (0.97 – 1.80)
	Tertile 3	1.26 (0.95 – 1.67)	1.71 (1.15 – 2.50)*	1.55 (1.11 – 2.16)*
	P for trend	0.090	0.003	0.006
	OR by 1 tertile	1.12 (0.98 – 1.29)	1.32 (1.09 – 1.59)*	1.25 (1.07 – 1.47)*
Poland	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.06 (0.79 – 1.42)	1.03 (0.59 – 1.80)	1.51 (0.99 – 2.31)
	Tertile 3	1.16 (0.85 – 1.59)	1.08 (0.63 – 1.81)	2.10 (1.36 – 3.23)*
	P for trend	0.361	0.887	0.001
	OR by 1 tertile	1.08 (0.92 – 1.26)	1.04 (0.79 – 1.35)	1.45 (1.17 – 1.79)*
Interaction country x OC	LR test	P= 0.922	P= 0.162	P= 0.788
Women				
Czech	Tertile 1	1.00	1.00	1.00
	Tertile 2	0.95 (0.65 – 1.39)	0.90 (0.47 – 1.72)	1.58 (0.71 – 3.56)
	Tertile 3	1.04 (0.73 – 1.47)	1.40 (0.72 – 2.69)	1.91 (0.85 – 4.39)
	P for trend	0.803	0.276	0.118
	OR by 1 tertile	0.99 (0.82 – 1.18)	1.18 (0.84 – 1.67)	1.37 (0.92 – 2.02)
Russia	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.05 (0.79 – 1.39)	1.17 (0.69 – 1.99)	1.39 (0.74 – 2.62)
	Tertile 3	1.17 (0.87 – 1.57)	1.22 (0.71 – 2.10)	1.47 (0.79 – 2.81)
	P for trend	0.594	0.485	0.237
	OR by 1 tertile	1.04 (0.90 – 1.19)	1.10 (0.85 – 1.42)	1.20 (0.89 – 1.63)
Poland	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.03 (0.74 – 1.44)	1.12 (0.48 – 2.61)	1.22 (0.55 – 2.70)
	Tertile 3	1.33 (0.94 – 1.87)	2.54 (1.16 – 5.42)*	2.75 (1.28 – 5.76)*
	P for trend	0.118	0.016	0.004
	OR by 1 tertile	1.15 (0.97 – 1.37)	1.62 (1.09 – 2.42)*	1.66 (1.12 – 2.44)*
Interaction country x OC	LR test	P= 0.537	P= 0.363	P= 0.402

▲ Country– and gender–specific tertiles of OC score. * P value < 0.05.

Bivariate analyses for the associations between covariates and three drinking outcomes are conducted. Chi–squared tests are used to examine the significance of differences between categories of the variable. P–values for heterogeneity are obtained in all categorical variables; p–values for trend are tested in ordinal categorical

variables (age and education). In men, bivariate analyses between covariates at wave 1 and three drinking outcomes at wave 2 are shown (Table 5.5). More heavy drinking is significantly ($p < 0.05$) associated with younger age. More binge drinking is significantly associated with younger age, less education, being manual workers, and higher deprivation. More problem drinking is significantly associated with younger age, less education, being manual workers, higher deprivation, and more depression.

Table 5.5 Bivariate analyses for relationships between covariates and drinking outcomes in men (n= 3782)

Covariates	Heavy drinking (%) n= 1120	Binge drinking (%) n= 376	Problem drinking (%) n= 517
Age: 45 – 49	34.4	12.1	16.7
50 – 54	28.9	8.8	13.2
55 – 59	29.0	9.2	12.3
60 – 69	25.1	7.7	10.1
P for heterogeneity	< 0.001	0.006	< 0.001
P for trend	< 0.001	0.004	< 0.001
Education: Primary/ less	29.4	11.9	20.1
Vocational	31.8	11.7	15.0
Secondary	28.7	9.6	14.0
University	28.6	7.6	10.4
P for heterogeneity	0.299	0.004	< 0.001
P for trend	0.150	0.001	< 0.001
Occupation class			
Manager/ profession	29.6	9.6	11.5
Non-manual worker	28.1	8.1	10.9
Manual worker	31.4	11.3	18.0
P value	0.146	0.015	< 0.001
Marital status			
Married/ cohabiting	29.3	9.3	13.4
Single	29.7	15.3	10.8
Divorce/ widowed	31.3	10.0	11.3
P value	0.760	0.098	0.455
Deprivation: Low (0–3.9)	28.9	8.9	11.9
High (4–9)	31.9	11.8	18.1
P value	0.086	0.012	< 0.001
Depression: CESD < 16	28.7	9.3	11.8
CESD >= 16	31.8	11.3	18.2
P value	0.121	0.179	< 0.001
Social isolation			
No (>= once a month)	29.9	9.8	13.0
Yes (< once a month)	29.0	9.2	13.4
P value	0.524	0.464	0.702
Self-rated health			
Very good – average	29.6	9.6	13.3
Poor – very poor	27.6	8.1	11.5
P value	0.471	0.416	0.403

Table 5.6 reports the bivariate analyses between covariates at wave 1 and drinking outcomes at wave 2 in women. More heavy drinking is significantly ($p < 0.05$) associated with younger age and higher education. More binge drinking is significantly associated with younger age. Finally, more problem drinking is significantly associated with younger age and more depression.

Table 5.6 Bivariate analyses for relationships between covariates and drinking outcomes in women (n= 3731)

Covariates	Heavy drinking (%) n= 648	Binge drinking (%) n= 154	Problem drinking (%) n= 153
Age: 45 – 49	23.0	6.3	4.5
50 – 54	22.4	5.1	4.1
55 – 59	20.8	3.3	3.6
60 – 69	13.2	0.7	0.7
P for heterogeneity	< 0.001	< 0.001	0.002
P for trend	< 0.001	< 0.001	0.001
Education: Primary/ less	16.2	3.5	3.0
Vocational	20.7	3.9	3.6
Secondary	20.3	4.9	4.0
University	23.8	4.9	3.5
P for heterogeneity	0.008	0.533	0.804
P for trend	0.029	0.196	0.957
Occupation class			
Manager/ profession	23.6	4.8	4.3
Non-manual worker	21.3	4.6	3.2
Manual worker	18.4	4.3	4.8
P value	0.060	0.897	0.098
Marital status			
Married/ cohabiting	20.9	4.6	3.9
Single	23.7	4.2	1.7
Divorce/ widowed	21.3	4.5	3.6
P value	0.598	0.958	0.219
Deprivation: Low (0–3.9)	21.0	4.8	3.6
High (4–9)	21.6	4.2	3.8
P value	0.705	0.410	0.843
Depression: CESD < 16	20.4	4.4	3.2
CESD >= 16	22.8	5.6	5.9
P value	0.125	0.142	< 0.001
Social isolation			
No (>= once a month)	21.8	4.8	3.8
Yes (< once a month)	20.4	4.4	3.7
P value	0.294	0.599	0.883
Self-rated health			
Very good – average	21.0	4.5	3.6
Poor – very poor	23.0	5.9	3.7
P value	0.339	0.184	0.982

5.2 Potential Role of OC in ERI–Drinking Relationship

The focus of this section is, as the first aim of the thesis, on the associations between ERI and drinking outcomes, on the associations between OC and drinking outcomes, and on assessing whether OC has the potential role of antecedent, mediator, modifier, or direct effect in the relationship between ERI and drinking. As there is no significant interaction between country and exposure variables, data for the three countries are pooled for further analyses.

5.2.1 Associations between ERI and drinking outcomes

The associations between ER ratio at wave 1 and three binary drinking outcomes (heavy drinking, binge drinking, and problem drinking) at wave 2 are assessed using three logistic regression analyses, separately for men and women. These associations are assessed after adjustment for age and country (Model 1) and after additionally adjustment for other covariates (Model 2).

Table 5.7 presents the associations between ER ratio at wave 1 and three drinking outcomes at wave 2 using three logistic regression analyses, respectively. In men, Model 1 shows that the odds of having heavy drinking, binge drinking and problem drinking are 1.41, 2.32 and 1.89, respectively, for highest versus lowest tertile of ER ratio. Model 2 reports that the odds of having heavy drinking, binge drinking and problem drinking are 1.33, 2.29 and 1.79 for highest versus lowest tertile of ER ratio. The adjusted OR changes for heavy drinking, binge drinking and problem drinking are 1.15, 1.49 and 1.35, respectively, by 1–tertile increase in ER ratio ($p < 0.05$).

In women, Model 1 shows that the odds of having heavy drinking, binge drinking and problem drinking are 1.23, 1.98 and 2.16, respectively, for highest versus lowest tertile of ER ratio. Model 2 shows the odds of heavy drinking, binge drinking and problem drinking are 1.29, 2.06 and 1.82 for highest versus lowest tertile of ER ratio. The adjusted OR changes for heavy drinking, binge drinking and problem drinking are

1.13, 1.42 and 1.36, respectively, by 1–tertile increase in ER ratio ($p < 0.05$).

Table 5.7 Associations between ER ratio at wave 1 and drinking outcomes at wave 2

Model	ER ratio Tertile▲	Heavy drinking OR (95% CI)	Binge drinking OR (95% CI)	Problem drinking OR (95% CI)
Men (n= 3782)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.24 (1.04 – 1.46)*	1.55 (1.16 – 2.07)*	1.25 (0.98 – 1.59)
	Tertile 3	1.41 (1.17 – 1.68)*	2.32 (1.73 – 3.09)*	1.89 (1.51 – 2.38)*
	P for trend	0.001	< 0.001	< 0.001
	OR by 1 tertile	1.19 (1.08 – 1.30)*	1.52 (1.32 – 1.75)*	1.39 (1.24 – 1.56)*
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.21 (1.01 – 1.46)*	1.51 (1.12 – 2.04)*	1.27 (0.99 – 1.63)
	Tertile 3	1.33 (1.12 – 1.57)*	2.29 (1.69 – 3.07)*	1.79 (1.40 – 2.28)*
	P for trend	0.002	< 0.001	< 0.001
	OR by 1 tertile	1.15 (1.06 – 1.26)*	1.49 (1.29 – 1.73)*	1.35 (1.19 – 1.52)*
Women (n= 3731)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.05 (0.87 – 1.27)	1.49 (0.99 – 2.24)	1.48 (0.93 – 2.36)
	Tertile 3	1.23 (1.02 – 1.49)*	1.98 (1.34 – 2.93)*	2.16 (1.34 – 3.47)*
	P for trend	0.054	0.001	0.002
	OR by 1 tertile	1.11 (1.00 – 1.22)	1.40 (1.15 – 1.69)*	1.42 (1.13 – 1.80)*
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.11 (0.91 – 1.37)	1.52 (0.97 – 2.37)	1.29 (0.82 – 2.15)
	Tertile 3	1.29 (1.04 – 1.58)*	2.06 (1.34 – 3.16)*	1.82 (1.13 – 2.94)*
	P for trend	0.025	0.001	0.010
	OR by 1 tertile	1.13 (1.02 – 1.26)*	1.42 (1.16 – 1.75)*	1.36 (1.08 – 1.72)*

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health. ▲ Gender–specific tertile of ER ratio: in men, tertile 1 (0.20–0.32), tertile 2 (0.32–0.47), and tertile 3 (> 0.47); in women, tertile 1 (0.20–0.31), tertile 2 (0.31–0.46), and tertile 3 (> 0.46). * P value < 0.05.

5.2.2 Associations between OC and drinking outcomes

The associations between OC at wave 1 and three drinking outcomes (heavy drinking, binge drinking, and problem drinking) at wave 2 are assessed using three logistic regression analyses, separately for men and women, after adjustment for age and country (Model 1) and after additionally adjustment for other covariates (Model 2).

Table 5.8 shows the associations between OC at wave 1 and three drinking outcomes at wave 2 using three logistic regression analyses, respectively. In men, Model 2 reports that the odds of having heavy drinking, binge drinking and problem

drinking are 1.18, 1.72 and 1.64, respectively, for highest versus lowest tertile of OC. The adjusted OR changes for heavy drinking, binge drinking and problem drinking are 1.08 (p= 0.081), 1.31 and 1.28 (p < 0.05), respectively, by 1–tertile increase in OC.

In women, Model 2 reports that the odds of having binge drinking and problem drinking are 1.52 and 1.63 for highest versus lowest tertile of OC. The adjusted OR changes for binge drinking and problem drinking are 1.24 and 1.27 (p < 0.05) by 1–tertile increase in OC score; nevertheless, the association between OC and heavy drinking does not reach statistical significance (p= 0.281).

Table 5.8 Associations between OC at wave 1 and drinking outcomes at wave 2

Model	OC score Tertile▲	Heavy drinking OR (95% CI)	Binge drinking OR (95% CI)	Problem drinking OR (95% CI)
Men (n= 3782)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.06 (0.90 – 1.25)	1.23 (0.93 – 1.62)	1.17 (0.91 – 1.50)
	Tertile 3	1.21 (1.01 – 1.44)*	1.68 (1.29 – 2.20)*	1.50 (1.20 – 1.88)*
	P for trend	0.034	< 0.001	0.001
	OR by 1 tertile	1.10 (1.01 – 1.20)*	1.30 (1.13 – 1.48)*	1.21 (1.08 – 1.36)*
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.02 (0.87 – 1.21)	1.24 (0.96 – 1.60)	1.19 (0.94 – 1.50)
	Tertile 3	1.18 (0.99 – 1.40)	1.72 (1.32 – 2.24)*	1.64 (1.29 – 2.07)*
	P for trend	0.081	< 0.001	0.001
	OR by 1 tertile	1.08 (0.99 – 1.18)	1.31 (1.15 – 1.50)*	1.28 (1.13 – 1.44)*
Women (n= 3731)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.04 (0.85 – 1.29)	1.14 (0.79 – 1.64)	1.35 (0.86 – 2.09)
	Tertile 3	1.07 (0.88 – 1.32)	1.60 (1.11 – 2.28)*	1.75 (1.15 – 2.68)*
	P for trend	0.375	0.012	0.008
	OR by 1 tertile	1.03 (0.95 – 1.14)	1.27 (1.05 – 1.51)*	1.34 (1.08 – 1.65)*
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.09 (0.90 – 1.33)	1.09 (0.75 – 1.61)	1.39 (0.90 – 2.14)
	Tertile 3	1.11 (0.91 – 1.37)	1.52 (1.03 – 2.25)*	1.63 (1.05 – 2.52)*
	P for trend	0.281	0.036	0.028
	OR by 1 tertile	1.06 (0.96 – 1.17)	1.24 (1.01 – 1.50)*	1.27 (1.03 – 1.58)*

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health. ▲ Gender–specific tertile of OC score: in men, tertile 1 (6–12), tertile 2 (12–15), and tertile 3 (15–24); in women, tertile 1 (6–12), tertile 2 (12–15), and tertile 3 (15–24).
* P value < 0.05.

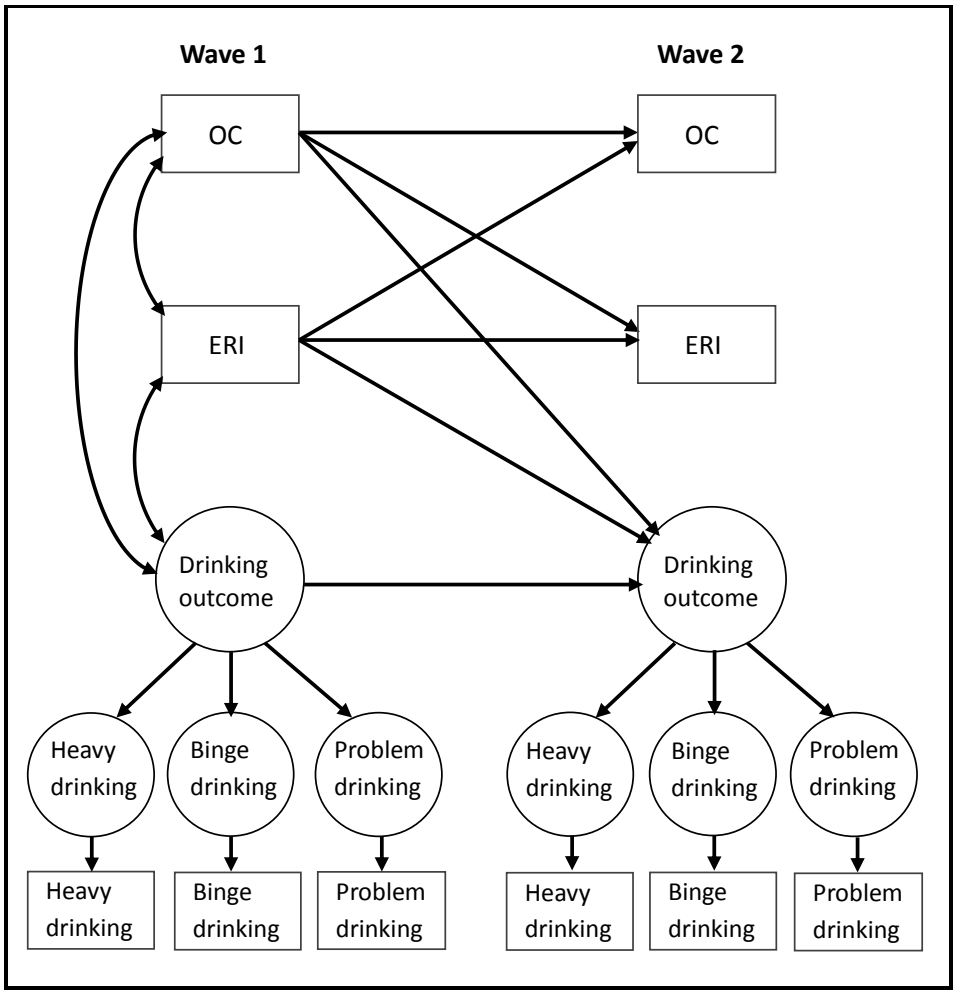
Previous analyses have found consistent and significant associations of exposure variables (ER ration and OC) with three drinking outcomes, respectively, with the same direction of causality but different magnitude of effect. Higher levels of ER ratio and

OC, respectively, are associated with higher levels of drinking outcomes.

5.2.3 Antecedent or mediator role of OC in ERI–drinking relationship

To assess antecedent or mediator roles of OC in ERI–drinking relationship, the structural equation modelling (SEM) for categorical outcomes with an autoregressive and cross–lagged model is adopted and applied in Mplus 7.[582] The measurement model and the structural model for the SEM are specified in Figure 5.1.

Figure 5.1 Measurement model and structural model specified for the SEM for antecedent or mediator role of OC in ERI–drinking relation



In the measurement model, a latent factor (drinking outcome) underlies the relationship among 3 continuous latent response variables, which underlie 3 observed binary variables (binge drinking, heavy drinking, and problem drinking), respectively.

Justification for adopting a latent variable is summarized. The results of path analyses for 3 separate drinking outcomes are compared (see Appendix 4). Despite different magnitudes of effect, there are consistent directions of causality between ERI and 3 drinking outcomes; for example, path coefficients are all positive – 0.146 for binge drinking, 0.116 for problem drinking, and 0.069 for heavy drinking in women. Similarly, there are consistent directions of causality but diverse magnitudes of effect between OC and 3 drinking outcomes. Other paths between OC and ERI are quite similar across 3 drinking outcomes. The objective of path analyses focuses on antecedent or mediator role of OC in ERI–drinking relationship, and I summarize 3 drinking outcomes by a latent variable in order to find an overall trend in the relationships between OC, ERI and drinking in one model. A data reduction definition views a latent variable as a way to reduce complexity or dimensionality of a set of data; a latent variable is viewed as an emergent property that summarizes the indicators. This method assumes an overabundance of data regarding the variables of interest and the need to find a parsimonious means of using the data to test relationships between these variables.[583]

The measurement model is shown in Table 5.9. Factor loadings estimate direct effects of a latent factor on the latent response variable and are interpreted as regression coefficients. For example, in women, 1–unit increase in drinking outcome at wave 2 is associated with 1.000, 0.657 and 0.762 unit of increase in latent response variables for binge drinking, heavy drinking and problem drinking at wave 2, respectively. Additionally, standardized factor loadings are estimated correlation between the latent response variable and the latent factor; squared standardized factor loadings are proportions of explained variance (R^2). Thus, residual variance (1 – R^2) are proportion of unexplained variance. Finally, the non–linear relationship between each latent response variable and corresponding observed categorical variable is linked by the probit model.

The acceptability of the measurement model is evaluated by the interpretability, size, and statistical significance of the model's parameter estimates. For interpretability,

the parameter estimates do not take out-of-range values (e.g. negative error variance). For statistical significance, all factor loadings are shown statistically significant. For size, when a standardized factor loading is > 0.7, the factor would explain the majority of variance of the indicator (R^2 > 0.5). Researchers suggest that standardized factor loadings are considered poor (0.32–0.45), fair (0.45–0.55), good (0.55–0.63), very good (0.63–0.71), and excellent (> 0.71).[584] In my measurement model, the lowest standardized factor loading is still considered to be good (> 0.55).

Table 5.9 Measurement model of the SEM for antecedent or mediator role of OC in ERI–drinking relation

Parameter	Factor loadings			Residual variance
	Unstandardized coefficient	Standard Error	Standardized coefficient	
Men (n= 3782)				
Latent factor – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.999	0.002
Heavy drinking, wave 1	0.863	0.038	0.862	0.257
Problem drinking, wave 1	0.620	0.029	0.619	0.616
Latent factor – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.932	0.131
Heavy drinking, wave 2	0.730	0.036	0.680	0.537
Problem drinking, wave 2	0.615	0.029	0.573	0.671
Women (n= 3731)				
Latent factor – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.957	0.084
Heavy drinking, wave 1	0.774	0.034	0.741	0.451
Problem drinking, wave 1	0.807	0.037	0.772	0.404
Latent factor – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.974	0.051
Heavy drinking, wave 2	0.657	0.042	0.640	0.590
Problem drinking, wave 2	0.762	0.049	0.742	0.449

In the structural model, an autoregressive and cross–lagged model is adopted in this 2–wave cohort study (Figure 5.1). First, “autoregressive” means regressed on itself, so each variable is predicted by the same variable at an early wave. Second, the cross–lagged effects of “OC at wave 1 on ERI at wave 2” and “ERI at wave 1 on OC at wave 2” are measured, respectively, in order to identify causal directionality between OC and ERI. Bidirectional relationship between OC and ERI is possible based on my hypotheses.

Third, as mediation is a causal chain involving at least two causal relations, these causal relations can be tested separately using two phases of data. The mediator role of ERI in OC–drinking relation is assessed by two cross–lagged effects: (1) OC at wave 1 on ERI at wave 2; (2) ERI at wave 1 on drinking at wave 2. The mediator role of OC in ERI–drinking relation is estimated by two cross–lagged effects: (1) ERI at wave 1 on OC at wave 2; (2) OC at wave 1 on drinking at wave 2. Partial mediation applies if both causal relations are confirmed; the product of two path coefficients (two cross–lagged effects) can estimate the strength of mediator effect.[585] For a mediator effect, the effect size measure is the product of two path coefficients. The bootstrap method is used for significance testing of the mediator effect with 5000 bootstrap samples to yield valid estimates for the mediator effect by Mplus 7; this method is adopted due to complicated models (categorical outcomes and multiple mediators).[586]

In terms of predictors, the tertiles of ERI and OC are firstly transformed into a series of dummy variables (one dummy variable for each tertile) to compare between tertile groups. Next, ERI tertile and OC tertile are treated as continuous variables to estimate assumed linear trend between the predictor and drinking outcome. In terms of model fit, three indexes are used: Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Tucker–Lewis Index (TLI). RMSEA < 0.06, CFI > 0.95 or TLI > 0.95 indicate “good model fit”.

Table 5.10 presents the structural model in men, with the results illustrated in Figure 5.2. First, the mediator effect of ERI in OC–drinking relationship is estimated by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 2 (unstandardized β= 0.142; standard error= 0.020); (2) higher ERI at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.152; standard error= 0.033). This mediator effect of ERI is significant (0.022= 0.142 x 0.152; standard error= 0.006 and p= 0.001 estimated by bootstrap method).

Second, the mediator effect of OC in ERI–drinking relationship is estimated by

multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.083; standard error= 0.020); (2) higher OC at wave 1 non-significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.050; standard error= 0.033; p= 0.104). This mediator effect of OC is not significant (0.004= 0.083 x 0.050; standard error= 0.003 and p= 0.155 by bootstrap method). Third, the fit indexes are considered good fit (RMSEA= 0.049 < 0.06) or close to cutoffs for good fit (CFI= 0.945; TLI= 0.923).

Table 5.10 Structural model of the SEM for antecedent or mediator role of OC in ERI–drinking relation in men (n= 3782)

Parameter	Unstandardized coefficient	Standardized coefficient	P value
Drinking wave 1 → Drinking wave 2	0.467	0.428	< 0.001
OC wave 1 → OC wave 2			
OC tertile 2 vs tertile 1	0.320	0.174	< 0.001
OC tertile 3 vs tertile 1	0.681	0.335	< 0.001
1–tertile increase	0.346	0.316	< 0.001
ERI wave 1 → ERI wave 2			
ERI tertile 2 vs tertile 1	0.216	0.126	< 0.001
ERI tertile 3 vs tertile 1	0.473	0.277	< 0.001
1–tertile increase	0.242	0.238	< 0.001
OC → ERI → Drinking	0.022	0.017	0.001
OC wave 1 → ERI wave 2			
OC tertile 2 vs tertile 1	0.113	0.066	0.001
OC tertile 3 vs tertile 1	0.279	0.148	< 0.001
1–tertile increase	0.142	0.132	< 0.001
ERI wave 1 → Drinking wave 2			
ERI tertile 2 vs tertile 1	0.162	0.071	0.003
ERI tertile 3 vs tertile 1	0.306	0.135	< 0.001
1–tertile increase	0.152	0.117	< 0.001
ERI → OC → Drinking	0.004	0.003	0.155
ERI wave 1 → OC wave 2			
ERI tertile 2 vs tertile 1	0.095	0.051	0.011
ERI tertile 3 vs tertile 1	0.166	0.089	< 0.001
1–tertile increase	0.083	0.078	< 0.001
OC wave 1 → Drinking wave 2			
OC tertile 2 vs tertile 1	0.049	0.021	0.298
OC tertile 3 vs tertile 1	0.096	0.037	0.145
1–tertile increase	0.050	0.035	0.104
Tests of model fit	RMSEA= 0.049	CFI= 0.945	TLI= 0.923

Figure 5.2 Results of the SEM for antecedent or mediator role of OC in ERI–drinking relation in men

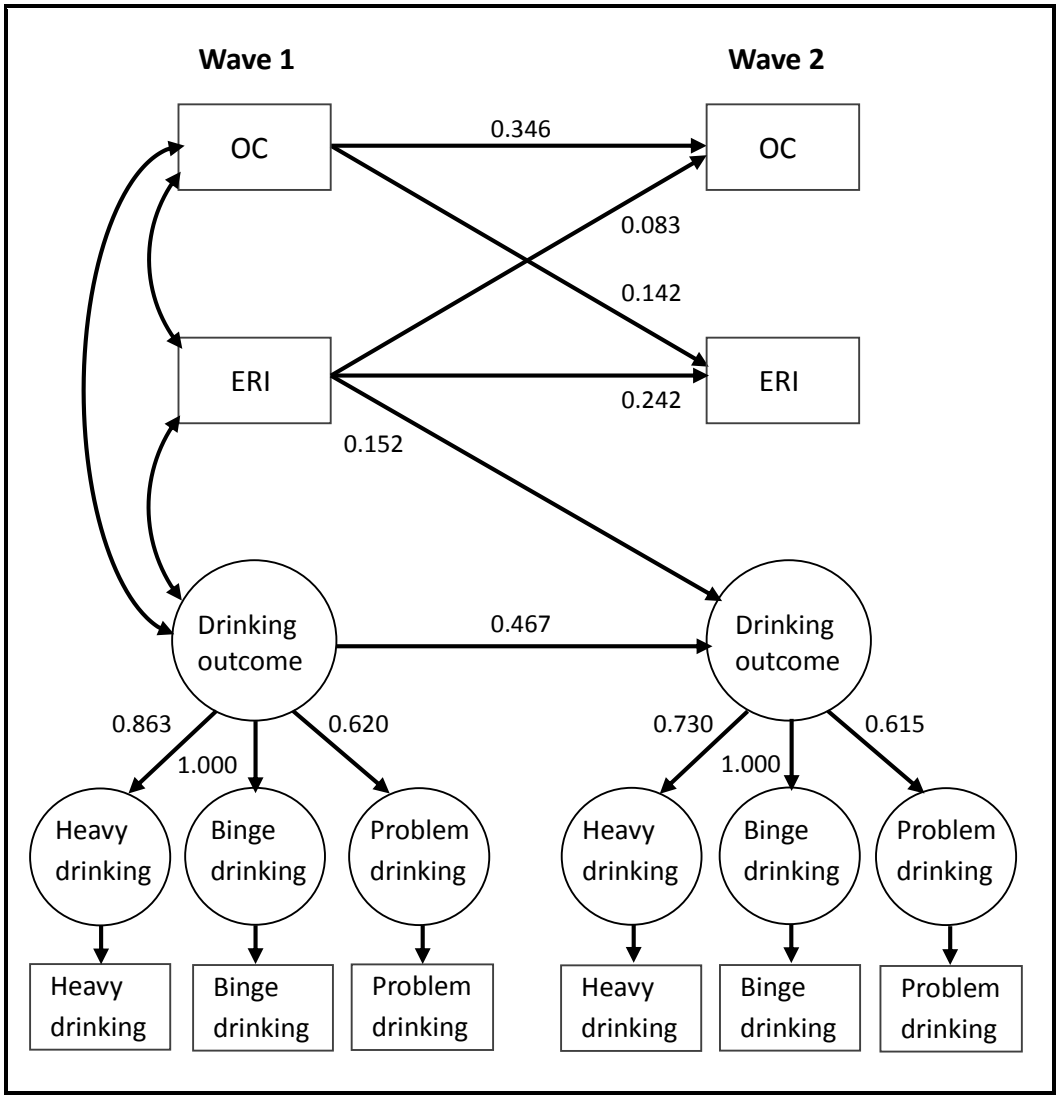


Table 5.11 presents the structural model in women; the results are illustrated in Figure 5.3. First, the mediator effect of ERI in OC–drinking relationship is estimated by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 2 (unstandardized β= 0.148; standard error= 0.019); (2) higher ERI at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.138; standard error= 0.040). This mediator effect of ERI is significant (0.020= 0.148 x 0.138; standard error= 0.006 and p= 0.002).

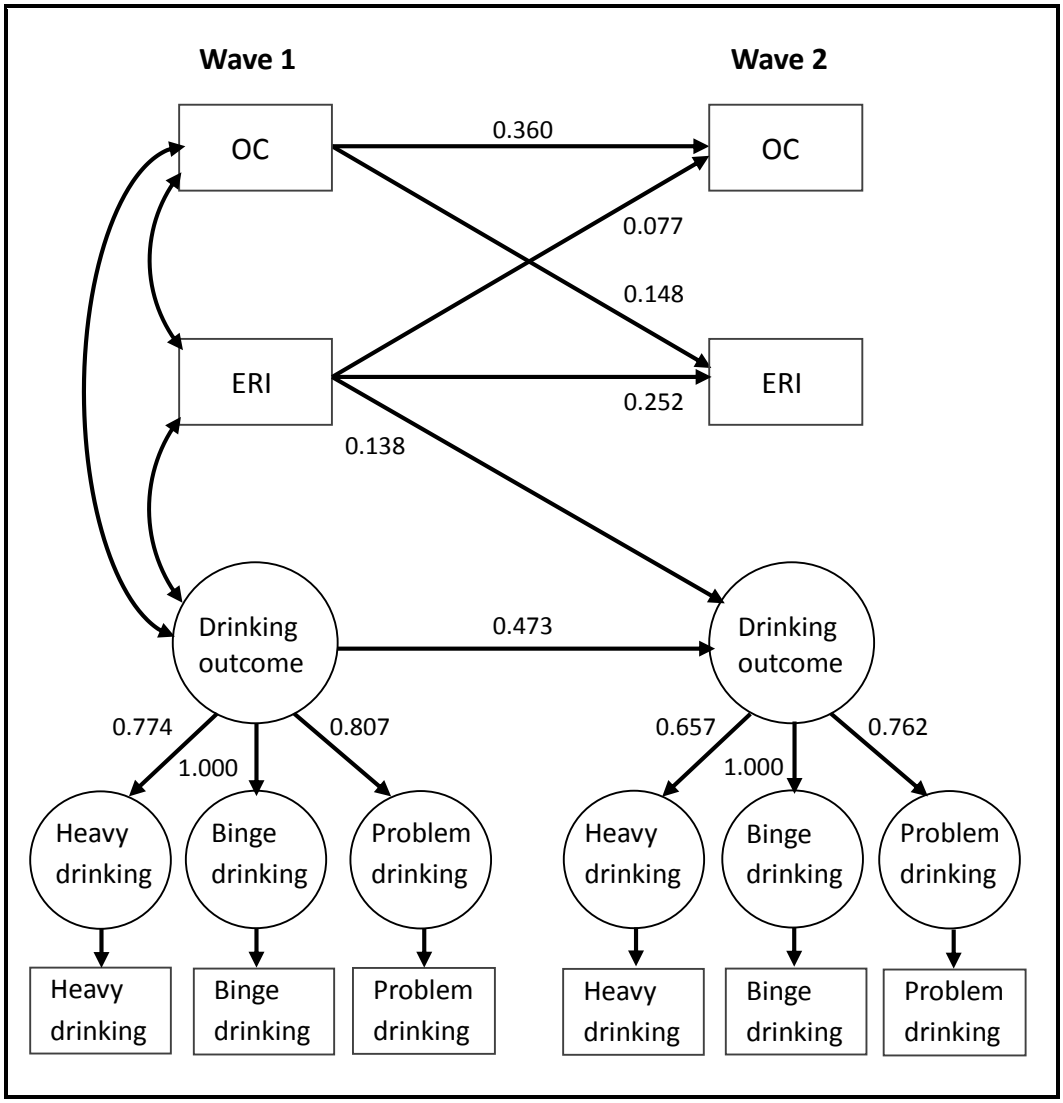
Second, the mediator effect of OC in ERI–drinking relationship is estimated by

multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.077; standard error= 0.019); (2) higher OC at wave 1 non-significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.040; standard error= 0.038; p= 0.162). This mediator effect of OC is not significant (0.003; standard error= 0.003 and p= 0.308). Third, the fit indexes are considered good fit (RMSEA= 0.050 < 0.06) or close to cutoffs for good fit (CFI= 0.925; TLI= 0.908).

Table 5.11 Structural model of the SEM for antecedent or mediator role of OC in ERI–drinking relation in women (n= 3731)

Parameter	Unstandardized coefficient	Standardized coefficient	P value
Drinking wave 1 → Drinking wave 2	0.473	0.468	< 0.001
OC wave 1 → OC wave 2			
OC tertile 2 vs tertile 1	0.292	0.170	< 0.001
OC tertile 3 vs tertile 1	0.730	0.400	< 0.001
1–tertile increase	0.360	0.358	< 0.001
ERI wave 1 → ERI wave 2			
ERI tertile 2 vs tertile 1	0.293	0.174	< 0.001
ERI tertile 3 vs tertile 1	0.484	0.290	< 0.001
1–tertile increase	0.252	0.242	< 0.001
OC → ERI → Drinking	0.020	0.016	0.002
OC wave 1 → ERI wave 2			
OC tertile 2 vs tertile 1	0.155	0.093	< 0.001
OC tertile 3 vs tertile 1	0.308	0.173	< 0.001
1–tertile increase	0.148	0.141	< 0.001
ERI wave 1 → Drinking wave 2			
ERI tertile 2 vs tertile 1	0.142	0.063	0.068
ERI tertile 3 vs tertile 1	0.280	0.126	0.001
1–tertile increase	0.138	0.108	0.001
ERI → OC → Drinking	0.003	0.002	0.308
ERI wave 1 → OC wave 2			
ERI tertile 2 vs tertile 1	0.054	0.031	0.123
ERI tertile 3 vs tertile 1	0.154	0.090	< 0.001
1–tertile increase	0.077	0.074	< 0.001
OC wave 1 → Drinking wave 2			
OC tertile 2 vs tertile 1	0.034	0.016	0.568
OC tertile 3 vs tertile 1	0.075	0.033	0.268
1–tertile increase	0.040	0.032	0.162
Tests of model fit	RMSEA= 0.050	CFI= 0.925	TLI= 0.908

Figure 5.3 Results of the SEM for antecedent or mediating role of OC in ERI–drinking relation in women



Interpretation of path coefficient in the SEM is summarized. The effect of ERI at wave 1 on drinking outcome at wave 2 in women is taken for example (Table 5.11). Unstandardized path coefficient for this effect is 0.138, which means that 1–tertile increase in ER ratio results in 0.138 unit of increase in drinking outcome. Next, the measurement model is considered (Table 5.9). In women, 1–unit increase in drinking outcome at wave 2 is associated with 1.000, 0.657 and 0.762 unit of increase in latent response variables for binge drinking, heavy drinking and problem drinking at wave 2. Thus, 1–tertile increase in ER ratio results in 0.138, 0.091 (0.138 x 0.657) and 0.105

(0.138 x 0.762) unit of increase in latent response variables for binge drinking, heavy drinking and problem drinking at wave 2. The nonlinear relationship between each latent response variable and observed binary variable is linked by the probit model. The OR is the exponential (antilog) of estimated logistic coefficient, which is derived from probit coefficient multiplied by 1.8. Thus, 1–tertile increase in ER ratio results in OR changes of 1.28 (exponential function 0.138 x 1.8), 1.18 and 1.21 for observed binary variables of binge drinking, heavy drinking and problem drinking at wave 2.

5.2.4 Modifying role of OC in ERI–drinking relationship

To evaluate modifying effect of OC in ERI–drinking relationship, two approaches are adopted. In the first approach, the associations between ER ratio at wave 1 and drinking outcomes at wave 2 in different strata of OC tertile are assessed after adjustment for covariates. Next, logistic regression analyses are conducted for 3 drinking outcomes at wave 2, respectively, regressed by OC tertile, ER–ratio tertile, and their interaction term at wave 1 after adjustment for covariates. By comparing the log likelihoods for the model with this interaction term and the model without, likelihood–ratio (LR) test is adopted to test significance of this interaction term.

Table 5.12 Evaluation for modifying role of OC in ERI–drinking relationship

Approaches	Strata	Heavy drinking	Binge drinking	Problem drinking
Men		OR of outcome by 1–tertile increase in ER ratio (95% CI)		
1. ERI–drinking relation	OC tertile 1	1.06 (0.92 – 1.23)	1.39 (1.10 – 1.75)	1.21 (0.99 – 1.49)
in different strata of OC	OC tertile 2	1.23 (1.05 – 1.44)	1.51 (1.17 – 1.94)	1.22 (0.98 – 1.53)
tertile	OC tertile 3	1.17 (0.95 – 1.43)	1.45 (1.05 – 2.00)	1.46 (1.09 – 1.96)
2. Interaction OC x ERI	P–value	P= 0.362	P= 0.853	P= 0.196
Women		OR of outcome by 1–tertile increase in ER ratio (95% CI)		
1. ERI–drinking relation	OC tertile 1	1.06 (0.90 – 1.24)	1.36 (0.95 – 1.93)	1.03 (0.68 – 1.57)
in different strata of OC	OC tertile 2	1.19 (0.99 – 1.42)	1.68 (1.09 – 2.57)	1.48 (0.97 – 2.26)
tertile	OC tertile 3	1.00 (0.82 – 1.22)	1.10 (0.75 – 1.63)	1.49 (0.91 – 2.44)
2. Interaction OC x ERI	P–value	P= 0.932	P= 0.851	P= 0.312

In Table 5.12, LR tests show that the interaction term between OC tertile and ER–ratio tertile is not significant for heavy drinking in men (p= 0.362) and women (p= 0.932), not significant for binge drinking in men (p= 0.853) and women (p= 0.851), and not significant for problem drinking in men (p= 0.196) and women (p= 0.312).

The second approach adopts a measurement model (Table 5.13) similar to previous SEM (Table 5.9), and a path analysis is conducted for the latent drinking outcome at wave 2 regressed by OC tertile, ER–ratio tertile, and the interaction term between OC tertile and ER–ratio tertile at wave 1 after adjustment for covariates. The significance of this interaction term is evaluated in the SEM by Mplus 7. This interaction term is not significant in men (p= 0.324; unstandardized β= 0.038; standard error= 0.039) and women (p= 0.282; unstandardized β= 0.044; standard error= 0.040). My results show that OC has no significantly modifying role in ERI–drinking relationship.

Table 5.13 Measurement model of the SEM for modifying role of OC in ERI–drinking relation

Parameter	Factor loadings			Residual variance
	Unstandardized coefficient	Standard Error	Standardized coefficient	
Men (n= 3782)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.997	0.006
Heavy drinking, wave 1	0.861	0.038	0.858	0.264
Problem drinking, wave 1	0.617	0.029	0.615	0.622
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.934	0.128
Heavy drinking, wave 2	0.735	0.037	0.686	0.529
Problem drinking, wave 2	0.611	0.029	0.571	0.674
Women (n= 3731)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.954	0.090
Heavy drinking, wave 1	0.782	0.033	0.746	0.443
Problem drinking, wave 1	0.806	0.035	0.769	0.409
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.977	0.045
Heavy drinking, wave 2	0.642	0.042	0.627	0.607
Problem drinking, wave 2	0.768	0.052	0.750	0.438

5.3 Potential Role of PC in Relation between OC, ERI, and Drinking

The focus of this section is, according to the second aim of the thesis, on the associations between PC and drinking outcomes, and on the potential role of PC in the relationship between ERI, OC, and drinking outcomes.

5.3.1 Associations between PC and drinking outcomes

The associations between PC at wave 1 and three drinking outcomes at wave 2 are assessed following the same steps as for ERI–drinking associations.

Table 5.14 Associations of PC at wave 1 with drinking outcomes at wave 2

Model	Perceived control tertile▲	Heavy drinking OR (95% CI)	Binge drinking OR (95% CI)	Problem drinking OR (95% CI)
Men (n= 3782)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.01 (0.82 – 1.22)	0.99 (0.77 – 1.27)	0.82 (0.66 – 1.02)
	Tertile 3	0.86 (0.71 – 1.04)	0.67 (0.51 – 0.89)*	0.67 (0.53 – 0.84)*
	P for trend	0.179	0.010	0.006
	OR by 1 tertile	0.94 (0.85 – 1.03)	0.84 (0.74 – 0.96)*	0.82 (0.73 – 0.92)*
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	1.00 (0.83 – 1.20)	0.98 (0.76 – 1.27)	0.83 (0.66 – 1.05)
	Tertile 3	0.81 (0.67 – 0.97)*	0.63 (0.47 – 0.86)*	0.64 (0.49 – 0.85)*
	P for trend	0.033	0.006	0.004
	OR by 1 tertile	0.90 (0.83 – 0.99)*	0.82 (0.71 – 0.94)*	0.81 (0.71 – 0.93)*
Women (n= 3731)				
Model 1★	Tertile 1	1.00	1.00	1.00
	Tertile 2	0.93 (0.76 – 1.13)	0.84 (0.60 – 1.17)	0.88 (0.60 – 1.26)
	Tertile 3	0.87 (0.70 – 1.06)	0.62 (0.43 – 0.90)*	0.67 (0.44 – 1.04)
	P for trend	0.155	0.023	0.073
	OR by 1 tertile	0.93 (0.83 – 1.03)	0.80 (0.66 – 0.96)*	0.84 (0.70 – 1.01)
Model 2★★	Tertile 1	1.00	1.00	1.00
	Tertile 2	0.90 (0.73 – 1.11)	0.88 (0.61 – 1.25)	0.81 (0.54 – 1.22)
	Tertile 3	0.83 (0.66 – 1.02)	0.61 (0.41 – 0.94)*	0.62 (0.40 – 1.00)*
	P for trend	0.083	0.035	0.046
	OR by 1 tertile	0.91 (0.81 – 1.01)	0.80 (0.65 – 0.98)*	0.80 (0.63 – 1.00)*

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates.
▲ Gender–specific tertile of PC: in men, tertile 1 (0–34), tertile 2 (34–41), and tertile 3 (41–55); in women, tertile 1 (0–33), tertile 2 (33–40), and tertile 3 (40–55). * P value < 0.05.

Table 5.14 shows the associations between PC at wave 1 and three drinking outcomes at wave 2 using logistic regression analyses, respectively. In men, Model 2 shows that the adjusted odds of having heavy drinking, binge drinking and problem

drinking are 0.81, 0.63 and 0.64, respectively, for highest versus lowest tertile of PC. The adjusted OR changes for heavy drinking, binge drinking and problem drinking are 0.90, 0.82 and 0.81, respectively, by 1–tertile increase in PC (all p-values < 0.05).

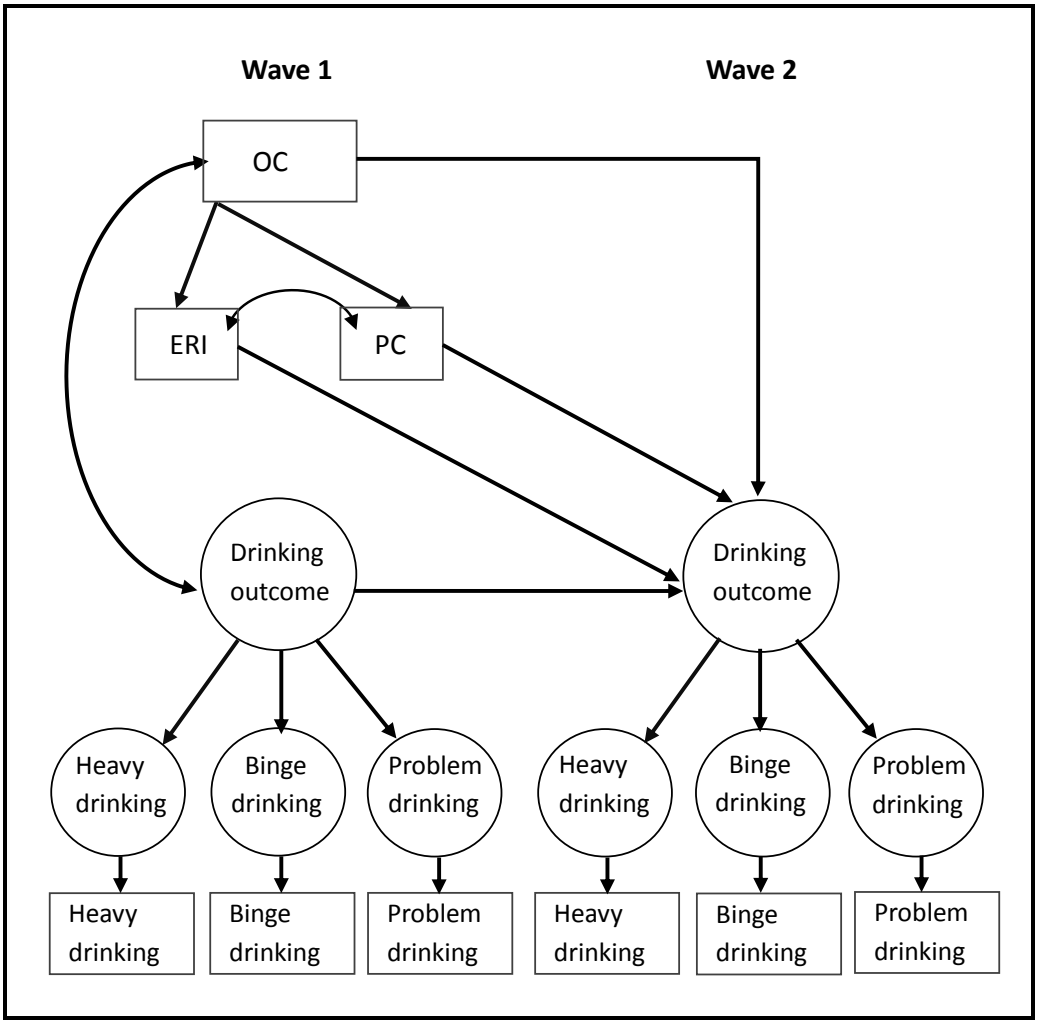
In women, Model 2 shows that the adjusted odds of heavy drinking, binge drinking and problem drinking are 0.83, 0.61 and 0.62, respectively, for highest versus lowest tertile of PC. The adjusted OR changes for heavy drinking, binge drinking and problem drinking are 0.91, 0.80 and 0.80 by 1–tertile increase in PC (all p-values < 0.1).

5.3.2 Mediator roles of PC and ERI in OC–drinking relationship

To assess the potential role of PC in the relationship between OC, ERI and drinking, the SEM for categorical outcomes with an autoregressive and cross–lagged model is adopted and applied by Mplus 7. The measurement model and the structural model for the SEM are specified in Figure 5.4.

The measurement model is specified in a similar way as the previous SEM model (Section 5.2.3). A latent drinking outcome is denoted by 3 continuous latent response variables, which underlie 3 observed binary variables (binge drinking, heavy drinking, and problem drinking) at wave 1 and 2, respectively. The justification for adopting a latent variable is summarized. The results of path analyses for 3 separate drinking outcomes are compared (see Appendix 4). Despite different magnitudes of effect, there are consistent directions of causality between ERI and 3 drinking outcomes. Similarly, there are consistent directions of causality but diverse magnitudes of effect between PC and 3 drinking outcomes. Other paths are quite similar across 3 drinking outcomes. I summarize 3 drinking outcomes by a parsimonious means (latent variable) in order to find an overall trend in the relations between OC, ERI, PC and drinking in one model. A data reduction definition views a latent variable as a way to reduce complexity or dimensionality of a set of data.

Figure 5.4 Measurement model and structural model specified for the SEM for mediator roles of PC and ERI in OC–drinking relationship



The results of the measurement model are shown in Table 5.15. The acceptability of the measurement model is evaluated by interpretability, size, and statistical significance of the model's parameter estimates. For interpretability, the parameter estimates do not take out-of-range values (e.g. negative error variance). For statistical significance, all factor loadings are statistically significant. For size, standardized factor loadings are considered poor (0.32–0.45), fair (0.45–0.55), good (0.55–0.63), very good (0.63–0.71), and excellent (> 0.71). In my measurement model, the lowest standardized factor loading is still considered to be good (> 0.55).

Table 5.15 Measurement model of the SEM for mediator roles of PC and ERI in OC–drinking relation

Parameter	Factor loadings			Residual variance
	Unstandardized coefficient	Standard Error	Standardized coefficient	
Men (n= 3782)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.985	0.030
Heavy drinking, wave 1	0.844	0.039	0.831	0.309
Problem drinking, wave 1	0.616	0.031	0.607	0.631
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.934	0.127
Heavy drinking, wave 2	0.738	0.036	0.689	0.525
Problem drinking, wave 2	0.612	0.030	0.572	0.673
Women (n= 3731)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.955	0.088
Heavy drinking, wave 1	0.768	0.033	0.733	0.462
Problem drinking, wave 1	0.807	0.037	0.771	0.405
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.985	0.030
Heavy drinking, wave 2	0.662	0.042	0.652	0.575
Problem drinking, wave 2	0.783	0.050	0.771	0.406

The structural model is specified in a different way from the previous SEM model (Section 5.2.3). There are two potential mediators (PC and ERI) between the effects of OC at wave 1 on drinking outcomes at wave 2 (Figure 5.4). Because the HAPIEE study is limited by no measurement of PC at wave 2, the cross–sectional associations between OC, ERI, and PC at wave 1 are used for this analysis. Thus, PC and ERI are only specified to be correlated, although bi-directional relationship between PC and ERI has been hypothesized.

An autoregressive and cross–lagged model for drinking outcomes is adopted. First, drinking outcomes at wave 2 are predicted by drinking outcomes at wave 1. Second, the cross–lagged effects of OC, PC, and ERI at wave 1 on drinking outcomes at wave 2 are measured, respectively. Third, the mediator effects can only be assessed by a half–longitudinal design. For example, the mediator role of PC in OC–drinking relation is assessed by two effects: (1) the cross–sectional association of OC at wave 1 on PC at wave 1; (2) the cross–lagged effect of PC at wave 1 on drinking at

wave 2. Partial mediation applies if both causal relations are confirmed; the product of two path coefficients might estimate the strength of mediator effect.[587]

Table 5.16 presents the structural model in men; the results are illustrated in Figure 5.5. First, the mediator effect of ERI in OC–drinking relation is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 1 (unstandardized β= 0.249; standard error= 0.020); (2) higher ERI at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.138; standard error= 0.032). This mediator effect of ERI is significant (0.034= 0.249 x 0.138; standard error= 0.008 and $p < 0.001$ by bootstrap method).

Second, the mediator effect of PC in OC–drinking relation in men is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.097; standard error= 0.017); (2) lower PC at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= –0.098; standard error= 0.031). This mediator effect of PC is significant (0.010= -0.097 x -0.098; standard error= 0.003 and p= 0.006). Third, ERI at wave 1 is inversely associated with PC at wave 1 (unstandardized β= –0.047; p= 0.002). Finally, the fit indexes are considered good fit (RMSEA= 0.053 < 0.06) or close to cutoffs for good fit (CFI= 0.917; TLI= 0.888).

Table 5.16 Structural model of the SEM for mediator roles of PC and ERI in OC–drinking relation in men (n= 3782)

Parameter	Unstandardized coefficient	Standardized coefficient	P value
Drinking wave 1 → Drinking wave 2	0.465	0.458	< 0.001
OC wave 1 → Drinking wave 2			
OC tertile 2 vs tertile 1	0.050	0.022	0.254
OC tertile 3 vs tertile 1	0.098	0.040	0.125
1–tertile increase	0.051	0.036	0.097
OC → ERI → Drinking	0.034	0.024	< 0.001
OC wave 1 → ERI wave 1			
OC tertile 2 vs tertile 1	0.253	0.151	< 0.001
OC tertile 3 vs tertile 1	0.494	0.270	< 0.001
1–tertile increase	0.249	0.231	< 0.001
ERI wave 1 → Drinking wave 2			
ERI tertile 2 vs tertile 1	0.165	0.070	0.005
ERI tertile 3 vs tertile 1	0.312	0.136	< 0.001
1–tertile increase	0.138	0.103	< 0.001
OC → PC → Drinking	0.010	0.008	0.006
OC wave 1 → PC wave 1			
OC tertile 2 vs tertile 1	- 0.012	- 0.007	0.724
OC tertile 3 vs tertile 1	- 0.198	- 0.109	< 0.001
1–tertile increase	- 0.097	- 0.095	< 0.001
PC wave 1 → Drinking wave 2			
PC tertile 2 vs tertile 1	- 0.030	- 0.013	0.484
PC tertile 3 vs tertile 1	- 0.208	- 0.095	< 0.001
1–tertile increase	- 0.098	- 0.072	0.003
ERI correlates with PC	- 0.047	- 0.071	0.002
Tests of model fit	RMSEA= 0.053	CFI= 0.917	TLI= 0.888

Figure 5.5 Results of the SEM for mediator roles of PC and ERI in OC–drinking relation in men

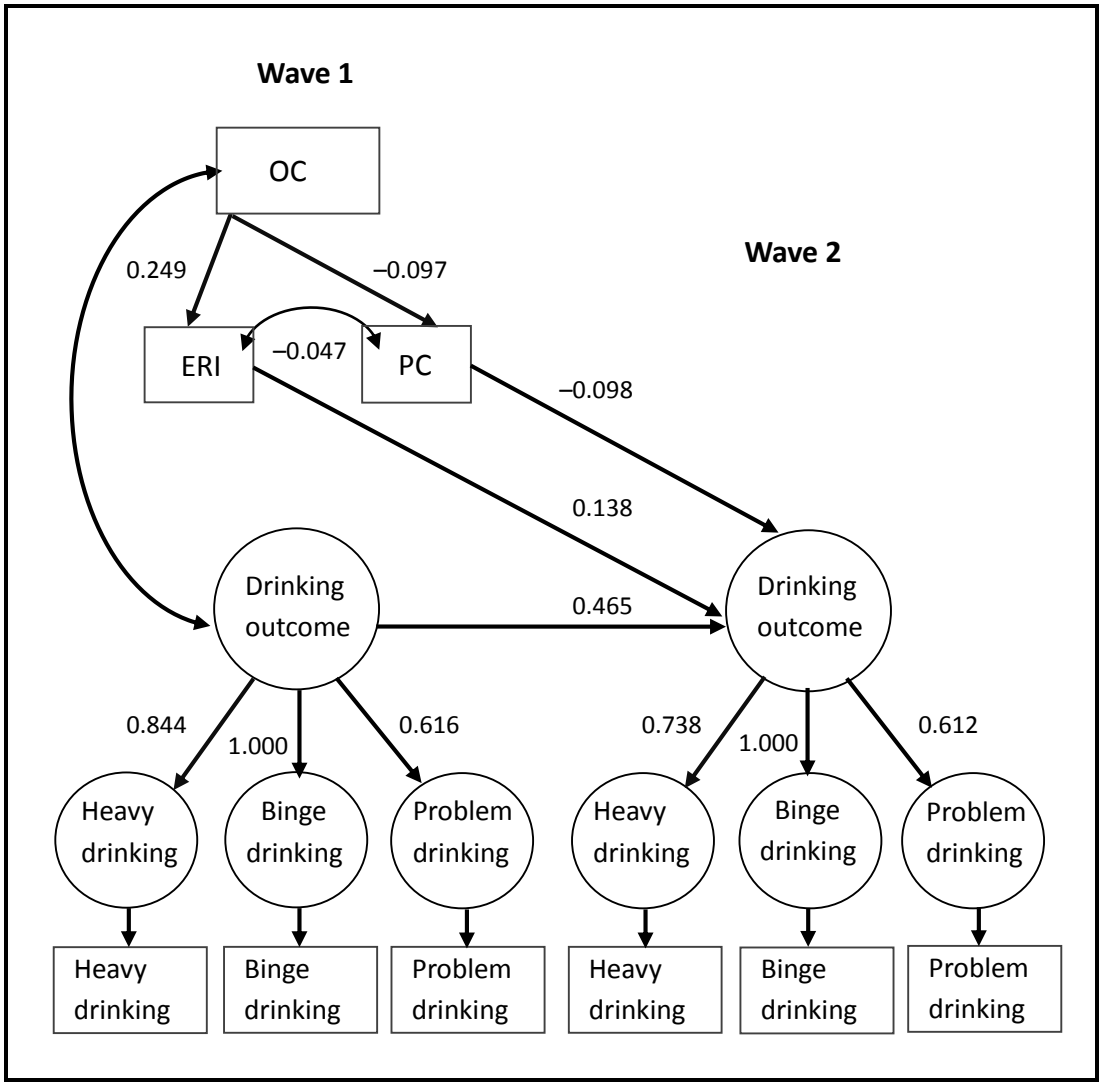


Table 5.17 shows the structural model in women, with the results illustrated in Figure 5.6. First, the mediator effect of ERI in OC–drinking relation is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 1 (unstandardized β= 0.240; standard error= 0.019); (2) higher ERI at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= 0.122; standard error= 0.039). This mediator effect of ERI is significant (0.029= 0.240 x 0.122; standard error= 0.009 and p= 0.002).

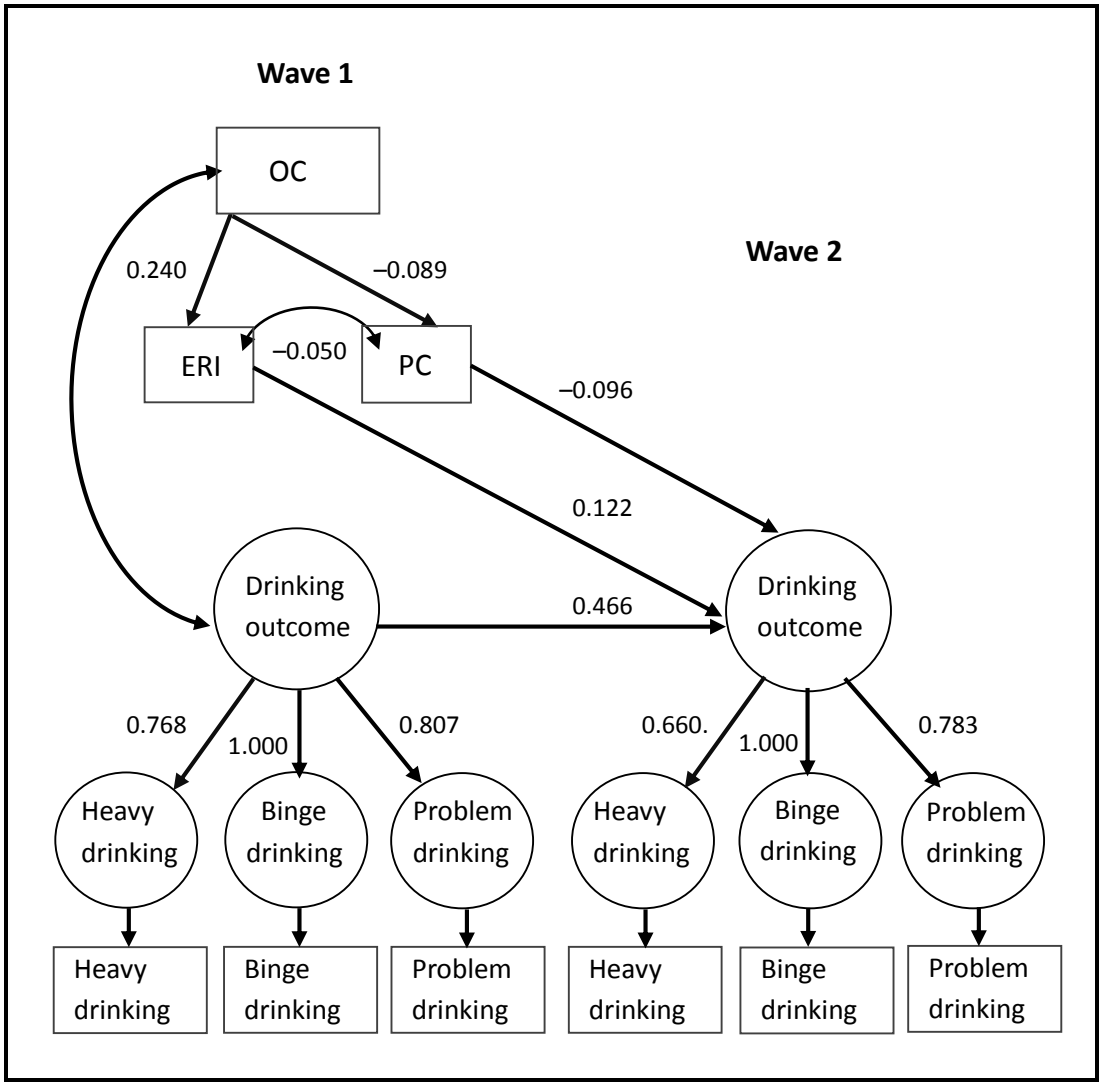
Second, the mediator effect of PC in OC–drinking relation is estimated by

multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.089; standard error= 0.017); (2) lower PC at wave 1 significantly associated with higher levels of drinking at wave 2 (unstandardized β= –0.096; standard error= 0.036). This mediator effect of PC is significant (0.009= -0.089 x -0.096; standard error= 0.004 and p= 0.017). Third, ERI at wave 1 is inversely associated with PC at wave 1 (unstandardized β= –0.050; p= 0.001). Finally, the fit indexes are considered good fit (RMSEA= 0.057 < 0.06) or close to cutoffs for good fit (CFI= 0.886; TLI= 0.875).

Table 5.17 Structural model of the SEM for mediator roles of PC and ERI in OC–drinking relation in women (n= 3731)

Parameter	Unstandardized coefficient	Standardized coefficient	P value
Drinking wave 1 → Drinking wave 2	0.466	0.470	< 0.001
OC wave 1 → Drinking wave 2			
OC tertile 2 vs tertile 1	0.021	0.010	0.646
OC tertile 3 vs tertile 1	0.091	0.039	0.252
1–tertile increase	0.052	0.040	0.186
OC → ERI → Drinking	0.029	0.022	0.002
OC wave 1 → ERI wave 1			
OC tertile 2 vs tertile 1	0.248	0.146	< 0.001
OC tertile 3 vs tertile 1	0.479	0.268	< 0.001
1–tertile increase	0.240	0.231	< 0.001
ERI wave 1 → Drinking wave 2			
ERI tertile 2 vs tertile 1	0.147	0.065	0.052
ERI tertile 3 vs tertile 1	0.274	0.124	< 0.001
1–tertile increase	0.122	0.096	0.002
OC → PC → Drinking	0.009	0.007	0.017
OC wave 1 → PC wave 1			
OC tertile 2 vs tertile 1	- 0.016	- 0.010	0.610
OC tertile 3 vs tertile 1	- 0.180	- 0.106	< 0.001
1–tertile increase	- 0.089	- 0.090	< 0.001
PC wave 1 → Drinking wave 2			
PC tertile 2 vs tertile 1	- 0.073	- 0.033	0.274
PC tertile 3 vs tertile 1	- 0.196	- 0.090	< 0.001
1–tertile increase	- 0.096	- 0.073	0.006
ERI correlates with PC	- 0.050	- 0.085	0.001
Tests of model fit	RMSEA= 0.057	CFI= 0.886	TLI= 0.875

Figure 5.6 Results of the SEM for mediator roles of PC and ERI in OC–drinking relation in women



5.3.3 Modifying role of PC in ERI–drinking relationship

Modifying effect of PC in ERI–drinking relationship is evaluated by two approaches. In the first approach, logistic regression analyses are conducted for 3 drinking outcomes at wave 2, respectively, regressed by PC tertile, ER–ratio tertile, and interaction term between PC tertile and ER–ratio tertile at wave 1 after adjustment for covariates. The log likelihoods for the model with this interaction term and the model without are compared, and LR tests show that this interaction term is not

significant for heavy drinking in men (p= 0.182) and women (p= 0.443), not significant for binge drinking in men (p= 0.523) and women (p= 0.206), and not significant for problem drinking in men (p= 0.175) and women (p= 0.284) (Table 5.18).

Table 5.18 Evaluation for modifying role of perceived control in ERI–drinking relationship

Approaches	Strata	Heavy drinking	Binge drinking	Problem drinking
Men		OR of outcome by 1–tertile increase in ER ratio (95% CI)		
1. ERI–drinking relation	PC tertile 1	1.10 (0.96 – 1.25)	1.40 (1.14 – 1.71)	1.20 (0.99 – 1.43)
in different strata of PC	PC tertile 2	1.10 (0.94 – 1.27)	1.47 (1.17 – 1.85)	1.22 (0.98 – 1.52)
tertile	PC tertile 3	1.27 (1.06 – 1.52)	1.45 (1.07 – 1.97)	1.55 (1.18 – 2.03)
2. Interaction PC x ERI	P–value	P= 0.182	P= 0.523	P= 0.175
Women		OR of outcome by 1–tertile increase in ER ratio (95% CI)		
1. ERI–drinking relation	PC tertile 1	1.09 (0.93 – 1.26)	1.51 (1.10 – 2.07)	1.14 (0.82 – 1.60)
in different strata of PC	PC tertile 2	1.14 (0.96 – 1.35)	1.34 (0.96 – 1.86)	1.44 (0.95 – 2.17)
tertile	PC tertile 3	0.97 (0.81 – 1.16)	1.04 (0.72 – 1.52)	1.49 (0.90 – 2.45)
2. Interaction PC x ERI	P–value	P= 0.443	P= 0.206	P= 0.284

Second, by a measurement model (Table 5.19) similar to previous SEM (Table 5.15), a path analysis is conducted for the latent drinking outcome at wave 2 regressed by PC tertile, ER–ratio tertile, and the interaction term between PC tertile and ER–ratio tertile at wave 1 after adjustment for covariates. In the SEM, this interaction term is not significant in men (unstandardized β= 0.049; standard error= 0.037 and p= 0.218) and in women (unstandardized β= 0.039; standard error= 0.040 and p= 0.315). My results based on the two approaches show no significantly modifying effect of PC in ERI–drinking relationship.

Table 5.19 Measurement model of the SEM for modifying role of PC in ERI–drinking relation

Parameter	Factor loadings			Residual variance
	Unstandardized coefficient	Standard Error	Standardized coefficient	
Men (n= 3782)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.990	0.020
Heavy drinking, wave 1	0.858	0.039	0.849	0.279
Problem drinking, wave 1	0.617	0.030	0.611	0.627
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.939	0.118
Heavy drinking, wave 2	0.738	0.037	0.693	0.520
Problem drinking, wave 2	0.615	0.030	0.577	0.667
Women (n= 3731)				
Latent variable – Drinking outcome				
Binge drinking, wave 1	1.000	0.000	0.964	0.071
Heavy drinking, wave 1	0.778	0.033	0.750	0.438
Problem drinking, wave 1	0.814	0.036	0.785	0.384
Latent variable – Drinking outcome				
Binge drinking, wave 2	1.000	0.000	0.975	0.049
Heavy drinking, wave 2	0.652	0.042	0.636	0.595
Problem drinking, wave 2	0.771	0.051	0.752	0.434

5.4 Main Findings for Drinking Outcomes

The analyses based on this 2–wave cohort study (3782 men and 3731 women aged 45–69) from the HAPIEE study report the following findings, which are in line with specific objectives and relevant hypotheses listed in Chapter 3.

In terms of the associations between ER ratio and drinking outcomes, Hypothesis 1 that higher ER ratio (wave 1) is associated with higher levels of alcohol drinking (wave 2) after adjustment for covariates is supported. In terms of the associations between OC and drinking outcomes, Hypothesis 4 that higher OC (wave 1) is associated with higher levels of alcohol drinking (wave 2) after adjustment for covariates is partially supported in binge drinking and problem drinking; however, the associations between OC and heavy drinking are marginally significant in men but non–significant in women.

With regards to the potential role of OC (antecedent, mediator, modifier, or direct

effect) in ERI–drinking relationship, Hypothesis 7 is supported. OC and ERI have bi–directional relationship, but the effect of OC on ERI is stronger than the other direction in the middle-aged and older populations. Antecedent role of OC in ERI–drinking relationship is found significant, but mediator role of OC is not significant. Direct effect of OC on drinking is not significant. Finally, modifying role of OC in ERI–drinking relation is not significant.

In terms of the associations between PC and drinking outcomes, Hypothesis 10 that lower PC (wave 1) is associated with higher levels of alcohol drinking (wave 2) after adjustment for covariates is supported.

With regards to the potential role of PC (mediator or modifier) in the relationship between ERI, OC and drinking, Hypothesis 13 is partially supported. PC and ERI partially mediate the effects of OC on alcohol drinking. In addition, PC and ERI may have bi–directional relationship. PC and ERI are negatively associated with each other in the cross–sectional analyses; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in cross–sectional analyses. Finally, modifying role of PC in ERI–drinking relation is non–significant.

Note that the methodological issues and interpretation of the main findings for drinking outcomes will be addressed in detail in Chapter 8, and their implications for practice, policy and research in Chapter 9.

Chapter 6. Smoking Outcomes

The aims of analyses for smoking outcomes in Chapter 6, based on the aims listed in Chapter 3, are as follows: (1) to examine the potential role of OC in ERI–smoking relationship, including modifying, antecedent, mediator, or direct effects; (2) to investigate the potential role of PC in the relationship between OC, ERI and smoking outcomes. The analyses use data from a 2–wave cohort study (3782 men and 3731 women aged 45–69), part of the HAPIEE study, which has been described in detail in Chapter 4 Methodology.

The results are presented in the following three parts. First, descriptive statistics for covariates and smoking outcomes by country and by gender are reported. Second, the associations of ERI and OC at wave 1 with smoking outcomes at wave 2, respectively, are evaluated by binary or ordinal logistic regression. The path analysis with an autoregressive and cross–lagged model is applied to examine antecedent or mediator roles of OC in ERI–smoking relationship. Modifying roles of OC in ERI–smoking relation are also examined. Third, the associations between PC at wave 1 and smoking outcomes at wave 2 are assessed by binary or ordinal logistic regression. The path analysis with an autoregressive and cross–lagged model is used to examine the relationship between OC, ERI, PC, and smoking outcomes. Modifying roles of PC in ERI–smoking relation are also tested.

6.1 Descriptive Statistics

6.1.1 Descriptive characteristics of study populations

In this sample of 7513 subjects (3782 men and 3731 women), the means of age assessed at wave 1 are 54.8 years in men (standard deviation= 6.0) and 53.2 years in women (standard deviation= 5.4). The average follow–up periods between wave 1 and wave 2 are 3.5 years in men (standard deviation= 0.7) and 3.6 years in women

(standard deviation= 0.6). Note that the descriptive statistics of covariates in this sample are the same as those described in Table 5.1 for drinking outcomes. Across the three countries, men generally have higher proportions in age group over 55, university–educated, manager/profession and manual workers, married/cohabiting, lower deprivation, less depression, and more social isolation than women.

6.1.2 Descriptive characteristics of smoking outcomes

Table 6.1 summarizes the descriptive statistics of smoking outcomes (smoking status in all subjects and smoking intensity in current smokers) by country and by gender at wave 1 and wave 2, respectively. There are gender differences across three countries. Men have dramatically higher proportions in current smokers and in heavy smokers than women.

There are country differences noted in Table 6.1. At wave 1, the percentages of current smokers are highest for Russian sample (46.4%) in men; in women, these percentages are highest for Polish sample (33.8%). Next, the percentages of heavy smokers (>= 20 cigarettes per day) in current smokers are highest for Polish men (59.2%) with averagely 19.1 (SD= 10.8) cigarettes smoked per day; these percentages are highest for Polish women (33.2%) with averagely 13.9 (SD= 8.0) cigarettes smoked per day. At wave 2, the percentages of current smokers are highest for Russian sample (44.7%) in men; in women, these percentages are highest for Polish sample (30.5%). The percentages of heavy smokers are highest for Russian men (58.7%) with averagely 18.7 (SD= 9.0) cigarettes per day; these percentages are highest for Polish women (31.7%) with averagely 13.4 (SD= 7.4) cigarettes per day.

Table 6.1 Descriptive statistics of smoking outcomes by country and gender (N= 7513)

Smoking outcomes	Czech		Russia		Poland	
	Men (n= 1082)	Women (n= 1099)	Men (n= 1402)	Women (n= 1394)	Men (n= 1298)	Women (n=1238)
Wave 1						
Smoking status, n (%)						
Lifetime non-smokers	403 (37.3)	548 (49.9)	421 (30.0)	1146 (82.2)	418 (32.2)	547 (44.2)
Former smokers	354 (32.7)	244 (22.2)	331 (23.6)	71 (5.1)	437 (33.7)	272 (22.0)
Current smokers	325 (30.0)	307 (27.9)	650 (46.4)	177 (12.7)	443 (34.1)	419 (33.8)
Smoking intensity in current smoker, n (%)						
Light smoker (1–9 /day)	68 (21.1)	122 (39.8)	74 (11.4)	89 (50.2)	72 (16.2)	104 (24.8)
Medium smoker (10–19/day)	118 (36.2)	128 (41.6)	203 (31.3)	66 (37.2)	109 (24.6)	176 (42.0)
Heavy smoker (>= 20/day)	139 (42.7)	57 (18.6)	373 (57.3)	22 (12.6)	262 (59.2)	139 (33.2)
Average cigarettes smoked per day, mean (s.d.)	15.5 (8.3)	10.6 (6.6)	18.2 (9.0)	9.5 (6.3)	19.1 (10.8)	13.9 (8.0)
Wave 2						
Smoking status, n (%)						
Lifetime non-smokers	415 (38.4)	602 (54.8)	395 (28.2)	1154 (82.8)	410 (31.6)	545 (44.0)
Former smokers	362 (33.5)	224 (20.4)	379 (27.0)	70 (5.0)	473 (36.4)	312 (25.2)
Current smokers	304 (28.1)	273 (24.8)	628 (44.8)	170 (12.2)	415 (32.0)	381 (30.8)
Smoking intensity in current smoker, n (%)						
Light smoker (1–9 /day)	65 (21.4)	102 (37.2)	68 (10.9)	77 (45.3)	62 (14.9)	99 (26.1)
Medium smoker (10–19/day)	127 (41.9)	127 (46.6)	191 (30.4)	73 (42.9)	123 (29.7)	161 (42.2)
Heavy smoker (>= 20/day)	112 (36.7)	44 (16.2)	369 (58.7)	20 (11.8)	230 (55.4)	121 (31.7)
Average cigarettes smoked per day, mean (s.d.)	15.1 (8.0)	10.7 (6.2)	18.7 (9.0)	9.5 (6.1)	18.6 (10.2)	13.4 (7.4)

The ways of pooling the data in subsequent analyses are described. First, men and women are analysed separately, as most studies on the relationships between psychosocial factors and health outcomes. Second, crude associations between exposure variables (ER ratio and OC) and smoking outcomes in country–specific strata are assessed. Binary and ordinal logistic regression analyses are conducted for two smoking outcomes (smoking status and smoking intensity) at wave 2, respectively, regressed by ER–ratio tertile, country, and interaction term between country and ER–ratio tertile at wave 1. By comparing the log likelihoods for the model with this interaction term and the model without, LR test is used to test significance of this

interaction term (Table 6.2). Similarly, the interaction term between country and OC tertile is evaluated (Table 6.3).

There are country differences seen in Table 6.2. In both sexes, the associations between ER ratio and smoking intensity in Poland are the strongest of all countries. Generally, crude associations between ER ratio at wave 1 and smoking outcomes at wave 2 are not very different across country–specific strata (all p–values by LR test > 0.32); no significant interaction between country and ER ratio is reported.

Table 6.2 Crude associations between ER ratio and smoking outcomes in country–specific strata

Strata	ER ratio Tertile▲	Smoking status: current smoker vs non–smoker OR (95% CI)	Smoking intensity in current smokers OR (95% CI)
Men			
Czech	Tertile 1	1.00	1.00
	Tertile 2	1.02 (0.72 – 1.45)	1.50 (0.88 – 2.56)
	Tertile 3	1.26 (0.90 – 1.76)	1.67 (0.93 – 2.99)
	P for trend	0.128	0.197
	OR by 1 tertile	1.14 (0.96 – 1.35)	1.19 (0.91 – 1.55)
Russia	Tertile 1	1.00	1.00
	Tertile 2	1.31 (1.04 – 1.64)*	1.05 (0.75 – 1.47)
	Tertile 3	1.65 (1.26 – 2.16)*	1.34 (0.91 – 1.98)
	P for trend	< 0.001	0.166
	OR by 1 tertile	1.29 (1.12 – 1.47)*	1.14 (0.95 – 1.39)
Poland	Tertile 1	1.00	1.00
	Tertile 2	1.31 (0.97 – 1.77)	1.43 (0.88 – 2.32)
	Tertile 3	1.47 (1.11 – 1.94)*	2.05 (1.30 – 3.25)*
	P for trend	0.008	0.002
	OR by 1 tertile	1.21 (1.05 – 1.38)*	1.42 (1.13 – 1.79)*
Interaction	Country x ERI	P= 0.669	P= 0.322
Women			
Czech	Tertile 1	1.00	1.00
	Tertile 2	1.27 (0.89 – 1.80)	0.88 (0.51 – 1.54)
	Tertile 3	1.40 (1.00 – 1.96)*	1.03 (0.61 – 1.76)
	P for trend	0.057	0.934
	OR by 1 tertile	1.17 (0.99 – 1.39)	1.01 (0.77 – 1.33)
Russia	Tertile 1	1.00	1.00
	Tertile 2	1.37 (0.94 – 1.99)	0.82 (0.46 – 1.58)
	Tertile 3	1.84 (1.22 – 2.75)*	1.24 (0.60 – 2.56)
	P for trend	0.003	0.603
	OR by 1 tertile	1.35 (1.10 – 1.66)*	1.10 (0.77 – 1.58)
Poland	Tertile 1	1.00	1.00
	Tertile 2	1.22 (0.90 – 1.66)	1.43 (0.91 – 2.24)
	Tertile 3	1.46 (1.09 – 1.94)*	1.48 (0.92 – 2.42)
	P for trend	0.009	0.148
	OR by 1 tertile	1.21 (1.05 – 1.39)*	1.20 (0.94 – 1.50)
Interaction	Country x ERI	P= 0.866	P= 0.451

▲ Country– and gender–specific tertiles of ER ratio. * P value < 0.05.

There are country differences observed in Table 6.3. In men, the association between OC and smoking status in Poland is the strongest of all countries. Overall, crude associations between OC at wave 1 and smoking outcomes at wave 2 are not very different across country–specific strata (all p–values by LR test > 0.26); no significant interaction between country and OC is reported. Due to no significant interactions between country and exposure variables (ER ratio and OC), data for the three countries are pooled for further analyses.

Table 6.3 Crude associations between OC and smoking outcomes in country–specific strata

Strata	OC score Tertile▲	Smoking status: current smoker vs non–smoker OR (95% CI)	Smoking intensity in current smokers OR (95% CI)
Men			
Czech	Tertile 1	1.00	1.00
	Tertile 2	0.97 (0.74 – 1.27)	1.02 (0.62 – 1.70)
	Tertile 3	1.03 (0.75 – 1.43)	1.17 (0.69 – 1.99)
	P for trend	0.968	0.557
	OR by 1 tertile	1.00 (0.85 – 1.19)	1.08 (0.83 – 1.41)
Russia	Tertile 1	1.00	1.00
	Tertile 2	1.17 (0.89 – 1.53)	1.03 (0.72 – 1.47)
	Tertile 3	1.20 (0.94 – 1.55)	1.12 (0.74 – 1.68)
	P for trend	0.149	0.614
	OR by 1 tertile	1.10 (0.97 – 1.26)	1.05 (0.86 – 1.28)
Poland	Tertile 1	1.00	1.00
	Tertile 2	1.19 (0.91 – 1.55)	1.18 (0.77 – 1.80)
	Tertile 3	1.48 (1.11 – 1.98)*	1.59 (1.00 – 2.54)*
	P for trend	0.008	0.056
	OR by 1 tertile	1.22 (1.05 – 1.41)*	1.25 (0.99 – 1.58)
Interaction	Country x OC	P= 0.262	P= 0.825
Women			
Czech	Tertile 1	1.00	1.00
	Tertile 2	1.17 (0.84 – 1.61)	0.90 (0.51 – 1.50)
	Tertile 3	1.42 (1.02 – 1.98)*	0.93 (0.53 – 1.61)
	P for trend	0.040	0.792
	OR by 1 tertile	1.19 (1.01 – 1.41)*	0.97 (0.74 – 1.27)
Russia	Tertile 1	1.00	1.00
	Tertile 2	1.19 (0.79 – 1.78)	1.14 (0.55 – 2.36)
	Tertile 3	1.33 (0.91 – 1.96)	1.21 (0.61 – 2.42)
	P for trend	0.129	0.571
	OR by 1 tertile	1.16 (0.96 – 1.40)	1.10 (0.78 – 1.55)
Poland	Tertile 1	1.00	1.00
	Tertile 2	0.93 (0.71 – 1.23)	1.17 (0.76 – 1.79)
	Tertile 3	1.12 (0.83 – 1.50)	1.62 (1.03 – 2.56)*
	P for trend	0.533	0.041
	OR by 1 tertile	1.05 (0.90 – 1.21)	1.27 (1.01 – 1.59)*
Interaction	country x OC	P= 0.756	P= 0.606

▲ Country– and gender–specific tertiles of OC score. * P value < 0.05.

Table 6.4 shows the bivariate analyses between covariates at wave 1 and smoking outcomes at wave 2 in men. Chi–squared tests and p–values for heterogeneity are obtained to examine the differences between categories of the variable. Smoking status as current smokers is significantly ($p < 0.05$) associated with younger age, less education, manual worker, divorce or widowed, higher deprivation, and poorer self-rated health. Besides, the association of higher smoking intensity with lower education is significant ($p < 0.05$), and the association of higher smoking intensity with manual workers is marginally significant ($p < 0.1$).

Table 6.4 Bivariate analyses between covariates and smoking outcomes in men

	Smoking status		Smoking intensity in current smokers		
Covariates	Non–smokers (%) n= 2435	Current smokers (%) n= 1347	Light smokers (%) n= 195	Medium smokers (%) n= 441	Heavy smokers (%) n= 711
Age: 45 – 49	58.4	41.6	13.9	31.8	54.3
50 – 54	63.4	36.6	14.4	32.6	53.0
55 – 59	67.7	32.3	13.0	34.2	52.8
60 – 69	74.0	26.0	16.3	33.0	50.7
P value	P < 0.001		P = 0.519		
Education: Primary/ less	44.4	55.6	3.4	32.6	64.0
Vocational	60.2	39.8	10.2	37.7	52.1
Secondary	61.7	38.3	17.2	30.6	52.2
University	74.9	25.1	17.3	30.5	52.2
P value	P < 0.001		P < 0.001		
Occupation class					
Manager/ profession	68.1	31.9	13.2	35.9	50.9
Non-manual worker	70.4	29.6	17.1	32.0	50.9
Manual worker	56.2	43.8	11.9	31.6	56.4
P value	P < 0.001		P = 0.066		
Marital status					
Married/ cohabiting	66.2	33.8	14.8	32.6	52.5
Single	69.4	30.6	8.8	32.4	58.8
Divorce/ widowed	56.7	43.3	10.0	34.6	55.4
P value	P = 0.003		P = 0.523		
Deprivation: Low (0– 3.9)	68.0	32.0	14.5	33.7	51.8
High (4– 9)	56.1	43.9	13.7	30.1	56.3
P value	P < 0.001		P = 0.323		
Depression: CESD < 16	67.1	32.9	14.5	33.7	51.8
CESD >= 16	64.6	35.4	13.7	30.1	56.3
P value	P = 0.299		P = 0.313		
Social isolation					
No (>= once a month)	65.9	34.1	15.3	33.0	51.8
Yes (< once a month)	65.2	34.8	13.1	32.6	54.3
P value	P = 0.596		P = 0.434		
Self-rated health					
Very good– average	66.2	33.8	14.5	32.9	52.7
Poor– very poor	56.6	43.4	11.9	32.2	55.9
P value	P = 0.001		P = 0.691		

Table 6.5 shows the bivariate analyses between covariates at wave 1 and smoking outcomes at wave 2 in women. Being current smokers is significantly ($p < 0.05$) associated with younger age, less education, manual worker, more depression, and less social isolation. In current smokers, higher smoking intensity is significantly associated with being single, more depression, and more social isolation, respectively.

Table 6.5 Bivariate analyses between covariates and smoking outcomes in women

	Smoking status		Smoking intensity in current smokers		
Covariates	Non–smokers (%) n= 2907	Current smokers (%) n= 824	Light smokers (%) n= 278	Medium smokers (%) n= 361	Heavy smokers (%) n= 185
Age: 45 – 49	73.4	26.6	35.2	42.7	22.1
50 – 54	76.6	23.5	32.2	44.4	23.4
55 – 59	83.7	16.3	32.8	45.6	21.6
60 – 69	92.1	7.9	35.1	43.4	21.4
P value	P < 0.001		P = 0.686		
Education					
Primary or less	73.2	26.8	34.0	39.6	26.4
Vocational	76.9	23.1	33.4	44.0	22.6
Secondary	77.3	22.7	31.7	46.1	22.2
University	82.8	17.2	35.9	41.3	22.9
P value	P < 0.001		P = 0.697		
Occupation class					
Manager/ profession	80.1	19.9	37.2	42.1	20.7
Non-manual worker	79.3	20.7	33.3	42.9	23.8
Manual worker	74.8	25.2	32.3	50.0	17.7
P value	P = 0.027		P = 0.340		
Marital status					
Married/ cohabiting	79.7	20.3	34.1	46.7	19.3
Single	77.5	22.5	29.5	35.9	34.6
Divorce/ widowed	76.4	23.6	34.7	39.6	25.7
P value	P = 0.069		P = 0.005		
Deprivation					
Low (0 – 3.9)	78.5	21.5	35.6	43.6	20.8
High (4 – 9)	79.2	20.8	29.1	44.8	26.1
P value	P = 0.653		P = 0.098		
Depression					
CESD < 16	78.8	21.2	30.0	47.8	22.3
CESD >= 16	74.9	25.1	40.8	32.6	26.6
P value	P = 0.016		P = 0.001		
Social isolation					
No (>= once a month)	77.2	22.8	36.8	44.9	18.4
Yes (< once a month)	79.7	20.3	29.3	42.2	28.5
P value	P = 0.006		P < 0.001		
Self-rated health					
Very good– average	78.6	21.4	33.2	44.2	22.6
Poor– very poor	79.9	20.1	38.3	40.7	21.0
P value	P = 0.533		P = 0.656		

6.2 Potential Role of OC in ERI–Smoking Relationship

This section focuses on the associations between ERI and smoking outcomes, on the associations between OC and smoking outcomes, and on assessing whether OC has the potential role of antecedent, mediator, modifier or direct effect in the relationship between ERI and smoking. Data for three countries are pooled for further analyses due to no significant interaction between country and exposure variables.

6.2.1 Associations between ERI and smoking outcomes

The association between ER ratio at wave 1 and smoking status (binary outcome: current smokers versus non-smokers) at wave 2 is assessed using binary logistic regression. Additionally, the association between ER ratio at wave 1 and smoking intensity among current smokers (ordinal categorical outcome: light smoker, medium smoker, and heavy smoker) is evaluated using ordinal logistic regression. These associations are assessed after adjustment for age and country (Model 1) and after additionally adjustment for other covariates (Model 2).

Table 6.6 reports the associations between ER ratio at wave 1 and two smoking outcomes at wave 2. In men, the odds of being current smokers are 1.40 for highest versus lowest tertile of ER ratio in Model 2; the adjusted OR changes for being current smokers are 1.18 by 1–tertile increase in ER ratio (p= 0.001). Among current smokers, the odds of a higher versus a lower outcome of smoking intensity (>= 20 versus < 20 cigarettes/day; >= 10 versus < 10 cigarettes/day) are 1.41 for highest versus lowest tertile of ER ratio in Model 2; the adjusted OR changes for a higher versus a lower outcome of smoking intensity are 1.19 by 1–tertile increase in ER ratio (p= 0.015).

In women, the odds of being current smokers are 1.48 for highest versus lowest tertile of ER ratio in Model 2; the adjusted OR changes for being current smokers are 1.21 by 1–tertile increase in ER ratio (p < 0.001). Among current smokers, the odds of a higher versus a lower outcome of smoking intensity are 1.33 for highest versus

lowest tertile of ER ratio in Model 2; the adjusted OR changes for a higher versus a lower outcome of smoking intensity are 1.16 by 1–tertile increase in ER ratio (p= 0.088).

Table 6.6 Associations between ER ratio at wave 1 and smoking outcomes at wave 2

Model	ER ratio Tertile▲	Smoking status – current smokers vs non-smokers OR (95% CI)	Smoking intensity in current smokers OR (95% CI)
Men		N = 3782	N = 1347
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	1.26 (1.07 – 1.49)*	1.32 (1.02 – 1.70)*
	Tertile 3	1.39 (1.18 – 1.63)*	1.49 (1.14 – 1.93)*
	P for trend	0.001	0.002
	OR by 1 tertile	1.18 (1.09 – 1.28)*	1.22 (1.07 – 1.39)*
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	1.33 (1.09 – 1.61)*	1.28 (0.97 – 1.69)
	Tertile 3	1.40 (1.16 – 1.68)*	1.41 (1.06 – 1.87)*
	P for trend	0.001	0.015
	OR by 1 tertile	1.18 (1.08 – 1.30)*	1.19 (1.03 – 1.37)*
Women		N = 3731	N = 824
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	1.32 (1.09 – 1.61)*	1.10 (0.80 – 1.51)
	Tertile 3	1.44 (1.18 – 1.74)*	1.45 (1.06 – 1.97)*
	P for trend	0.001	0.042
	OR by 1 tertile	1.20 (1.08 – 1.31)*	1.18 (1.01 – 1.38)*
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	1.18 (0.92 – 1.51)	1.06 (0.74 – 1.50)
	Tertile 3	1.48 (1.15 – 1.90)*	1.33 (0.95 – 1.88)*
	P for trend	< 0.001	0.088
	OR by 1 tertile	1.21 (1.08 – 1.36)*	1.16 (0.98 – 1.37)

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health. ▲ Gender–specific tertile of ER ratio: in men, tertile 1 (0.20–0.32), tertile 2 (0.32–0.47), and tertile 3 (>= 0.47); in women, tertile 1 (0.20–0.31), tertile 2 (0.31–0.46), and tertile 3 (>= 0.46). * P value < 0.05.

6.2.2 Associations between OC and smoking outcomes

The association between OC at wave 1 and smoking status at wave 2 is assessed using binary logistic regression. Additionally, the association between OC at wave 1 and smoking intensity among current smokers is evaluated using ordinal logistic regression. These associations are assessed after adjustment for age and country (Model 1) and after additionally adjustment for other covariates (Model 2).

Table 6.7 reports the associations between OC score at wave 1 and two smoking

outcomes at wave 2, respectively. In men, the odds of being current smokers are 1.33 for highest versus lowest tertile of OC in Model 2; the adjusted OR changes for being current smokers are 1.14 by 1–tertile increase in OC (p= 0.008). For current smokers, the odds of a higher versus a lower outcome of smoking intensity are 1.20 for highest versus lowest tertile of OC in Model 2; the adjusted OR changes for a higher versus a lower outcome of smoking intensity are 1.11 by 1–tertile increase in OC (p= 0.171).

In women, the odds of being current smokers are 1.32 for highest versus lowest tertile of OC in Model 2; the adjusted OR changes for being current smokers are 1.15 by 1–tertile increase in OC (p= 0.024). Among current smokers, the odds of a higher versus a lower outcome of smoking intensity are 1.29 for highest versus lowest tertile of OC in Model 2; the adjusted OR changes for a higher versus a lower outcome of smoking intensity are 1.14 by 1–tertile increase in OC (p= 0.145).

Table 6.7 Associations between OC at wave 1 and smoking outcomes at wave 2

Model	OC score Tertile▲	Smoking status – current smokers vs non-smokers OR (95% CI)	Smoking intensity in current smokers OR (95% CI)
Men		N = 3782	N = 1347
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	1.07 (0.91 – 1.26)	1.21 (0.93 – 1.56)
	Tertile 3	1.20 (1.01 – 1.40)*	1.25 (0.96 – 1.63)
	P for trend	0.061	0.054
	OR by 1 tertile	1.08 (1.00 – 1.18)	1.13 (1.00 – 1.30)
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	1.06 (0.89 – 1.26)	1.17 (0.90 – 1.52)
	Tertile 3	1.33 (1.07 – 1.60)*	1.20 (0.90 – 1.61)
	P for trend	0.008	0.171
	OR by 1 tertile	1.14 (1.03 – 1.28)*	1.11 (0.96 – 1.28)
Women		N = 3731	N = 824
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	1.24 (1.04 – 1.49)*	1.13 (0.84 – 1.53)
	Tertile 3	1.35 (1.11 – 1.62)*	1.21 (0.89 – 1.65)
	P for trend	0.001	0.220
	OR by 1 tertile	1.16 (1.06 – 1.28)*	1.10 (0.94 – 1.28)
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	1.15 (0.91 – 1.47)	1.09 (0.79 – 1.52)
	Tertile 3	1.32 (1.03 – 1.69)*	1.29 (0.92 – 1.82)
	P for trend	0.024	0.145
	OR by 1 tertile	1.15 (1.01 – 1.30)*	1.14 (0.96 – 1.35)

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates. ▲ Gender–specific tertile of OC score in men and women: tertile 1 (6–12), tertile 2 (12–15), and tertile 3 (15–24). * P value < 0.05.

Previous analyses have shown consistent associations of exposure variables (ER ratio and OC) with two smoking outcomes. Most associations between exposure variables and smoking outcomes are significant, but OC–smoking intensity associations in men and women do not reach statistical significance.

6.2.3 Antecedent or mediator role of OC in ERI–smoking relationship

To assess antecedent or mediator roles of OC in ERI–smoking relationship, the path analyses for binary categorical outcome (smoking status) and for ordinal categorical outcome (smoking intensity) are applied by Mplus 7. Each path coefficient is obtained by probit regression for an outcome on a predictor after adjustment for covariates. The odds ratio (OR) is the exponential (antilog) of estimated logistic coefficient, which is calculated from probit coefficient multiplied by 1.8.[588] In terms of predictors, first, the tertiles of ERI and OC are transformed into a series of dummy variables to compare between tertile groups in each predictor. Second, ERI tertile and OC tertile are treated as continuous variables to estimate assumed linear trend between the exposure and odds of smoking outcomes.

Path analysis with an autoregressive and cross–lagged model is specified in this 2–wave cohort study (Figure 6.1). First, each variable is predicted by the same variable at an early wave. Second, the cross–lagged effects of “OC at wave 1 on ERI at wave 2” and “ERI at wave 1 on OC at wave 2” are assessed, respectively, to identify causal directionality between OC and ERI. Bidirectional relationship between OC and ERI is possible based on my hypotheses.

Third, as mediation is a causal chain involving at least two causal relations, these causal relations can be tested separately using two phases of data. The mediator role of ERI in OC–smoking relation is assessed by two cross–lagged effects: (1) OC at wave 1 on ERI at wave 2; (2) ERI at wave 1 on smoking at wave 2. The mediator role of OC in ERI–smoking relation is estimated by two cross–lagged effects: (1) ERI at

wave 1 on OC at wave 2; (2) OC at wave 1 on smoking at wave 2. Partial mediation applies if both causal relationships are confirmed; the product of two path coefficients can estimate the strength of mediator effect.

For a mediator effect, the effect size measure is the product of two path coefficients. The bootstrap method is used for significance testing of the mediator effect with 5000 bootstrap samples to yield valid estimates for the mediator effect by Mplus 7. This method is adopted due to complicated models (categorical outcomes and multiple mediators) in the SEM.

For tests of model fit, three indexes are adopted: Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Tucker–Lewis Index (TLI). RMSEA < 0.06, CFI > 0.95 or TLI > 0.95 indicate “good model fit”.

Figure 6.1 Hypothetical model specified for the path analysis for antecedent or mediator role of OC in ERI–smoking relation

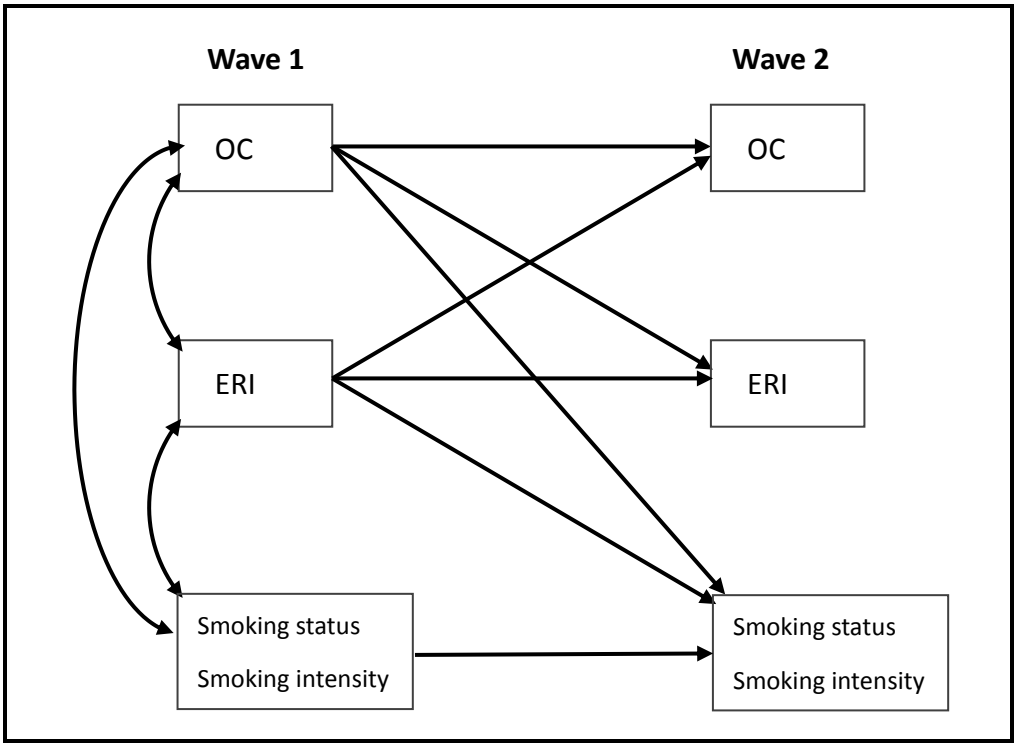


Table 6.8 reports the results of path analysis for the outcome of smoking status in men, with the results shown in Figure 6.2. First, the mediator effect of ERI in OC–smoking relation is estimated by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 2 (unstandardized β= 0.155; standard error= 0.020); (2) higher ERI at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= 0.075; standard error= 0.028). Thus, this mediator effect of ERI is significant (0.012= 0.155 x 0.075; standard error= 0.005 and p= 0.011 by bootstrap method).

Second, the mediator effect of OC in ERI–smoking relation is estimated by multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.084; standard error= 0.020); (2) higher OC at wave 1 non-significantly associated with being current smokers at wave 2 (unstandardized β= 0.028; standard error= 0.028; p= 0.321). This mediator effect of OC is not significant (0.002= 0.084 x 0.028; standard error= 0.002 and p= 0.330). Third, the fit indexes are considered good fit (RMSEA= 0.048 < 0.06; CFI= 0.953 > 0.95) or close to cutoffs for good fit (TLI= 0.843).

Table 6.8 Results of path analysis for antecedent or mediator role of OC in ERI–smoking status relationship in men (N= 3782)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Autoregressive model				
Smoking status wave 1 → wave 2	4.68	0.908	0.554	< 0.001
OC wave 1 → OC wave 2				
OC tertile 2 vs tertile 1	1.66	0.290	0.157	< 0.001
OC tertile 3 vs tertile 1	3.14	0.674	0.323	< 0.001
OR change by 1 tertile	1.79	0.337	0.303	< 0.001
ERI wave 1 → ERI wave 2				
ERI tertile 2 vs tertile 1	1.46	0.211	0.123	< 0.001
ERI tertile 3 vs tertile 1	2.28	0.484	0.283	< 0.001
OR change by 1 tertile	1.53	0.238	0.242	< 0.001
OC → ERI → Smoking status	1.02	0.012	0.009	0.011
OC wave 1 → ERI wave 2				
OC tertile 2 vs tertile 1	1.32	0.156	0.091	< 0.001
OC tertile 3 vs tertile 1	1.70	0.304	0.158	< 0.001
OR change by 1 tertile	1.32	0.155	0.151	< 0.001
ERI wave 1 → Smoking status wave 2				
ERI tertile 2 vs tertile 1	1.27	0.131	0.048	0.020
ERI tertile 3 vs tertile 1	1.35	0.168	0.062	0.008
OR change by 1 tertile	1.14	0.075	0.058	0.010
ERI → OC → Smoking status	1.00	0.002	0.002	0.330
ERI wave 1 → OC wave 2				
ERI tertile 2 vs tertile 1	1.26	0.128	0.070	< 0.001
ERI tertile 3 vs tertile 1	1.38	0.179	0.096	< 0.001
OR change by 1 tertile	1.16	0.084	0.078	< 0.001
OC wave 1 → Smoking status wave 2				
OC tertile 2 vs tertile 1	1.02	0.010	0.003	0.725
OC tertile 3 vs tertile 1	1.17	0.088	0.029	0.132
OR change by 1 tertile	1.05	0.028	0.021	0.321
Tests of model fit	RMSEA=	0.048	CFI= 0.953	TLI= 0.843

Figure 6.2 Results of path analysis for antecedent or mediator role of OC in ERI–smoking status relationship in men

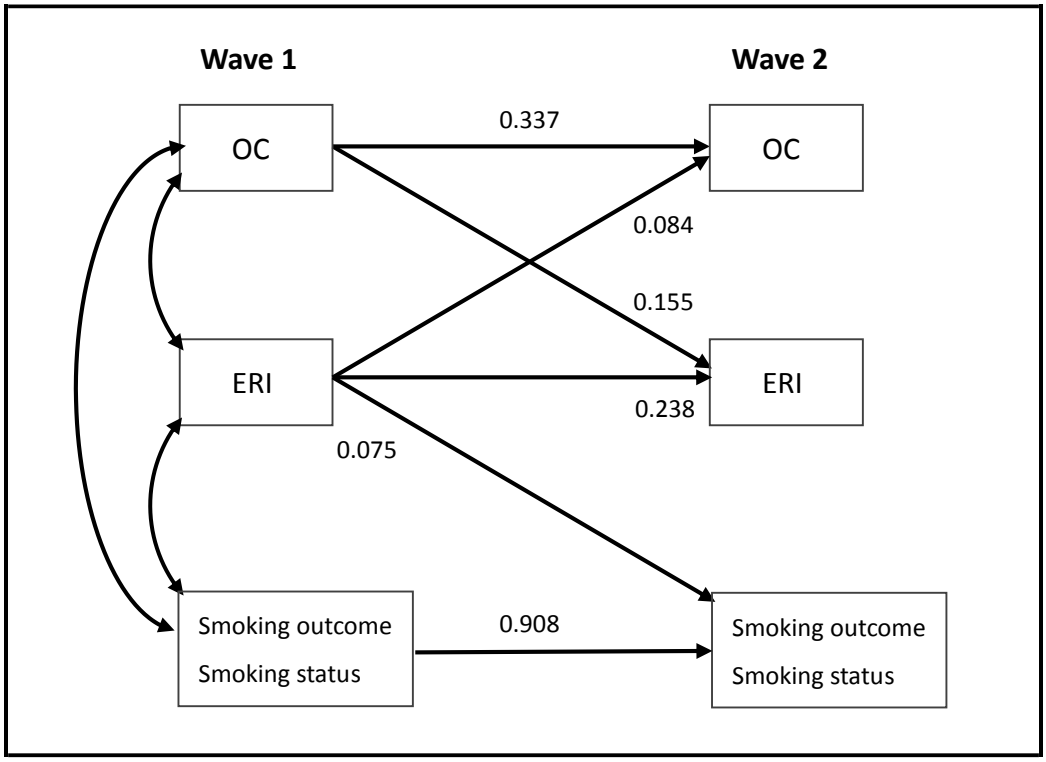


Table 6.9 shows the results of path analysis for the outcome of smoking status in women, with the results illustrated in Figure 6.3. First, the mediator effect of ERI in OC–smoking relation is estimated by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 2 (unstandardized β= 0.155; standard error= 0.019); (2) higher ERI at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= 0.085; standard error= 0.024). This mediator effect of ERI is significant (0.013= 0.155 x 0.085; standard error= 0.004 and p= 0.002 by bootstrap method).

Second, the mediator effect of OC in ERI–smoking relationship is evaluated by multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.075; standard error= 0.020); (2) higher OC at wave 1 non-significantly associated with being current smokers at wave 2 (unstandardized β= 0.037; standard error= 0.025; p= 0.113). This mediator effect of

OC is not significant (0.003; standard error= 0.002 and p= 0.168). Third, the fit indexes show good fit (RMSEA= 0.055 < 0.06) or close to good fit (CLI= 0.926; TLI= 0.825).

Table 6.9 Results of path analysis for antecedent or mediator role of OC in ERI–smoking status relationship in women (N= 3731)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Autoregressive model				
Smoking status wave 1 → wave 2	4.75	0.917	0.562	< 0.001
OC wave 1 → OC wave 2				
OC tertile 2 vs tertile 1	1.74	0.326	0.175	< 0.001
OC tertile 3 vs tertile 1	3.27	0.698	0.357	< 0.001
OR change by 1 tertile	1.83	0.357	0.340	< 0.001
ERI wave 1 → ERI wave 2				
ERI tertile 2 vs tertile 1	1.62	0.275	0.163	< 0.001
ERI tertile 3 vs tertile 1	2.37	0.507	0.301	< 0.001
OR change by 1 tertile	1.56	0.252	0.262	< 0.001
OC → ERI → Smoking status	1.02	0.013	0.010	0.002
OC wave 1 → ERI wave 2				
OC tertile 2 vs tertile 1	1.27	0.131	0.076	< 0.001
OC tertile 3 vs tertile 1	1.76	0.334	0.185	< 0.001
OR change by 1 tertile	1.32	0.155	0.149	< 0.001
ERI wave 1 → Smoking status wave 2				
ERI tertile 2 vs tertile 1	1.12	0.063	0.022	0.395
ERI tertile 3 vs tertile 1	1.45	0.211	0.074	< 0.001
OR change by 1 tertile	1.17	0.085	0.066	0.002
ERI → OC → Smoking status	1.01	0.003	0.002	0.168
ERI wave 1 → OC wave 2				
ERI tertile 2 vs tertile 1	1.16	0.083	0.046	0.022
ERI tertile 3 vs tertile 1	1.35	0.166	0.091	< 0.001
OR change by 1 tertile	1.07	0.075	0.071	< 0.001
OC wave 1 → Smoking status wave 2				
OC tertile 2 vs tertile 1	1.07	0.037	0.012	0.569
OC tertile 3 vs tertile 1	1.13	0.066	0.021	0.364
OR change by 1 tertile	1.07	0.037	0.030	0.113
Tests of model fit	RMSEA=	0.055	CFI= 0.926	TLI= 0.825

Figure 6.3 Results of path analysis for antecedent or mediator role of OC in ERI–smoking status relationship in women

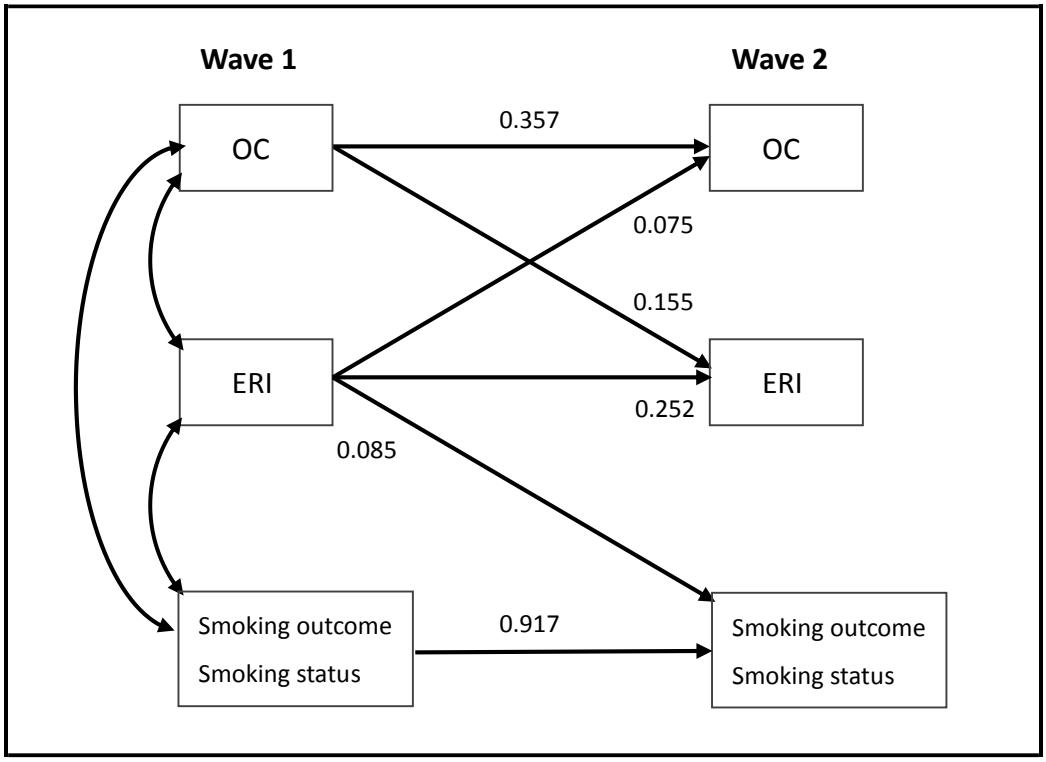


Table 6.10 reports the results of path analysis for the outcome of smoking intensity among current smokers in men, with the results illustrated in Figure 6.4. First, the mediator effect of ERI in OC–smoking relation is assessed by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 is significantly associated with higher ERI at wave 2 (unstandardized β= 0.167; standard error= 0.030); (2) higher ERI at wave 1 is marginally significantly associated with higher levels of smoking intensity at wave 2 (unstandardized β= 0.084; standard error= 0.039; p= 0.080). This mediator effect of ERI appears significant (0.014= 0.167 x 0.084; standard error= 0.007 and p= 0.042).

Second, the mediator effect of OC in ERI–smoking relation is estimated by multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.091; standard error= 0.033); (2) higher OC at wave 1 non-significantly related to higher levels of smoking intensity at wave 2 (unstandardized β= 0.028; standard error= 0.040; p= 0.358). This mediator effect of

OC is not significant (0.003; standard error= 0.004 and p= 0.497). Third, the fit indexes show good fit (RMSEA= 0.044 < 0.06; CFI= 0.962 > 0.95) or close to cutoffs of good fit (TLI= 0.905).

Table 6.10 Results of path analysis for antecedent or mediator role of OC in ERI–smoking intensity relationship in men (N= 1347)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Autoregressive model				
Smoking intensity wave 1 → wave 2	4.49	0.884	0.616	< 0.001
OC wave 1 → OC wave 2				
OC tertile 2 vs tertile 1	1.58	0.260	0.138	< 0.001
OC tertile 3 vs tertile 1	3.28	0.699	0.342	< 0.001
OR change by 1 tertile	1.80	0.346	0.313	< 0.001
ERI wave 1 → ERI wave 2				
ERI tertile 2 vs tertile 1	1.52	0.235	0.140	< 0.001
ERI tertile 3 vs tertile 1	2.05	0.442	0.271	< 0.001
OR change by 1 tertile	1.47	0.223	0.235	< 0.001
OC → ERI → Smoking intensity	1.03	0.014	0.009	0.042
OC wave 1 → ERI wave 2				
OC tertile 2 vs tertile 1	1.45	0.208	0.125	< 0.001
OC tertile 3 vs tertile 1	1.74	0.327	0.178	< 0.001
OR change by 1 tertile	1.35	0.167	0.170	< 0.001
ERI wave 1 → Smoking intensity wave 2				
ERI tertile 2 vs tertile 1	1.26	0.129	0.046	0.185
ERI tertile 3 vs tertile 1	1.35	0.168	0.061	0.088
OR change by 1 tertile	1.16	0.084	0.054	0.080
ERI → OC → Smoking intensity	1.00	0.003	0.002	0.497
ERI wave 1 → OC wave 2				
ERI tertile 2 vs tertile 1	1.19	0.097	0.051	0.129
ERI tertile 3 vs tertile 1	1.41	0.190	0.103	< 0.001
OR change by 1 tertile	1.18	0.091	0.086	0.006
OC wave 1 → Smoking intensity wave 2				
OC tertile 2 vs tertile 1	1.06	0.035	0.013	0.620
OC tertile 3 vs tertile 1	1.14	0.075	0.028	0.253
OR change by 1 tertile	1.05	0.028	0.016	0.358
Tests of model fit	RMSEA=	0.044	CFI= 0.962	TLI= 0.905

Figure 6.4 Results of path analysis for antecedent or mediator role of OC in ERI–smoking intensity relationship in current smokers in men

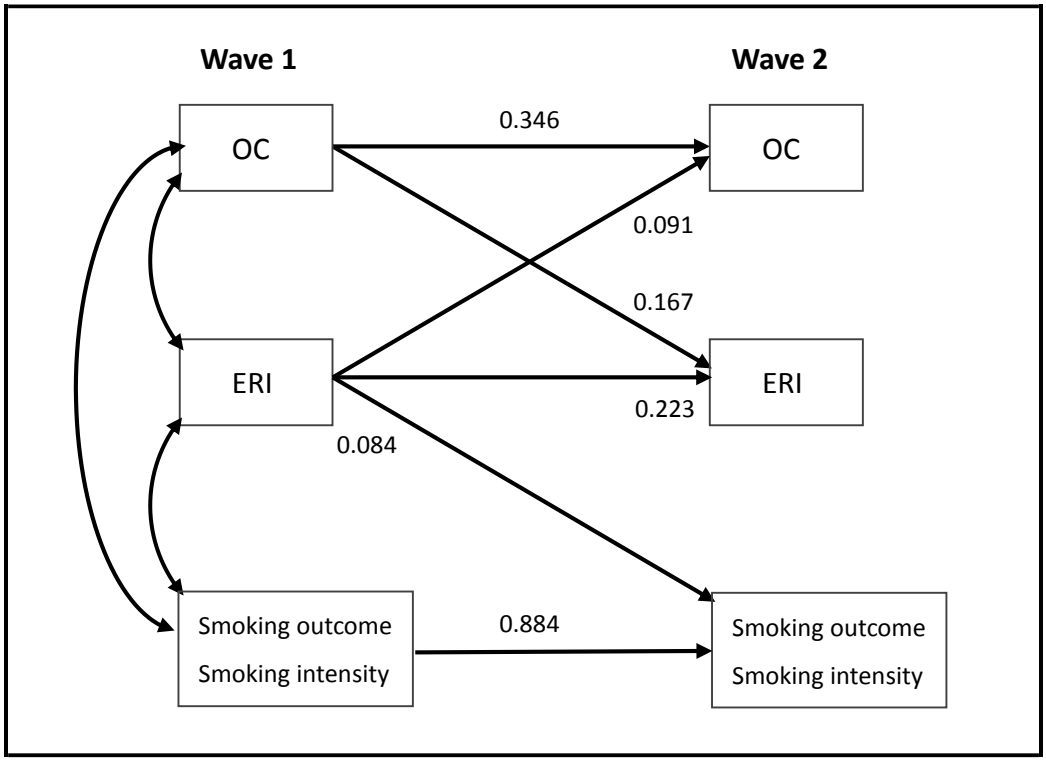


Table 6.11 presents the results of path analysis for the outcome of smoking intensity among current smokers in women, with the results shown in Figure 6.5. First, the mediator effect of ERI in OC–smoking relation is assessed by multiplying 2 cross–lagged effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 2 (unstandardized β= 0.170; standard error= 0.039); (2) higher ERI at wave 1 non-significantly associated with higher levels of smoking intensity at wave 2 (unstandardized β= 0.068; standard error= 0.046; p= 0.221). This mediator effect of ERI is not significant (0.012= 0.170 x 0.068; standard error= 0.008 and p= 0.161).

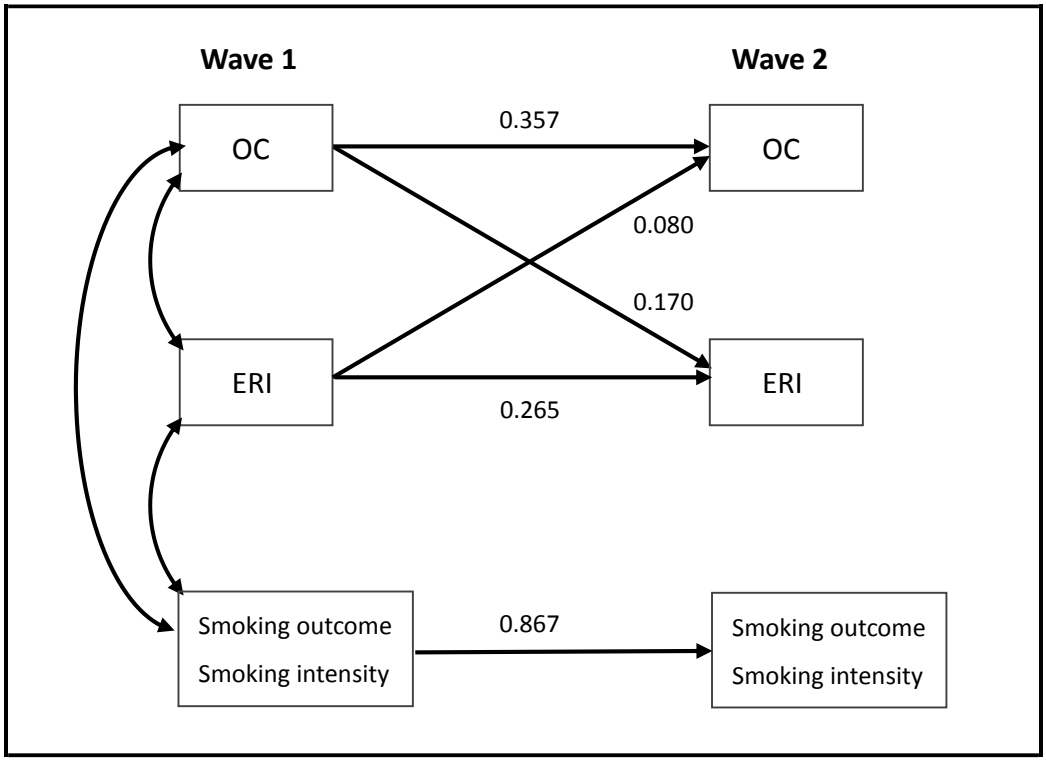
Second, the mediator effect of OC in ERI–smoking relation is estimated by multiplying 2 cross–lagged effects: (1) higher ERI at wave 1 significantly associated with higher OC at wave 2 (unstandardized β= 0.080; standard error= 0.041); (2) higher OC at wave 1 non-significantly related to higher levels of smoking intensity at wave 2 (unstandardized β= 0.036; standard error= 0.045; p= 0.408). This mediator effect of

OC is not significant (0.003; standard error= 0.004 and p= 0.459). Third, the fit indexes show good fit (RMSEA= 0.057 < 0.06; CFI= 0.957 > 0.95) or close to cutoffs for good fit (TLI= 0.865).

Table 6.11 Results of path analysis for antecedent or mediator role of OC in ERI–smoking intensity relationship in women (N= 824)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Autoregressive model				
Smoking intensity wave 1 → wave 2	4.37	0.867	0.605	< 0.001
OC wave 1 → OC wave 2				
OC tertile 2 vs tertile 1	1.80	0.346	0.193	< 0.001
OC tertile 3 vs tertile 1	3.37	0.715	0.391	< 0.001
OR change by 1 tertile	1.83	0.357	0.352	< 0.001
ERI wave 1 → ERI wave 2				
ERI tertile 2 vs tertile 1	1.36	0.170	0.100	0.020
ERI tertile 3 vs tertile 1	2.40	0.516	0.313	< 0.001
OR change by 1 tertile	1.59	0.265	0.270	< 0.001
OC → ERI → Smoking intensity	1.02	0.012	0.007	0.161
OC wave 1 → ERI wave 2				
OC tertile 2 vs tertile 1	1.27	0.131	0.075	0.059
OC tertile 3 vs tertile 1	1.80	0.347	0.199	< 0.001
OR change by 1 tertile	1.36	0.170	0.176	< 0.001
ERI wave 1 → Smoking intensity wave 2				
ERI tertile 2 vs tertile 1	1.03	0.022	0.010	0.867
ERI tertile 3 vs tertile 1	1.26	0.129	0.049	0.297
OR change by 1 tertile	1.13	0.068	0.044	0.221
ERI → OC → Smoking intensity	1.01	0.003	0.002	0.459
ERI wave 1 → OC wave 2				
ERI tertile 2 vs tertile 1	1.25	0.126	0.072	0.087
ERI tertile 3 vs tertile 1	1.33	0.161	0.095	0.053
OR change by 1 tertile	1.15	0.080	0.078	0.053
OC wave 1 → Smoking intensity wave 2				
OC tertile 2 vs tertile 1	1.05	0.029	0.011	0.764
OC tertile 3 vs tertile 1	1.15	0.078	0.030	0.431
OR change by 1 tertile	1.07	0.036	0.023	0.408
Tests of model fit	RMSEA=	0.057	CFI= 0.957	TLI= 0.865

Figure 6.5 Results of path analysis for antecedent or mediator role of OC in ERI–smoking intensity relationship in current smokers in women



6.2.4 Modifying role of OC in ERI–smoking relationship

For evaluating modifying role of OC in ERI–smoking relationship, the associations between ER ratio at wave 1 and smoking outcomes at wave 2 in different strata of OC tertile are assessed after adjustment for covariates. Next, binary and ordinal logistic regression analyses are conducted for two smoking outcomes (smoking status and smoking intensity) at wave 2, respectively, regressed by OC tertile, ER–ratio tertile, and the interaction term between OC tertile and ER–ratio tertile at wave 1 after adjustment for covariates. The log likelihoods for the model with this interaction term and the model without are compared, and LR test is used to test significance of the interaction term. In Table 6.12, LR tests show that this interaction term is not significant for smoking status in men (p= 0.371) and women (p= 0.874), and not significant for

smoking intensity in men (p= 0.243) and women (p= 0.988). My results show that OC has no significantly modifying role in ERI–smoking relation.

Table 6.12 Evaluation for modifying role of OC in ERI–smoking relationship

Approaches	Strata	Smoking status: current smokers vs non-smokers	Smoking intensity among current smokers
Men		OR of outcome by 1–tertile increase in ERI (95% CI)	
1. ERI–smoking relation	OC tertile 1	1.24 (1.06 – 1.45)	1.15 (0.91 – 1.45)
in different strata of OC	OC tertile 2	1.11 (0.92 – 1.33)	1.09 (0.83 – 1.43)
tertile	OC tertile 3	1.04 (0.82 – 1.32)	0.88 (0.61 – 1.22)
2. Interaction OC x ERI			
Likelihood–ratio test	P–value	P= 0.371	P= 0.243
Women		OR of outcome by 1–tertile increase in ERI (95% CI)	
1. ERI–smoking relation	OC tertile 1	1.28 (1.04 – 1.59)	1.14 (0.86 – 1.52)
in different strata of OC	OC tertile 2	1.01 (0.80 – 1.26)	0.92 (0.65 – 1.30)
tertile	OC tertile 3	1.34 (0.97 – 1.84)	1.15 (0.78 – 1.69)
2. Interaction OC x ERI			
Likelihood–ratio test	P–value	P= 0.874	P= 0.988

6.3 Potential Role of PC in Relation between OC, ERI, and Smoking

The focus of this section is, according to the second aim of the thesis, on the associations between PC and smoking outcomes, and on the potential role of PC in the relationship between ERI, OC, and smoking outcomes.

6.3.1 Associations between PC and smoking outcomes

The associations between PC at wave 1 and two smoking outcomes at wave 2 are assessed following the same steps as for ERI–smoking associations.

Table 6.13 reports the associations between PC at wave 1 and two smoking outcomes at wave 2, respectively. In men, the odds of being current smokers are 0.66 for highest versus lowest tertile of PC in Model 2; the adjusted OR changes of being current smokers are 0.81 by 1–tertile increase in PC ($p < 0.001$). Among current smokers, the odds of a higher versus a lower outcome of smoking intensity are 0.63

for highest versus lowest tertile of PC in Model 2; the adjusted OR changes of a higher versus a lower outcome of smoking intensity are 0.79 by 1–tertile increase in PC.

In women, the odds of being current smokers are 0.65 for highest versus lowest tertile of PC in Model 2; the adjusted OR changes of being current smokers are 0.81 by 1–tertile increase in PC (p= 0.001). Among current smokers, the odds of a higher outcome versus a lower outcome of smoking intensity are 0.65 for highest versus lowest tertile of PC in Model 2; the adjusted OR changes of a higher outcome versus a lower outcome of smoking intensity are 0.80 by 1–tertile increase in PC.

Table 6.13 Associations between perceived control at wave 1 and smoking outcomes at wave 2

Model	Perceived control Tertile▲	Smoking status – current smokers vs non-smokers OR (95% CI)	Smoking intensity in current smokers OR (95% CI)
Men		N = 3782	N = 1347
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	0.81 (0.68 – 0.97)*	0.74 (0.58 – 0.96)*
	Tertile 3	0.69 (0.59 – 0.84)*	0.68 (0.53 – 0.89)*
	P for trend	< 0.001	0.002
	OR by 1 tertile	0.83 (0.76 – 0.91)*	0.82 (0.72 – 0.93)*
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	0.84 (0.70 – 1.00)	0.75 (0.57 – 0.98)*
	Tertile 3	0.66 (0.55 – 0.82)*	0.63 (0.48 – 0.83)*
	P for trend	< 0.001	0.001
	OR by 1 tertile	0.81 (0.74 – 0.90)*	0.79 (0.68 – 0.91)*
Women		N = 3731	N = 824
Model 1★	Tertile 1	1.00	1.00
	Tertile 2	0.87 (0.72 – 1.04)	0.72 (0.52 – 0.97)*
	Tertile 3	0.70 (0.58 – 0.86)*	0.68 (0.50 – 0.92)*
	P for trend	0.001	0.023
	OR by 1 tertile	0.84 (0.76 – 0.92)*	0.81 (0.69 – 0.95)*
Model 2★★	Tertile 1	1.00	1.00
	Tertile 2	0.79 (0.62 – 1.01)	0.75 (0.54 – 1.04)
	Tertile 3	0.65 (0.50 – 0.84)*	0.65 (0.47 – 0.91)*
	P for trend	0.001	0.011
	OR by 1 tertile	0.81 (0.72 – 0.92)*	0.80 (0.67 – 0.96)*

★ Model 1: adjusted for age and country. ★★ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health.

▲ Gender–specific tertile of PC: in men, tertile 1 (0–34), tertile 2 (34–41), and tertile 3 (41–55); in women, tertile 1 (0–33), tertile 2 (33–40), and tertile 3 (40–55).

* P value < 0.05.

6.3.2 Mediator roles of PC and ERI in OC–smoking relationship

To assess mediator roles of PC and ERI in OC–smoking relationship, path analyses for binary outcome (smoking status) and for ordinal categorical outcome (smoking intensity in current smokers) are applied in Mplus 7. Each path coefficient is obtained by probit regression for an outcome on a predictor after adjustment for covariates. There are two potential mediators (PC and ERI) between the effects of OC at wave 1 on smoking outcomes at wave 2. As the HAPIEE study is limited by no measurement of PC at wave 2, the cross–sectional associations between OC, ERI, and PC at wave 1 are analyzed. Thus, PC and ERI are only specified to be correlated, while bi-directional relationship between PC and ERI has been hypothesized.

Path analysis with an autoregressive and cross–lagged model for smoking outcomes is specified (Figure 6.6). First, smoking outcomes at wave 2 are predicted by corresponding smoking outcomes at wave 1. Second, the cross–lagged effects of OC, PC, and ERI at wave 1 on smoking outcomes at wave 2 are measured.

Third, the mediator effect can only be assessed by a half–longitudinal design. For example, the mediator role of PC in OC–smoking relation is assessed by two effects: (1) the cross–sectional association of OC at wave 1 on PC at wave 1; (2) the cross–lagged effect of PC at wave 1 on smoking at wave 2. Partial mediation applies if both causal relation are confirmed; the product of two path coefficients can estimate the strength of mediator effect.

Figure 6.6 Hypothetical model specified for the path analysis for mediator roles of PC and ERI in OC–smoking relation

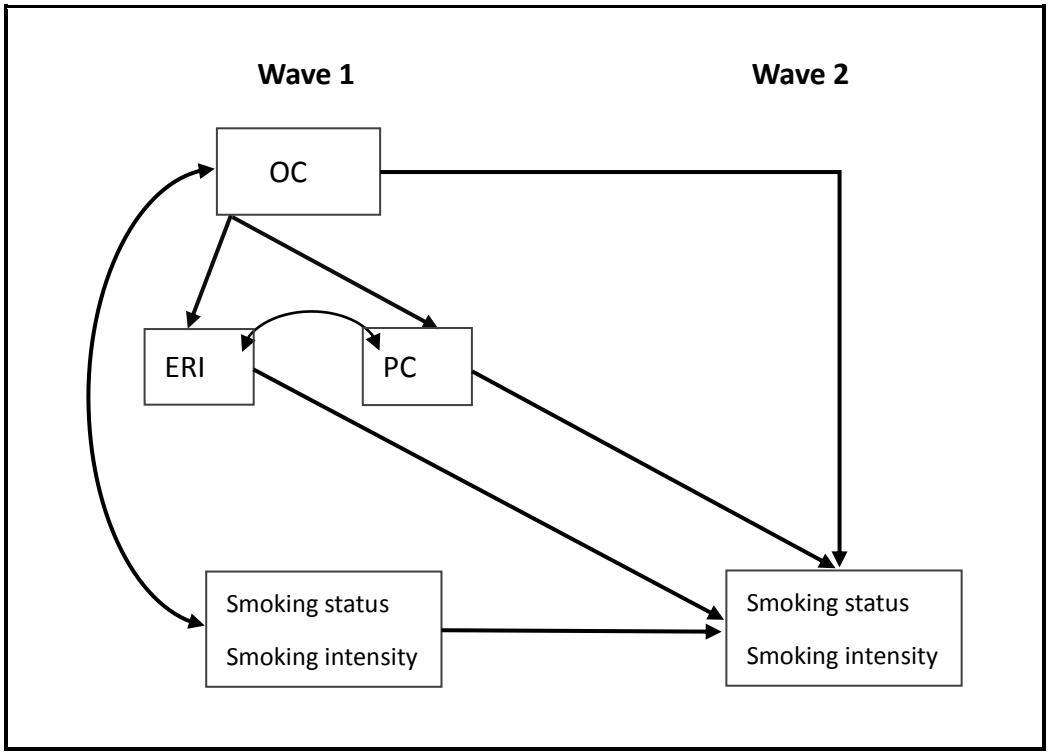


Table 6.14 presents the results of path analysis for the outcome of smoking status in men; the results are illustrated in Figure 6.7. First, the mediator effect of ERI in the relationship between OC and smoking status is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 1 (unstandardized β= 0.253; standard error= 0.020); (2) higher ERI at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= 0.082; standard error= 0.028). The mediator effect of ERI is significant (0.021= 0.253 x 0.082; standard error= 0.007 and p= 0.004 by bootstrap method).

Second, the mediator effect of PC in the relationship between OC and smoking status is assessed by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.110; standard error= 0.020); (2) lower PC at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= –0.112; standard error= 0.025). The mediator effect of PC is

significant (0.012= -0.110 x -0.112; standard error= 0.004 and p= 0.001). Third, ERI at wave 1 is significantly and inversely associated with PC at wave 1 (unstandardized β= –0.052; p< 0.001). Finally, the fit indexes are considered good fit (RMSEA= 0.052 < 0.06) or close to cutoffs for good fit (CFI= 0.921; TLI= 0.834).

Table 6.14 Results of path analysis for mediator roles of PC and ERI in OC–smoking status relationship in men (N= 3782)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Smoking status wave 1 → wave 2	4.70	0.911	0.556	< 0.001
OC wave 1 → Smoking status wave 2				
OC tertile 2 vs tertile 1	1.02	0.012	0.004	0.696
OC tertile 3 vs tertile 1	1.17	0.085	0.028	0.179
OR change by 1 tertile	1.05	0.027	0.021	0.338
OC → ERI → Smoking status	1.04	0.021	0.016	0.004
OC wave 1 → ERI wave 1				
OC tertile 2 vs tertile 1	1.55	0.250	0.146	< 0.001
OC tertile 3 vs tertile 1	2.28	0.485	0.256	< 0.001
OR change by 1 tertile	1.56	0.253	0.235	< 0.001
ERI wave 1 → Smoking status wave 2				
ERI tertile 2 vs tertile 1	1.25	0.129	0.060	0.002
ERI tertile 3 vs tertile 1	1.41	0.192	0.087	< 0.001
OR change by 1 tertile	1.16	0.082	0.066	0.004
OC → PC → Smoking status	1.02	0.012	0.009	0.001
OC wave 1 → PC wave 1				
OC tertile 2 vs tertile 1	0.92	- 0.044	- 0.025	0.127
OC tertile 3 vs tertile 1	0.66	- 0.238	- 0.121	< 0.001
OR change by 1 tertile	0.82	- 0.110	- 0.105	< 0.001
PC wave 1 → Smoking status wave 2				
PC tertile 2 vs tertile 1	0.82	- 0.116	- 0.054	0.004
PC tertile 3 vs tertile 1	0.67	- 0.227	- 0.100	< 0.001
OR change by 1 tertile	0.82	- 0.112	- 0.088	< 0.001
ERI correlates with PC		- 0.052	- 0.089	< 0.001
Tests of model fit	RMSEA =	0.052	CFI= 0.921	TLI= 0.834

Figure 6.7 Results of path analysis for mediator roles of PC and ERI in OC–smoking status relationship in men

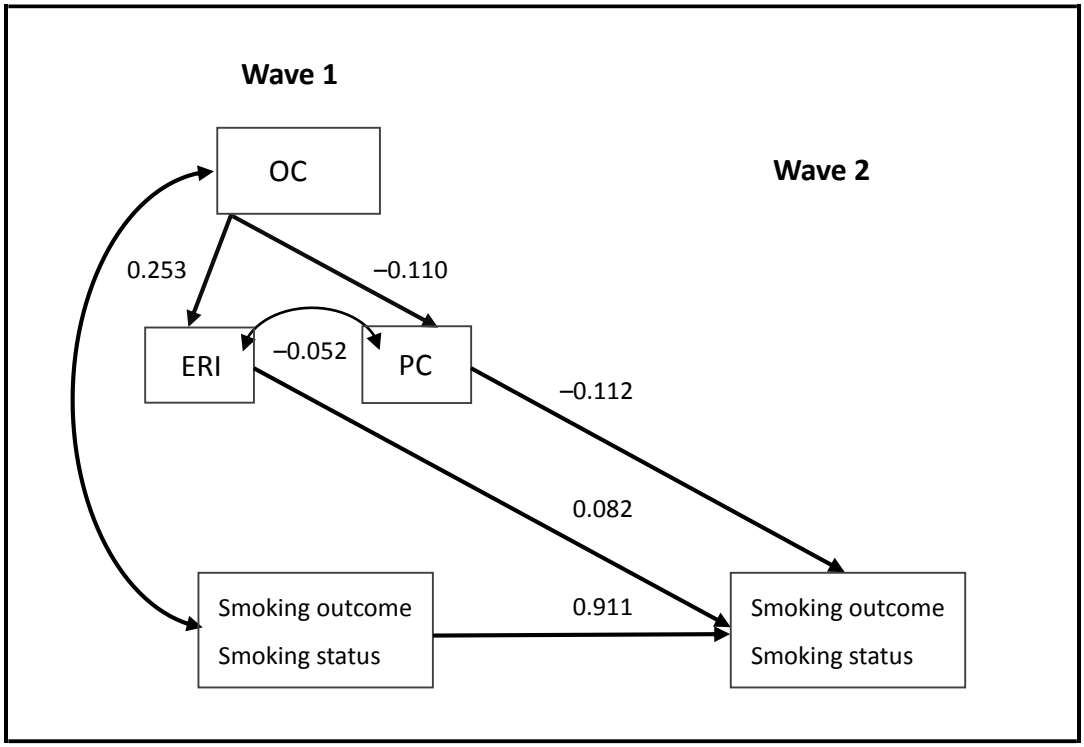


Table 6.15 shows the results of path analysis for the outcome of smoking status in women, with the results shown in Figure 6.8. First, the mediator effect of ERI in the relationship between OC and smoking status is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 1 (unstandardized β= 0.244; standard error= 0.020); (2) higher ERI at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= 0.084; standard error= 0.031). This mediator effect of ERI is significant (0.020= 0.244 x 0.084; standard error= 0.008 and p= 0.008).

Second, the mediator effect of PC in the relationship between OC and smoking status is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.122; standard error= 0.019); (2) lower PC at wave 1 significantly associated with being current smokers at wave 2 (unstandardized β= –0.116; standard error= 0.033). This mediator effect of PC

is significant (0.014; standard error= 0.005 and p= 0.002). Third, ERI at wave 1 is significantly and inversely associated with PC at wave 1 (unstandardized β= –0.056; p< 0.001). Finally, the fit indexes are considered good fit (RMSEA= 0.055 < 0.06) or close to cutoffs for good fit (CFI= 0.931; TLI= 0.819).

Table 6.15 Results of path analysis for mediator roles of PC and ERI in OC–smoking status relationship in women (N= 3731)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Smoking status wave 1 → wave 2	4.77	0.920	0.563	< 0.001
OC wave 1 → Smoking status wave 2				
OC tertile 2 vs tertile 1	1.08	0.043	0.015	0.520
OC tertile 3 vs tertile 1	1.12	0.062	0.020	0.407
OR change by 1 tertile	1.06	0.033	0.020	0.390
OC → ERI → Smoking status	1.04	0.020	0.015	0.008
OC wave 1 → ERI wave 1				
OC tertile 2 vs tertile 1	1.58	0.262	0.147	< 0.001
OC tertile 3 vs tertile 1	2.25	0.476	0.255	< 0.001
OR change by 1 tertile	1.53	0.244	0.236	< 0.001
ERI wave 1 → Smoking status wave 2				
ERI tertile 2 vs tertile 1	1.14	0.073	0.025	0.290
ERI tertile 3 vs tertile 1	1.36	0.170	0.059	0.022
OR change by 1 tertile	1.16	0.084	0.052	0.017
OC → PC → Smoking status	1.03	0.014	0.010	0.002
OC wave 1 → PC wave 1				
OC tertile 2 vs tertile 1	0.85	- 0.093	- 0.052	0.002
OC tertile 3 vs tertile 1	0.64	- 0.251	- 0.133	< 0.001
OR change by 1 tertile	0.80	- 0.122	- 0.120	< 0.001
PC wave 1 → Smoking status wave 2				
PC tertile 2 vs tertile 1	0.79	- 0.133	- 0.048	0.024
PC tertile 3 vs tertile 1	0.67	- 0.224	- 0.077	< 0.001
OR change by 1 tertile	0.81	- 0.116	- 0.071	0.001
ERI correlates with PC		- 0.056	- 0.094	< 0.001
Tests of model fit	RMSEA =	0.055	CFI= 0.931	TLI= 0.819

Figure 6.8 Results of path analysis for mediator roles of PC and ERI in OC–smoking status relationship in women

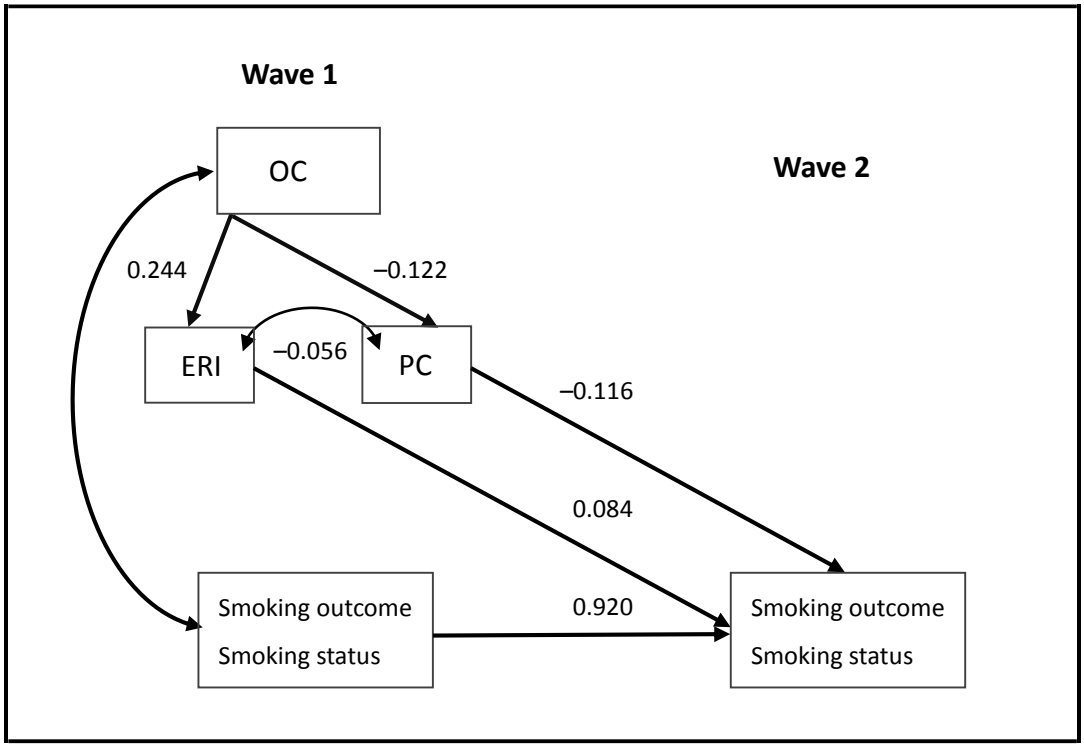


Table 6.16 shows the results of path analysis for the outcome of smoking intensity among current smokers in men, with the results illustrated in Figure 6.9. First, the mediator effect of ERI in the relationship between OC and smoking intensity is estimated by multiplying 2 effects: (1) higher OC at wave 1 is significantly associated with higher ERI at wave 1 (unstandardized β= 0.260; standard error= 0.033); (2) higher ERI at wave 1 is marginally significantly associated with higher levels of smoking intensity at wave 2 (unstandardized β= 0.078; standard error= 0.038; p= 0.097). This mediator effect of ERI is significant (0.020; standard error= 0.010 and p= 0.042).

Second, the mediator effect of PC in the relationship between OC and smoking intensity is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.114; standard error= 0.028); (2) lower PC at wave 1 significantly associated with higher levels of smoking intensity at wave 2 (unstandardized β= –0.125; standard error= 0.039). This mediator effect of

PC is significant (0.014; standard error= 0.006 and p= 0.017). Third, ERI at wave 1 is significantly and inversely associated with PC at wave 1 (unstandardized β= –0.055; p< 0.001). Finally, the fit indexes are considered good fit (RMSEA= 0.050 < 0.06; CFI= 0.962 > 0.95) or close to cutoffs for good fit (TLI= 0.916).

Table 6.16 Results of path analysis for mediator roles of PC and ERI in OC–smoking intensity relationship in men (N= 1347)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Smoking intensity wave 1 → wave 2	4.45	0.878	0.612	< 0.001
OC wave 1 → Smoking intensity wave 2				
OC tertile 2 vs tertile 1	1.02	0.011	0.004	0.732
OC tertile 3 vs tertile 1	1.12	0.064	0.021	0.504
OR change by 1 tertile	1.04	0.024	0.015	0.611
OC → ERI → Smoking intensity	1.04	0.020	0.012	0.042
OC wave 1 → ERI wave 1				
OC tertile 2 vs tertile 1	1.65	0.288	0.164	< 0.001
OC tertile 3 vs tertile 1	2.39	0.513	0.269	< 0.001
OR change by 1 tertile	1.58	0.260	0.254	< 0.001
ERI wave 1 → Smoking intensity wave 2				
ERI tertile 2 vs tertile 1	1.28	0.136	0.049	0.134
ERI tertile 3 vs tertile 1	1.35	0.166	0.059	0.057
OR change by 1 tertile	1.15	0.078	0.049	0.097
OC → PC → Smoking intensity	1.03	0.014	0.009	0.017
OC wave 1 → PC wave 1				
OC tertile 2 vs tertile 1	0.85	- 0.090	- 0.051	0.077
OC tertile 3 vs tertile 1	0.66	- 0.234	- 0.123	< 0.001
OR change by 1 tertile	0.81	- 0.114	- 0.113	< 0.001
PC wave 1 → Smoking intensity wave 2				
PC tertile 2 vs tertile 1	0.77	- 0.143	- 0.050	0.063
PC tertile 3 vs tertile 1	0.64	- 0.258	- 0.086	0.003
OR change by 1 tertile	0.80	- 0.125	- 0.078	0.001
ERI correlates with PC		- 0.055	- 0.091	0.001
Tests of model fit	RMSEA =	0.050	CFI = 0.962	TLI = 0.916

Figure 6.9 Results of path analysis for mediator roles of PC and ERI in OC–smoking intensity relationship in current smokers in men

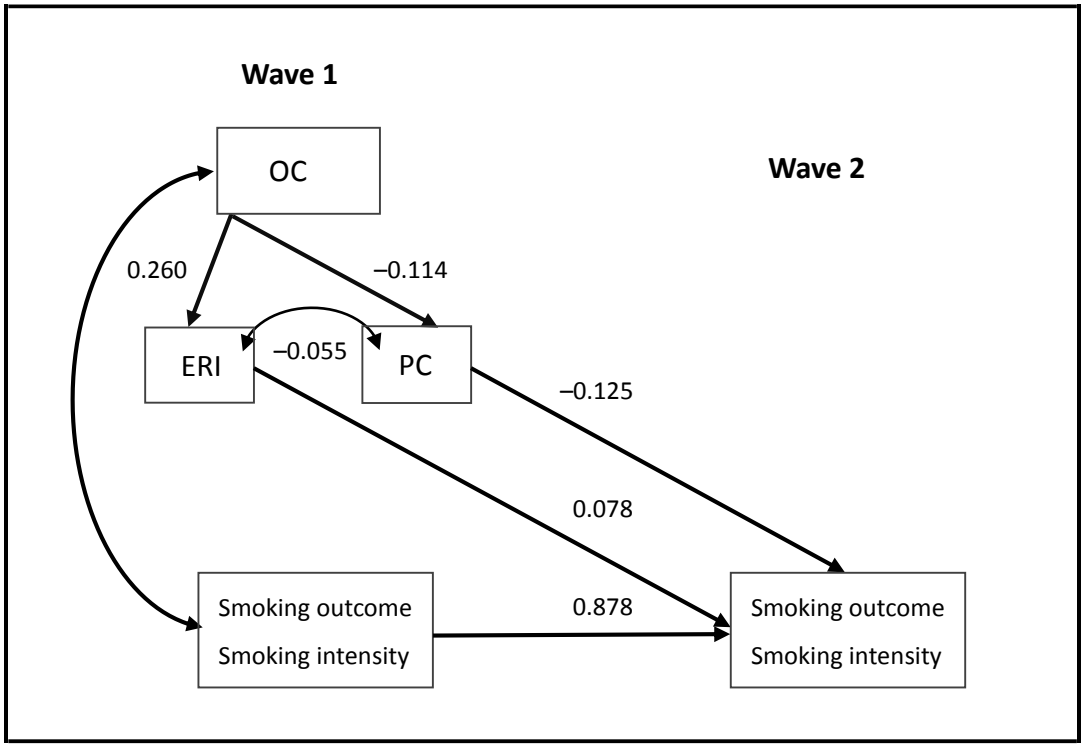


Table 6.17 reports the results of path analysis for the outcome of smoking intensity among current smokers in women, with the results illustrated in Figure 6.10. First, the mediator effect of ERI in the relationship between OC and smoking intensity is assessed by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with higher ERI at wave 1 (unstandardized β= 0.258; standard error= 0.038); (2) higher ERI at wave 1 non-significantly related to higher levels of smoking intensity at wave 2 (unstandardized β= 0.064; standard error= 0.041; p= 0.276). This mediator effect of ERI appears not significant (0.017= 0.258 x 0.064; standard error= 0.011 and p= 0.117).

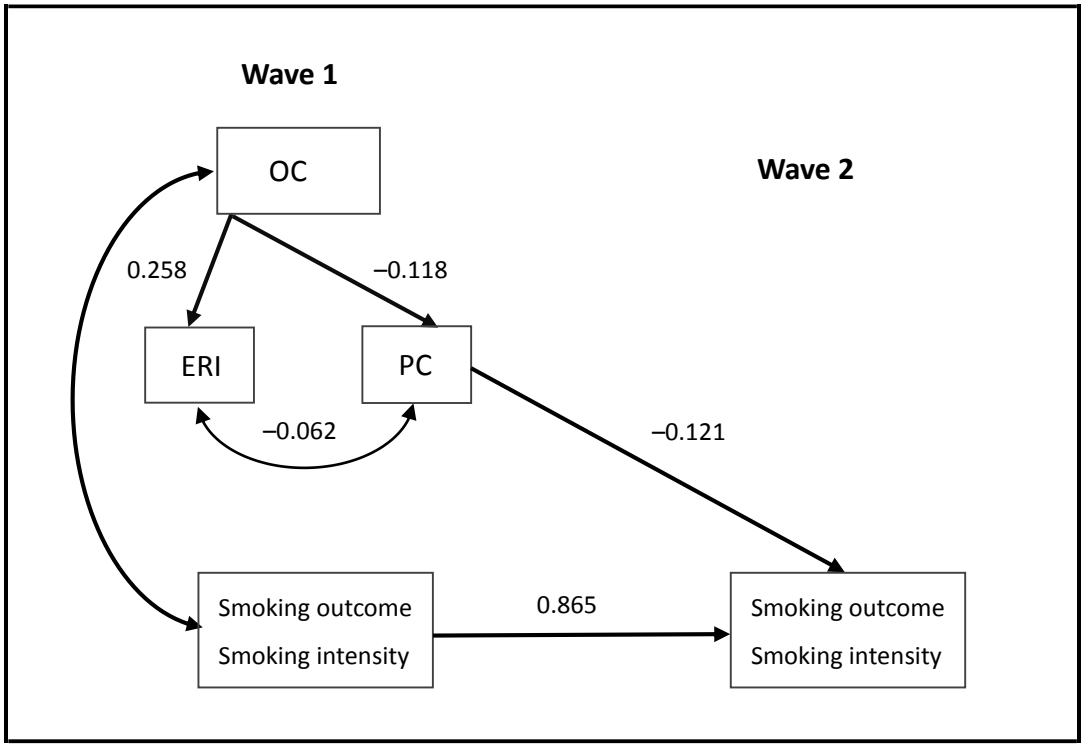
Second, the mediator effect of PC in the relationship between OC and smoking intensity is estimated by multiplying 2 effects: (1) higher OC at wave 1 significantly associated with lower PC at wave 1 (unstandardized β= –0.118; standard error= 0.037); (2) lower PC at wave 1 significantly associated with higher levels of smoking intensity at wave 2 (unstandardized β= –0.121; standard error= 0.043). This mediator effect of

PC is significant (0.014; standard error= 0.007 and p= 0.040). Third, ERI at wave 1 is significantly and inversely associated with PC at wave 1 (unstandardized β= –0.062; p< 0.001). Finally, the fit indexes are considered good fit (RMSEA= 0.058 < 0.06) or close to cutoffs for good fit (CFI= 0.929; TLI= 0.815).

Table 6.17 Results of path analysis for mediator roles of PC and ERI in OC–smoking intensity relationship in women (N= 824)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
Smoking intensity wave 1 → wave 2	4.35	0.865	0.603	< 0.001
OC wave 1 → Smoking intensity wave 2				
OC tertile 2 vs tertile 1	1.05	0.027	0.011	0.774
OC tertile 3 vs tertile 1	1.14	0.072	0.028	0.455
OR change by 1 tertile	1.07	0.035	0.023	0.462
OC → ERI → Smoking intensity	1.03	0.017	0.010	0.117
OC wave 1 → ERI wave 1				
OC tertile 2 vs tertile 1	1.41	0.190	0.109	< 0.001
OC tertile 3 vs tertile 1	2.43	0.522	0.292	< 0.001
OR change by 1 tertile	1.57	0.258	0.260	< 0.001
ERI wave 1 → Smoking intensity wave 2				
ERI tertile 2 vs tertile 1	1.07	0.038	0.014	0.774
ERI tertile 3 vs tertile 1	1.23	0.113	0.042	0.297
OR change by 1 tertile	1.12	0.064	0.040	0.276
OC → PC → Smoking intensity	1.03	0.014	0.009	0.040
OC wave 1 → PC wave 1				
OC tertile 2 vs tertile 1	0.94	- 0.035	- 0.020	0.601
OC tertile 3 vs tertile 1	0.65	- 0.247	- 0.140	< 0.001
OR change by 1 tertile	0.81	- 0.118	- 0.120	0.001
PC wave 1 → Smoking intensity wave 2				
PC tertile 2 vs tertile 1	0.75	- 0.159	- 0.057	0.073
PC tertile 3 vs tertile 1	0.66	- 0.241	- 0.084	0.029
OR change by 1 tertile	0.80	- 0.121	- 0.077	0.018
ERI correlates with PC		- 0.062	- 0.103	0.002
Tests of model fit	RMSEA =	0.058	CFI = 0.929	TLI = 0.815

Figure 6.10 Results of path analysis for mediator roles of PC and ERI in OC–smoking intensity relationship in current smokers in women



6.3.3 Modifying role of PC in ERI–smoking relationship

The modifying role of PC in ERI–smoking relationship is evaluated following the same steps as for the modifying role of OC in ERI–smoking relationship.

Table 6.18 shows the results of evaluation for modifying effect of PC in ERI–smoking relationship. Binary and ordinal logistic regression analyses are conducted for two smoking outcomes at wave 2, respectively, regressed by PC tertile, ER–ratio tertile, and interaction term between PC tertile and ER–ratio tertile at wave 1 after adjustment for covariates. LR tests reveal that this interaction term is not significant for smoking status in men (p= 0.363) and women (p= 0.780); this interaction term is not significant for smoking intensity in men (p= 0.221) and women (p= 0.961). My results show that PC has no significantly modifying role in ERI–smoking relationship.

Table 6.18 Evaluation for modifying role of PC in ERI–smoking relationship

Approaches	Strata	Smoking status: current smokers vs non-smokers	Smoking intensity among current smokers
Men		OR of outcome by 1–tertile increase in ERI (95% CI)	
1. ERI–smoking relation	PC tertile 1	1.04 (0.90 – 1.19)	0.97 (0.78 – 1.20)
in different strata of PC	PC tertile 2	1.23 (1.04 – 1.47)	1.01 (0.77 – 1.31)
tertile	PC tertile 3	1.25 (1.04 – 1.52)	1.18 (0.88 – 1.57)
2. Interaction PC x ERI			
Likelihood–ratio test	P–value	P= 0.363	P= 0.221
Women		OR of outcome by 1–tertile increase in ERI (95% CI)	
1. ERI–smoking relation	PC tertile 1	1.17 (0.96 – 1.44)	1.18 (0.91 – 1.52)
in different strata of PC	PC tertile 2	1.28 (1.02 – 1.61)	1.16 (0.85 – 1.59)
tertile	PC tertile 3	1.09 (0.86 – 1.39)	1.20 (0.84 – 1.71)
2. Interaction PC x ERI			
Likelihood–ratio test	P–value	P= 0.780	P= 0.961

6.4 Main Findings for Smoking Outcomes

The analyses based on a 2–wave cohort study (3782 men and 3731 women aged 45–69) from the HAPIEE study show the following findings, which are in line with specific objectives and relevant hypotheses listed in Chapter 3.

In terms of the associations between ER ratio and smoking outcomes, Hypothesis 2 that higher ER ratio (wave 1) is associated with higher levels of smoking outcomes (wave 2) after adjustment for covariates is supported. In terms of the associations between OC and smoking outcomes, Hypothesis 5 that higher OC (wave 1) is associated with higher levels of smoking outcomes (wave 2) after adjustment for covariates is partially supported in smoking status, but the associations between OC and smoking intensity do not reach statistical significance in both sexes.

With regards to the potential role of OC (antecedent, mediator, modifier, or direct effect) in ERI–smoking relation, Hypothesis 8 is partially supported. OC and ERI have bi–directional relationship, but the effect of OC on ERI is stronger than the other direction in the middle-aged and older populations. Antecedent role of OC in ERI–

smoking relationship is found significant among two smoking outcomes in both sexes (except smoking intensity in women), but mediator role of OC is not significant. Direct effect of OC on smoking is not significant. Finally, modifying role of OC in ERI–smoking relationship is non–significant.

In terms of the associations between PC and smoking outcomes, Hypothesis 11 that lower PC (wave 1) is associated with higher levels of smoking (wave 2) after adjustment for covariates is supported.

With regards to the potential role of PC (mediator or modifier) in the relationship between ERI, OC, and smoking, Hypothesis 14 is partially supported. PC and ERI partially mediate the effects of OC on all smoking outcomes in both sexes (except smoking intensity in women). In addition, PC and ERI may have bi–directional relationship. PC and ERI are negatively associated with each other in the cross–sectional analyses; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in the cross–sectional analyses. Finally, modifying role of PC in ERI–smoking relationship is non–significant.

Note that the methodological issues and interpretation of the main findings for smoking outcomes will be addressed in detail in Chapter 8, and their implications for practice, policy, and research in Chapter 9.

Chapter 7. Dietary Outcomes

The aims of analyses for dietary outcomes are: (1) to examine the relationship between ERI, OC, and dietary outcomes; (2) to evaluate the potential role of PC in the relationship between ERI, OC and dietary outcomes. The analyses only use data from the cross–sectional study (wave 1) of the HAPIEE study, as PC and dietary outcomes were not collected at wave 2. The subsample consists of 11012 subjects (5735 men / 5277 women). Antecedent role of OC in ERI–diet relation was specified according to previous findings in drinking and smoking outcomes, as bidirectional relationship between OC and ERI (Hypothesis 9) cannot be tested in a cross–sectional study.

The results are presented in three parts. First, descriptive statistics for covariates and dietary outcomes by country and by gender are presented. Second, for assessing the associations between ERI, OC and dietary outcomes, the associations of exposure variables (ERI and OC) and dietary outcomes (HDI components and HDI) are assessed using binary and ordinal logistic regression, respectively. Third, to examine the potential role of PC in the relationship between ERI, OC and dietary outcomes, the path analysis with an ordinal categorical outcome (HDI outcome) is used.

7.1 Descriptive Statistics

7.1.1 Descriptive characteristics of study populations

In this sample of 11012 subjects (5735 men and 5277 women), the mean age is 55.0 years in men (standard deviation= 6.0) and 53.0 years in women (standard deviation= 5.3). Descriptive statistics for covariates are presented by country and gender in Table 7.1. First, gender differences are found across three countries; men have higher proportions in age group over 55, university–educated, manager and manual worker, married and cohabiting, lower deprivation, less depression, and more social isolation than women. Second, country differences are reported in both sexes.

Russian samples are older than Czech and Polish samples. University education in Czech samples is less common than other countries, and Czech samples have the largest gender inequality in education. The proportions of social isolation in Czech Republic are the lowest among all countries The proportions of high deprivation and poor to very poor self-rated health in Russia are the highest of all countries.

Table 7.1 Descriptive statistics of study sample by country and gender (N= 11012)

Variable	Czech Republic		Russia		Poland	
	Men (n= 1645)	Women (n= 1560)	Men (n= 2297)	Women (n= 2121)	Men (n= 1793)	Women (n= 1596)
Age, N (%)						
45 – 49	446 (27.1)	558 (35.8)	489 (21.3)	657 (31.0)	500 (27.9)	600 (37.6)
50 – 54	498 (30.3)	602 (38.6)	583 (25.4)	689 (32.5)	529 (29.5)	539 (33.8)
55 – 59	454 (27.6)	275 (17.6)	616 (26.8)	469 (22.1)	422 (23.5)	303 (19.0)
60 – 69	247 (15.0)	125 (8.0)	609 (26.5)	306 (14.4)	342 (19.1)	154 (9.6)
Education, N (%)						
Primary or less	54 (3.3)	137 (8.8)	158 (6.9)	83 (3.9)	70 (3.9)	73 (4.6)
Vocational	654 (39.7)	416 (26.7)	508 (22.1)	702 (33.1)	400 (22.3)	174 (10.9)
Secondary	549 (33.4)	769 (49.3)	797 (34.7)	619 (29.2)	554 (30.9)	669 (41.9)
University	388 (23.6)	237 (15.2)	836 (36.4)	717 (33.8)	769 (42.9)	680 (42.6)
Occupation class, N (%)						
Manager/profession	444 (27.0)	268 (17.2)	611 (26.6)	424 (20.0)	545 (30.4)	314 (19.7)
Non-manual worker	668 (40.6)	1011 (64.8)	781 (34.0)	1277 (60.2)	862 (48.1)	1071 (67.1)
Manual worker	533 (32.4)	281 (18.0)	905 (39.4)	420 (19.8)	386 (21.5)	211 (13.2)
Marital status, N (%)						
Married/cohabiting	1383 (84.1)	1129 (72.4)	2030 (88.4)	1332 (62.8)	1631 (91.0)	1116 (69.9)
Single	52 (3.2)	44 (2.8)	74 (3.2)	123 (5.8)	65 (3.6)	142 (8.9)
Divorce/widowed	209 (12.7)	387 (24.8)	193 (8.4)	666 (31.4)	97 (5.4)	337 (21.1)
Deprivation, N (%)						
Low (0 – 3.9)	1423 (86.5)	1279 (82.0)	1629 (70.9)	1126 (53.1)	1449 (80.8)	1171 (73.4)
High (4 – 9)	222 (13.5)	281 (18.0)	668 (29.1)	995 (46.9)	344 (19.2)	425 (26.6)
Depression, N (%)						
CESD < 16	1439 (87.5)	1237 (79.3)	1996 (86.9)	1525 (71.9)	1540 (85.9)	1285 (80.5)
CESD >= 16	206 (12.5)	323 (20.7)	301 (13.1)	596 (28.1)	253 (14.1)	311 (19.5)
Social isolation, N (%)						
No (>= once a month)	1076 (65.4)	1114 (71.4)	1015 (44.2)	982 (46.3)	809 (45.1)	776 (48.6)
Yes (< once a month)	569 (34.6)	446 (28.6)	1282 (55.8)	1139 (53.7)	984 (54.9)	820 (51.4)
Self-rated health, N (%)						
Very good – average	1538 (93.5)	1476 (94.6)	2099 (91.4)	1731 (81.6)	1678 (93.6)	1495 (93.7)
Poor – very poor	107 (6.5)	84 (5.4)	198 (8.6)	390 (18.4)	115 (6.4)	101 (6.3)

7.1.2 Descriptive characteristics of dietary outcomes

Dietary data are collected using the Food Frequency Questionnaire (FFQ) developed by Willett et al and adapted from the Whitehall II Study.[589] Table 7.2 shows descriptive characteristics of dietary outcomes by country and by gender. Absolute nutrient/food intakes (quantity per day) are presented by means and medians; medians are shown as the distributions are often skewed. Nutrient density is presented by percentage of total energy intakes without energy provided by alcohol.

Table 7.2 Descriptive statistics of dietary outcomes by country and gender

Dietary outcomes	Czech		Russia		Poland	
	Men (n= 1645)	Women (n= 1560)	Men (n= 2297)	Women (n= 2121)	Men (n= 1793)	Women (n= 1596)
Saturated fat						
Mean (sd), g/day	32 (13)	29 (13)	48 (20)	40 (16)	40 (16)	35 (14)
Median, g/day	30	27	45	38	37	33
Nutrient density (%)	13.2	13.0	14.8	14.5	15.3	14.6
Polyunsaturated fat						
Mean (sd), g/day	15 (7)	14 (6)	26 (10)	25 (10)	13 (6)	12 (5)
Median, g/day	14	13	24	24	12	11
Nutrient density (%)	6.2	6.3	8.0	9.1	5.0	5.0
Total carbohydrate						
Mean (sd), g/day	240 (96)	238 (96)	287 (85)	253 (82)	267 (86)	262 (87)
Median, g/day	223	222	279	243	257	248
Nutrient density (%)	43.7	47.8	39.8	40.8	45.5	48.7
Free sugars						
Mean (sd), g/day	110 (57)	128 (67)	126 (49)	125 (48)	124 (54)	133 (59)
Median, g/day	101	115	118	118	116	123
Nutrient density (%)	20.2	25.5	17.3	20.1	21.0	24.7
Protein						
Mean (sd), g/day	96 (35)	87 (30)	125 (38)	107 (33)	106 (32)	95 (29)
Median, g/day	91	83	121	103	101	92
Nutrient density (%)	17.6	17.4	17.1	17.2	18.0	17.7
Cholesterol						
Mean (sd), mg/day	326 (141)	283 (123)	544 (253)	413 (165)	424 (195)	357 (144)
Median, mg/day	304	266	492	391	390	337
Sodium						
Mean (sd), mg/day	3013 (1123)	2562 (1022)	4020 (1358)	3379 (1171)	3756 (1306)	3256 (1155)
Median, mg/day	2854	2416	3847	3262	3570	3078
Fruit and vegetable						
Mean (sd), g/day	452 (396)	678 (582)	379 (255)	450 (305)	456 (267)	559 (347)
Median, g/day	369	537	314	369	408	492
Non-starch polysaccharide						
Mean (sd), g/day	17 (9)	19 (10)	18 (6)	18 (6)	19 (7)	19 (8)
Median, g/day	15	17	18	17	18	18
Total energy intake						
Mean (sd), MJ/day	9.1 (3.1)	8.4 (3.0)	12.2 (3.6)	10.4 (3.2)	9.9 (3.0)	9.0 (2.7)
Median, MJ/day	8.7	7.9	11.7	10.0	9.5	8.6

The Healthy Diet Indicator (HDI) is constructed to reflect the adherence to dietary recommendations of WHO for the prevention of chronic diseases (2003).[590] Nine selected nutrient/food intakes are: (1) nutrient density from saturated fat, polyunsaturated fat, total carbohydrate, free sugars, and protein; (2) nutrient intakes of non–starch polysaccharides (NSP), cholesterol, and sodium; (3) food intakes of fruit and vegetable. A dichotomous variable is generated for each nutrient/food intake; if one's intake is within the WHO recommended range this variable is coded as 1 (healthy intake), otherwise it is coded as 0 (unhealthy intake). The HDI score is the sum of 9 dichotomous variables, ranged from 0 to 9.[591]

Table 7.3 reports the percentage of subjects who meet the WHO dietary recommendations. Across three countries, the proportions of women who meet the WHO recommended ranges are generally higher than those of men, except polyunsaturated fat (in Russia and Poland), free sugars, and NSP (in Russia).

Table 7.3 Percentage of subjects who meet dietary recommendations of WHO for the prevention of chronic diseases (2003)[592]

Proportion meeting WHO suggested ranges (%)	WHO suggested ranges	Czech Republic		Russia		Poland	
		Men (n= 1645)	Women (n= 1560)	Men (n= 2297)	Women (n= 2121)	Men (n= 1793)	Women (n= 1596)
Saturated fat	< 10 %	5.6	9.7	2.8	3.3	2.0	4.9
Polyunsaturated fat	6–10 %	56.7	58.8	70.8	62.2	17.2	15.8
Total carbohydrate	55–75 %	5.4	16.0	1.1	2.1	7.3	17.2
Free sugars	< 10 %	4.3	1.0	5.2	1.9	2.8	1.2
Protein	10–15 %	9.0	13.9	11.8	19.3	6.5	9.6
Cholesterol	< 300 mg	48.6	63.1	10.9	24.3	21.7	35.8
Sodium	< 2000 mg	19.0	36.2	4.4	12.8	5.5	12.6
Fruit & vegetable	> 400 g	55.8	75.1	51.7	64.6	65.2	75.4
NSP	> 20 g	19.1	28.8	22.2	18.7	27.4	31.5

Table 7.4 shows the means and medians of overall HDI score and the proportions of subjects meeting different HDI scores by gender and by country. There are gender differences across three countries; mean HDI scores in women are higher than those in men. Country differences are observed in both sexes; mean HDI scores in Czech

Republic are the highest of all countries, but mean HDI scores in Poland are the lowest of all countries. Due to very low proportions of those having HDI 5 to 9, six categories of HDI outcome (HDI= 0, 1, 2, 3, 4, and 5–9) are adopted for further analyses.

Table 7.4 Overall scores of Healthy Diet Indicator and proportions of subjects in different scores

Healthy Diet Indicator (HDI)	Czech Republic		Russia		Poland	
	Men (n= 1645)	Women (n= 1560)	Men (n= 2297)	Women (n= 2121)	Men (n= 1793)	Women (n= 1596)
Different score	N (%)		N (%)		N (%)	
0	100 (6.1)	34 (2.2)	221 (9.6)	121 (5.7)	298 (16.6)	145 (9.1)
1	357 (21.7)	136 (8.7)	733 (31.9)	534 (25.2)	609 (34.0)	405 (25.4)
2	551 (33.5)	388 (24.9)	744 (32.4)	774 (36.5)	579 (32.3)	557 (34.9)
3	423 (25.7)	516 (33.1)	487 (21.2)	473 (22.3)	217 (12.1)	297 (18.6)
4	155 (9.4)	306 (19.6)	87 (3.8)	176 (8.3)	63 (3.5)	118 (7.4)
5–9	59 (3.6)	179 (11.5)	25 (1.1)	42 (2.0)	27 (1.5)	74 (4.6)
Overall score						
Mean (sd)	2.2 (1.2)	3.0 (1.3)	1.8 (1.1)	2.1 (1.1)	1.6 (1.1)	2.0 (1.2)

In terms of the way of pooling the data, crude associations between exposure variables (ER ratio and OC) and HDI outcome in country–specific strata are assessed by ordinal logistic regression, respectively (Table 7.5). By comparing the log likelihoods for the model with the interaction term (ERI x country; OC x country) and the model without, likelihood–ratio (LR) test is used to test significance of the interaction term.

There are small country differences in crude associations between exposure variables and HDI outcome. The associations between ER ratio and HDI outcome in Czech Republic are slightly stronger than those in other countries; a similar pattern of country differences is observed in OC–HDI associations. Overall, these associations between exposure variables and HDI outcome are not very different across country–specific strata (all p–values > 0.128). The interaction terms between country and exposure variables do not reach statistical significance. Thus, data for three countries are pooled for further analyses.

Table 7.5 Crude associations between exposure variables and HDI outcome in country–specific strata

Strata	Tertile of exposure variables▲	HDI outcome OR (95% CI)	HDI outcome OR (95% CI)
ERI–HDI relation	ER–ratio tertile	Men	Women
Czech	Tertile 1	1.00	1.00
	Tertile 2	0.83 (0.66 – 1.04)	0.71 (0.57 – 0.89)*
	Tertile 3	0.65 (0.53 – 0.81)*	0.64 (0.51 – 0.79)*
	P for trend	< 0.001	< 0.001
	OR by 1 tertile	0.81 (0.73 – 0.90)*	0.80 (0.72 – 0.89)*
Russia	Tertile 1	1.00	1.00
	Tertile 2	0.80 (0.68 – 0.94)*	0.96 (0.81 – 1.14)
	Tertile 3	0.68 (0.56 – 0.82)*	0.75 (0.62 – 0.90)*
	P for trend	0.001	0.005
	OR by 1 tertile	0.83 (0.76 – 0.91)*	0.87 (0.80 – 0.96)*
Poland	Tertile 1	1.00	1.00
	Tertile 2	0.93 (0.76 – 1.15)	0.96 (0.77 – 1.19)
	Tertile 3	0.88 (0.72 – 1.08)	0.90 (0.74 – 1.10)
	P for trend	0.337	0.309
	OR by 1 tertile	0.94 (0.85 – 1.04)	0.94 (0.85 – 1.05)
Interaction country x ERI	LR test	P= 0.152	P= 0.135
OC–HDI relation	OC tertile	Men	Women
Czech	Tertile 1	1.00	1.00
	Tertile 2	0.87 (0.70 – 1.06)	0.76 (0.62 – 0.94)*
	Tertile 3	0.67 (0.54 – 0.84)*	0.64 (0.51 – 0.80)*
	P for trend	0.001	< 0.001
	OR by 1 tertile	0.83 (0.74 – 0.92)*	0.81 (0.72 – 0.91)*
Russia	Tertile 1	1.00	1.00
	Tertile 2	0.94 (0.77 – 1.14)	1.01 (0.84 – 1.22)
	Tertile 3	0.78 (0.66 – 0.94)*	0.91 (0.76 – 1.09)
	P for trend	0.108	0.360
	OR by 1 tertile	0.91 (0.81 – 1.02)	0.95 (0.87 – 1.04)
Poland	Tertile 1	1.00	1.00
	Tertile 2	0.98 (0.82 – 1.18)	0.86 (0.70 – 1.06)
	Tertile 3	0.85 (0.69 – 1.04)	0.85 (0.69 – 1.04)
	P for trend	0.150	0.143
	OR by 1 tertile	0.93 (0.84 – 1.03)	0.92 (0.83 – 1.03)
Interaction country x OC	LR test	P= 0.203	P= 0.128

▲ Country– and gender–specific tertiles of ER ratio or OC score. * P value < 0.05.

After the data of three countries are pooled, crude associations between exposure variables (ER ratio and OC) and HDI outcome in gender–specific strata are assessed by ordinal logistic regression, respectively (Table 7.6). By comparing the log likelihoods for the model with the interaction term (ERI x gender; OC x gender) and the model without, LR test is used to test significance of the interaction term. The crude associations between exposure variables and HDI outcome are not very

different across gender–specific strata (all p–values > 0.17). The interaction terms between gender and exposure variables do not reach statistical significance. Although men and women are initially assessed separately as most literature on psychosocial factors and diet, data from both sexes will be pooled in final analyses.

Table 7.6 Crude associations between exposure variables and HDI outcome in gender–specific strata

Strata	Tertile of exposure variables▲	HDI outcome OR (95% CI)
ERI–HDI relationship	ER–ratio tertile	
Men	Tertile 1	1.00
	Tertile 2	0.87 (0.78 – 0.97)*
	Tertile 3	0.80 (0.72 – 0.89)*
	P for trend	0.001
	OR by 1 tertile	0.90 (0.85 – 0.95)*
Women	Tertile 1	1.00
	Tertile 2	0.97 (0.86 – 1.08)
	Tertile 3	0.93 (0.83 – 1.04)
	P for trend	0.231
	OR by 1 tertile	0.96 (0.91 – 1.02)
Interaction term: gender x ERI	LR test	P= 0.176
OC–HDI relationship	OC tertile	
Men	Tertile 1	1.00
	Tertile 2	0.95 (0.85 – 1.05)
	Tertile 3	0.85 (0.76 – 0.95)*
	P for trend	0.007
	OR by 1 tertile	0.93 (0.88 – 0.98)*
Women	Tertile 1	1.00
	Tertile 2	1.03 (0.93 – 1.16)
	Tertile 3	0.90 (0.81 – 1.01)
	P for trend	0.132
	OR by 1 tertile	0.96 (0.90 – 1.01)
Interaction term: gender x OC	LR test	P= 0.253

▲ Gender–specific tertiles of ER ratio or OC score. * P value < 0.05.

The associations between covariates and HDI outcome by ordinal logistic regression are summarized (Table 7.7). In men, age 55–59 (OR= 1.12, P= 0.088) and age 60–69 (OR= 1.27, P= 0.001) are associated with higher HDI; in contrast, high deprivation (OR= 0.80, P < 0.001), depression (OR= 0.88, P= 0.084) and social isolation (OR= 0.85, P= 0.001) are associated with lower HDI. In women, age 50–54 (OR= 1.18, P= 0.004), age 55–59 (OR= 1.29, P < 0.001), age 60–69 (OR= 1.18, P= 0.074), and university-educated (OR= 1.29, P= 0.048) are associated with higher HDI. High deprivation (OR= 0.75, P < 0.001) and social isolation (OR= 0.79, P <

0.001) are associated with lower HDI.

Table 7.7 Associations between HDI outcome and covariates by ordinal logistic regression

Variables	Men (n = 5735)		Women (n = 5277)	
	OR (95% CI)	P-value	OR (95% CI)	P-value
Age				
45 – 49	1.00		1.00	
50 – 54	1.06 (0.93 – 1.21)	0.355	1.18 (1.05 – 1.34)	0.004
55 – 59	1.12 (0.98 – 1.28)	0.088	1.29 (1.12 – 1.49)	< 0.001
60 – 69	1.27 (1.10 – 1.47)	0.001	1.18 (0.98 – 1.41)	0.074
Education				
Primary or less	1.00		1.00	
Vocational	1.17 (0.88 – 1.56)	0.271	1.15 (0.89 – 1.29)	0.289
Secondary	1.10 (0.83 – 1.46)	0.488	1.08 (0.84 – 1.38)	0.534
University	1.22 (0.90 – 1.64)	0.194	1.29 (1.00 – 1.66)	0.048
Occupation class				
Manager/ professional	1.00		1.00	
Non-manual worker	0.91 (0.81 – 1.03)	0.134	0.96 (0.85 – 1.09)	0.564
Manual worker	0.94 (0.82 – 1.07)	0.370	0.95 (0.80 – 1.13)	0.557
Marital status				
Married/ cohabiting	1.00		1.00	
Single	1.12 (0.84 – 1.49)	0.437	1.06 (0.92 – 1.23)	0.388
Divorced/ widowed	1.01 (0.85 – 1.20)	0.833	0.90 (0.80 – 1.04)	0.118
Self-rated health				
Very good/ average	1.00		1.00	
Very poor/ poor	0.96 (0.79 – 1.17)	0.747	0.92 (0.78 – 1.09)	0.356
Deprivation				
low (0 – 3.9)	1.00		1.00	
High (4 – 9)	0.80 (0.70 – 0.90)	< 0.001	0.75 (0.66 – 0.84)	< 0.001
Depression				
No	1.00		1.00	
Yes	0.88 (0.76 – 1.02)	0.084	0.92 (0.82 – 1.04)	0.185
Social isolation				
No	1.00		1.00	
Yes	0.85 (0.77 – 0.94)	0.001	0.79 (0.71 – 0.87)	< 0.001

7.2 Associations between ERI, OC, and Dietary Outcomes

7.2.1 Associations between ERI and dietary outcomes

The associations between ER–ratio tertile and dietary outcomes are evaluated by the following two steps. First, binary logistic regression is used to assess the associations between ER–ratio tertile and 9 dichotomous variables (nutrient/food intakes), respectively, after adjustment for age and country (Model 1) and after

additionally adjustment for other covariates (Model 2). Second, ordinal logistic regression is used to assess the associations between ER–ratio tertile and HDI as an ordinal categorical variable. Due to low proportions of subjects in HDI 5 to 9, six categories of HDI outcome (HDI= 0, 1, 2, 3, 4, and 5–9) are used. The ERI–HDI associations are assessed after adjustment for age and country (Model 1) and after additionally adjustment for other covariates (Model 2). Ordinal logistic regression assumes that the coefficient for relationship between, for example, the lowest versus all higher categories of outcome variable is the same as that coefficient for relationship between the next lowest category and all higher categories (parallel regression assumption); this assumption is found not violated by Brant test ($p > 0.05$).

In Table 7.8, binary logistic regression analyses for men indicate that higher ER ratio is significantly ($p < 0.05$) associated with less healthy intakes of saturated fat (OR changes by 1–tertile increase in ER ratio= 0.81), free sugars (OR= 0.80), protein (OR= 0.88), cholesterol (OR= 0.91), sodium (OR= 0.83), and fruit/vegetable (OR= 0.93) in Model 2. For women, higher ER ratio is significantly associated with less healthy intakes of saturated fat (OR= 0.80), total carbohydrate (OR= 0.89), free sugars (OR= 0.74), cholesterol (OR= 0.92), sodium (OR= 0.88), fruit/vegetable (OR= 0.92), and NSP (OR= 0.91) in Model 2.

At the bottom of Table 7.8, ordinal logistic regression analyses demonstrate a consistent and significant ($p < 0.001$) association between higher ER ratio and lower HDI score. For 1–tertile increase in ER ratio, the adjusted odds of being in a higher HDI category (e.g. HDI > 0 versus <= 0, HDI > 1 versus <= 1, or HDI > 4 versus <= 4) are changed by 0.86 and 0.88 in men and women, respectively. For highest versus lowest tertile of ER ratio, the adjusted odds of being in a higher HDI category are 0.73 and 0.78 in men and women, respectively.

Table 7.8 Associations between ER ratio and dietary outcomes

		Men (n= 5735)		Women (n= 5277)	
Outcome Variables	ER ratio tertile **	Model 1 † OR (95% CI)	Model 2 ‡ OR (95% CI)	Model 1 † OR (95% CI)	Model 2 ‡ OR (95% CI)
Saturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.75 (0.53–1.05)	0.74 (0.51–1.08)	0.75 (0.57–0.98)*	0.77 (0.58–1.02)
	Tertile 3	0.67 (0.47–0.96)*	0.66 (0.44–0.98)*	0.64 (0.49–0.88)*	0.63 (0.48–0.87)*
	OR by 1 tertile	0.82 (0.68–0.98)*	0.81 (0.66–0.99)*	0.80 (0.69–0.92)*	0.80 (0.69–0.93)*
	P for trend	0.029	0.039	0.002	0.003
Poly-unsaturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.00 (0.87–1.15)	0.97 (0.83–1.13)	1.03 (0.89–1.19)	1.01 (0.86–1.20)
	Tertile 3	0.94 (0.81–1.08)	0.90 (0.77–1.06)	0.99 (0.85–1.15)	1.00 (0.84–1.19)
	OR by 1 tertile	0.97 (0.90–1.04)	0.95 (0.88–1.03)	1.01 (0.94–0.18)	1.00 (0.92–1.09)
	P for trend	0.382	0.207	0.699	0.881
Total carbo-hydrate	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.90 (0.65–1.25)	0.92 (0.65–1.29)	0.99 (0.79–1.23)	0.98 (0.77–1.24)
	Tertile 3	0.88 (0.64–1.22)	0.90 (0.64–1.27)	0.83 (0.67–1.03)	0.81 (0.64–1.02)
	OR by 1 tertile	0.94 (0.81–1.11)	0.95 (0.80–1.13)	0.91 (0.82–1.01)	0.89 (0.79–1.00)*
	P for trend	0.469	0.584	0.077	0.048
Free sugars	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.71 (0.52–0.98)*	0.67 (0.48–0.93)*	0.72 (0.43–1.20)	0.74 (0.41–1.26)
	Tertile 3	0.69 (0.51–0.93)*	0.65 (0.46–0.92)*	0.54 (0.30–0.96)*	0.53 (0.28–1.00)*
	OR by 1 tertile	0.83 (0.71–0.98)*	0.80 (0.67–0.96)*	0.74 (0.56–0.98)*	0.74 (0.54–0.98)*
	P for trend	0.030	0.014	0.032	0.030
Protein	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.86 (0.70–1.06)	0.87 (0.70–1.10)	0.90 (0.75–1.07)	0.91 (0.75–1.10)
	Tertile 3	0.81 (0.65–1.01)	0.78 (0.61–0.99)*	0.88 (0.73–1.05)	0.84 (0.69–1.03)
	OR by 1 tertile	0.90 (0.81–1.01)	0.88 (0.78–0.99)*	0.93 (0.85–1.02)	0.92 (0.83–1.01)
	P for trend	0.062	0.044	0.148	0.108
Cholesterol	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.88 (0.75–1.03)	0.84 (0.71–0.99)*	0.93 (0.81–1.07)	0.89 (0.77–1.04)
	Tertile 3	0.87 (0.74–1.02)	0.82 (0.69–0.99)*	0.91 (0.79–1.04)	0.86 (0.74–1.00)*
	OR by 1 tertile	0.94 (0.87–1.02)	0.91 (0.84–0.99)*	0.95 (0.88–1.02)	0.92 (0.85–1.00)*
	P for trend	0.125	0.041	0.225	0.050
Sodium	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.75 (0.60–0.94)*	0.75 (0.59–0.96)*	0.91 (0.77–1.07)	0.90 (0.75–1.07)
	Tertile 3	0.71 (0.56–0.89)*	0.69 (0.54–0.88)*	0.84 (0.71–1.00)*	0.78 (0.64–0.94)*
	OR by 1 tertile	0.84 (0.75–0.94)*	0.83 (0.73–0.94)*	0.92 (0.84–1.00)*	0.88 (0.80–0.97)*
	P for trend	0.003	0.003	0.049	0.008
Fruit and vegetable	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.99 (0.88–1.12)	0.96 (0.84–1.11)	0.95 (0.83–1.09)	0.96 (0.82–1.12)
	Tertile 3	0.86 (0.76–0.98)*	0.87 (0.75–1.00)*	0.84 (0.73–0.97)*	0.85 (0.72–1.00)
	OR by 1 tertile	0.93 (0.87–0.99)*	0.93 (0.86–1.00)*	0.92 (0.85–0.98)*	0.92 (0.85–1.00)*
	P for trend	0.029	0.050	0.016	0.048
NSP	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.91 (0.78–1.05)	0.90 (0.76–1.07)	0.94 (0.82–1.09)	0.94 (0.80–1.11)
	Tertile 3	0.87 (0.75–1.02)	0.89 (0.76–1.05)	0.81 (0.70–0.94)*	0.83 (0.70–0.98)*
	OR by 1 tertile	0.93 (0.87–1.01)	0.95 (0.87–1.03)	0.90 (0.84–0.97)*	0.91 (0.84–0.99)*
	P for trend	0.082	0.233	0.006	0.027
HDI score	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.84 (0.76–0.94)*	0.81 (0.72–0.92)*	0.91 (0.81–1.02)	0.91 (0.81–1.04)
	Tertile 3	0.75 (0.67–0.84)*	0.73 (0.65–0.83)*	0.80 (0.71–0.90)*	0.78 (0.69–0.90)*
	OR by 1 tertile	0.87 (0.81–0.92)*	0.86 (0.80–0.91)*	0.89 (0.84–0.95)*	0.88 (0.83–0.94)*
	P for trend	< 0.001	< 0.001	0.001	< 0.001

† Model 1: adjusted for age and country.
‡ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health.
* P value < 0.05. ** Gender-specific tertile of ER ratio: in men, tertile 1 (0.20–0.32), tertile 2 (0.32–0.47), and tertile 3 (>= 0.47); in women, tertile 1 (0.20–0.31), tertile 2 (0.31–0.46), and tertile 3 (>= 0.46).

7.2.2 Associations between OC and dietary outcomes

The associations between OC and dietary outcomes are assessed following the same two steps as for ERI–diet associations.

In Table 7.9, binary logistic regression analyses for men indicate that higher OC is significantly ($p < 0.05$) associated with less healthy intakes of saturated fat (OR changes by 1–tertile increase in OC score= 0.78), polyunsaturated fat (OR= 0.91), free sugars (OR= 0.81), protein (OR= 0.89), and fruit/vegetable (OR= 0.92) in Model 2. For women, higher OC is significantly ($p < 0.05$) associated with less healthy intakes of saturated fat (OR= 0.86), polyunsaturated fat (OR= 0.91), protein (OR= 0.89), and NSP (OR= 0.90) in Model 2. The associations between high OC and less healthy intakes of sodium (OR= 0.92) and fruit/vegetable (OR= 0.93) reach marginal significance ($p < 0.1$).

At the bottom of Table 7.9, ordinal logistic regression analyses show a consistent and significant (p= 0.001) association between higher OC and lower HDI score. For 1–tertile increase in OC score, the adjusted odds of being in a higher HDI category are changed by 0.90 in both men and women. For highest versus lowest tertile of OC score, the adjusted odds of being in a higher HDI category are 0.81 and 0.80 in men and women, respectively.

Table 7.9 Associations between OC and dietary outcomes

		Men (n= 5735)		Women (n= 5277)	
Outcome Variables	OC score tertile **	Model 1 † OR (95% CI)	Model 2 ‡ OR (95% CI)	Model 1 † OR (95% CI)	Model 2 ‡ OR (95% CI)
Saturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.83 (0.60–1.16)	0.82 (0.57–1.17)	0.80 (0.62–1.04)	0.85 (0.64–1.11)
	Tertile 3	0.57 (0.39–0.84)*	0.60 (0.40–0.90)*	0.72 (0.54–0.96)*	0.75 (0.55–1.00)*
	OR by 1 tertile	0.76 (0.63–0.92)*	0.78 (0.63–0.96)*	0.85 (0.73–0.97)*	0.86 (0.73–1.00)*
	P for trend	0.006	0.018	0.019	0.045
Poly-unsaturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.01 (0.88–1.16)	1.00 (0.87–1.16)	0.97 (0.84–1.11)	0.94 (0.81–1.09)
	Tertile 3	0.85 (0.74–0.99)*	0.82 (0.70–0.97)*	0.83 (0.72–0.95)*	0.82 (0.70–0.96)*
	OR by 1 tertile	0.93 (0.87–1.00)	0.91 (0.84–0.99)*	0.91 (0.85–0.98)*	0.91 (0.84–0.98)*
	P for trend	0.061	0.026	0.011	0.017
Total carbo-hydrates	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.83 (0.62–1.11)	0.83 (0.61–1.12)	0.94 (0.76–1.15)	0.96 (0.77–1.19)
	Tertile 3	0.82 (0.60–1.13)	0.82 (0.58–1.16)	0.90 (0.72–1.13)	0.90 (0.71–1.14)
	OR by 1 tertile	0.90 (0.77–1.06)	0.90 (0.76–1.07)	0.95 (0.85–1.06)	0.95 (0.84–1.07)
	P for trend	0.212	0.245	0.372	0.382
Free sugars	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.92 (0.69–1.22)	0.87 (0.64–1.18)	0.65 (0.37–1.13)	0.67 (0.35–1.24)
	Tertile 3	0.68 (0.48–0.96)*	0.65 (0.44–0.94)*	0.61 (0.35–1.05)	0.60 (0.32–1.10)
	OR by 1 tertile	0.84 (0.71–0.99)*	0.81 (0.68–0.98)*	0.78 (0.59–1.03)	0.79 (0.58–1.06)
	P for trend	0.033	0.026	0.084	0.106
Protein	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.80 (0.65–0.98)*	0.85 (0.67–1.07)	0.79 (0.66–0.94)*	0.84 (0.69–1.03)
	Tertile 3	0.80 (0.64–0.99)*	0.82 (0.65–1.03)	0.77 (0.64–0.92)*	0.79 (0.65–0.96)*
	OR by 1 tertile	0.88 (0.79–0.99)*	0.89 (0.79–1.00)*	0.87 (0.79–0.95)*	0.89 (0.80–0.99)*
	P for trend	0.032	0.048	0.003	0.045
Cholesterol	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.06 (0.91–1.23)	1.04 (0.89–1.22)	0.97 (0.84–1.11)	0.95 (0.82–1.10)
	Tertile 3	0.99 (0.84–1.16)	0.93 (0.79–1.11)	1.05 (0.91–1.21)	1.02 (0.87–1.19)
	OR by 1 tertile	1.00 (0.92–1.08)	0.97 (0.89–1.05)	1.02 (0.95–1.09)	1.01 (0.93–1.09)
	P for trend	0.970	0.499	0.511	0.840
Sodium	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.09 (0.88–1.34)	1.13 (0.90–1.41)	0.91 (0.77–1.06)	0.86 (0.72–1.02)
	Tertile 3	0.89 (0.70–1.12)	0.83 (0.64–1.07)	0.93 (0.78–1.09)	0.85 (0.70–1.03)
	OR by 1 tertile	0.95 (0.85–1.06)	0.92 (0.82–1.04)	0.96 (0.88–1.05)	0.92 (0.83–1.01)
	P for trend	0.385	0.209	0.423	0.091
Fruit and vegetable	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.88 (0.77–1.00)	0.89 (0.77–1.02)	1.00 (0.87–1.15)	1.01 (0.87–1.18)
	Tertile 3	0.81 (0.71–0.91)*	0.81 (0.71–0.92)*	0.85 (0.74–0.98)*	0.86 (0.73–1.00)*
	OR by 1 tertile	0.92 (0.87–0.99)*	0.92 (0.86–1.00)*	0.93 (0.86–0.99)*	0.93 (0.86–1.01)
	P for trend	0.018	0.048	0.036	0.085
NSP	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.90 (0.78–1.03)	0.93 (0.79–1.10)	0.87 (0.75–1.01)	0.93 (0.80–1.08)
	Tertile 3	0.88 (0.75–1.03)	0.89 (0.76–1.04)	0.78 (0.67–0.91)*	0.81 (0.68–0.95)*
	OR by 1 tertile	0.93 (0.87–1.01)	0.96 (0.88–1.04)	0.88 (0.82–0.96)*	0.90 (0.83–0.98)*
	P for trend	0.084	0.330	0.002	0.013
HDI score	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.89 (0.80–0.99)*	0.88 (0.79–0.99)*	0.89 (0.80–1.00)*	0.91 (0.81–1.03)
	Tertile 3	0.81 (0.72–0.91)*	0.81 (0.71–0.92)*	0.82 (0.73–0.92)*	0.80 (0.70–0.91)*
	OR by 1 tertile	0.90 (0.85–0.95)*	0.90 (0.84–0.96)*	0.91 (0.86–0.97)*	0.90 (0.84–0.96)*
	P for trend	0.001	0.001	0.008	0.001

† Model 1: adjusted for age and country.
‡ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health.
* P value < 0.05. ** Gender- specific tertile of OC score in men and women: tertile 1 (6–11), tertile 2 (11–15), tertile 3 (15–24)

7.2.3 Modifying role of OC in ERI–diet relationship

To evaluate modifying role of OC in ERI–diet relationship, the associations between ER ratio and HDI outcome in different strata of OC tertile are assessed after adjustment for age, country and other covariates. Next, ordinal logistic regression is conducted for HDI outcome regressed by OC tertile, ER–ratio tertile, and interaction term between OC tertile and ER–ratio tertile after adjustment for covariates. By comparing the log likelihoods for the model with this interaction term and the model without, LR test is adopted to test the significance of this interaction term.

Table 7.10 shows the results for modifying effect of OC in ERI–diet relationship. LR tests show that the interaction term between OC tertile and ER–ratio tertile does not reach significance in men (p= 0.140) and women (p= 0.146), respectively. OC has no significantly modifying role in the relationship between ERI and diet.

Table 7.10 Evaluation for modifying role of OC in ERI–diet relationship

Approaches	Strata	Healthy Diet Indicator (HDI) outcome
Men		OR of outcome by 1–tertile increase in ERI (95% CI)
1. ERI–diet relationship in	OC tertile 1	0.85 (0.76 – 0.93)
different strata of OC tertile	OC tertile 2	0.94 (0.84 – 1.04)
	OC tertile 3	0.97 (0.84 – 1.11)
2. Interaction term OC x ERI	P–value	P= 0.140
Women		OR of outcome by 1–tertile increase in ERI (95% CI)
1. ERI–diet relationship in	OC tertile 1	0.89 (0.80 – 1.00)
different strata of OC tertile	OC tertile 2	0.96 (0.86 – 1.08)
	OC tertile 3	1.06 (0.93 – 1.22)
2. Interaction term OC x ERI	P–value	P= 0.146

7.3 Potential Role of PC in Relation between OC, ERI, and Diet

7.3.1 Associations between PC and dietary outcomes

The associations between PC and dietary outcomes are assessed following the same two steps as for ERI–diet associations.

In Table 7.11, binary logistic regression analyses for men indicate that higher PC is significantly ($p < 0.05$) associated with more healthy intakes of saturated fat (OR changes by 1–tertile increase in PC= 1.29), fruit/vegetable (OR= 1.17), and NSP (OR= 1.17) in Model 2; in addition, the associations of PC with total carbohydrate (OR= 1.15) and sodium (OR= 1.10) reach marginal significance ($p < 0.1$). For women, higher PC is significantly ($p < 0.05$) associated with more healthy intakes of saturated fat (OR changes by 1–tertile increase in PC= 1.32), total carbohydrate (OR= 1.16), fruit/vegetable (OR= 1.13), and NSP (OR= 1.10); the association of PC with cholesterol reaches marginal significance ($p < 0.1$).

At the bottom of Table 7.11, ordinal logistic regression analyses show a consistent and significant ($p= 0.001$) association between higher PC and higher levels of HDI outcome. For 1–tertile increase in PC, the adjusted odds of being in a higher HDI category are changed by 1.12 in both men and women. For highest verus lowest tertile of PC, the adjusted odds of being in a higher HDI category are 1.26 in both men and women, respectively.

Table 7.11 Associations between perceived control and dietary outcomes

Outcome Variables	Perceived Control tertile **	Men (n= 5735) Model 1 † OR (95% CI)	Men (n= 5735) Model 2 ‡ OR (95% CI)	Women (n= 5277) Model 1 † OR (95% CI)	Women (n= 5277) Model 2 ‡ OR (95% CI)
Saturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.11 (0.77–1.59)	1.18 (0.79–1.67)	1.18 (0.90–1.56)	1.29 (0.95–1.74)
	Tertile 3	1.58 (1.12–2.21)*	1.67 (1.12–2.32)*	1.56 (1.18–2.05)*	1.74 (1.25–2.39)*
	OR by 1 tertile	1.27 (1.06–1.49)*	1.29 (1.06–1.52)*	1.25 (1.09–1.44)*	1.32 (1.13–1.54)*
	P for trend	0.008	0.007	0.002	< 0.001
Poly-unsaturated fat	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.98 (0.86–1.12)	0.95 (0.82–1.10)	0.98 (0.86–1.13)	0.97 (0.83–1.13)
	Tertile 3	1.01 (0.88–1.15)	1.01 (0.86–1.17)	0.94 (0.82–1.09)	0.96 (0.82–1.14)
	OR by 1 tertile	1.00 (0.94–1.07)	1.00 (0.93–1.08)	0.97 (0.91–1.04)	0.98 (0.90–1.07)
	P for trend	0.923	0.963	0.364	0.661
Total carbo-hydrates	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.14 (0.82–1.57)	1.10 (0.78–1.54)	1.01 (0.81–1.26)	1.00 (0.80 –1.27)
	Tertile 3	1.35 (0.99–1.84)	1.29 (0.91–1.79)	1.29 (1.04–1.60)*	1.33 (1.04–1.69)*
	OR by 1 tertile	1.17 (1.00–1.36)	1.15 (0.98–1.35)	1.14 (1.02–1.27)*	1.16 (1.03–1.32)*
	P for trend	0.056	0.098	0.017	0.017
Free sugars	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.87 (0.65–1.16)	0.90 (0.67–1.19)	0.79 (0.50–1.27)	0.88 (0.52–1.54)
	Tertile 3	1.01 (0.72–1.20)	1.02 (0.73–1.23)	0.94 (0.58–1.56)	1.23 (0.60–2.32)
	OR by 1 tertile	0.99 (0.84–1.17)	1.00 (0.84–1.19)	0.96 (0.73–1.26)	1.12 (0.80–1.55)
	P for trend	0.930	0.956	0.751	0.459
Protein	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	0.89 (0.73–1.09)	0.90 (0.72–1.12)	0.84 (0.71–0.99)*	0.86 (0.71–1.03)
	Tertile 3	0.87 (0.71–1.07)	0.87 (0.69–1.11)	0.97 (0.82–1.16)	1.04 (0.84–1.28)
	OR by 1 tertile	0.93 (0.84–1.03)	0.93 (0.83–1.05)	0.98 (0.89–1.07)	1.01 (0.91–1.12)
	P for trend	0.142	0.258	0.592	0.875
Cholesterol	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.03 (0.88–1.20)	1.00 (0.85–1.17)	0.98 (0.86–1.12)	1.03 (0.89–1.20)
	Tertile 3	1.14 (0.98–1.33)	1.10 (0.93–1.30)	1.07 (0.93–1.23)	1.15 (0.97–1.35)
	OR by 1 tertile	1.07 (0.99–1.15)	1.06 (0.97–1.15)	1.03 (0.96–1.11)	1.08 (0.99–1.17)
	P for trend	0.077	0.168	0.385	0.097
Sodium	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.01 (0.81–1.24)	1.04 (0.83–1.32)	0.98 (0.84–1.15)	1.03 (0.86–1.23)
	Tertile 3	1.11 (0.90–1.38)	1.19 (0.94–1.50)	1.03 (0.87–1.22)	1.07 (0.87–1.30)
	OR by 1 tertile	1.06 (0.95–1.18)	1.10 (0.99–1.24)	1.01 (0.93–1.10)	1.03 (0.93–1.14)
	P for trend	0.331	0.062	0.747	0.525
Fruit and vegetable	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.35 (1.19–1.52)*	1.24 (1.08–1.41)*	1.27 (1.11–1.45)*	1.14 (0.98–1.32)
	Tertile 3	1.59 (1.40–1.79)*	1.34 (1.17–1.54)*	1.60 (1.38–1.82)*	1.28 (1.07–1.51)*
	OR by 1 tertile	1.26 (1.18–1.35)*	1.17 (1.08–1.25)*	1.26 (1.18–1.36)*	1.13 (1.04–1.23)*
	P for trend	< 0.001	< 0.001	< 0.001	0.006
NSP	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.33 (1.15–1.55)*	1.23 (1.04–1.44)*	1.10 (0.95–1.27)	1.05 (0.89–1.22)
	Tertile 3	1.61 (1.39–1.85)*	1.37 (1.16–1.60)*	1.33 (1.14–1.54)*	1.21 (1.02–1.43)*
	OR by 1 tertile	1.27 (1.18–1.36)*	1.17 (1.08–1.26)*	1.15 (1.07–1.24)*	1.10 (1.01–1.20)*
	P for trend	< 0.001	< 0.001	< 0.001	0.032
HDI score	Tertile 1	1.00	1.00	1.00	1.00
	Tertile 2	1.16 (1.04–1.29)*	1.07 (0.95–1.20)	1.03 (0.92–1.15)	1.03 (0.91–1.16)
	Tertile 3	1.40 (1.26–1.56)*	1.26 (1.12–1.43)*	1.30 (1.15–1.46)*	1.26 (1.10–1.44)*
	OR by 1 tertile	1.18 (1.12–1.24)	1.12 (1.06–1.20)*	1.14 (1.07–1.21)*	1.12 (1.05–1.20)*
	P for trend	< 0.001	0.001	< 0.001	0.001

† Model 1: adjusted for age and country.
‡ Model 2: additionally adjusted for other covariates such as education, occupation, marital status, deprivation, depression, social isolation, and self-rated health.
* P value < 0.05. ** Gender– specific tertile of PC: in men, tertile 1 (0–34), tertile 2 (34–41), and tertile 3 (41–55); in women, tertile 1 (0–33), tertile 2 (33–40), and tertile 3 (40–55).

7.3.2 Modifying role of PC in ERI–diet relationship

For evaluating the modifying role of PC in ERI–diet relationship, ordinal logistic regression is conducted for HDI outcome regressed by PC tertile, ER–ratio tertile, and interaction term between PC tertile and ER–ratio tertile after adjustment for covariates. The log likelihoods for the model with this interaction term and the model without are compared. By comparing the log likelihoods for the model with this interaction term and the model without, LR test is used to test the significance of this interaction term.

As shown in Table 7.12, LR tests report that this interaction term does not reach significance in men (p= 0.960) and women (p= 0.714), respectively. PC has no significantly modifying role in the relationship between ERI and diet.

Table 7.12 Evaluation for modifying role of PC in ERI–diet relationship

Approaches	Strata	Healthy Diet Indicator (HDI) outcome
Men		OR of outcome by 1–tertile increase in ERI (95% CI)
1. ERI–diet relationship in	PC tertile 1	0.91 (0.82 – 1.01)
different strata of PC tertile	PC tertile 2	0.88 (0.79 – 0.98)
	PC tertile 3	0.90 (0.81 – 1.00)
2. Interaction term PC x ERI	P–value	P= 0.960
Women		OR of outcome by 1–tertile increase in ERI (95% CI)
1. ERI–diet relationship in	PC tertile 1	0.95 (0.86 – 1.06)
different strata of PC tertile	PC tertile 2	0.96 (0.86 – 1.07)
	PC tertile 3	0.99 (0.88 – 1.11)
2. Interaction term PC x ERI	P–value	P= 0.714

7.3.3 Mediator roles of PC and ERI in OC–diet relationship

To assess mediator roles of PC and ERI in OC–diet relationship, path analysis for an ordinal categorical outcome (HDI outcome) is adopted in Mplus 7. Each path coefficient is obtained by probit regression for HDI outcome on a predictor after adjustment for covariates. The odds ratio (OR) is the antilog of estimated logistic coefficient, calculated from probit coefficient multiplied by 1.8. In terms of exposures, the tertiles of ERI and OC are transformed into a series of dummy variables to compare between tertile groups in each exposure. Next, ERI tertile and OC tertile are treated as continuous variables to estimate assumed linear trend between the exposure and odds of HDI outcome (OR changes by 1–tertile increase in the exposure).

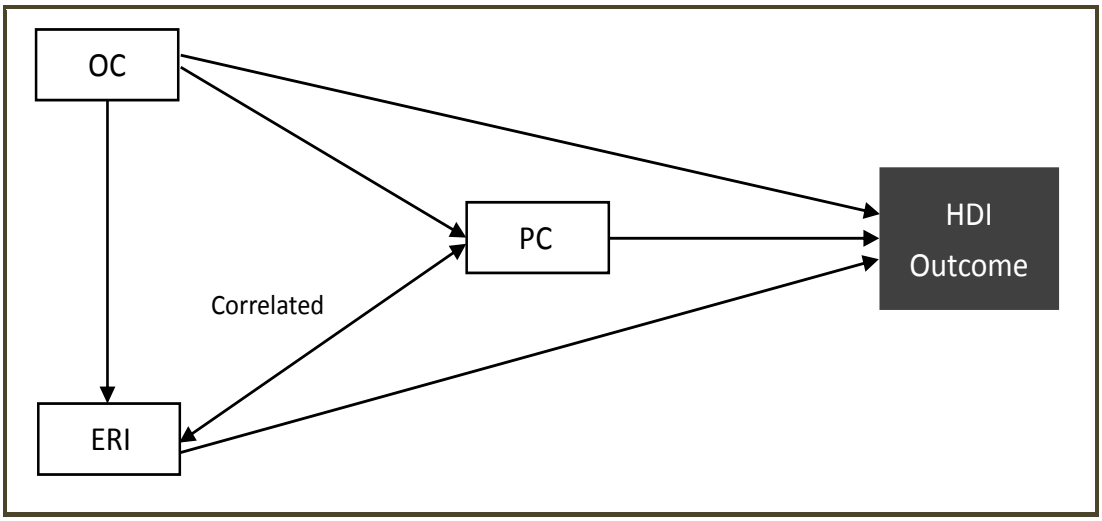
The path model is specified in Figure 7.1. First, there are two potential mediators (PC and ERI) between the effects of OC on HDI outcome. As the HAPIEE study is limited by no measurement of PC and dietary outcomes at wave 2, the cross–sectional associations between OC, ERI, PC and dietary outcomes (all at wave 1) are analyzed. Thus, PC and ERI are only specified to be correlated, although bi-directional relationship between PC and ERI had been hypothesized. Second, antecedent roles of OC in ERI–diet and PC–diet associations are specified based on previous results in the cohort studies on drinking (Chapter 5) and smoking outcomes (Chapter 6).

Third, the mediator effect of PC in OC–diet relation is assessed by two effects: (1) the effect of OC on PC; (2) the effect of PC on diet. The partially mediator effect of ERI in OC–diet relation is estimated by two effects: (1) the effect of OC on ERI; (2) the effect of ERI on diet. Partial mediation applies if both causal relations are confirmed; the product of two path coefficients can estimate the strength of mediator effect.

The bootstrap method is used for significance testing of a mediator effect due to complicated models (an ordinal categorical outcome and two mediators), with 5000 bootstrap samples used to yield more valid estimates for indirect effects by Mplus 7.[593] Finally, three indexes for model fit are used: Root Mean Square Error of Approximation

(RMSEA), Comparative Fit Index (CFI), and Tucker–Lewis Index (TLI). RMSEA < 0.06, CFI > 0.95 or TLI > 0.95 indicate “good model fit”.[594]

Figure 7.1 Hypothetical model specified for path analysis for mediator roles of PC and ERI in OC–diet relation



In the results of path analysis in men (Table 7.13; Figure 7.2), first, the mediator effect of ERI in the relationship between OC and HDI outcome is estimated by multiplying two effects: (1) higher OC significantly associated with higher ERI (unstandardized β= 0.286; standard error= 0.016); (2) higher ERI significantly associated with lower levels of HDI outcome (unstandardized β= –0.057; standard error= 0.019). This mediator effect of ERI is significant (–0.016= 0.286 x –0.057; standard error= 0.006 and p= 0.003 by bootstrap method).

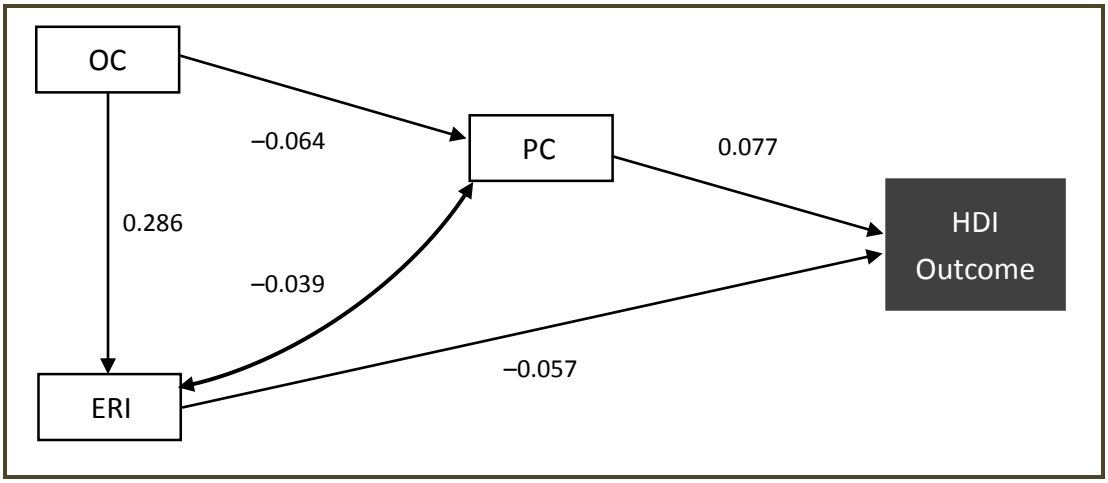
Second, the mediator effect of PC in the relationship between OC and HDI outcome is estimated by multiplying two effects: (1) higher OC significantly associated with lower PC (unstandardized β= –0.064; standard error= 0.014); (2) lower PC significantly associated with lower levels of HDI outcome (unstandardized β= 0.077; standard error= 0.018). This mediator effect of PC is significant (–0.005; standard error= 0.002 and p= 0.004). Third, the inverse association between PC and ERI reaches statistical significance (unstandardized β= –0.039; p= 0.001). Finally, the fit

indexes (RMSEA= 0.066, CFI= 0.826, and TLI= 0.796) are not far from the cutoffs for good fit (the cutoffs are RMSEA < 0.06, CFI > 0.95, and TLI > 0.95).

Table 7.13 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in men (N= 5735)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC → HDI outcome				
OC tertile 2 vs tertile 1	0.97	- 0.016	- 0.007	0.646
OC tertile 3 vs tertile 1	0.92	- 0.048	- 0.020	0.223
OR change by 1 tertile	0.96	- 0.023	- 0.018	0.232
OC → ERI → HDI outcome	0.97	- 0.016	- 0.013	0.003
OC → ERI				
OC tertile 2 vs tertile 1	1.74	0.316	0.187	< 0.001
OC tertile 3 vs tertile 1	2.62	0.567	0.308	< 0.001
OR change by 1 tertile	1.65	0.286	0.288	< 0.001
ERI → HDI outcome				
ERI tertile 2 vs tertile 1	0.85	- 0.093	- 0.042	0.006
ERI tertile 3 vs tertile 1	0.81	- 0.114	- 0.053	0.003
OR change by 1 tertile	0.90	- 0.057	- 0.044	0.003
OC → PC → HDI outcome	0.99	- 0.005	- 0.004	0.004
OC → PC				
OC tertile 2 vs tertile 1	0.98	- 0.012	- 0.007	0.684
OC tertile 3 vs tertile 1	0.80	- 0.144	- 0.081	< 0.001
OR change by 1 tertile	0.91	- 0.064	- 0.066	< 0.001
PC → HDI outcome				
PC tertile 2 vs tertile 1	1.13	0.067	0.030	0.060
PC tertile 3 vs tertile 1	1.33	0.159	0.072	< 0.001
OR change by 1 tertile	1.15	0.077	0.058	< 0.001
ERI correlates with PC		- 0.039	- 0.065	0.001
Tests of model fit	RMSEA=	0.066	CFI= 0.826	TLI= 0.796

Figure 7.2 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in men



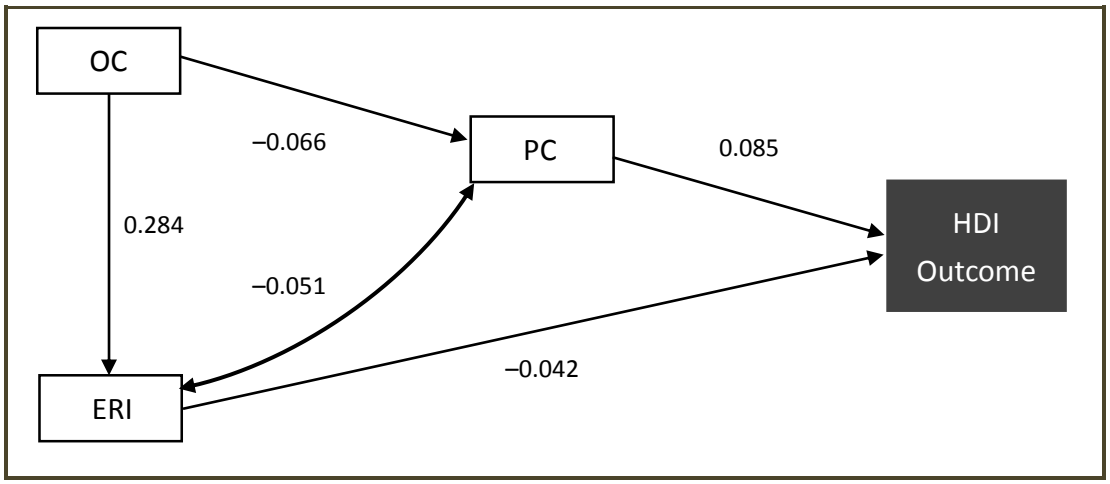
In the results of path analysis in women (Table 7.14; Figure 7.3), first, the mediator effect of ERI in the relationship between OC and HDI outcome is estimated by multiplying two effects: (1) higher OC significantly associated with higher ERI (unstandardized β= 0.284; standard error= 0.016); (2) higher ERI significantly associated with lower levels of HDI outcome (unstandardized β= –0.042; standard error= 0.020). This mediator effect of ERI is significant (–0.012= 0.284 x –0.042; standard error= 0.006 and p= 0.004).

Second, the mediator effect of PC in the relationship between OC and HDI outcome is assessed by multiplying two effects: (1) higher OC significantly associated with lower PC (unstandardized β= –0.066; standard error= 0.014); (2) lower PC significantly associated with lower levels of HDI outcome (unstandardized β= 0.085; standard error= 0.020). The mediator effect of PC is significant (–0.006; standard error= 0.002 and p= 0.001). Third, the inverse association between PC and ERI reaches statistical significance (unstandardized β= –0.051; $p < 0.001$). Finally, the fit indexes (RMSEA= 0.068, CFI= 0.811, and TLI= 0.782) are not far from the cutoffs for good fit (the cutoffs are RMSEA < 0.06, CFI > 0.95, and TLI > 0.95).

Table 7.14 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in women (N= 5277)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC → HDI outcome				
OC tertile 2 vs tertile 1	0.98	- 0.011	- 0.006	0.750
OC tertile 3 vs tertile 1	0.96	- 0.025	- 0.012	0.426
OR change by 1 tertile	0.98	- 0.015	- 0.008	0.412
OC → ERI → HDI outcome	0.98	- 0.012	- 0.009	0.004
OC → ERI				
OC tertile 2 vs tertile 1	1.66	0.291	0.171	< 0.001
OC tertile 3 vs tertile 1	2.62	0.567	0.317	< 0.001
OR change by 1 tertile	1.64	0.284	0.288	< 0.001
ERI → HDI outcome				
ERI tertile 2 vs tertile 1	0.90	- 0.050	- 0.024	0.224
ERI tertile 3 vs tertile 1	0.86	- 0.088	- 0.039	0.054
OR change by 1 tertile	0.93	- 0.042	- 0.032	0.045
OC → PC → HDI outcome	0.99	- 0.006	- 0.004	0.001
OC → PC				
OC tertile 2 vs tertile 1	0.99	- 0.008	- 0.005	0.777
OC tertile 3 vs tertile 1	0.81	- 0.140	- 0.085	< 0.001
OR change by 1 tertile	0.90	- 0.066	- 0.073	< 0.001
PC → HDI outcome				
PC tertile 2 vs tertile 1	1.01	0.004	0.002	0.906
PC tertile 3 vs tertile 1	1.37	0.174	0.075	< 0.001
OR change by 1 tertile	1.17	0.085	0.060	< 0.001
ERI correlates with PC		- 0.051	- 0.093	< 0.001
Tests of model fit	RMSEA=	0.068	CFI= 0.811	TLI= 0.782

Figure 7.3 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in women



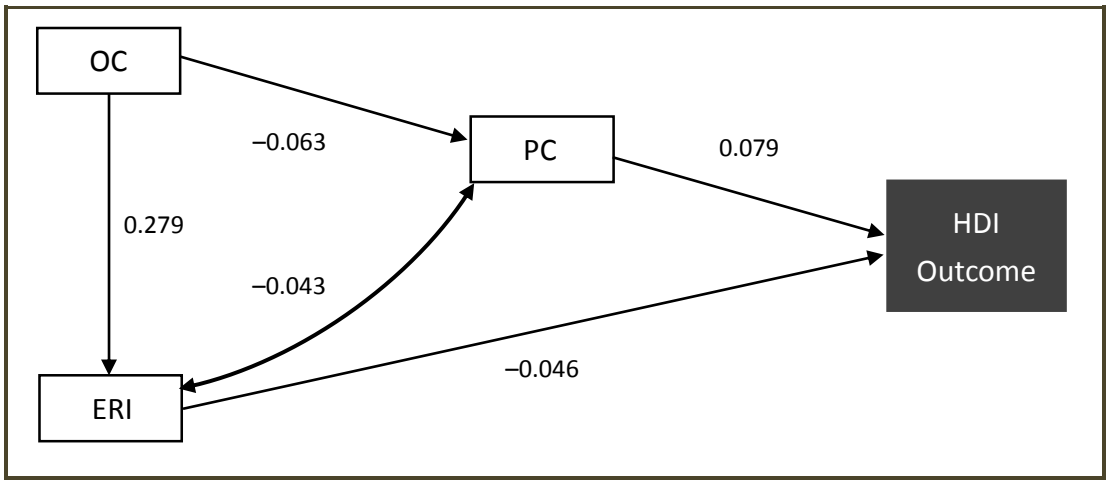
As mentioned earlier, the interactions between gender and exposure variables (ERI and OC) are not significant. Thus, the data from men and women are pooled for final path analysis for mediator roles of PC and ERI in OC–diet relationship (Table 7.15; Figure 7.4). First, the mediator effect of ERI in the relationship between OC and HDI outcome is estimated by multiplying two effects: (1) higher OC significantly associated with higher ERI (unstandardized β= 0.279; standard error= 0.012); (2) higher ERI significantly associated with lower levels of HDI outcome (unstandardized β= –0.046; standard error= 0.014). The mediator effect of ERI is significant (–0.013; standard error= 0.004 and p= 0.001 by bootstrap method).

Second, the mediator effect of PC in the relationship between OC and HDI outcome is estimated by multiplying two effects: (1) higher OC significantly associated with lower PC (unstandardized β= –0.063; standard error= 0.011); (2) lower PC significantly associated with lower levels of HDI outcome (unstandardized β= 0.079; standard error= 0.013). The mediator effect of PC is significant (–0.005; standard error= 0.001 and $p < 0.001$). Third, the inverse association between PC and ERI reaches statistical significance (unstandardized β= –0.043; $p < 0.001$). Finally, the fit indexes (RMSEA= 0.066, CFI= 0.819, and TLI= 0.790) are not far from the cutoffs for good fit (RMSEA < 0.06, CFI > 0.95 or TLI > 0.95).

Table 7.15 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in pooled data of men and women (N= 11012)

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC → HDI outcome				
OC tertile 2 vs tertile 1	0.99	- 0.006	- 0.003	0.814
OC tertile 3 vs tertile 1	0.94	- 0.037	- 0.016	0.193
OR change by 1 tertile	0.97	- 0.017	- 0.013	0.237
OC → ERI → HDI outcome	0.98	- 0.013	- 0.010	0.001
OC → ERI				
OC tertile 2 vs tertile 1	1.68	0.295	0.174	< 0.001
OC tertile 3 vs tertile 1	2.56	0.554	0.306	< 0.001
OR change by 1 tertile	1.63	0.279	0.282	< 0.001
ERI → HDI outcome				
ERI tertile 2 vs tertile 1	0.88	- 0.068	- 0.031	0.009
ERI tertile 3 vs tertile 1	0.85	- 0.091	- 0.042	0.001
OR change by 1 tertile	0.92	- 0.046	- 0.035	0.001
OC → PC → HDI outcome	0.99	- 0.005	- 0.004	< 0.001
OC → PC				
OC tertile 2 vs tertile 1	0.98	- 0.010	- 0.006	0.735
OC tertile 3 vs tertile 1	0.78	- 0.136	- 0.078	< 0.001
OR change by 1 tertile	0.91	- 0.063	- 0.066	< 0.001
PC → HDI outcome				
PC tertile 2 vs tertile 1	1.08	0.041	0.018	0.108
PC tertile 3 vs tertile 1	1.34	0.163	0.072	< 0.001
OR change by 1 tertile	1.15	0.079	0.059	< 0.001
ERI correlates with PC		- 0.043	- 0.075	< 0.001
Tests of model fit	RMSEA=	0.066	CFI= 0.819	TLI= 0.790

Figure 7.4 Results of path analysis for mediator roles of PC and ERI in OC–diet relationship in pooled data of men and women



7.4 Main Findings for Dietary Outcomes

The analyses are based on the cross–sectional study of 11012 subjects (5735 men and 5277 women) aged 45–69 from the wave 1 of HAPIEE study. The aims of analyses are: (1) to examine the relationship between ERI, OC, and dietary outcomes; (2) to evaluate the potential role of PC in the relationship between ERI, OC and dietary outcomes. Antecedent role of OC in ERI–diet association was specified according to previous findings in drinking and smoking outcomes, as potentially bidirectional relationship between OC and ERI (Hypothesis 9) cannot be disentangled in the cross–sectional study. The following findings are reported according to specific objectives and hypotheses listed in Chapter 3.

In terms of the associations between ERI, OC and dietary outcomes, Hypothesis 3 that higher ER ratio is associated with less healthy diet (lower levels of HDI) after adjustment for covariates is supported. Hypothesis 6 that higher OC is associated with less healthy diet after adjustment for covariates is supported. Although there are inconsistent associations between exposure variables and individual HDI components, the overall effects of exposure variables on HDI (the sum of 9 components) appear more robust. This finding implies that exposure variables are associated with overall diet quality that is linked to the risks of chronic diseases. In addition, modifying role of OC in ERI–diet relationship is found non–significant.

With regards to the potential role of PC in the relationship between ERI, OC and dietary outcomes, Hypothesis 12 that lower PC is associated with less healthy diet after adjustment for covariates is supported.

Hypothesis 15 is partially supported. PC and ERI partially mediate the effects of OC on dietary outcomes. Additionally, PC and ERI may have bi–directional relationship. PC and ERI are negatively associated with each other; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in the cross–sectional study. Finally, modifying role of PC in ERI–diet

relation is non–significant.

Note that the methodological issues and interpretation of the main findings for dietary outcomes will be addressed in detail in Chapter 8, and their implications for practice, policy, and research in Chapter 9. The analyses for the associations between OC, ERI and dietary outcomes have been peer reviewed and published in the British Journal of Nutrition (please see Appendix 5).

Chapter 8. General Discussion

In this chapter, I will firstly summarize the results in terms of all objectives and hypotheses listed in Chapter 3. This part will be followed by the discussion of various methodological issues of the thesis. Finally, in the last part of Chapter 8, I will compare my findings with those from other existing studies.

8.1 Summary for Results

This section provides the summary of results for drinking, smoking, and dietary outcomes, respectively, according to objectives and hypotheses listed in Chapter 3.

Objective 1

To assess crude and adjusted associations between ERI and three health behaviours – alcohol drinking, smoking, and diet, respectively.

According to Hypothesis 1, higher ER ratio is associated with higher levels of alcohol drinking after adjustment for covariates. In men, the OR changes for heavy drinking, binge drinking and problem drinking were 1.15, 1.49 and 1.35, respectively, by 1–tertile increase in ER ratio. In women, these OR changes were 1.13, 1.42 and 1.36, respectively. All p–values were significant ($p < 0.05$). My findings suggest that Hypothesis 1 is supported.

Based on Hypothesis 2, higher ER ratio is associated with higher levels of smoking after adjustment for covariates. The OR changes for being current smokers were 1.18 and 1.21 by 1–tertile increase in ER ratio in men and women, respectively. In current smokers, the OR changes for being in a higher level of smoking intensity were 1.19 and 1.16 ($p= 0.088$) by 1–tertile increase in ER ratio in men and women, respectively. All p–values were significant ($p < 0.05$) except that identified as marginally significant. My findings suggest that Hypothesis 2 is supported.

According to Hypothesis 3, higher ER ratio is associated with less healthy diet

(lower levels of HDI) after adjustment for covariates. The OR changes of being in a higher HDI category were 0.86 and 0.88 by 1–tertile increase in ER ratio in men and women, respectively. Both p–values were significant ($p < 0.05$). My findings indicate that Hypothesis 3 is supported.

Objective 2

To assess crude and adjusted associations between OC and three health behaviours.

Based on Hypothesis 4, higher OC is associated with higher levels of alcohol drinking after adjustment for covariates. In men, the OR changes for heavy drinking, binge drinking and problem drinking were 1.08 ($p= 0.081$), 1.31 and 1.28, respectively, by 1–tertile increase in OC; all p–values were significant ($p < 0.05$) except that identified as marginally significant. In women, these OR changes were 1.06 ($p= 0.281$), 1.24 and 1.27, respectively; all p–values were significant except that identified as non–significant. My findings suggest that Hypothesis 4 is partially supported.

According to Hypothesis 5, higher OC is associated with higher levels of smoking after adjustment for covariates. The OR changes for being current smokers were 1.14 and 1.15 by 1–tertile increase in OC in men and women, respectively; both p–values were significant ($p < 0.05$). In current smokers, the OR changes for being in a higher level of smoking intensity were 1.11 ($p= 0.171$) and 1.14 ($p= 0.145$) by 1–tertile increase in OC in men and women, respectively; these associations did not reach statistical significance. My findings indicate that Hypothesis 5 is partially supported.

Based on Hypothesis 6, higher OC is associated with less healthy diet (lower levels of HDI) after adjustment for covariates. The OR changes of being in a higher HDI category were 0.90 by 1–tertile increase in OC in both sexes; both p–values were significant ($p < 0.05$). My findings suggest that Hypothesis 6 is supported.

Objective 3

To evaluate the potential role of OC (antecedent, mediator, modifier, or direct effect) in

the relationship between ERI and health behaviours.

As predicted by Hypothesis 7, OC and ERI had bi–directional relationship, but the effect of OC on ERI was stronger than the other direction in the middle-aged and older populations. Antecedent role of OC in ERI–drinking relationship was found significant, but mediator role of OC was not significant. Direct effect of OC on drinking was not significant. Finally, modifying role of OC in ERI–drinking relation was non–significant. My findings show that Hypothesis 7 is supported.

As suggested by Hypothesis 8, OC and ERI had bi–directional relationship, but the effect of OC on ERI was stronger than the other direction in the middle-aged and older populations. Antecedent role of OC in ERI–smoking relationship was found significant among all smoking outcomes in both sexes (except smoking intensity in women), but mediator role of OC was not significant. Direct effect of OC on smoking was not significant. Finally, modifying role of OC in ERI–smoking relation was non–significant. My findings suggest that Hypothesis 8 is partially supported.

Hypothesis 9 suggested that OC and ERI had bi–directional relationship; however, bidirectional relationship between OC and ERI cannot be disentangled in this cross–sectional study (diet was only available at wave 1 whereas drinking and smoking were available at wave 1 and wave 2). Antecedent role of OC in ERI–diet relationship was significant, but mediator role of OC was not tested. Direct effect of OC on diet was not significant. Finally, modifying role of OC in ERI–diet relationship was non–significant. Hypothesis 9 is partially supported.

Objective 4

To assess crude and adjusted associations between PC and three health behaviours.

According to Hypothesis 10, lower PC is associated with higher levels of alcohol drinking after adjustment for covariates. In men, the OR changes for heavy drinking, binge drinking and problem drinking were 0.90, 0.82 and 0.81, respectively, by 1–tertile increase in PC. In women, these OR changes were 0.91 ($p= 0.083$), 0.80 and 0.80,

respectively. All p–values were significant ($p < 0.05$) except that identified as marginally significant. My findings suggest that Hypothesis 10 is supported.

Based on Hypothesis 11, lower PC is associated with higher levels of smoking after adjustment for covariates. The OR changes for being current smokers were 0.81 by 1–tertile increase in PC in both sexes. In current smokers, the OR changes for being in a higher level of smoking intensity were 0.79 and 0.80 by 1–tertile increase in PC in men and women, respectively. All p–values were significant ($p < 0.05$). My findings indicate that Hypothesis 11 is supported.

According to Hypothesis 12, lower PC is associated with less healthy diet after adjustment for covariates. The OR changes for being in a higher HDI category were 1.12 by 1–tertile increase in PC in both sexes; both p–values were significant ($p < 0.05$). My findings show that Hypothesis 12 is supported.

Objective 5

To examine the potential role of PC (mediator or modifier) in the relationship between ERI, OC, and health behaviours.

As predicted by Hypothesis 13, ERI and PC partially mediated the effects of OC on alcohol drinking. Additionally, PC and ERI were negatively associated with each other; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in the cross–sectional analyses. Finally, modifying role of PC in ERI–drinking relation was non–significant. My findings show that Hypothesis 13 is partially supported.

As suggested by Hypothesis 14, ERI and PC partially mediated the effects of OC on all smoking outcomes in both sexes (except smoking intensity in women). Additionally, PC and ERI were negatively associated with each other; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in the cross–sectional analyses. Finally, modifying role of PC in ERI–smoking relation was non–significant. My findings indicate that Hypothesis 14 is

partially supported.

As predicted by Hypothesis 15, ERI and PC partially mediated the effects of OC on diet. In addition, PC and ERI were negatively associated with each other; bi–directional relationship between PC and ERI is possible, but causal directionality cannot be established in the cross–sectional study. Finally, modifying role of PC in ERI–diet relation was non–significant. Hypothesis 15 is partially supported.

8.2 Methodological Issues

Before comparing my findings summarized above with those from previous studies, it is essential to focus on several methodological issues and to highlight potential strengths and limitations of the current project. The strengths of this thesis are a population–based approach with random community samples from three CEE countries, a large sample size with strong statistical power, a 2–wave cohort study design, a central protocol across all study centres (such as questionnaires or training), data collected on diverse aspects of health behaviours, and application of advanced statistical methods (such as SEM and path analysis). However, the results described in this thesis should be interpreted carefully, because as with all studies, there are several methodological features of study population and of study design whose limitations need to be taken into account. These methodological issues are discussed in detail in the following sections.

8.2.1 Representativeness of study sample and selection bias

External validity refers to the extent to which the results of a study can be extrapolated or generalized to the reference population; this concept is related to the representativeness of the study, and thus depends on the procedure of selection of participants. Selection bias is systematic error that results from procedures used to

select subjects and from factors that influence study participation.[595]

The participants in the HAPIEE study were randomly selected from population registers in Novosibirsk, Krakow and six towns in the Czech Republic, and they are considered representative of urban populations. While the selected urban centers cannot be entirely representative for the whole countries, available indicators of socioeconomic characteristics, health behaviours, and mortality suggest that these selected urban populations approximately represented their national populations in the WHO systematic reviews and reports.[596,597]

Overall response rate in the HAPIEE study was 60%, which was typical for contemporary cohort studies, such as the Survey of Health, Ageing and Retirement in Europe (SHARE) and the English Longitudinal Study of Ageing (ELSA).[598,599] In all three countries, short questionnaires from subsample of those who refused participation were analyzed. As a small proportion of non–respondents had died or moved away after the sample was selected but before being invited to the study, they were ineligible for inclusion. Extrapolating from the proportion of incorrect addresses identified in home visits and from the evaluation of accuracy of the population register, real response rates were estimated to be 71% in Novosibirsk, 68% in Krakow, and 60% in Czech Republic. The comparison of respondents and non–respondents in the HAPIEE study was conducted; non–respondents were more likely to be younger age, male, with lower levels of education, with higher prevalence of smoking, and with poorer self–rated health.[600]

The approach of handling missing values in this thesis is “complete case analysis”; only subjects with complete data on exposures and outcomes were analyzed. In some cases, a complete case analysis can provide unbiased estimates; for example, fitting the regression model to complete cases would be unbiased if missingness is independent of outcome, after adjusting for predictors. This assumption may not be entirely true in this project, particularly for drinking outcome, but should not influence results substantially. A disadvantage of this approach is that excluding observations

with missing values may reduce sample size of analytical sample.[601] Notably, there are alternative methods such as imputation methods – which predict missing values based on observed data and missing–data pattern. This approach assumes that missing is dependent on observed data but not on unobserved data (missing at random; MAR). However, multiple imputation might make some statistical analyses (e.g. SEM) too complex and MAR assumption might result in biased results.

As a form of sensitivity analysis, bivariate analyses were conducted among study samples and excluded subjects due to missing values in exposures, outcomes and covariates (for more details, see Section 4.2). In the subsample for drinking/smoking outcomes, excluded subjects (n= 5758) were more likely to be male, with lower educational level, with higher alcohol consumption, with more current smokers, and with poorer self–rated health than the study sample. In the subsample for dietary outcomes, excluded subjects (n= 2259) were more likely to be older and male, with lower educational level, with more current smokers, and with poorer self–rated health than the study sample. There were no major systemic differences between complete cases and incomplete cases. Those with worse profiles of health behaviours were more likely to drop out from follow–up or have missing values; the levels of risky health behaviours were probably underestimated in this thesis, but this bias should not substantially affect the associations between exposures and health behaviours.

8.2.2 Information bias

Information bias is systematic error in estimating an effect caused by measurement error in the needed information. Differential misclassification is dependent on exposure or outcome status. Non–differential misclassification is independent from exposure or outcome status. Non–differential misclassification in exposures occurs when the proportion of subjects misclassified on exposure does not depend on outcome status of subjects; non–differential misclassification in outcomes

occurs when the proportion of subjects misclassified on outcome does not depend on exposure status of subjects.[602]

Common scenarios of differential misclassification were not found in this thesis, because original aims of the HAPIEE study were to investigate psychosocial risk factors for chronic diseases in CEE – different from the aims of this thesis.[603] However, analytical procedures such as collapsing continuous or categorical exposures into fewer categories can change non–differential error to differential misclassification, thereby exaggerating or underestimating an effect.[604]

In terms of non–differential misclassification in exposures, ER ratio, OC and PC were evaluated by self–reported measurements that were potentially subjective to recall bias. In addition, in CEE countries, people may tend to answer questions in psychosocial measures around the middle of the scale, rather than using the extreme points of this scale, thereby leading to overestimation of the effect.[605]

Non–differential misclassifications in outcomes (drinking, smoking, and diet) are described in the following paragraphs. For drinking outcomes, self–reported measures of alcohol drinking typically underestimate actual consumption.[606] In particular, social stigma associated with alcohol affects women more than men, and systematic underreporting of alcohol is probably greater in women than in men.[607] Nevertheless, GFQ method appears less prone to underreporting among available alcohol measures. In the HAPIEE study, both GFQ–based variables and problem drinking were strongly associated with separately taken measures of alcohol consumption and serum gamma–glutamyl transferase, indicating acceptable validity of drinking outcomes.[608]

For smoking outcomes, self–reported measures of smoking are often subject to underreporting. As smoking is considered more socially acceptable in men than in women, smoking status and smoking intensity may be underestimated particularly in women. In the HAPIEE study, the validity of smoking outcomes has not been tested by estimating correlation with plasma biomarkers.[609]

For dietary outcomes, FFQ is the primary method to gather dietary information

from large population samples, as it is inexpensive and representative for average long–term diet. However, the following limitations should be considered. First, the FFQ method tends to be semi–quantitative, rather than fully quantitative, probably resulting in overestimation or underestimation of dietary intakes.[610] Assigning HDI scores may introduce some misclassification, but the ranking of subjects in terms of HDI should be unbiased. Second, local but internationally comparable food composition tables are unavailable for these CEE countries. The inclusion of local food tables to capture country–specific foods may introduce misclassification. Besides, the FFQ components differed slightly in three CEE countries, thereby leading to imprecision in comparing dietary intakes between these countries. However, added items of country–specific foods were approved by local nutritionists to ensure that diet was assessed properly in each country. Third, the validity of the FFQ regarding fruit, vegetable and micronutrient intakes was found acceptable by estimating correlation with plasma biomarkers in a random HAPIEE subsample; these correlations were similar to other large studies. However, other HDI components have not been tested for validity.[611] Fourth, the HDI was constructed by Huijbregts' original approach consisting of dietary components coded as dichotomous variables. Note that Jankovic et al proposed a new HDI approach which applied continuous scoring to obtain greater variation between individuals, providing more precise estimation of diet quality.[612]

8.2.3 Confounding

Confounding means that the apparent effect of the exposure of interest is distorted because the effect of extraneous factor (confounding factor) is mistaken for or mixed with the actual exposure effect, leading to overestimation or underestimation of an effect. For a variable to be a confounding factor, it should meet three necessary criteria: it must be a risk factor for the outcome; it must be associated with the exposure in the source population of the study; it cannot be a mediator in the causal path between the

exposure and the outcome.

In this thesis, potential confounding factors were selected from the HAPIEE data and then adjusted as covariates in regression analyses, including age, country, social position (education and occupation), material factors (deprivation), psychosocial factors (marital status, depression, and social isolation), and self–rated health. Although possible confounding factors were controlled in the analyses, there may be residual confounding factors not taken into account. For example, chronic stressors outside workplace (e.g. daily hassles), psychological constructs (e.g. attitude and subjective norm in Theory of Planned Behaviour), and community–level factors (e.g. access to neighborhood resources) are known risk factors for health behaviours but unavailable in the HAPIEE study. These confounding factors may lead to underestimation or overestimation of exposure–outcome relationships, depending on direction of associations of the confounding factor with the exposure and outcome.[613]

8.2.4 Limitations of two–wave cohort study designs

The ideal condition for mediation analyses is a 3–wave cohort study design, in which the exposure variable (wave 1) precedes the mediator (wave 2) in time and the mediator precedes the outcome (wave 3) in time. Although a 3–wave cohort study provides the best estimation for mediation, a 2–wave cohort study still can offer indication for the presence of partial mediation, but not full mediation, thereby yielding better evidence than a cross–sectional study or a half–longitudinal design (one of the associations is cross–sectional).[614]

As three waves of relevant measurements for this thesis were unavailable in the HAPIEE study, a 2–wave cohort design (an autoregressive and cross–lagged model) was adopted in path analyses for the potential role of OC in ERI–drinking relation and ERI–smoking relation, respectively. Note that only partial mediation (e.g. ERI partially mediated the effects of OC on drinking), rather than full mediation, can be examined

in these analyses. Moreover, the effect size measure for a mediated effect (the proportion of total effect that is mediated) cannot be measured as precisely as that in a 3–wave cohort study; the mediated effect, if not strong, would tend to be underestimated in a 2–wave cohort study.[615]

8.2.5 Limitations of cross–sectional study designs

As PC and dietary outcomes were unavailable at wave 2 in the HAPIEE study, two parts of the analyses were actually in cross–sectional design: (1) the relationship between OC, ERI and PC at wave 1 in the analyses of drinking or smoking outcomes at wave 2 (a half–longitudinal design); (2) the relationship between exposure variables at wave 1 and dietary outcomes at wave 1. A cross–sectional study often has difficulty in determining the time order between the exposure and the outcome, unless the exposure is defined prior to recruitment and measurement of the outcome.

In the cross–sectional analysis for the associations between OC, ERI and PC, the parsimonious hypothesis that OC causes ERI and PC, rather than the other causal direction, has been supported by theoretical explanation (e.g. Transactional Model of Stress or personality psychology) and empirical studies (including the 2–wave cohort analysis in this thesis). However, potentially bidirectional relationship between OC, ERI and PC still cannot be disentangled in the cross–sectional analysis.

In the cross–sectional analysis for exposure–diet associations, the difficulty in determining time sequence between events would introduce bias into the analysis. For example, those with unhealthy diet may tend to have more mental and physical problems, thereby causing them to perceive or encounter higher levels of work stress. Thus, the reverse causality that unhealthy diet causes high levels of work stress cannot be ruled out by the cross–sectional design.

8.2.6 Random error

Random error (chance or random variation) is the divergence, due to chance alone, of an observation on a sample from the true population value, leading to imprecise measurement of an association. Random error is often induced by the process of selecting study subjects and the unpredictable inaccuracies in occurrence measures. A common way to reduce random error in an epidemiologic study is to enlarge the size of the study, which is planned based on statistical sample–size formulas. In addition, significance testing in epidemiology focuses on deciding whether chance or random error could be solely responsible for an observed association.

In this thesis, the subsamples were generated from the HAPIEE study and the data was stratified by gender in the analyses, it is not at risk of being underpowered to detect small differences. In the subsample on dietary outcomes (sample size= 6000), power calculation shows that statistical power is over 99% for odds ratio larger than 1.3 in baseline probability of 0.05. In the subsample on drinking/smoking outcomes (sample size= 4000), power calculation reports that statistical power is over 95% for odds ratio larger than 1.3 in baseline probability of 0.05. However, the study was sometimes overpowered to investigate the proposed research questions, and small effects may have been detected as statistically significant. Thus, the interpretation of results should not rely entirely on statistical significance of an effect, but the magnitude of an effect estimate and previous work should also be considered in order to make careful interpretation of statistics.

8.3 Discussion of Results

Taking all the methodological issues of the thesis into account, it is possible to compare the findings of this thesis with previous literature. This discussion of results covers the following topics: (1) work stress and health behaviours; (2) OC personality and health behaviours; (3) the potential role of OC in the relationship between ERI and

health behaviours; (4) PC and health behaviours; (5) the potential role of PC in the relationship between OC, ERI and health behaviours.

8.3.1 Work stress and health behaviours

In terms of work stress and drinking outcomes, this thesis found that higher ER ratio at wave 1 was associated with higher levels of drinking outcomes (heavy drinking, binge drinking and problem drinking) at wave 2 after adjustment for covariates in both sexes. This finding is in line with previous literature on work stress and alcohol drinking. Several reviews and prospective studies have supported the associations between the DC model and alcohol drinking (for more details, see Section 2.3.3).[616,617,618] Moreover, existing studies have showed promising results to support the links between the ERI model and alcohol drinking. For example, Head et al reported that high ER ratio predicted alcohol dependence in a British cohort study (n= 7,372); this association was stronger in men than in women.[619] In the pilot HAPIEE study (n= 694) in the same CEE populations as this thesis, Bobak et al found that high ER ratio was associated with binge drinking (OR= 1.36), problem drinking (OR= 1.37), high annual intake of alcohol (OR= 1.29), and high annual number of drinking sessions (OR= 1.34) in the cross–sectional analyses.[620] Thus, my finding provides evidence for consistency of longitudinal associations between ERI and drinking outcomes in the CEE populations.

With regard to work stress and smoking outcomes, this thesis reported that higher ER ratio at wave 1 was associated with higher levels of smoking outcomes (smoking status and smoking intensity) at wave 2 after adjustment for covariates in both sexes. This finding is consistent with previous reviews on the associations of the DC model with smoking status and smoking intensity (for more details, see Section 2.3.3).[621,622] Additionally, several cross–sectional studies have supported the links between the ERI model and smoking outcomes. For example, Kouvonen et al reported that high ER ratio was associated with being current smokers (OR= 1.28) in a Finnish cross–

sectional study (n= 46,190); among current smokers, high ER ratio was associated with high smoking intensity (OR= 1.19).[623] Radi et al showed that higher ER ratio was associated with higher smoking intensity among current smokers in women, but not in men in an Australian cross–sectional study (n= 1,101).[624] However, Ota et al found that ER ratio did not significantly predict smoking cessation at 2–year follow-up in 1,423 middle-aged men in Japan.[625] Thus, my finding provides further evidence for longitudinal associations of ERI with smoking outcomes in a population–based study.

In terms of work stress and dietary outcomes, this thesis found that higher ER ratio was cross–sectionally associated with less healthy diet (HDI) after adjustment for covariates in both sexes. While there are inconsistent associations between ER ratio and individual HDI components (9 nutrient/food intakes), which may reflect sex or individual differences in dietary responses to work stress, the overall effects of ER ratio on HDI (the sum of 9 nutrient/food intakes) appeared more robust. This finding implies that high ER ratio is associated with poor diet quality linked to the risks of chronic diseases. My finding is generally in line with previous literature on work stress and diet. Many studies have supported the links between the DC model and dietary outcomes (see Section 2.3.3).[626,627,628] To my best knowledge, this cross–sectional analysis is the first study to demonstrate the links between the ERI model and dietary outcomes. Moreover, the measurements of dietary outcomes varied considerably between previous studies on the DC model and diet. The method of diet quality takes into account intakes of various foods and nutrients, thereby providing more accurate pictures of diet than single food/nutrient intake.[629] Thus, this thesis adopting the outcome of diet quality can offer more solid evidence for the associations between work stress and diet.

8.3.2 Overcommitment personality and health behaviours

The associations of OC personality with three health behaviours were examined

in this thesis. Firstly, higher OC at wave 1 was associated with higher levels of drinking outcomes (heavy drinking, binge drinking and problem drinking) at wave 2 after adjustment for covariates in both sexes, except non-significant association between OC and heavy drinking in women. Secondly, higher OC at wave 1 was associated with higher levels of smoking outcomes (smoking status) at wave 2 after adjustment for covariates, but the association between OC and smoking intensity did not reach statistical significance in both sexes. Thirdly, higher OC was cross–sectionally associated with less healthy diet (HDI) after adjustment for covariates in both sexes. The associations of OC with individual HDI components were inconsistent, but the overall effects of OC on HDI appeared remarkable.

There has been very limited literature regarding the effect of or the potential role of OC (main or modifying effect) on health behaviours. Importantly, two studies from Japan and Australia have reported negative findings on main effects of OC on smoking outcomes; however, modifying effect of OC was not examined.[630,631] Although this thesis is probably the first to show the effect of OC on health behaviours, there have been many studies supporting the effects of Type A behaviour and related personality constructs (Neuroticism and Hostility) on health behaviours (for more details, see Section 2.4.2),[632,633] together with numerous studies supporting the effects of PC on health behaviours (see Section 2.4.4).[634,635] As the origins of OC are traced to Type A behaviour and PC, the aforementioned studies can be used to partially support the links between OC and health behaviours identified in this thesis.

8.3.3 Potential role of OC in relationship between ERI and health behaviours

The potential role of OC in ERI–outcome relationship was examined in the thesis, including modifying, antecedent, mediator or direct effects. The potential role of OC in ERI–outcome relationship was originally suggested as main or modifying effect by Siegrist.[636] The review by Van Vegchel et al found that main effect of OC was

supported in 17 of 27 studies (63%), but modifying effect was only supported in 3 of 12 studies (25%).[637] The role of OC in ERI–outcome relationship remains inconclusive in existing literature, and originally assumed role of OC (main or modifying effect) appears relatively simple compared to accumulated research on diverse roles of personality in stress processes. For instance, the Michigan model proposed that objective work environments influence subjective perceptions of work stress, which affect short–term psycho–biological responses, leading to long–term health problems; personality can operate at several points in the stress process, including modifying, antecedent, mediator, or direct effects.[638]

Antecedent role of OC (OC predicts ERI which subsequently affects outcome) has been supported by theories and empirical studies. Personality can influence work stress via theoretical mechanisms: selection, stressor creation, and perception.[639] Several studies have supported antecedent roles of OC and related personality constructs (e.g. Type A behaviour and Neuroticism) in the relationship between work stress and outcomes (for more details, see Section 2.5.2).[640,641] Siegrist also implied the possibility of antecedent role of OC in ERI–outcome relationship; individuals with high OC might expose themselves more often to high efforts at work, or they exaggerate their efforts beyond what is formally needed, thereby resulting in continued imbalance between high effort and low reward.[642]

Mediator role of OC (ERI predicts OC which then affects outcome) appears not impossible based on theories and empirical studies. The meta-analysis of 92 longitudinal studies found that personality continues to change throughout adulthood but only modestly after age 50.[643] Several studies supported mediator roles of OC and related personality in the relation between work stress and outcomes (For more details, see Section 2.5.3).[644,645] Notably, there may be bidirectional relationship between personality and work environment across life course; antecedent and mediator roles of OC might coexist (see Section 2.5.4).[646]

In the path analyses in drinking and smoking outcomes, I found that OC and ERI

may have bi–directional relationship; the effect of OC on ERI is stronger than the other causal direction in the middle-aged and older populations. Antecedent role of OC in the relation between ERI and health behaviours is significant, but mediator role of OC is not. Finally, modifying role of OC in ERI–outcome relationship is not significant.

In my analyses, the traditional approach (logistic regression) initially found "main effects" of OC on health behaviours. Similarly, the review by Van Vegchel et al supported "main effects" of OC in 63% of studies. However, previous evidence may not really support main effects in a more critical appraisal, as possibilities of antecedent and mediator roles had not been tested simultaneously. Confirmation of OC–outcome relationship is just a first step for either OC–ERI–outcome (OC as antecedent) or ERI–OC–outcome (OC as mediator) causal chains in mediation analysis.[647] It is possible that previously reported "main effect" of OC actually represents the "snapshot" of dynamic relationship between OC, ERI and outcomes.

Bidirectional relationship between OC and ERI was found in this thesis; OC at wave 1 predicted ER ratio at wave 2, and ER ratio at wave 1 predicted OC at wave 2. To my best knowledge, this cohort study is the first to demonstrate the bidirectional relationship between OC personality and work stress in the ERI model; this finding corresponds to increased recognition for bidirectional relationship between personality and environment across life course.[648] Social environments in childhood and adulthood (e.g. workplace) may alter an individual's personality; conversely, personality influences an individual to select, create and perceive environmental stressors.[649,650] High OC personality may perceive, select and create high levels of ERI work stress, which further aggravate their vulnerable personality, resulting in a vicious circle. Thus, intervention should focus on both work environment and person in order to disrupt the cumulated effects in the reciprocal relationship.

This thesis also found that the effect of OC personality on work stress is much stronger than the other causal direction in this sample aged 45–69. As noted previously, the meta-analysis reported that personality changes only modestly after age 50.[651]

The effect of work stress on personality appeared significant but modest in the middle-aged and older populations, but this effect is expected to be stronger if younger populations would be examined. Although bidirectional relationship between OC and ERI may exist, only antecedent role of OC, not mediator role, reached statistical significance; this finding that OC acts mainly as an antecedent in ERI–outcome relationship is crucial for the implications discussed later.

8.3.4 Perceived control and health behaviours

The associations of PC with three health behaviours were examined in this thesis. Firstly, higher PC at wave 1 was associated with lower levels of drinking outcomes (heavy drinking, binge drinking and problem drinking) at wave 2 after adjustment for covariates in both sexes. Secondly, higher PC at wave 1 was associated with lower levels of smoking outcomes (smoking status and smoking intensity) at wave 2 after adjustment for covariates in both sexes. Thirdly, higher PC was cross–sectionally associated with more healthy diet (HDI) after adjustment for covariates in both sexes.

The findings in this thesis are consistent with previous studies, which have extensively supported that PC and its components (e.g. self-efficacy or PBC in TPB) can predict health behaviours – drinking, smoking, poor diet and physical inactivity (for more details, see Section 2.4.4).[652,653,654] In fact, self-efficacy is the common construct across all Social Cognitive Models – the most commonly used theories in predicting health behaviours at intrapersonal level.[655] Moreover, PC is suggested to influence various health outcomes directly by psychobiological processes and indirectly via health behaviours (for more details, see Section 2.4.3).[656,657]

8.3.5 Potential role of PC in relationship between OC, ERI and health behaviours

By the Transactional Model of Stress, the potential role of PC is considered in the

relationship between ERI, OC and health behaviours. When a person faces a stressor, one would evaluate potential threat (primary appraisal – perceived work stress ERI) and one's ability to alter the situation and manage negative emotion (secondary appraisal – PC). Both cognitive appraisals affect one's coping efforts (problem management and emotional regulation) and then influence health behaviours.[658] This model has been enriched by accumulating literature on personality psychology; personality traits (e.g. OC) are suggested to influence the transactional process at several points – primary appraisal, secondary appraisal and coping efforts (for more details, see Section 2.6.1).[659]

In Transactional Model of Stress, Lazarus suggested that primary appraisal and secondary appraisal engage in a dynamic and reciprocal relationship with each other.[660] The potentially bidirectional relationship between perceived work stress (primary appraisal) and PC (secondary appraisal) has been supported by two types of empirical evidence: (1) PC acts as a mediator in the effects of work stress on outcomes; (2) work stress acts as a mediator in the effects of PC on outcomes (for more details, see Section 2.6.2).[661,662] On the other hand, the interaction between work stress and PC has sometimes been supported in previous studies, and this possibility was tested in this thesis (for more details, see Section 2.6.3).[663,664]

In this thesis, path analyses were conducted for potential relationship between OC, ERI, PC and health behaviours. The results showed that both ERI and PC partially mediated the effects of OC on health behaviours. Additionally, ERI and PC were negatively associated with each other; ERI and PC may have bi–directional relationship, but causal directionality cannot be established in the cross–sectional study. Finally, PC had no significantly modifying role in the relationship between ERI and health behaviours.

Previous analyses for the potential role of OC in ERI–outcome relationship found significant antecedent role of OC; high OC persons tend to select, create, and perceive high levels of ERI works stress. Transactional Model of Stress provides another

explanation for the effect of OC personality on health behaviours; high OC persons tend to perceive high levels of ERI work stress (primary appraisal) and feel low levels of PC to alter the situation (secondary appraisal); both cognitive appraisals affect their use of emotion–focused coping (e.g. engaging in risky health behaviours). Moreover, high PC might decrease ERI work stress, and vice versa. Cognitive appraisal appears essential to understand the dynamic stress processes.[665] Whether the influences come from environment (workplace) or person (personality), primary and secondary appraisals are main cognitive processes associated with subsequent coping outcomes. Thus, both cognitive appraisals can serve as modifiable targets by treatments such as cognitive–behavioral therapy.[666]

Work stress research has a long and rich history of identifying work factors potentially causing stress, but individual differences have not been paid enough attention. The ERI model addressed individual differences by OC personality, but the mechanisms via which OC can influence stress processes remained unclear. Transactional Model of Stress provided mediating pathways linking environment and personality to health, thereby recognizing possible mechanisms of individual differences (personality, cognitive appraisal, and coping). This thesis attempts to integrate the ERI model with Transactional Model of Stress in order to enrich work stress research by psychological literature and to identify potential causal pathways in guiding effective interventions for work stress in the future.

Chapter 9. Implications for Research, Practice and Policy

Implications for research, practice and policy are proposed based on the three main findings in the thesis: (1) associations between work stress (ERI) and health behaviours; (2) potential role of OC in the relation between ERI and health behaviours; (3) potential role of PC in the relation between OC, ERI and health behaviours.

Published evidence of organisation interventions to reduce work stress has yielded mixed findings so far; most interventions focused on changing objective work characteristics, rather than personality factors.[667] My findings showed that personality constructs (OC and PC) can play active and crucial roles in the relationship between work stress and health behaviours, thereby providing potential targets for interventions. Thus, I propose several types of interventions for work stress, personality and health behaviours, including: organisational interventions for health behaviours and the ERI model, individual intervention targeting mechanisms via which OC influences work stress, and stress management intervention for the ERI model. Finally, a multiple–level and integrated approach combining organisational intervention for work stress and individual intervention for vulnerable personality is recommended to improve health behaviours at workplace.

9.1 Associations between Work Stress and Health Behaviours

In this thesis using prospective data, I found that high work stress defined by the ERI model was associated with worse profiles of health behaviours. Based on the following literature, I propose that work stress should be a main target for organisational intervention designed to improve health behaviours; organisational intervention based on the ERI model appears a promising approach to reduce employees' work stress.

9.1.1 Organisational intervention for health behaviours

During the 1980s and 1990s, most interventions for health behaviours were grounded in psychological theories for behaviour change, including perceived control. However, such individual interventions did not address the upstream social contexts influencing health behaviours. Increased recognition that prevention requires efforts beyond the individual level resulted in the development of community interventions at workplace or school.[668] Workplace has emerged as an important medium for delivering behaviour change interventions. Health behaviours such as smoking, diet and physical activity have been targeted through organisational interventions.[669,670,671] For instance, workplace may offer smoke–free office buildings, smoking cessation classes, facilities for physical activity, and healthy foods in cafeterias.

In the earlier organisational interventions, employees' health behaviours were treated as factors unrelated to occupational hazards. However, evidence showed that occupational hazards influenced employees to adopt and maintain risky health behaviours.[672] Thus, Sorenson et al tested an integrated intervention to reduce exposure to occupational hazards and to improve health behaviours as opposed to another intervention that only focused on health behaviours; the rate of smoking cessation in the integrated program was twice as high as that in another program.[673] In fact, this integrated model addressing both occupational hazards and health behaviours is now the prevailing approach for workplace health promotion.[674]

Since the impacts of works stress (defined by the DC model and the ERI model) on health behaviours have been repeatedly reported in empirical studies and confirmed in this thesis, organisational interventions for health behaviours should also address the potential occupational exposures – work stressors – in order to increase the opportunity of successful changes in health behaviours.

9.1.2 Organisational intervention for the ERI model

Based on the ERI model, restoring the balance between effort and reward at work is considered the best intervention strategy. Tsutsumi and Kawakami proposed an approach of organisational intervention for the ERI model. In terms of extrinsic effort, interventions can focus on reduction of overtime work and long working time, even distribution of workload and responsibility among employees, and provision of holidays and sufficient rest time. In terms of reward, social skill training can improve supervisors' leadership behaviours in praising employees' good performance and providing support for employees, resulting in increased esteem reward. Introduction of additional benefits for employees such as welfare facilities or recreational facilities can increase non–monetary reward. Provision of vocational training and steps for promotion might ensure employees' sense of job security.[675]

Meta-analyses of organisational interventions to reduce work stress have yielded mixed findings so far.[676,677] Montano et al reviewed 39 studies of organisational interventions based on various work stress models. The interventions frequently reporting significant effects included: reduction of workloads and rotation schedules, improvement of communication between workers and supervisors, introduction of employee training, or improvement in material conditions; these strategies were quite similar to Tsutsumi and Kawakami's approach based on the ERI model, and they can be adopted to design organisational interventions.[678] Importantly, there have been several studies reporting that the organisational interventions based on the ERI model appeared effective and promising.[679,680,681]

9.2 Potential Role of OC in Relationship between ERI and Health Behaviours

This thesis found that OC and ERI have bi–directional relationship; antecedent role of OC in ERI–outcome relation is significant, but mediator role of OC is not. Antecedent role of OC personality in the relation between work stress and health

behaviours appears crucial for clinical practice, but personality is often neglected in interventions to reduce work stress. Based on the following literature, I propose individual intervention targeting cognitive–behaviour mechanisms via which OC may influence work stress.

9.2.1 Cognitive behaviour therapy and personality

Cognitive behaviour therapy (CBT) is one form of psychotherapy that has been empirically tested in many clinical trials for different psychiatric diseases such as depression, anxiety or personality disorders.[682] The cognitive model describes how people's perceptions or thoughts about situations influence their emotional and behavioural reactions. CBT uses a wide range of techniques to help individuals change cognitive appraisal of stressors and their coping responses. Cognitive restructuring encourages individuals to become aware of negative thoughts or irrational beliefs, to recognize distortion and irrationality in thought processes, and to substitute positive thoughts or rational beliefs. In addition, behavioural techniques are used to challenge specific dysfunctional beliefs or to change coping responses, including behavioural rehearsal, modelling, relaxation training, and time management.

In cognitive psychology, personality is conceptualized as a relatively stable organisation composed of schemas (responsible for the sequence from selecting and synthesizing stimulus to a behavioural response) and modes (network of cognitive, affective, and motivational components that organize response patterns).[683] The goal of CBT for personality disorder is to decrease valence of dysfunctional schemas (e.g. irrational beliefs) and to strengthen availability of benevolent schemas (e.g. rational beliefs); the patient gradually reinterprets schemas and modes in a more functional way. However, considerably more time and effort are required to produce changes in personality than depression or anxiety.[684]

Despite extensive support on CBT for personality disorders, there has been no

literature on CBT targeting OC personality itself. Nevertheless, several intervention studies have targeted “cognitive–behaviour mechanisms” via which OC and related personality (Hostility or Type A behaviour) can influence work stress and health (see Section 9.2.2). Although it is not easy to induce strong changes in personality itself by an intervention, it appears rather practical to change individual’s specific tendency in cognition and behaviour.

9.2.2 Individual intervention targeting cognitive–behaviour mechanisms via which personality influences work stress

A meta-analysis from 36 intervention studies to reduce work stress found that CBT consistently produced larger effects than other individual interventions.[685] Several intervention studies have targeted “cognitive–behaviour mechanisms” via which OC and related personality can influence work stress and health. For example, Aust, Peter, and Siegrist conducted a 12–week intervention program in bus drivers; this program included 90–min sessions of self–observation for perception of arousal, relaxation training, management of conflict with supervisors, and coping with anger. The mean OC levels significantly reduced in the intervention group, and the effects persisted after 3 months.[686] William and William reported that CBT reduced adverse effects of Hostility on stress; hostile people tend to interpret neutral situations as threatening and become angry easily. The program included early recognition of angry feelings, cognitive restructuring for negative thoughts, relaxation training, and communication skill training.[687] Furthermore, several interventions have targeted similar cognitive–behaviour mechanisms to change negative effects of Type A behaviour on health.[688,689]

This thesis is probably the first to support antecedent role of OC personality in the relation between work stress (ERI) and health behaviours based on a 2–wave cohort study, thereby providing fundamental basis and further support for practical applications of CBT targeting cognitive–behaviour mechanisms. Based on theories,

future interventions are suggested to target the following “cognitive–behaviour mechanisms” via which OC may influence work stress: (1) Perception: individual’s cognitive appraisals of stressful situation – mismatch between effort and reward can be changed by cognitive restructuring. (2) Selection: high OC persons may select themselves into stressful jobs or tasks; unrealistic high goal can be changed by cognitive restructuring, and time management would help. (3) Stressor creation: high OC persons may create real work stressors for themselves by conflicting with colleagues and by anxiety on time pressure; social skill training and relaxation training would help. (4) Reaction: high OC persons might react exaggeratedly to work stressors in their psycho–biological processes and health behaviours; behavioural therapy targeted at coping efforts would be beneficial.[690,691] To match individual need, the intervention can be designed by evaluating one’s personality traits and specific cognitive–behaviour mechanisms before the CBT.

9.3 Potential Role of PC in Relation between OC, ERI, and Health Behaviours

This thesis attempted to link the ERI model with Transactional Model of Stress by demonstrating the relationship between OC (personality), ERI (primary appraisal), PC (secondary appraisal), and health behaviours (coping effort and outcome). By the following review, I suggest that stress management intervention can be applied to the ERI model by their common theoretical platform – Transactional Model of Stress.

9.3.1 Stress management intervention and Transactional Model of Stress

A stress management intervention (SMI) is a program initiated by an organisation that focuses on reducing work stressors or on assisting individuals to minimize negative outcomes of exposure to work stressors.[692] A SMI can be implemented in the form of individual intervention or organisational intervention. Despite extensive

application in organisational settings, SMIs were criticized by lack of theoretical basis.[693] Since the 1990s, SMIs have been highly influenced by Transactional Model of Stress, which provided a theoretical platform for the design of interventions.[694] Thus, contemporary SMI programs often target three points in stress processes: the intensity of work stressors, the cognitive appraisal of stressful situations, and the ability to cope with stressful situations.[695]

The components of SMI encompass a broad array of treatments. Cognitive behaviour therapy (CBT) – the most effective component in SMI – is intended to change individuals' cognitive appraisal of stressful situations and coping responses. Meditation and relaxation interventions – the most popular components adopted in 69% of SMIs – are designed to reduce employees' adverse reactions to stress by inducing psychological and physiological status opposite to stress reactions.[696] Time management interventions are designed to help employees manage time when working on multiple tasks. Time management provides skills training in defining one's goals to achieve in a specified time period, prioritizing tasks to ensure that important ones receive attention, self–monitoring, and problem solving.[697]

9.3.2 Stress management intervention for the ERI model

This thesis is probably the first study showing that the ERI model can be integrated well with Transactional Model of Stress; the path analyses demonstrated the relationship between OC (personality), ERI (primary appraisal), PC (secondary appraisal), and health behaviours (coping effort and outcome). Thus, it is plausible that SMI can be applied to design interventions for the ERI model by their common theoretical platform – Transactional Model of Stress.

In fact, one intervention study have adopted the SMI based on the ERI model. Limm et al conducted a randomized controlled trial to test the effectiveness of a SMI based on the ERI model in 174 German managers.[698] The SMI was conducted by a

group–orientated prevention program, including: (1) to foster awareness of and insight into stress situations at workplace – high effort and low reward; (2) to provide tools to cope with stressful situations such as work overload, social conflicts, negative emotion, or failure at work; (3) individual resources were promoted in group processes. The SMI was found to reduce perceived stress reactivity, sympathetic activation, and ER ratio; these effects persisted for 1 year. Although this SMI targeted primary appraisal and coping effort, other potential pathways in Transactional Model of Stress (e.g. secondary appraisal or personality) were not incorporated. To change potential pathways in Transactional Model of Stress in a more significant way, future research can incorporate more treatment components into SMIs.

Note that the interventions to enhance PC (secondary appraisal) have often been adopted in individual interventions for health behaviours rather than SMIs. These interventions were proposed by Social Cognitive Models, with techniques such as changing existing beliefs, introducing new beliefs, role modelling, or enactive mastery experience.[699,700] As my findings implied that PC might change causal pathways in Transactional Model of Stress, the intervention to enhance PC can be adopted as a SMI component in future research.[701]

9.4 A Multi–Level and Integrated Perspective for Psychosocial Factors at Work and Health Behaviours

I propose several types of organisational and individual interventions for work stress, personality and health behaviours in future research and hopefully in practice if results are favourable. My findings of bidirectional relationship between ERI and OC imply that interventions should focus on both work environments and individuals in order to disrupt the cumulated effects in the reciprocal relationship.

Okechukwu et al in Harvard School of Public Health proposed that it is critical to address the “dualism” of individual versus organisational approaches to intervention

design and delivery; they argued that focusing on impact and reach is more useful than putting individual and organisational approaches against each other.[702] Individual interventions and organisational interventions are complementary; individual interventions are effective at individual–level outcomes such as health behaviours and health outcomes, but organisational interventions have favorable impacts at organisational–level outcomes such as reducing exposure in working conditions. Thus, LaMontagne et al suggested that superior results would be expected from combining individual and organisational interventions over a single type.[703]

Mellor et al in the Health and Safety Laboratory UK suggested that tackling the impacts of psychosocial factors at work on health should be considered from a multi–level perspective (interplay between work factors and individual differences); multi–level interventions combining organisational and individual interventions had the strongest effects on health. Given that many employees' diseases are often linked to health behaviours, management of psychosocial factors at work needs to be integrated with health promotion to improve health behaviours; this integration has been recommended by public health authorities like the World Health Organisation.[704]

Based on my evidence, the best strategy for addressing psychosocial factors at work and health behaviours is a multi–level and integrated perspective combining organisational and individual interventions. My opinions for the multi–level interventions are that, first, organisational interventions for work stress and health behaviours can be implemented if organisational resources are available; second, for identified individuals with personality vulnerable to work stress, individual interventions targeting cognitive–behaviour mechanisms or stress management interventions for the ERI model can be adopted according to individual needs.

Chapter 10. Conclusions

This thesis examines the relationship between OC, ERI and health behaviours, together with the relationship between OC, ERI, PC and health behaviours, through a two–wave cohort study for drinking and smoking outcomes (n= 7513) and a cross–sectional study for dietary outcomes (n= 11012) conducted in the middle–aged and older populations in Central and Eastern Europe.

The results of this thesis are summarized as follows. First, higher ER ratio (work stress) was associated with higher levels of drinking (heavy drinking, binge drinking and problem drinking), higher levels of smoking (smoking status and smoking intensity), and less healthy diet (diet quality) after adjustment for covariates. Second, higher OC score was generally associated with higher levels of drinking, higher levels of smoking, and less healthy diet after adjustment for covariates. However, these associations of OC with health behaviours existed but did not reach statistical significance in heavy drinking in women and in smoking intensity in both sexes.

Third, the potential role of OC in the relationship between ERI and health behaviours was examined, including modifying, antecedent, mediator, and direct effect of OC. I found that OC and ERI may have bi–directional relationship, but the effect of OC on ERI was stronger than the other direction in the middle-aged and older populations. Thus, antecedent role of OC in the relationship between ERI and health behaviours was found significant, but mediator role of OC was not. Direct effect of OC on health behaviours was not significant. Finally, OC had no significantly modifying effect in the relationship between ERI and health behaviours.

Fourth, lower PC was associated with higher levels of drinking, higher levels of smoking, and less healthy diet after adjustment for covariates. Fifth, the potential role of PC in the relationship between OC, ERI and health behaviours was assessed based on Transactional Model of Stress. I found that both ERI and PC partially mediated the effects of OC on health behaviours. ERI and PC were negatively associated with each

other in the cross–sectional analyses; it is possible that ERI and PC have bi–directional relationship. Finally, PC had no significantly modifying effect in the relationship between ERI and health behaviours.

Work stress research has a long and rich history of identifying those work factors potentially causing stress, but individual differences have not been paid enough attention. This thesis contributes to deeper understanding of intersecting pathways by which work stress (ERI) and personality constructs (OC and PC) jointly influence health behaviours. The ERI model can be integrated well with Transactional Model of Stress, which provides potential mechanisms to explain individual differences (personality, cognitive appraisal, and coping). Investigating psychosocial mechanisms may help to identify a broad set of intervention opportunities; I propose that the next steps are to develop, implement and evaluate several types of interventions for work stress, personality and health behaviours in order to translate my findings into practice. To further clarify the relationship between work stress, personality and health behaviours, future research should recruit samples from wider cultural bases and younger populations with at least three waves of data.

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Appendix 1.

Personal questionnaire in the HAPIEE study wave 1

The health consequences of the profound social and economic changes in our country that have started in 1989 are not well understood. The present study has been set up to investigate and monitor the health impacts of these changes.

The study has two principal components:

a) An assessment of your present health and your other characteristics that do or can influence health; this will be done by a questionnaire and a short medical examination, including a blood sample.
b) Monitoring the changes in your health in the future; this will be done by a short annual postal questionnaire and by using data available in national health statistics registers.

We would like to assure you that all your personal data will be kept confidential and will not be available to any individuals or institutions except the core investigators of this study. Any published results will not identify individuals.

If you agree to participate in the study, you can change your mind and leave the study at any time in the future without giving any reason for your withdrawal. You will be asked for another specific consent before we take a blood sample.

Do you agree to participate in the study and complete the questionnaires? *Please circle Yes or No.*

Yes No

Do you give your consent for us to use data on major changes in your health that are contained in national health statistics? *Please circle Yes or No.*

Yes No

To be able to use national data on major changes in your health, we need your ID number. Please enter your ID number. _____ / _____

Please date and sign this form below.

Signature of participant : _____

Print name: _____

Date: _____

Thank you for your time.

1. Place of birth (region): _____

2. Sex:

1. Male
2. Female

3. What is your highest completed level of education?

1. Incomplete primary or no formal education
2. Primary
3. Vocational (apprenticeship)
4. Secondary
5. University (degree)

4. What is your marital status?

1. Single
2. Married
3. Cohabiting
4. Divorced
5. Widowed

About your health

5. What is your height in cm? ☐☐☐ . ☐

6. What is your weight in kg? ☐☐☐ . ☐

7. Over the last 12 months, would you say your health has been:

1. Very good
2. Good
3. Average
4. Poor
5. Very poor

8. Here is a list of activities that you might do during a typical day. Does your health now limit your ability in these activities? If so, how much?

	Yes, limited a lot	*Yes, limited a little*	*No, not limited at all*
Vigorous activities, such as running, lifting heavy objects, participating in strenuous sports	☐ 1	☐ 2	☐ 3
Moderate activities, such as moving a table, pushing a vacuum cleaner	☐ 1	☐ 2	☐ 3
Lifting or carrying bag of groceries	☐ 1	☐ 2	☐ 3
Climbing several flights of stairs	☐ 1	☐ 2	☐ 3
Climbing one flight of stairs	☐ 1	☐ 2	☐ 3
Bending, kneeling or stooping	☐ 1	☐ 2	☐ 3

	Yes, limited a lot	*Yes, limited a little*	*No, not limited at all*
Walking two kilometres	☐ 1	☐ 2	☐ 3
Walking one kilometre	☐ 1	☐ 2	☐ 3
Walking one hundred metres	☐ 1	☐ 2	☐ 3
Bathing and dressing yourself	☐ 1	☐ 2	☐ 3

9. Do you have any long-term health problems for which medical treatment has been sought over last 12 months?

1. Yes
2. No

10. Have any of the following diseases ever been diagnosed in you <u>by a doctor</u> and have you ever been <u>hospitalised</u> for this disease?

	Yes, diagnosed and hospitalised	*Yes, diagnosed, never hospitalised*	*No or do not know*
heart attack / acute myocardial infarction	☐ 1	☐ 2	☐ 3
angina / ischaemic heart disease	☐ 1	☐ 2	☐ 3
Stroke	☐ 1	☐ 2	☐ 3
chronic respiratory disease	☐ 1	☐ 2	☐ 3
Cancer	☐ 1	☐ 2	☐ 3
stomach ulcer	☐ 1	☐ 2	☐ 3
gallbladder disease	☐ 1	☐ 2	☐ 3
kidney stones	☐ 1	☐ 2	☐ 3
Asthma	☐ 1	☐ 2	☐ 3
atopic eczema	☐ 1	☐ 2	☐ 3
other allergy	☐ 1	☐ 2	☐ 3
hay fever	☐ 1	☐ 2	☐ 3
disease of spine or joints	☐ 1	☐ 2	☐ 3

11. Do you usually **cough** on most days for as much as **3 months each year?**

1. Yes
2. No

12. Do you usually bring up any **phlegm** from your chest first thing in the morning for as much as **3 months each year**?

1. Yes
2. No

Injuries and accidents

13. In the past **12 months** have you been injured or have you had an accident serious enough to contact a doctor?

1. Yes
2. No, please go to the **question 20**

14. How many different times in the past **12 months** were you injured or have you had an accident serious enough to contact a doctor?

Please would you **tell us** about the **MOST SERIOUS INJURY OR ACCIDENT** you have had in **last 12 months.**

15. Place: Where were you when you were injured or had your accident?

1. Home (yours or someone else's home)
2. Work
3. Road
4. Other
5. Unknown

16. Mechanism: How were you hurt or how was the injury inflicted?

1. Traffic injury
2. Fall
3. Other blunt force
4. Stab or cut
5. Firearm
6. Fire or hot subject or substance (e.g. scald)
7. Chocking or hanging
8. Drowning or submersion
9. Suffocation
10. Poisoning
11. Machinery related
12. Struck by or against
13. Other
14. Unknown

17. Intent: Was this accident:

1. Unintentional
2. Self-harm
3. Intentional
4. Other
5. Unknown

18. What was the **nature of your injury?**

1. Fracture
2. Sprain or strain
3. Cut, bite or open wound
4. Bruise
5. Burn
6. Concussion
7. Organs system injury
8. Other – please specify
9. Unknown

_____

19. Did you require **medical treatment** as a result of your injury or accident?

1. No treatment required
2. Treated as outpatient, discharged
3. Admitted to hospital
4. Other – please specify
5. Unknown

_____

20. Have you ever been told by a doctor that you have **high blood pressure**?

1. Yes
2. No

If **YES**, have you been taking drugs for high blood pressure in the last 2 weeks?

1. Yes
2. No
3. Don't know

21. Have you every been told by a doctor that you have **diabetes**?

1. Yes
2. No

If **YES**, how are you treated?

1. Only by diet
2. By diet and insulin
3. By diet and tablets
4. By diet, tablets and insulin
5. No treatment

22. Have you ever been told by a doctor that you have **high blood cholesterol**?

1. Yes
2. No

If **YES**, how are you treated?

1. Only by diet
2. By diet and tablets
3. Tablets only
4. No treatment

23. Are you under **long-term treatment or medical care** for any medical condition, except for high blood pressure, high cholesterol or diabetes?

1. Yes
2. No

If **YES**, please give details:

_____

24. Do you take any **vitamins or mineral supplements**?

1. Yes (regularly, at least 3 times per week)
2. Yes (irregularly, less than 3 times per week)
2. No

25. If YES, do these supplements contain vitamin C?

1. Yes
2. No

26. Can you **seek medical advice** when you need it?

1. Anytime I want to and without any difficulty
2. Usually, but it can be complicated e.g. difficult to get to doctor, doctor busy, or can't
3. Not usually, too complicated and often I do not bother
4. No, it is either too difficult to get to the doctor, the doctor is too busy, or it is too expensive

27. Where do you go, when you want medical advice and it is not an emergency?

1. State funded general practitioner
2. State funded specialist
3. State hospital
4. Private general practitioner
5. Private funded specialist
6. Private hospital
7. Other

28. Do you have to **pay** to see the doctor?

1. Yes
2. No

29. At any time in the last 6 months, have you been prescribed a medicine and not been able to buy it?

1. No, I can always obtain the medicines that I need
2. Yes, it was unavailable
3. Yes, it was too expensive
4. No, I have not been prescribed any medicines

30. How many times in the **last 12 months** did you **seek medical advice?** ☐☐

31. Did any of your **parents or siblings suffer** from any of the **following diseases?**

	Did parents or siblings suffer from disease?		**IF YES**, did a parent or sibling have onset before the age of 60?	
	Yes	No	Yes	No
Heart disease (infarction, angina)	☐ 1	☐ 2	☐ 1	☐ 2
Stroke	☐ 1	☐ 2	☐ 1	☐ 2
Diabetes	☐ 1	☐ 2	☐ 1	☐ 2
Neoplasms	☐ 1	☐ 2	☐ 1	☐ 2
Allergy	☐ 1	☐ 2	☐ 1	☐ 2

32. Have you ever had any pain or discomfort in your chest?

1. Yes
2. No

If no, please, **women** proceed to **Question 39**, **men** proceed to **Question 45**.

33. Do you get it when you walk uphill or hurry or do physically demanding work?

1. Yes
2. No
3. Never hurries or walks uphill or does physically demanding work

34. Do you get it when you walk at an ordinary pace on the level?

1. Yes
2. No

35. What do you do if you get it while you are walking?

1. Stop or slow down
2. Carry on at the same pace
3. Take nitroglycerine

36. If you stand still, what happens to it?

1. Relieved
2. Not relieved

37. If relieved, how soon?

1. 10 minutes or less
2. More than 10 minutes

38. Can you specify where such pain or discomfort appeared? (*Please choose all appropriate options)*

1. Sternum (upper or middle)
2. Sternum (lower)
3. Left anterior chest
4. Left arm
5. Neck
6. Other Please specify: _____

Only for women

39. Do you still have periods?

1. Yes, regularly
2. Yes, irregularly **If YES**, go to question 42.
3. No

40. How old were you when the periods stopped? | | | Years

41. What was the cause of the menopause?

1. Natural
2. Surgical (operation)

42. Have you ever used hormonal contraception?

1. No, never
2. Yes, but I no longer use it
3. Yes and I still use it

43. Have you ever had hormonal replacement therapy?

1. Yes
2. No

44. If YES, are you still taking hormonal replacement therapy?

1. Yes
2. No

Health behaviours

45. How many hours during a typical week, except when at work, do you engage in physically demanding activities, such as housework, gardening, maintenance of the house (DIY) etc? | | | |

46. How many hours during a typical week do you engage in sports, games or hiking? | | | |

47. Do you smoke cigarettes?

1. Yes, regularly, at least one cigarette a day on average
2. Yes, occasionally, less than one cigarette a day
3. No, I smoked in the past but I stopped
4. No, I have never smoked

48. For current and past smokers: How many cigarettes a day do you smoke now (or you used to smoke, if you stopped)? | | | |

49. For current and past smokers: How old were you when you started smoking? | | | Years

50. For past smokers: How old were you when you stopped smoking? | | | Years

51. For past smokers: When did you stop smoking? | | | | | Calendar year

52. The next few questions are about how much wine, beer and spirits you may have had during the last 12 months. When we say one drink, we mean 0.5 litre of beer, 2 dl glass of wine, or 5 cl of spirits. Please answer each question below - ie. cross a square **in each row -** to indicate how often you had that amount of alcohol during one day.

Here is an example how to calculate correct amount of alcohol on a single occasion: if you had 0.7 l bottle of wine AND two 5cl measures of spirit in a single occasion you had 3.5 drinks of wine and 2 drinks of spirit which is a total of 5.5 drinks. Then you need to choose correct column to indicate how often in the last year you had such amount of alcohol.

	Every day or almost every day	***3-4 per week***	***1-2 per week***	***2-3 per month***	***About once a month***	***6-11 in past year***	***3-5 in past year***	***1-2 in past year***	***Never in past year***
1. How often in the last year did you have 10 drinks or more during one day?									
10 drinks or more 5 l (10 x 0.5 l) of beer or 2 l (10 x 2 dl) of wine or 0.5 l (10 x 5 cl) of spirits	☐	☐	☐	☐	☐	☐	☐	☐	☐
2. How often in the last year did you have 7-9 drinks during one day?									
7-9 drinks (7-9 x 0.5 l of beer or 7-9 x 2 dl of wine or 7-9 x 5 cl of spirits)	☐	☐	☐	☐	☐	☐	☐	☐	☐
3. How often in the last year did you have 5-6 drinks during one day?									
5-6 drinks (5-6 x 0.5 l of beer or 5-6 x 2 dl of wine or 5-6 x 5 cl of spirits)	☐	☐	☐	☐	☐	☐	☐	☐	☐
4. How often in the last year did you have 3-4 drinks during one day?									
3-4 drinks (3-4 x 0.5 l of beer or 3-4 x 2 dl of wine or 3-4 x 5 cl of spirits)	☐	☐	☐	☐	☐	☐	☐	☐	☐
5. How often in the last year did you have 1-2 drinks during one day?									
1-2 drinks (1-2 x 0.5 l of beer or 1-2 x 2 dl of wine or 1-2 x 5 cl of spirits)	☐	☐	☐	☐	☐	☐	☐	☐	☐
6. How often in the last year did you have about half drink during one day?									
About half drink	☐	☐	☐	☐	☐	☐	☐	☐	☐
	Every day or almost every day	***3-4 per week***	***1-2 per week***	***2-3 per month***	***About once a month***	***6-11 in past year***	***3-5 in past year***	***1-2 in past year***	***Never in past year***

53. How much beer (litres) do you usually drink during one week? ☐☐

54. How much wine (decilitres) do you usually drink during one week? ☐☐

55. How much spirits (decilitres) do you usually drink during one week? ☐☐

56. What was the largest amount of alcohol you had **on a single occasion** during the last 4 weeks?

0.5 L bottles or glasses of beer AND

2 dl glasses of wine AND

5 cl glasses of spirits (double shots)

57. During the **last 12 months**, how often did you drink enough to feel drunk?

1. every day or at least 5 times a week
2. about 1-4 times a week
3. about 1-3 times a month
4. 3-11 times a year
5. once or twice a year
6. never in the past year

58. In **last 12 months**, did your drinking cause you difficulties with the following aspects of your life?

Please cross appropriate box in each row:	Yes	No
marriage/partner or home life	☐ 1	☐ 2
friendships and social life	☐ 1	☐ 2
your work	☐ 1	☐ 2
Police or other authorities	☐ 1	☐ 2
your physical health	☐ 1	☐ 2
any injury or accident	☐ 1	☐ 2
your psychological or mental health	☐ 1	☐ 2
your financial circumstances	☐ 1	☐ 2

59. In the **last 12 months**, did you have any of the following experiences?

Please cross appropriate box in each row:	Yes	No
Have you ever felt you should cut down on your drinking?	☐ 1	☐ 2
Have people ever annoyed you by criticising your drinking?	☐ 1	☐ 2
Have you ever felt bad or guilty about your drinking?	☐ 1	☐ 2
Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover?	☐ 1	☐ 2

60. How do the following factors influence human health?

Please cross appropriate box in each row:	Improve		No effect	Make It Worse	
	Strongly	Slightly		Slightly	Strongly
Eating meat	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Eating fruit and vegetables	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Lack of physical activity	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Obesity	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Smoking	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Drinking alcohol	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Passive smoking	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Environmental pollution	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Lack of money	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Stress	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6
Exercise	☐ 1	☐ 2	☐ 3-4	☐ 5	☐ 6

61. Below is a list of the ways you might have felt or behaved during **the last week**.
For each of the following statements, please indicate how often you felt that way:

During the past week:	*Less than one day*	*1-2 days*	*3-4 days*	*5-7 days*
a) I was bothered by things that usually do not bother me	☐ 1	☐ 2	☐ 3	☐ 4
b) I did not feel like eating, my appetite was poor	☐ 1	☐ 2	☐ 3	☐ 4
c) I felt that I could not shake off the blues even with help from my family and friends	☐ 1	☐ 2	☐ 3	☐ 4
d) I felt that I was just as good as other people	☐ 1	☐ 2	☐ 3	☐ 4
e) I had trouble keeping my mind on what I was doing	☐ 1	☐ 2	☐ 3	☐ 4
f) I felt depressed.	☐ 1	☐ 2	☐ 3	☐ 4
g) I felt that everything I did was an effort	☐ 1	☐ 2	☐ 3	☐ 4
h) I felt hopeful about the future	☐ 1	☐ 2	☐ 3	☐ 4
i) I thought my life had been a failure	☐ 1	☐ 2	☐ 3	☐ 4
j) I felt fearful	☐ 1	☐ 2	☐ 3	☐ 4
k) My sleep was restless	☐ 1	☐ 2	☐ 3	☐ 4

During the past week:	*Less than one day*	*1-2 days*	*3-4 days*	*5-7 days*
l) I was happy	☐ 1	☐ 2	☐ 3	☐ 4
m) I talked less than usual	☐ 1	☐ 2	☐ 3	☐ 4
n) I felt lonely	☐ 1	☐ 2	☐ 3	☐ 4
o) People were unfriendly	☐ 1	☐ 2	☐ 3	☐ 4
p) I enjoyed life	☐ 1	☐ 2	☐ 3	☐ 4
q) I had crying spells	☐ 1	☐ 2	☐ 3	☐ 4
r) I felt sad	☐ 1	☐ 2	☐ 3	☐ 4
s) I felt people dislike me	☐ 1	☐ 2	☐ 3	☐ 4
t) I could not get going	☐ 1	☐ 2	☐ 3	☐ 4

62. Are you **a member of club or organisation** (sports club, church, political party)?

1. Yes
2. No

If **YES**, how often do you take part in common activities?

1. Several times a week
2. Several times a month
3. About once a month
4. Several times a year
5. Never or almost never

63. On whom do you rely first of all when having problems?

1. friends
2. relatives
3. employer
4. state
5. private / commercial companies
6. public organisations such as trade unions
7. charities, church
8. no one
9. other, please give details: _____

64. Are you regularly in **contact with your relatives** who do not live in your household?

1. several times a week
2. about once a week
3. several times a month
4. about once a month
5. less than once a month
6. I do not have relatives / no relatives outside my household

65. How many relatives who do not live in your household do you **see at least once a week**?

1. none
2. 1 or 2
3. 3 to 5
4. more than 5
5. I do not have relatives / no relatives outside my household

66. How often do you visit **friends**?

1. several times a week
2. about once a week
3. several times a month
4. about once a month
5. less than once a month
6. I do not have friends

67. How many friends do you see at least once a week?

1. none
2. 1 or 2
3. 3 to 5
4. more than 5
5. I do not have friends

68. We would like to ask about your area of residence and other people:

	Always	*Mostly*	*Some-times*	*Rarely*	*Never*
Do you feel safe in the area of your residence during the day?	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Do you feel safe in the area of your residence at night?	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Would your neighbours help you if you need it?	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Is there trust among people in your area of residence?	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Do you think that you can trust people?	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

69. Have the changes since 1989 been good or bad for you:

	Very good	*Good*	*No change*	*Bad*	*Very bad*
Occupational position	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Income	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
Material circumstances	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5
General social position	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5

70. How much do you agree or disagree with the following statements?

Measurement for perceived control	DISAGREE			AGREE		
	STRONGLY	MODERATELY	SLIGHTLY	SLIGHTLY	MODERATELY	STRONGLY
a) At home, I feel I have control over what happens in most situations	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
b) Keeping healthy depends on things that I can do	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
c) There are certain things I can do for myself to reduce the risk of a heart attack	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
d) There are certain things I can do for myself to reduce the risk of getting cancer	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
e) I feel that what happens in my life is often determined by factors beyond my control	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
f) Over the next 5-10 years I expect to have many more positive than negative experiences	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
g) I often have the feeling that I am being treated unfairly	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
h) In the past ten years my life has been full of changes without my knowing what will happen next	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
i) I very often have the feeling that there's little meaning in the things I do in my daily life	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
j) I sometimes feel as if I've done all there is to do in life	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6
k) I gave up trying to make big improvements or changes in my life a long time ago	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6

Social and economic conditions

71. How often does it happen that you do not have enough **money for food** which you and your family need? And how often did this happen before 1990?

	at present		before 1990
☐	1. all the time	☐	1. all the time
☐	2. often	☐	2. often
☐	3. sometimes	☐	3. sometimes
☐	4. rarely	☐	4. rarely
☐	5. never	☐	5. never

72. How often does it happen that you do not have enough **money for clothing** which you and your family need? And how often did this happen before 1990?

	at present		before 1990
☐	1. all the time	☐	1. all the time
☐	2. often	☐	2. often
☐	3. sometimes	☐	3. sometimes
☐	4. rarely	☐	4. rarely
☐	5. never	☐	5. never

73. Do you have difficulties with **paying bills** (for housing, electricity, heating etc)? And what was the situation before 1990?

	at present		before 1990
☐	1. all the time	☐	1. all the time
☐	2. often	☐	2. often
☐	3. sometimes	☐	3. sometimes
☐	4. rarely	☐	4. rarely
☐	5. never	☐	5. never

74. Are you in receipt of any of the following benefits at the moment? *Choose all that apply.*

1. Child benefit
2. Unemployment benefit
3. Care allowance (care for invalid)
4. Widow(er)'s pension
5. Social assistance (e.g. with food, fuel, clothes or medication)
6. Others – please specify: _____
7. Do not receive any state benefits

75. How many **rooms** does your house/flat have **(excluding kitchen and bathrooms)**? ☐☐

76. How many **adults** (18 years or older) live in your house/flat? ☐☐

77. How many **children** (under 18 years old) live in your house/flat? ☐☐

78. What was the highest completed **level of education** of your **parents**?

Your father:	Your mother:
1. Incomplete primary or no formal education	1. Incomplete primary or no formal education
2. Primary	2. Primary
3. Vocational (apprenticeship)	3. Vocational (apprenticeship)
4. Secondary	4. Secondary
5. University (degree)	5. University (degree)

79. Did you have any of the following items in your house when you were a child (about 10 years old)?

Cold tap water	1. Yes	2. No	3. I don't remember
Hot tap water	1. Yes	2. No	3. I don't remember
Radio	1. Yes	2. No	3. I don't remember
Fridge	1. Yes	2. No	3. I don't remember
Own kitchen	1. Yes	2. No	3. I don't remember
Own toilet	1. Yes	2. No	3. I don't remember

80. What is your current economic activity?

1. Employed
2. Entrepreneur (owner of a company)
3. Self-employed / freelance
4. Housewife
5. Farmer
6. Pensioner, still employed
7. Pensioner, not employed. At what age did you retire ? …….. ☐☐ years old
8. Unemployed

81. What was your main life-time occupation? _____

82. Have you ever experienced unemployment?

1. No
2. Yes, for up to 3 months in total
3. Yes, for 3 months to 1 year
4. Yes, for more than one year

83. If you are out of work, do you look for a job?

1. Yes
2. No, no hope
3. No, I choose not to work
4. No, I am too ill to work
5. No, I am retired
6. No, other reason: please specify _____

84. Now, would you tell us about your household? Below is a list of various items, which of the following do you have in your household?

	Yes	No, I do not want it	No, I can not afford it
Microwave	☐ 1	☐ 2	☐ 3
Video recorder	☐ 1	☐ 2	☐ 3
Television (colour)	☐ 1	☐ 2	☐ 3
Washing machine	☐ 1	☐ 2	☐ 3
Dishwasher	☐ 1	☐ 2	☐ 3
Car	☐ 1	☐ 2	☐ 3
Freezer	☐ 1	☐ 2	☐ 3
Cottage (for holidays / weekends etc.)	☐ 1	☐ 2	☐ 3
Video camera / camcorder	☐ 1	☐ 2	☐ 3
Satellite / cable TV	☐ 1	☐ 2	☐ 3
Telephone	☐ 1	☐ 2	☐ 3
Mobile phone	☐ 1	☐ 2	☐ 3

Appendix 2.

Questionnaire for working individuals in the HAPIEE study wave 1

1. **What is your current occupation?** _____
2. **How many hours do you spend at work in a typical week?** _____
3. **Is part of it overtime work**?
   1. Yes
   2. No
4. **If yes, how many hours do you spend at overtime work in a typical week ?** _____
5. **What is your position at your main job**?
   1. higher managerial post or director
   2. manager / supervisor / foreman, more than 25 inferiors
   3. manager / supervisor / foreman, 5-25 inferiors
   4. manager / supervisor / foreman, less than 5 inferiors
   5. employee, without inferiors
   6. self-employed (25+ employees)
   7. self-employed (1-24 employees)
   8. self-employed (no employees)
6. **Which of these best describes your work in your main job?**
   Please choose one answer only
   1. Sedentary occupation: You spend most of your time sitting (such as in an office)
   2. Standing occupation: You spend most of your time standing or walking. However the way you spend your time does not require intense physical effort (e.g. shop assistant, hairdresser, security guard etc.).
   3. Physical work: This involves some physical effort including handling of heavy objects and use of tools (e.g. plumber, cleaner, nurse, sports instructor, electrician, carpenter etc.)
   4. Manual: This involves very vigorous physical activity including handling of very heavy objects (e.g., miner, bricklayer, construction worker etc.)
7. **What is size of firm you work at?**
   1. Working alone
   2. 1-5 other people
   3. 6-24 other people
   4. 25-49 other people
   5. 50-499 other people
   6. 500+ other people
8. **Here are some statements about possible strenuous aspects of your current work situation.**
   Please cross the answer that best describes your job:

	strongly disagree	***disagree***	***agree***	***strongly agree***
I get easily overwhelmed by time pressures at work	☐ 1	☐ 2	☐ 3	☐ 4
As soon as I get up in the morning I start thinking about work problems	☐ 1	☐ 2	☐ 3	☐ 4
When I get home, I can easily relax and 'switch off' work	☐ 1	☐ 2	☐ 3	☐ 4
People close to me say I sacrifice too much for my job	☐ 1	☐ 2	☐ 3	☐ 4
Work rarely lets me go, it is still on my mind when I go to bed	☐ 1	☐ 2	☐ 3	☐ 4
If I postpone something that I was supposed to do today I'll have trouble sleeping at night	☐ 1	☐ 2	☐ 3	☐ 4

9. Here are some statements about possible strenuous aspects of your current work situation.
Please cross the answer that best describes your job:

	strongly disagree	***disagree***	***agree***	***strongly agree***
My job requires that I learn new things	☐ 1	☐ 2	☐ 3	☐ 4
My job requires a high level of skill	☐ 1	☐ 2	☐ 3	☐ 4
My job requires me to be creative	☐ 1	☐ 2	☐ 3	☐ 4
I get to do a variety of different things on my job	☐ 1	☐ 2	☐ 3	☐ 4
I have an opportunity to develop my own special abilities	☐ 1	☐ 2	☐ 3	☐ 4
My job allows me to make a lot of decisions on my own	☐ 1	☐ 2	☐ 3	☐ 4
On my job, I have very little freedom to decide how I do my work	☐ 1	☐ 2	☐ 3	☐ 4
I have a lot of say about what happens in my job	☐ 1	☐ 2	☐ 3	☐ 4
My job requires working very fast	☐ 1	☐ 2	☐ 3	☐ 4
My job requires working very hard	☐ 1	☐ 2	☐ 3	☐ 4
I have enough time to get the job done	☐ 1	☐ 2	☐ 3	☐ 4
I am not asked to do an excessive amount of work	☐ 1	☐ 2	☐ 3	☐ 4
My job requires long periods of intense concentration on the task	☐ 1	☐ 2	☐ 3	☐ 4
My job is very hectic	☐ 1	☐ 2	☐ 3	☐ 4
My tasks are often interrupted before they can be completed, requiring attention at a later time	☐ 1	☐ 2	☐ 3	☐ 4
I am free from conflicting demands that others make	☐ 1	☐ 2	☐ 3	☐ 4
Waiting on work from other people or departments often slows me down on my job	☐ 1	☐ 2	☐ 3	☐ 4
My work puts me in emotionally disturbing situations	☐ 1	☐ 2	☐ 3	☐ 4
My work is emotionally demanding	☐ 1	☐ 2	☐ 3	☐ 4
I get emotionally involved in my work	☐ 1	☐ 2	☐ 3	☐ 4
My supervisor is concerned about the welfare of those under him	☐ 1	☐ 2	☐ 3	☐ 4
My supervisor pays attention to what I am saying	☐ 1	☐ 2	☐ 3	☐ 4
My supervisor is helpful in getting the job done	☐ 1	☐ 2	☐ 3	☐ 4
My supervisor is successful in getting people to work together	☐ 1	☐ 2	☐ 3	☐ 4
People I work with are competent in doing their jobs	☐ 1	☐ 2	☐ 3	☐ 4
People I work with take a personal interest in me	☐ 1	☐ 2	☐ 3	☐ 4
People I work with are friendly	☐ 1	☐ 2	☐ 3	☐ 4
People I work with are helpful in getting the job done	☐ 1	☐ 2	☐ 3	☐ 4

10. Here are some questions about your current work situation.
Please cross the answer that best describes your job.

	Yes	No	IF YES			
			Not at all distressed	*Somewhat distressed*	*Rather distressed*	*Very distressed*
There is constant time pressure in my job due to a heavy workload	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
There are many interruptions and disturbances in my job	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
I have a lot of responsibility in my job	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
There is pressure in my job to work overtime	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
My job is physically demanding	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Over the past few years, my job has become more and more demanding	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Are you treated unfairly at work?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Are the promotion prospects in your job poor?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Have you experienced or do you expect to experience an undesirable change in your work situation?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Have job redundancies recently affected your work colleagues?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Is your own job security poor?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4

	Yes	No	IF NO			
			Not at all distressed	*Somewhat distressed*	*Rather distressed*	*Very distressed*
Do you receive the respect you deserve from your work colleagues?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Do you receive the respect you deserve from your supervisors?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Do you experience adequate support in difficult situations?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Does your current job adequately reflect your knowledge, skills and training?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Does your salary/income adequately reflect all your past efforts and achievements?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Considering all your efforts and achievements, do you receive the respect and prestige you deserve at work?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4
Considering all your efforts and achievements, are your work prospects adequate?	☐ 1	☐ 2	☐ 1	☐ 2	☐ 3	☐ 4

Appendix 3.

Dietary questionnaire in the HAPIEE study wave 1

We would like to ask you to estimate your average food use. Please cross the appropriate square in each row of the tables below a number indicating how often, on average, you have eaten the specified amount during the last 3 months.

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Bread and cereals										
White bread, rolls	Medium slice, 1 roll	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Dark bread, rolls	Medium slice, 1 roll	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cereals	Medium bowl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Potatoes, rice, pasta, dumplings										
Potatoes boiled or mashed	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Potatoes fried (chips) or roasted	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Rice	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pasta (spaghetti, noodles)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pizza	Medium slice	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Roll-dumplings	4 slices	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Potato-dumplings	4 slices	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Groats	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Dairy products and fats										
Cream, sour cream	50 ml	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
White yoghurt	1 carton (100-150 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fruit yoghurt	1 carton (100-150ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Milk desserts	1 carton (100-150 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Soft cottage cheese	Medium serving (about 30 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Hard cottage cheese	Medium serving (about 30 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Low fat soft cheese	Medium serving (about 30 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
High fat soft cheese	Medium serving (about 30g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Hard cheese, processed cheese	Medium serving (about 30 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Eggs	1 egg	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Margarine (on bread)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Margarine (in food)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Butter (on bread)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Butter (in food)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mixture of margarine and butter (on bread)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mixture of margarine and butter (in food)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Vegetable oil	1 tablespoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Lard (on bread)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Lard (in food)	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mayonnaise	1 tablespoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Soups, sauces and spreads										
Borsch, shiee, vegetable soup	Medium serving (about 250 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Bouillon	Medium serving (about 250 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Beetroot soup, white borsch	Medium serving (about 250 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cabbage soup	Medium serving (about 250 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Other soups	Medium serving (about 250 ml)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Ketchup	1 tablespoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sauces with meat, pasta, groats (such as gravy or white sauces)	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Marmalade, jam, honey	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sweets and snacks										
Biscuits	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cakes, pies (sweet)	medium slice	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Buns, pastries, doughnuts, muffins	1 piece	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sweets	1 bonbon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Chocolate	1 bar	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Ice cream	one scoop	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Milk pudding	medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Sweet rice	medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pancakes	1 pancake	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sweet (fruit) dumplings	4 pieces	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Crisps, crackers and other packet-snacks	1 small packet (25 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Peanuts and other nuts	1 small packet (50 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sugar into coffee, tea	1 teaspoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sweetener into coffee, tea	1 capsule, 1 tablet	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Drinks										
Milk	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cocoa	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fruit juice	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fizzy drinks (lemonade, coke, fanta)	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Diet/low calorie drinks	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Squash	one tablespoon	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Coffee	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Tea	2 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Wine	1 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Beer	0.25 l	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Port, sherry, vermouth	1 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Liqueurs	0.5 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Spirits	0.25 dl	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Meat and fish										
Beef : roast, steak, mince, stew or casserole	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Lamb: roast, chops or stew	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pork: roast, chops or stew	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Poultry	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Rabbit	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Offals (heart, kidney, liver)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Soft sausages	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Hard sausages	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Soft salami	50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Hard salami	50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Ham	about 50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Bacon	2 slices	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pate	50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Meat pie	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Luncheon meat	50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Canned meat	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Meat ravioli	Serving (10 pieces)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fish – fresh, frozen or canned (not in oil)										
Fresh water fish (e.g. carp, pike)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Salt water white fish (e.g. cod of haddock)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Oily fish (e.g. mackerel, tuna, salmon, sardines, herring, kippers)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Other fish										
Fish canned in oil	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fish fingers, fish Afilé	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Salted fish	25 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Crab, prawns, mussels (sea food)	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Fresh fruit										
Apples	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pears	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Oranges	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Grapefruit	½ medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mandarins	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Lemons	½ medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Peaches	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Apricots	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Plums	about 100 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Cherries	about 100 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Strawberries	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Raspberries	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Red currant	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Black currant	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Blueberries	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Gooseberry	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Kiwi	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Melon	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pineapple	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Bananas	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Grapes	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Tinned or bottled fruit	medium serving (about 100g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Dried fruit (e.g. raisins, apricots, apples)	medium serving (about 50g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Vegetables										
Green salad (lettuce)	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Spinach	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Brussels sprouts	5 sprouts	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cabbage	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Beans	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Lentils	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Dried peas	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Green beans	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Green peas	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Turnips, swedes, parsnips	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Radish	4 radishes	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Celeriac	50 g	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

	Amount	*6+ per day*	*4-5 per day*	*2-3 per day*	*1 per day*	*5-6 per week*	*2-4 per week*	*1 per week*	*1-3 per month*	*Never or less than 1 per month*
Parsley	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cauliflower	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Broccoli	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Carrots	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Onion	½ medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Leeks	½ medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Garlic	1 clove	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Peppers	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Tomatoes	1 medium	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Cucumbers	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Aubergine	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Courgette/marrow	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Corn	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Beet-root cooked Russian salad (RU)	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Sauerkraut	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Pickled vegetables, gherkins	Medium serving (about 50 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mushrooms	Medium serving	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Soya meat	Medium serving (about 100g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9
Mixed frozen vegetables	Medium serving (about 100 g)	☐ 1	☐ 2	☐ 3	☐ 4	☐ 5	☐ 6	☐ 7	☐ 8	☐ 9

1. Are the foods and drinks listed in the previous table representative of the foods and drinks that you consumed in the last 3 months?

1. Yes
2. No

2. Are there any other foods, which you ate more than once a week?

1. Yes
2. No

3. If yes, please list below

Food name	Usual serving size	Number of times eaten each week

4. What type of milk did you most often use?
1. Full cream (3% of fat and more)
2. Semi- skimmed (2% of fat)
3. Skimmed (about 0.5% of fat)
4. Soya milk
5. Cream into coffee, tea
6. I do not use milk
7. I do not know

5. How much milk do you drink each day, including milk with tea, coffee, cereals etc.?
1. None
2. Less than 250 ml
3. More than 250, less than 500 ml
4. More than 500 ml, less than 1000 ml
5. More than 1000 ml

6. How often do you have coffee or deserts with added cream?
1. Daily
2. 4-6 times a week
3. 1-3 times a week
4. 1-3 times a month
5. Less than once a month
6. I do not have coffee or deserts with cream
7. I do not have coffee or deserts
8. I do not know

7. How often do you eat soups with added cream?
1. Daily
2. 4-6 times a week
3. 1-3 times a week
4. 1-3 times a month
5. Less than once a month
6. I do not eat soup with cream
7. I do not eat soup
8. I do not know

8. How often do you use sour cream including when added to the food?
1. Daily
2. 4-6 times a week
3. 1-3 times a week
4. 1-3 times a month
5. Less than once a month
6. I do not use sour cream
7. I do not know

9. What kind of fat do you use most often for frying, roasting, grilling, baking etc?

1. vegetable oil
2. olive oil
3. butter
4. margarine, solid vegetable fat
5. lard
6. none
7. I do not know

10. How often do you eat fried food ?

1. Daily
2. 4-6 times a week
3. 1-3 times a week
4. 1-3 times a month
5. Less than once a month
6. I do not eat fried food
7. I do not know

11. What do you do with the visible fat on your meat?

1. I do not eat meat
2. I eat as little as possible of the fat
3. I eat some of the fat
4. I eat most of the fat

12. How often do you or your spouse add salt to food during cooking?

1. Never
2. Rarely
3. Sometimes
4. Usually
5. Always
6. I do not cook
7. I do not know

13. How often do you add salt to any food at the table?

1. Never
2. Rarely
3. Sometimes
4. Usually
5. Always
6. I do not know

14. What type of breakfast cereals do you most often eat?

1. I do not eat cereals
2. Corn flakes
3. Oat flakes
4. Corn and oat flakes
5. Other
6. I do not know

Appendix 4.

Path analyses for 3 separate drinking outcomes: antecedent or mediator role of OC in relationship between ERI and drinking outcomes

Table 1. Path analysis for antecedent or mediator role of OC in relationship between ERI and binge drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.80	0.346	0.316	< 0.001
ERI wave 1 → ERI wave 2	1.50	0.237	0.242	< 0.001
OC → ERI → Binge drinking	1.05	0.027	0.021	< 0.001
OC wave 1 → ERI wave 2	1.31	0.148	0.146	< 0.001
ERI wave 1 → Binge drinking wave 2	1.39	0.183	0.143	< 0.001
ERI → OC → Binge drinking	1.01	0.006	0.005	0.077
ERI wave 1 → OC wave 2	1.16	0.084	0.079	< 0.001
OC wave 1 → Binge drinking wave 2	1.15	0.076	0.058	0.054
Tests of model fit		RMSEA= 0.055	CFI= 0.861	TLI= 0.786

Table 2. Path analysis for antecedent or mediator role of OC in relationship between ERI and problem drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.80	0.346	0.316	< 0.001
ERI wave 1 → ERI wave 2	1.50	0.237	0.242	< 0.001
OC → ERI → Problem drinking	1.03	0.019	0.014	0.001
OC wave 1 → ERI wave 2	1.30	0.147	0.145	< 0.001
ERI wave 1 → Problem drinking wave 2	1.26	0.128	0.098	< 0.001
ERI → OC → Problem drinking	1.01	0.007	0.005	0.048
ERI wave 1 → OC wave 2	1.16	0.082	0.078	< 0.001
OC wave 1 → Problem drinking wave 2	1.16	0.084	0.061	0.031
Tests of model fit		RMSEA= 0.056	CFI= 0.882	TLI= 0.791

Table 3. Path analysis for antecedent or mediator role of OC in relationship between ERI and heavy drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.80	0.347	0.317	< 0.001
ERI wave 1 → ERI wave 2	1.50	0.238	0.242	< 0.001
OC → ERI → Heavy drinking	1.02	0.012	0.010	0.009
OC wave 1 → ERI wave 2	1.31	0.148	0.146	< 0.001
ERI wave 1 → Heavy drinking wave 2	1.15	0.081	0.065	0.005
ERI → OC → Heavy drinking	1.00	0.002	0.001	0.475
ERI wave 1 → OC wave 2	1.16	0.084	0.079	< 0.001
OC wave 1 → Heavy drinking wave 2	1.04	0.021	0.016	0.453
Tests of model fit		RMSEA= 0.056	CFI= 0.848	TLI= 0.742

Table 4. Path analysis for antecedent or mediator role of OC in relationship between ERI and binge drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.84	0.359	0.358	< 0.001
ERI wave 1 → ERI wave 2	1.51	0.242	0.251	< 0.001
OC → ERI → Binge drinking	1.04	0.023	0.018	0.007
OC wave 1 → ERI wave 2	1.32	0.155	0.159	< 0.001
ERI wave 1 → Binge drinking wave 2	1.30	0.146	0.113	0.005
ERI → OC → Binge drinking	1.01	0.004	0.003	0.341
ERI wave 1 → OC wave 2	1.15	0.076	0.077	< 0.001
OC wave 1 → Binge drinking wave 2	1.09	0.050	0.038	0.328
Tests of model fit		RMSEA= 0.062	CFI= 0.837	TLI= 0.739

Table 5. Path analysis for antecedent or mediator role of OC in relationship between ERI and problem drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.84	0.360	0.359	< 0.001
ERI wave 1 → ERI wave 2	1.51	0.242	0.252	< 0.001
OC → ERI → Problem drinking	1.03	0.018	0.015	0.048
OC wave 1 → ERI wave 2	1.32	0.156	0.160	< 0.001
ERI wave 1 → Problem drinking wave 2	1.23	0.116	0.091	0.042
ERI → OC → Problem drinking	1.01	0.005	0.004	0.276
ERI wave 1 → OC wave 2	1.15	0.076	0.077	< 0.001
OC wave 1 → Problem drinking wave 2	1.13	0.069	0.053	0.253
Tests of model fit		RMSEA= 0.061	CFI= 0.840	TLI= 0.726

Table 6. Path analysis for antecedent or mediator role of OC in relationship between ERI and heavy drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → OC wave 2	1.83	0.357	0.356	< 0.001
ERI wave 1 → ERI wave 2	1.51	0.244	0.253	< 0.001
OC → ERI → Heavy drinking	1.02	0.011	0.009	0.037
OC wave 1 → ERI wave 2	1.32	0.156	0.160	< 0.001
ERI wave 1 → Heavy drinking wave 2	1.13	0.069	0.056	0.033
ERI → OC → Heavy drinking	1.00	0.001	0.001	0.608
ERI wave 1 → OC wave 2	1.15	0.076	0.077	< 0.001
OC wave 1 → Heavy drinking wave 2	1.03	0.018	0.012	0.541
Tests of model fit		RMSEA= 0.062	CFI= 0.818	TLI= 0.708

Path analyses for 3 separate drinking outcomes: mediator roles of PC and ERI in relationship between OC and drinking outcomes

Table 7. Path analysis for mediator roles of PC and ERI in relationship between OC and binge drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Binge drinking wave 2	1.12	0.063	0.049	0.084
OC → ERI → Binge drinking	1.07	0.037	0.029	< 0.001
OC wave 1 → ERI wave 1	1.54	0.253	0.247	< 0.001
ERI wave 1 → Binge drinking wave 2	1.30	0.148	0.117	< 0.001
OC → PC → Binge drinking	1.02	0.010	0.008	0.013
OC wave 1 → PC wave 1	0.85	- 0.092	- 0.090	< 0.001
PC wave 1 → Binge drinking wave 2	0.82	- 0.107	- 0.084	0.006
ERI correlates with PC		- 0.051	- 0.085	< 0.001
Tests of model fit		RMSEA= 0.062	CFI= 0.857	TLI= 0.775

Table 8. Path analysis for mediator roles of PC and ERI in relationship between OC and problem drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Problem drinking wave 2	1.13	0.067	0.051	0.055
OC → ERI → Problem drinking	1.07	0.035	0.027	< 0.001
OC wave 1 → ERI wave 1	1.52	0.246	0.240	< 0.001
ERI wave 1 → Problem drinking wave 2	1.29	0.142	0.112	< 0.001
OC → PC → Problem drinking	1.02	0.010	0.007	0.007
OC wave 1 → PC wave 1	0.85	- 0.089	- 0.086	< 0.001
PC wave 1 → Problem drinking wave 2	0.82	- 0.109	- 0.085	0.003
ERI correlates with PC		- 0.047	- 0.078	< 0.001
Tests of model fit		RMSEA= 0.064	CFI= 0.863	TLI= 0.782

Table 9. Path analysis for mediator roles of PC and ERI in relationship between OC and heavy drinking in men

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Heavy drinking wave 2	1.03	0.018	0.014	0.526
OC → ERI → Heavy drinking	1.03	0.019	0.015	0.010
OC wave 1 → ERI wave 1	1.52	0.248	0.242	< 0.001
ERI wave 1 → Heavy drinking wave 2	1.15	0.077	0.061	0.009
OC → PC → Heavy drinking	1.01	0.006	0.005	0.032
OC wave 1 → PC wave 1	0.84	- 0.094	- 0.092	< 0.001
PC wave 1 → Heavy drinking wave 2	0.89	- 0.067	- 0.053	0.025
ERI correlates with PC		- 0.045	- 0.075	< 0.001
Tests of model fit		RMSEA= 0.065	CFI= 0.861	TLI= 0.766

Table 10. Path analysis for mediator roles of PC and ERI in relationship between OC and binge drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Binge drinking wave 2	1.11	0.059	0.046	0.208
OC → ERI → Binge drinking	1.06	0.035	0.026	0.003
OC wave 1 → ERI wave 1	1.51	0.242	0.236	< 0.001
ERI wave 1 → Binge drinking wave 2	1.30	0.145	0.112	0.003
OC → PC → Binge drinking	1.01	0.008	0.006	0.048
OC wave 1 → PC wave 1	0.86	- 0.086	- 0.087	< 0.001
PC wave 1 → Binge drinking wave 2	0.84	- 0.098	- 0.072	0.037
ERI correlates with PC		- 0.049	- 0.084	< 0.001
Tests of model fit		RMSEA= 0.062	CFI= 0.834	TLI= 0.745

Table 11. Path analysis for mediator roles of PC and ERI in relationship between OC and problem drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Problem drinking wave 2	1.13	0.070	0.055	0.158
OC → ERI → Problem drinking	1.06	0.030	0.023	0.026
OC wave 1 → ERI wave 1	1.50	0.238	0.233	< 0.001
ERI wave 1 → Problem drinking wave 2	1.25	0.124	0.097	0.021
OC → PC → Problem drinking	1.02	0.009	0.007	0.045
OC wave 1 → PC wave 1	0.85	- 0.090	- 0.091	< 0.001
PC wave 1 → Problem drinking wave 2	0.83	- 0.102	- 0.079	0.029
ERI correlates with PC		- 0.048	- 0.083	< 0.001
Tests of model fit		RMSEA= 0.060	CFI= 0.855	TLI= 0.752

Table 12. Path analysis for mediator roles of PC and ERI in relationship between OC and heavy drinking in women

Parameter	Odds Ratio	Unstandardized coefficient	Standardized coefficient	P value
OC wave 1 → Heavy drinking wave 2	1.03	0.018	0.012	0.533
OC → ERI → Heavy drinking	1.02	0.013	0.010	0.095
OC wave 1 → ERI wave 1	1.51	0.241	0.236	< 0.001
ERI wave 1 → Heavy drinking wave 2	1.10	0.054	0.043	0.091
OC → PC → Heavy drinking	1.01	0.005	0.004	0.070
OC wave 1 → PC wave 1	0.86	- 0.085	- 0.086	< 0.001
PC wave 1 → Heavy drinking wave 2	0.90	- 0.061	- 0.043	0.065
ERI correlates with PC		- 0.049	- 0.082	< 0.001
Tests of model fit		RMSEA= 0.064	CFI= 0.837	TLI= 0.726

Appendix 5.
Published paper relevant for the thesis

During the period of my PhD study, I have published three academic papers in the field of social epidemiology (as first author), mainly focusing on work stress and health. In particular, one published paper relevant for the thesis has focused on the associations between OC, ERI and dietary outcomes in the HAPIEE study.

Published paper relevant for the thesis:

Chen SW, Peasey A, Stefler D, Malyutina S, Pajak A, Kubinova R, Chan JH, Bobak M, Pikhart H* (2016) Effort–reward imbalance at work, overcommitment personality and diet quality in Central and Eastern European populations. *British Journal of Nutrition* 115 (7): 1254–1264.